Postinfection Activity of Selected Late Blight Fungicides

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ABSTRACT

Johnson, D. A., Cummings, T. F., and Geary, B. 2000. Postinfection activity of selected late blight fungicides. *Plant Dis.* 84:1116-1120.

Two sets of experiments were done in the greenhouse with 'Russet Burbank' potatoes infected with *Phytophthora infestans*. The first determined the effectiveness of selected late blight fungicides in restricting lesion length and spore production when applied after stem lesions had occurred on potted plants. The second quantified the curative activity of selected fungicides applied 12 to 48 h after inoculation of excised leaves with *P. infestans*. Such information may be beneficial in selecting an effective fungicide for late blight management when symptoms of the disease are present in a field. Expansion of established stem lesions was consistently restricted by one application of either dimethomorph plus mancozeb or metiram plus triphenyltin hydroxide. Sporulation on established stem lesions was consistently inhibited by either one application of dimethomorph plus mancozeb or cymoxanil plus mancozeb. Two applications of propamocarb hydrochloride plus chlorothalonil were required to restrict lesion expansion and inhibit sporulation. Size and number of lesions and sporulation on leaflets were reduced when propamocarb hydrochloride plus chlorothalonil was applied up to 48 h after inoculation.

Late blight of potato, *Solanum tuberosum* L., caused by *Phytophthora infestans* (Mont.) de Bary, increases very rapidly in commercial fields when favored by cool, wet weather (13,15,20). In the Columbia Basin of Washington and Oregon in 1995, when environmental conditions favored the disease, the estimated cost of managing late blight on 65,000 ha (160,000 acres) was \$30 million (15). One aggressive strain, US-8, of *P. infestans* has caused more recent outbreaks in the Columbia Basin (17, 18). More applications of protectant fungicide are required to suppress outbreaks caused by the US-8 strain compared with US-1, as determined by computer simulation (16) and inoculation tests (8,9). Thus, effective disease management in eastern Washington and Oregon depends on a combination of several practices, including planting disease-free seed tubers, eliminating inoculum on potato tuber refuse, proper irrigation management, adequate coverage of developing tubers with soil to reduce tuber infections, and timely and thorough applications of effective fungicides. A regional late blight forecasting system that employs logistic models derived from historical weather and disease data is used to assist in fungicide scheduling (13,14).

Fungicides are most effective in late blight management when applications are

made before infection (2,19,20), because most fungicides have protective modes of action and are not effective after *P. infestans* enters plant tissues (2,19). However, dimethomorph (4) and triphenyltin hydroxide (19) have been reported to inhibit sporulation. Cymoxanil and propamocarb hydrochloride also may have some activity after infection (11,19). Even though fungicides usually are intended to be applied before infection occurs, this does not always happen, because canopy growth may be rapid, extensive areas may need to be covered, custom applicators may need to be scheduled, or wind and rain may preclude application. As a result, lesions in various stages of development may exist in a field before fungicides are applied. Furthermore, a protectant fungicide application may not thoroughly cover all foliage within a canopy, leaving sections of the canopy unprotected. For example, lower canopy levels may not initially receive sufficient fungicide when application is made by airplane after closure between rows (10). A knowledge of fungicide characteristics and whether they restrict disease development when applied after infection may help determine the most effective materials to use under such conditions. Such knowledge may be particularly important in the Columbia Basin, where early season plant growth is rapid, the crop canopy rapidly closes, and the growing season is long, up to 180 days.

The amount of sporulation and extent of lesion expansion on infected plants are major components of late blight epidemics (1,20). Lesions are capable of continually producing sporangia during expansion if humidity and temperature are favorable, whereas new infections must pass through

a latent period before lesions develop and sporulate. After initial infection, a fungicide that restricts lesion expansion and sporulation would be helpful in retarding continued development of late blight and might reduce tuber infections. Little information has been published on the effectiveness of currently used late blight fungicides in restricting various components of late blight development. The primary purpose of our experiments was to determine the effectiveness of selected late blight fungicides in inhibiting lesion expansion and sporulation after stem lesions had occurred on potted plants in the greenhouse. A second set of experiments was done to quantify the curative activity of selected fungicides 12 to 48 h after inoculation of excised leaves.

