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January 1980

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COMPENSATORY RESPONSE OF MATURING CORN KERNELS FOLLOWING SIMULATED DAMAGE BY BIRDS

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SUMMARY

(1) A study was made to measure compensatory growth responses and to estimate losses associated with bird damage to maturing corn.

(2) Corn kernels contain 20–40% of their final biomass at the time they are usually consumed by blackbirds.

(3) Very slight compensation of kernel weight occurred following simulated bird damage to tip kernels.

(4) Heavy bird damage, early in kernel development, increased fungal, sprouting, and insect damage before harvest.

(5) Estimates of bird damage, subsequent secondary damage, and compensation were affected by the amount of damage, maturity of the kernels at the time of damage, and environmental factors before, during, and after damage.

(6) Visual estimates of weight change were closely correlated with actual loss of the total kernel weight.

(7) Studies of food habits and bioenergetics that have used feeding rates and numbers of birds to estimate the impact of blackbirds on corn crops may have underestimated reductions in corn yields.

INTRODUCTION

Red-winged blackbird (*Agelaius phoeniceus*) damage to maturing corn (*Zea mays*) in the milk and dough stages has often been considered a severe problem in localized areas of the United States (Stone *et al.* 1972). Most estimates of primary loss to corn yields have been based on visual or measured surface area estimates of damage to individual ears (Linehan 1967; De Grazio *et al.* 1969; DeHaven 1974; Granett *et al.* 1974). These methods of estimation of loss ignore any compensatory growth in the undamaged kernels and any secondary losses from insects and disease that may occur following damage.

Numerous studies of fruit, small grain, and hay have shown that moderate injury or loss to stems, leaves, roots, or fruit can occur before yield is actually affected (Stern 1973; Harriss 1974; Smith & Kendall 1975; Webster & Walker 1977; Dyer & Bokhari 1976; Allen 1977; Summers & Pollock 1978; Free & Williams 1978; Wolff 1978). Compensation in maturing corn has been suggested by Linehan (1967), Dawson (1970), Dyer (1975, 1976) and Woronecki, Ingram & Dolbeer (1976). However, studies by Duncan & Hatfield (1964), although not addressing the question of compensation following bird damage *per se*, indicated that removal of non-apical kernels during development had no effect on the growth of the remaining kernels.

Our previous study (Woronecki, Ingram & Dolbeer 1976) suggested that net loss of shelled corn weight was a function of both the level of damage and the maturity of the ear at the time of damage. It also showed that low levels of bird damage could be partially compensated by increased kernel weight. Regardless of maturity, damage of less than six kernels per ear resulted in no net loss and in some instances in increased yield. The conclusions from that experiment are now reconsidered because primary and secondary damage had not been adequately quantified. Modifications were made in the present experiment to eliminate some of the disparities contributing to weight loss of shelled corn and to reduce sampling errors so both compensatory growth responses and secondary losses associated with bird damage to maturing corn could be accurately measured.

METHODS

Experimental design and simulated bird damage

The experiment was conducted in Huron County, Ohio, in a 0.2 ha plot within a 32.4 ha field planted on 1 May 1977 with Pioneer 3518 (120 day) field corn. The plot, 60 m from any field edge, contained plants of uniform height and maturity. At the time of silking (when cobs, husk, and shank are nearly developed), the plot was gridded into eighty subplots (six rows by 5 m). Subplots with voids, gaps, or fewer than seventy plants with silking ears of corn were not included in the test.

The plot, in addition to normal commercial agricultural practices, was aerially treated with carbaryl insecticide (Sevin) 7 and 14 days after silking to reduce insect damage. A propane exploder and shotgun patrol were maintained in the field to ensure that damage by real birds was minimized.

Experimental units (fifty-six subplots and ten alternates) were randomly selected from all acceptable subplots. At the blister stage of ear growth (76 days after emergence), the first seventy top ears within each experimental unit (including alternates) that met the following criteria were tagged: at least 11 cm in length from the base of the corn ear to tip of ear; not irregular in shape or improperly developed or pollinated; and not showing any signs of tip emergence, fungus, insect, or bird damage. Within each experimental unit, thirty of the tagged ears were randomly selected to be artificially damaged and another thirty tagged ears were controls. The ten alternate ears remained to be used if any of the sixty selected ears were unacceptable at the time damage was to be inflicted.

We used two stages of corn maturity and seven levels of simulated bird damage to form a total of fourteen treatments (Table 1). The two maturity stages were: milk (22 days after silking) and dough (7 days after milk). These stages of corn maturity encompass the

TABLE 1. Description of the seven levels of simulated damage

Damage level	Number of apical kernels damaged	ca. % of total kernels that were damaged*	Pattern of damage (kernels across × kernels down)
1	0†	0.0	—
2	3	0.5	1 × 2
3	6	1.0	2 × 3
4	12	2.0	3 × 4
5	24	4.0	4 × 6
6	48	8.0	6 × 8
7	72	12.0	6 × 12

* Based on Woronecki, Ingram & Dolbeer (1976).

† 2 cm longitudinal slit made in husk at tip end but no kernels damaged.

period when most damage to corn by red-winged blackbirds occurs (Bridgeland 1979). Each treatment was replicated four times so that a total of fifty-six maturity and damage combinations were randomly assigned to the fifty-six subplots (experimental units).

Damage was inflicted with fine-tipped forceps, simulating actual bird damage. First the husk was shredded on the upper side of the ear near the tip to expose only the necessary number of apical kernels. Then, beginning with tip kernels and proceeding in a predetermined pattern (Table 1), the kernels were damaged by pinching and removing most of the internal kernel biomass. To further simulate bird damage activity, any insects encountered were removed.

Biomass accumulation determination

At weekly intervals, starting with first damage until harvest, thirty acceptable top ears of corn were collected from randomly selected alternate subplots to determine dry matter accumulation of cobs and kernels. The ears were husked, weighed, and dried (60 °C) for 20 days until moisture level reached 3 to 5%. The ears were then stored at room temperature (18–20 °C for 6 days) until ears reached an equilibrium moisture level between 5 and 9%. Each sample was weighed in seven separate aliquots composed of those kernels removed from each ear at each of the six levels of damage and then all remaining kernels. Cobs were weighed and % moisture was recorded for the kernels. In addition, data were recorded on the incidence and surface area of fungus, insect and bird damage, and abnormal kernel development.

Ear measurements

Damaged, undamaged, and bottom ears from each of the fifty-six experimental units were harvested 160 days after planting, husked, and placed in burlap bags. Bottom ears were subsequently ignored in all analyses because only nine poorly developed bottom ears were present on the 3360 plants from which the top ears were taken. Bags were placed in a corn drier (60–71 °C) and the ears dried for 10 days and then stored at room temperature (18–20 °C) for 14 days until ears reached an equilibrium moisture level between 9 and 11%. The damaged and undamaged samples from each unit were examined, shelled, and weighed in random order, from randomly selected plots.

Before net kernel weight was obtained for a sample, total weight, length of ear from cob butt to cob tip, and maximum kernel row length were recorded. A visual estimate was made of percentage surface area damaged and categorized as: abnormal kernel development; fungus; sprouting; insects; birds; simulated damage; and unknown. Before the undamaged sample was shelled, the kernels from the same location and pattern as were

TABLE 2. The activities associated with the design and data collection for the experiment

Activity	Date(s)	
Cornfield planted (Pioneer 3518)	1 May	1977
Ears silking	11 July	1977
Kernels at blister stage	21 July	1977
Damage at milk stage of kernel development	2 August	1977
Kernels at dough stage	4 August	1977
Damage at dough stage of kernel development	9 August	1977
Corn plants at physiological maturity	29 August	1977
Harvest of experimental units	6 & 7 October	1977
Commercial harvest of field	22 October	1977

previously destroyed in the damaged sample were removed from all thirty ears and weighed. After shelling the thirty ears, the following data were collected: total weight; kernel weight; cob weight; and % moisture. Net weight loss (hereafter called net loss) was computed as the difference between the kernel weight of the thirty-ear damaged sample and the thirty-ear undamaged sample for each plot. Expected loss, assuming no compensation, was obtained from the dry weight of the same number of kernels removed at the same ear locations after harvest from the undamaged thirty-ear samples. Table 2 summarizes the activities associated with the experiment.