MATERIALS AND METHODS

'Russet Burbank' potato plants were propagated from growing buds cut with a disk-shaped cutter from certified seed tubers that produced a tissue sphere weighing ≈ 13 g. Plants were grown in 15-cm-diameter pots in the greenhouse; 10 g of 16-16-16 N-P-K fertilizer was incorporated into the potting mix (3:1:1, vol/vol/vol) before planting.

Fungicides and rates used in the tests were azoxystrobin (Quadris) at 0.9 g a.i./liter; chlorothalonil (Bravo WS) at 2.4 g a.i./liter; metiram (Polyram 80 DF) at 4.6 g a.i./liter; metiram + triphenyltin hydroxide (Super Tin 80 WP) at 4.6 g a.i./liter + 0.4 g a.i./liter, respectively; dimethomorph + mancozeb (Acrobat MZ) at 0.6 g a.i./liter + 4.0 g a.i./liter, respectively; propamocarb hydrochloride + chlorothalonil (Tattoo C) at 2.5 g a.i./liter + 2.5 g a.i./liter, respectively; cymoxanil + mancozeb (Curzate 50 WP + Manzate 200 DF) at 0.37 g a.i./liter + 3.7 g a.i./liter, respectively; and cymoxanil + mancozeb (Manex C-8) at 0.34 g a.i./liter + 2.8 g a.i./liter, respectively.

Potato stems were inoculated in three trials with two isolates of *P. infestans* to produce lesions for postsymptom application of fungicide. Isolate CF-1, (US-8, A-2 mating type) was used in the first and second trials, and 6-366 (no US designation, A-1 mating type, metalaxyl insensitive, *glucose-6-phosphate isomerase* [*GPI*; 100/111/122]) was used in the third. Inoculum was increased on excised leaves of Russet Burbank at 18°C in humid chambers. Preflowering plants ≈ 55 cm tall were inoculated by placing 0.05 ml of a sporangium suspension containing 10,000 sporangia per ml on a 1-cm filter paper square and transferring the square to the main stem ≈ 30 to 40 cm above the soil line. The

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Accepted for publication 5 July 2000.

Publication no. D-2000-0817-01R
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filter paper surface receiving inoculum was placed against one side of the stem; surface tension held the square in place. Inoculated plants were placed in a mist chamber for 18 h at 17 to 21°C and moved to a greenhouse for 6 days at 23 to 27°C during the day and 18 to 21°C at night until lesions developed. Inoculations were done in the spring and late summer when the natural photoperiod was at least 14 h; supplemental lighting was not used in the greenhouse.

Single lesions developed on inoculated stems within 5 days after inoculation. Lengths of visible lesions 6 days after inoculation were 3 to 5 cm on stems inoculated with isolate CF-1 in the first trial, 1 to 2 cm on stems inoculated with isolate CF-1 in the second trial, and 5 to 8 cm on stems inoculated with isolate 6-366. (Dif-

ferences in initial lesion size between trials and isolates may be due to differences in inoculum viability and isolate aggressiveness.)

Plants with stem lesions were sprayed to runoff with various fungicides 6 days after inoculation. Five fungicides or fungicide combinations were used in trial one, and seven were used in trials two and three (Tables 1 and 2). Plants with stem lesions not treated with fungicides served as non-treated controls. Plants were again placed in a mist chamber for 18 h to provide conditions for sporulation. Infected stems sporulated within 18 h. Plants were moved to the greenhouse, and lesion length was measured and number of sporangia was counted on infected stems. Plants with stem lesions were again treated with the same fungicides 5 to 7 days after the first ap-

plication (11 to 15 days after original inoculation), placed in the mist chamber for 18 h, and moved to the greenhouse, where lesion length and sporangia number were again determined. Fungicides were applied, and treated plants were arranged in randomized complete blocks with eight replicates in the mist chamber and greenhouse.

Timing of the second fungicide application and placement in the mist chamber varied among the three experiments used to preliminarily assess their effects on lesion expansion and sporulation. In the first trial with isolate CF-1, the second set of fungicide applications was made 15 days after inoculation (9 days after first fungicide application). Plants were placed in the mist chamber 7 h after both applications, and lesion expansion and sporulation were determined 30 h after each fungicide application. In the second trial with isolate CF-1, the second fungicide applications were made 11 days after inoculation (5 days after first application). Plants were placed in the mist chamber 24 h after each application, and lesion length and sporulation were determined 2 days after applications. In the third trial (isolate 6-366), the second fungicide application was made 13 days after inoculation (7 days after first application). Plants were placed in the mist chamber 24 h after each application, and lesion length and sporulation were determined 4 days after applications.