RESULTS

Dry matter accumulation

Maximum dry matter accumulation in the cob occurred about 7 weeks and in kernels 10 weeks after silking (Fig. 1). At the time of milk- and dough-stage damage, 26 and 44%, respectively, of the dry matter had accumulated in all the kernels although only 19 and 41%, respectively, had accumulated in the damaged tip kernels (Table 3). Thus, from 81 to 59% of the dry matter that would have been incorporated into those kernels which were damaged was potentially still available for translocation into the remaining undamaged kernels.

Visual assessment of bird damage

In spite of attempts to minimize real bird damage, 6.5% of the ears in the undamaged sample showed bird or bird-like damage at the time of shelling. Surprisingly, 10% of the 1440 ears which had received simulated bird damage showed no sign of bird damage. Apparently low levels of damage, especially early in development, can be obscured by the growth of adjacent kernels. For example, 38 and 17% of the ears that had six kernels removed at the milk and dough stages, respectively, showed no visual evidence of damage at the time of shelling (Table 4). At harvest, the visual assessment underestimated both the incidence of damage (Table 4) and the kernel surface area damaged (Tables 3 & 4).

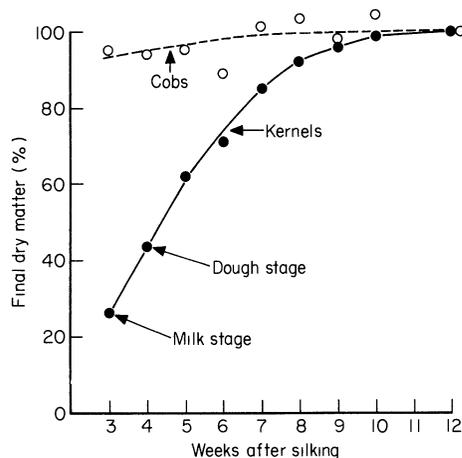


FIG. 1. Biomass accumulation in corn cobs and kernels after silking.

TABLE 3. Percentage of the total dry matter of corn contained in the damaged kernels and the percentage of total kernel dry matter potentially available for translocation after damage

No. of kernels damaged	% of total dry matter at maturity represented by damaged kernels	% of final dry matter accumulated in damaged kernels at time of damage		% of total kernel dry matter potentially available for translocation	
		milk	dough	milk	dough
0	0.00	—	—	—	—
3	0.42	16.56	40.09	0.35	0.25
6	0.86	18.17	39.95	0.70	0.52
12	1.80	18.76	40.48	1.46	1.07
24	3.79	19.13	39.68	3.06	2.29
48	7.79	20.15	40.88	6.22	4.61
72	12.05	21.78	42.20	9.42	6.96
Mean 23.6	3.82	19.09	40.55	3.54	2.62

TABLE 4. Visual assessment of % surface area of kernels damaged and % of ears showing bird-like damage by maturity level (undamaged samples in parentheses), 1977

No. of kernels damaged	Milk stage		Dough stage	
	% of kernel surface area damaged	% of ears showing damage	% of kernel surface area damaged	% of ears showing damage
0	0.15	20 (8)	0.02	3 (2)
3	0.32	49 (11)	0.38	73 (5)
6	0.55	62 (14)	0.58	83 (2)
12	1.27	94 (11)	1.40	96 (3)
24	2.25	99 (6)	2.61	100 (3)
48	6.24	100 (7)	7.74	100 (3)
72	9.59	100 (13)	11.07	100 (3)
Mean 23.6	2.91	75 (10)	3.40	79 (3)

Net loss

Net losses are shown for each damage and maturity level in Fig. 2. In only eleven of the fifty-six plots did the thirty damaged ears weigh more than undamaged ears. Expected losses are shown in Table 3. Analysis of variance of the difference between net loss and expected loss showed no significant effects for damage level ($F = 0.336$) or time of damage ($F = 0.044$).