Lesion length and sporulation after the wet period were determined on infected stems. Lesion expansion was determined by marking the upper edge of the lesion with a permanent marker before placement in the mist chamber and measurement of the length of the new lesion growth with a ruler after plants were removed. Density of sporangia was estimated visually, using a scale of 0 to 4, with 0 through 4 indicating none, slight, moderate, abundant, and very

Table 1. Mean length of stem lesion expansion (LE) and spore production when 'Russet Burbank' potato stems with late blight lesions were sprayed with selected fungicides at 6 days or at 6 and 15 days after inoculation with isolate CF-1 (US-8) of *Phytophthora infestans*[†]

Fungicide	Time of Fungicide Application ^u			
	6 days ^v		6 and 15 days ^w	
	LE length (cm) ^x	Sporulation index ^y	LE length (cm)	Sporulation index
Control	2.0 a	4.0 a	2.8 a	4.0 a
Cymoxanil + mancozeb ^z	1.9 a	3.0 b	2.4 ab	2.6 b
Chlorothalonil	1.9 a	3.0 b	2.1 ab	3.2 ab
Propamocarb hydrochloride + chlorothalonil	1.8 ab	3.0 b	2.5 ab	2.8 b
Metiram + triphenyltinhydroxide	1.4 bc	3.0 b	1.9 b	3.0 ab
Dimethomorph + mancozeb	1.1 c	1.0 b	2.4 ab	1.0 c

[†] First of two experiments with isolate CF-1.

^u Values followed by the same letter are not significantly different at $P = 0.05$, according to Fisher's protected LSD.

^v Values are means of eight replicates.

^w Values are means of four to six replicates.

^x Distance of expansion up stems. Lesions expanded down stems about the same distance.

^y Sporulation index: 1 through 4; 4,000 $\pm$ 2,400; 15,000 $\pm$ 500; 23,000 $\pm$ 4,700; and 50,000 $\pm$ 7,600 (mean $\pm$ standard error) sporangia per cm of infected stem, respectively.

^z Curzate 50 WP plus Manzate 200 DF.

Table 2. Mean length of stem lesion expansion (LE), sporulation index (SI), and number of sporangia per cm of stem (S/cm) when 'Russet Burbank' potato stems with late blight lesions were sprayed with selected fungicides at 6 days or at 6 and 11 to 13 days after inoculation with one of two isolates of *Phytophthora infestans* (CF-1 [US-8] or 6-366)[†]

Fungicide	LE length (cm)				Sporulation index and no. of sporangia						
	CF-1 ^u		6-366 ^v		CF-1			6-366			
	6 days ^w	6 and 11 days	6 days	6 and 13 days	6 days	6 and 11 days		6 days		6 and 13 days	
					SI ^x	SI	S/cm ^y	SI	S/cm	SI	S/cm
Control	4.4 a	3.1 a	2.4 a	2.6 a	4 a	4 a	54 a	4 a	117 a	4 a	77 a
Azoxystrobin	...	...	2.2 ab	1.9 bc	...	...	...	4 a	99 a	4 a	36 a
Chlorothalonil	4.3 a	3.4 a	2.1 ab	2.1 b	4 a	3.2 b	22 a	4 a	129 a	4 a	51 a
Metiram	...	...	2.1 ab	2.3 ab	...	...	...	4 a	74 a	4 a	42 a
Propamocarb hydrochloride + chlorothalonil	3.9 ab	1.2 bc	1.9 bc	0 e	3.6 a	1.3 c	2 b	4 a	111 a	3.1 b	19 a
Metiram + triphenyltin hydroxide	2.7 bc	1.2 bc	1.6 d	1.4 d	1.2 b	1.3 c	1 bc	4 a	117 a	3.1 b	41 a
Dimethomorph + mancozeb	2.0 cd	1.8 b	1.7 cd	1.6 cd	0.3 b	0.3 d	0.3 bc	1.3 c	14 b	1.6 c	3 b
Cymoxanil + mancozeb ^z	1.5 d	0.3 c	1.5 d	0.3 e	0.3 b	0.1 d	0 c	1.9 b	14 b	0.8 d	2 b

[†] Values are means of eight replicates. Values followed by the same letter are not significantly different at $P = 0.05$, according to Fisher's protected LSD.

^u Second of two experiments with isolate CF-1.

^v A-1 mating type, metalaxyl insensitive, *GPI 100/111/122*.

^w Time of fungicide application after inoculation.

^x Sporulation index: 0 through 4: none, slight, moderate, abundant, and very abundant sporulation, respectively.

^y Sporangia per cm of stem $\times 10^{-3}$.

^z Manex C-8.

abundant sporangiophores and sporangia, respectively. Sporangia also were quantified by cutting a 2-cm section of the stem, where sporulation was visually judged to be most abundant, and placing it in a 130-ml bottle with 10 ml of a fixative solution (90:5:5 [vol/vol/vol] 95% ethanol, glacial acetic acid, 37% formalin). Stem diameters of 9 to 10 mm at inoculation sites were fairly uniform. The bottles were shaken to dislodge sporangia. Sporangia in three ali-

quot subsamples per bottle were counted with a hemacytometer. Quantity per linear centimeter of sporulating stem was calculated for all stems inoculated with isolate 6-366 and the second inoculation with isolate CF-1. For the first trial with isolate CF-1, density of sporangia was estimated using the 0 to 4 scale, and sporangia were quantified for 12 stems, with visual ratings ranging from 1 to 4. Viability of sporangia was not tested.

Curative activity of fungicides 12 to 48 h after inoculation. Potato leaves of 'Russet Burbank' were grown in the greenhouse from certified seed as previously described. Petioles of excised leaves with three leaflets were placed in a sucrose nutrient solution in test tubes. Leaflets were inoculated with isolate CF-1 by placing a 1-cm filter paper with a 0.05-ml drop of inoculum at 10,000 sporangia per ml in the center of each leaflet. Inoculated leaflets were placed in a mist chamber for 12 h at 18 to 20°C and moved to a greenhouse at 23 to 27°C during the day and 18 to 21°C during the night. At 12, 24, 36, and 48 h postinoculation, leaves were sprayed until runoff with one of the four fungicides (Fig. 1). Treatments were arranged as a randomized complete block with four replicates and three subsamples (leaflets) per replicate. Disease severity, incidence of infected leaflets, and quantity of sporangia were determined based on all inoculated leaflets. Quantity of sporangia per square centimeter of leaf was determined with a cork borer used to cut two 1.5-cm-diameter disks from each leaf where sporulation was visually judged to be the greatest. Leaf disks were placed in a vial with 3 ml of fixative solution and shaken, and sporangia in six aliquot subsamples were counted with a hemacytometer. The experiment was repeated.

Data were analyzed by analysis of variance using the GLM procedure, (version 6.12, SAS Institute, Cary, NC). Fisher's protected LSD at $P = 0.05$ was used to compare treatment means.

RESULTS

Lesions on control plants not sprayed with fungicide expanded a mean distance of 2 to 4.4 cm up stems from the inoculation point after test plants received one fungicide application. Lesions on control stems expanded another 2.6 to 3.1 cm after test plants received the second fungicide application (Tables 1 and 2). Expansion down the stem was not measured, but lesion lengths were similar from the inoculation point in both directions.

Lesion length was significantly ($P < 0.05$) restricted when established stem lesions caused by *P. infestans* were treated with one application of either dimethomorph plus mancozeb or metiram plus triphenyltin hydroxide (Tables 1 and 2). Lesion length was significantly ($P < 0.05$) restricted by two applications of metiram plus triphenyltin hydroxide in three trials and by two applications of dimethomorph plus mancozeb in two of three trials (Tables 1 and 2). One application of propamocarb hydrochloride plus chlorothalonil did not restrict lesion length in two of three trials compared with the nontreated control. When propamocarb hydrochloride plus chlorothalonil did restrict lesion length (isolate 6-366), the mixture did not ($P > 0.05$) restrict lesion length more than azoxystrobin,

Fig. 1. Lesion incidence, percentage of leaf area infected, and number of sporangia produced on 'Russet Burbank' potato leaflets treated with chlorothalonil (Bravo), propamocarb hydrochloride plus chlorothalonil (Tattoo), cymoxanil plus mancozeb (Cymxl), dimethomorph plus mancozeb (Acrobat), or not treated 12, 24, 36, and 48 h after inoculation with *Phytophthora infestans*.

chlorothalonil, or metiram (Table 2). Two applications of propamocarb hydrochloride plus chlorothalonil restricted lesion length in two of three trials (Tables 1 and 2).