This analysis indicated no significant compensation in kernel weight per plot following bird damage. Two confounding problems in the experimental design and the analysis became apparent during examination of the data; therefore, the conclusion of no compensation, based on the above analysis, remained tentative. First, the amount of secondary damage (fungus, sprouting, and insect loss) was influenced by the damage treatment. Second, the experimental design with paired thirty-ear samples of damaged and undamaged ears from each plot did not reduce sampling variation enough to allow sensitive statistical tests for low levels of compensation. The variation in the average kernel weight of each sample was largely due to the between ear variation in the thirty ears comprising the sample, therefore a more accurate method for predicting the weight of the damaged

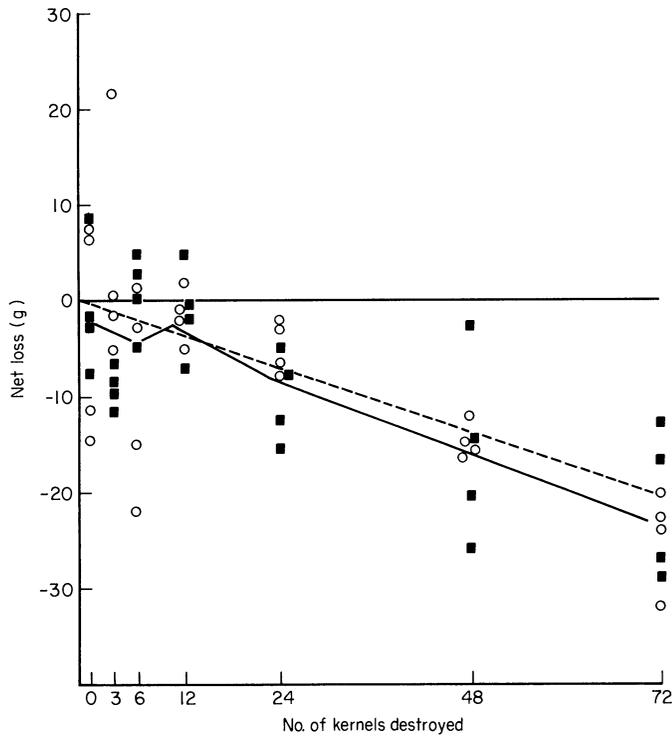


FIG. 2. Net loss of corn per ear (weight of thirty damaged ears minus thirty undamaged ears) for individual plots at each damage level. The circles represent milk-stage damage and the squares dough-state damage. The solid line is the average net loss for the eight plots at each damage level and the broken line is the expected loss based on the weight of kernels removed at harvest from the undamaged sample.

sample was needed. These two factors are now considered and then followed by a re-analysis for any compensatory response.

Incidence of secondary damage

The incidence of secondary damage differed between control and damaged ears at different damage and maturity levels (Table 5). Simulated bird damage increased the frequency of fungal infection, especially when damage was inflicted at the milk stage. Sprouting of kernels was increased only in ears having high simulated damage, twenty-four or more kernels removed, and was greatest for milk stage damage. The frequency of insect damage increased following simulated bird damage inflicted at the milk stage of maturity. The frequency of abnormal kernel development did not differ among treatments.

Since secondary damage was confounded with simulated damage it is of interest to estimate the kernel weight losses due to fungi, sprouting, or insect damage. The dry kernel weight of each thirty-ear sample was adjusted upward using a visual estimation of the proportion of the total kernel surface area showing secondary loss. In the damaged samples, secondary losses averaged 0.78% and 0.32% of the total kernel surface area at milk and dough stages, respectively, which was equivalent to 34.1 g (range 3.7 to 95.4 g) and 14.1 g (range 4.1 to 26.6 g) for the thirty-ear samples. For the undamaged samples, secondary losses averaged 0.19% of the total kernel surface area and 8.8 g (range 0.7 to 18.6 g) per sample.

TABLE 5. Percentage of ears with secondary damage following artificial bird damage

	Number of kernels removed	Damaged ears		Undamaged ears
		Milk	Dough	
Fungus	0-12	16.9*	6.7*	3.0
	24-72	25.3*	6.7*	
Sprouted kernels	0-12	4.8	6.2	4.2
	24-72	22.8*	10.8*	
Insect damage	0-12	15.0*	10.4	10.5
	24-72	15.0*	9.7	
Abnormal kernel development	0-12	1.7	1.9	2.2
	24-72	1.1	1.4	

* Significant difference in frequency from undamaged ears ($P < 0.05$).