Lesion length was not significantly ($P > 0.05$) restricted when stem lesions were sprayed with one or two applications of either chlorothalonil or metiram (Tables 1 and 2). The two formulations of cymoxanil plus mancozeb (Manex C-8 and Curzate plus Manzate) were inconsistent in reducing lesion length on stems. Lesion length was restricted by Manex C-8 in two trials (Table 2) but not by Curzate plus Manzate in a third trial (Table 1).

Sporulation of *P. infestans* on control stems after removal from the mist chamber was considered very abundant, and mean numbers of sporangia ranged from 50,000 to 117,000 per linear centimeter of stem length. Sporulation was significantly ($P < 0.05$) reduced when established stem lesions were treated with either one or two applications of dimethomorph plus mancozeb or cymoxanil plus mancozeb. Sporulation was significantly ($P < 0.05$) reduced with one application of metiram plus triphenyltin hydroxide on stems infected with isolate CF-1 (US-8) but not isolate 6-366 (Tables 1 and 2). Sporulation was significantly ($P < 0.05$) reduced with two applications of metiram plus triphenyltin hydroxide when stems were infected with isolate 6-366 and in one of two trials with isolate CF-1. Sporulation was usually not significantly ($P > 0.05$) reduced with one application of propamocarb hydrochloride plus chlorothalonil, whereas it was significantly ($P < 0.05$) reduced with two applications of the mixture (Tables 1 and 2). Sporulation was not significantly ($P > 0.05$) reduced when stem lesions were sprayed with either one or two applications of chlorothalonil, azoxystrobin, or metiram (Tables 1 and 2).

Curative activity of fungicides applied 12 to 48 h after inoculation. Incidence of leaflet lesions was significantly ($P < 0.05$) reduced when propamocarb hydrochloride plus chlorothalonil was applied 24 and 48 h after inoculation and when cymoxanil plus mancozeb was applied 12, 24, and 36 h after inoculation (Fig. 1). Lesion size (disease severity) was significantly ($P < 0.05$) reduced when propamocarb hydrochloride plus chlorothalonil was applied up to 48 h after inoculation and when cymoxanil plus mancozeb was applied up to 36 h after inoculation (Fig. 1). Sporulation on leaflets was significantly ($P < 0.05$) inhibited when either propamocarb hydrochloride plus chlorothalonil or cymoxanil plus mancozeb was applied up to 48 h after inoculation (Fig. 1). Incidence of leaflet lesions, lesion size, and sporulation were significantly ($P < 0.05$) reduced when dimethomorph plus mancozeb was applied 12 h after inoculation but not when applied 24, 36, and 48 h after inoculation. Chlorothalonil did not consistently reduce any of the three disease components

measured when applied 12 to 48 h after inoculation (Fig. 1). Data from the repeated experiment were similar to those reported.

DISCUSSION

Lesion expansion was suppressed, and sporulation was reduced when established lesions on potato stems caused by *P. infestans* were treated with dimethomorph plus mancozeb, cymoxanil plus mancozeb, and metiram plus triphenyltin hydroxide. Dimethomorph plus mancozeb and cymoxanil plus mancozeb reduced sporulation more consistently than did metiram plus triphenyltin hydroxide. These materials are fungicide mixtures that each contain an ethylene bis-dithiocarbamate (EBDC) fungicide. The active ingredients restricting disease development after initial disease establishment on stems were probably dimethomorph, cymoxanil, and triphenyltin hydroxide. Dimethomorph and organic tin fungicides, such as triphenyltin, have been reported to reduce sporulation (4,19). In another study (2), high concentrations of chlorothalonil and mancozeb were reported to reduce sporulation of *P. infestans* on potato leaves. Lesion expansion was less affected by the two fungicides in the previous study, whereas their greatest effect was on spore germination. In this study, chlorothalonil and metiram, an EBDC fungicide (as is mancozeb), did not restrict lesion expansion or inhibit sporulation by themselves. Chlorothalonil and the EBDC fungicides reduce spore germination and are effective in managing late blight when applied before infection (2,20).

Results generally were consistent among the three experiments when fungicides were applied postsymptom on stems. However, the two formulations of cymoxanil plus mancozeb used in this study were inconsistent in reducing lesion expansion on stems. Both mixtures contain cymoxanil and should have produced similar results. The inconsistency may have been due to trial differences, such as timing of fungicide applications in the three tests, or to differences in product formulation. Sporulation was inhibited on stem lesions treated with both cymoxanil mixtures in the three trials. Why sporulation was not consistently inhibited by triphenyltin hydroxide is unknown. The difference cannot be attributed to an isolate effect because the second metiram plus triphenyltin hydroxide application reduced sporulation when stems were infected with isolate 6-366, even though one application did not. Additional research is needed to resolve the difference and to more fully understand environmental and chemical factors affecting late blight lesion expansion and sporulation on potato stems.

Propamocarb hydrochloride plus chlorothalonil applied up to 48 h after inoculation reduced lesion development (lesion number and size) and sporulation on leaves. It is unknown based on this study if the post-

infection effectiveness of propamocarb hydrochloride plus chlorothalonil extends beyond 48 h. Cymoxanil plus mancozeb inhibited lesion development and sporulation on Russet Burbank leaflets up to 36 h after inoculation. These time periods present only a narrow window of opportunity to apply a fungicide after weather conditions favor infection. Windy or rainy weather during favorable periods for infection and the size and number of fields needing treatment frequently preclude fungicide applications after infection. An isolate of an aggressive strain, such as was used in this study, makes the window of opportunity narrower than does an isolate of a less aggressive strain (7). Growers generally do not know the precise timing of infection. Thus, it is in their best interest to use the most efficacious product with the broadest window of efficacy.

One application of propamocarb hydrochloride plus chlorothalonil prevented recent infections from developing further on leaves, whereas two applications were required to arrest lesion expansion and sporulation on stems. One explanation may be that stems take up the active ingredients of the fungicide at a slower rate. Furthermore, stages of disease development were different on leaves and stems and also could account for the amount of material needed to arrest further disease development.

Late blight is extremely hard to manage in the Columbia Basin once the disease is present in a potato field, because after row closure the crop canopy microclimate usually favors infection whenever sprinkler irrigation water is applied. Fields may be irrigated as frequently as every 1.5 to 3 days. In addition, duration of epidemic development is potentially long because the canopy closes relatively early and the growing season is long. *P. infestans* lesions on potato stems are important epidemiologically because they enable the late blight pathogen to perpetuate more readily during hot weather than leaf lesions do (D. A. Johnson, unpublished data). Fungicides that limit sporulation and lesion expansion on stems may help abate late blight outbreaks in fields, especially when disease levels are still relatively low. Inhibiting sporulation on potato foliage between tuber bulking and harvest may reduce the incidence of tuber infections. Fungicide application schemes that included cymoxanil, dimethomorph, or propamocarb hydrochloride provided slightly better control of late blight in a North American fungicide trial than schemes that did not include them (12). Restricting lesion expansion also may be beneficial in reducing tuber infection by limiting total sporulating plant tissue in the crop canopy.

Fungicides need to be integrated into a total disease management program and used in response to identified needs for maximum efficiency and benefit (5,6). However, disease may appear in a field before man-

agement practices are applied. Effective management of late blight in the Columbia Basin after the disease is found in a field includes using effective fungicides and application methods (3,10), shortening fungicide application intervals, decreasing the frequency of sprinkler irrigation applications, and irrigating when the crop canopy will be wet for the least amount of time. With regard to fungicides, however, the most economical and effective timing for application is before infection (5,19).

ACKNOWLEDGMENTS

PPNS No. 0287, Department of Plant Pathology, College of Agriculture and Home Economics Research Center, Project 0678, Washington State University, Pullman. We thank P. B. Hamm, D. A. Inglis, and two anonymous reviewers for critical reviews of the manuscript.

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