Predicted weight of destroyed kernels

Because of the large sampling error in the estimated mean of each thirty-ear sample, instead of using the paired thirty control ears in each plot to determine what the kernel weight on the damaged ears would have been without damage, we used two characteristics of the damaged ears themselves (cob weight and kernel row length) to predict this kernel weight. This approach was prompted by the reported consistent relation between rachis weight (cob minus floral bracts) and kernel weight (Mangelsdorf 1974). Multiple regression revealed that the sum of the lengths (cm) of the longest row of kernels on each ear and the sum of the dry cob weights (g) for each thirty-ear undamaged sample predicted the undamaged adjusted kernel weight (g) by the equation: kernel weight = $-548.7 + 8.129 \times \text{kernel row length} + 2.305 \times \text{cob weight}$ ($n = 56$, $r = 0.923$, $s_{y \cdot x}^2 = 5999$). Using this relationship, we calculated the predicted kernel weight without any damage of each sample of thirty damaged ears, based on the kernel row lengths and cob weights of the damaged ears. Increasing damage levels did not change the maximum kernel row length ($r = 0.054$, slope = 0.026 , $P \simeq 0.34$) or the cob weight ($r = 0.130$, slope = 0.144 , $P \simeq 0.17$). The 95% confidence interval for a predicted kernel weight for a thirty-ear sample was ± 156.9 at the mean weight of 4630.1 g, or expressed as a percentage, $\pm 3.39\%$.

Net loss based on predicted kernel weight

Net loss in yield due to damage can now be recalculated as the difference between the regression predicted kernel weight of each thirty-ear sample and the observed kernel weight adjusted for secondary damage. Figure 3 shows the results for each damage and maturity level. The expected loss with no compensation is based on a regression equation kernel weight = $-6.85 + 7.58 \times \text{kernels removed}$, relating the weight of the same number of kernels removed at harvest from the undamaged sample with the number of kernels damaged ($n = 56$, $r = 0.999$, $s_{y \cdot x}^2 = 102.7$). Compared with Fig. 2, the scatter between plots was reduced indicating that the thirty-ear kernel weights predicted by regression were less variable than those based on paired control ear samples. The variance estimates, unexplained variation after linear regression with damage (residual mean square = $s_{y \cdot x}^2$), were significantly different for Figs 2 and 3 ($F = 53.09/6.85 = 7.76$ with 54,54 d.f., $P < 0.001$).

Analysis of variance indicated that the difference between net loss (predicted weight minus actual weight) and expected loss was somewhat influenced by maturity ($F = 2.417$,

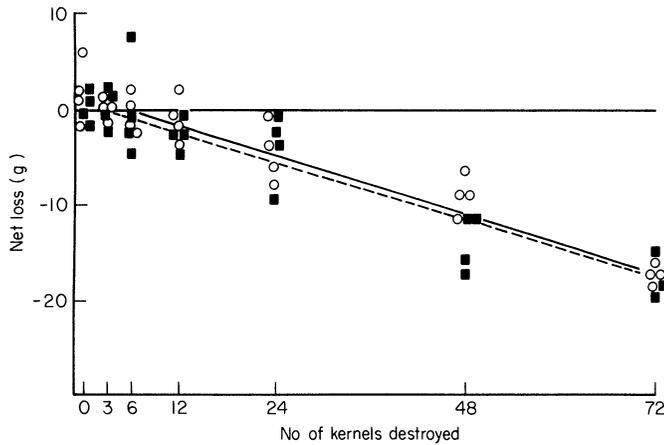


FIG. 3. Predicted net loss of corn per ear (difference between the regression predicted kernel weight of each thirty-ear sample and the actual kernel weight adjusted for secondary damage) for all damaged plots at each damage level. The circles represent milk-stage damage and the squares dough-stage damage. The solid line is the predicted average net loss for the eight plots at each damage level and the broken line is the expected loss based on a regression equation relating the weight of kernels removed at harvest from the undamaged sample with the number of kernels damaged.

$P \approx 0.124$) but not by damage level ($F = 0.250$). For milk stage of maturity, the difference between net loss and expected loss was 39.3 g (1.31 g per ear), significantly greater than zero ($t = 3.196$, $s_{\bar{d}} = 12.307$, $P < 0.005$, d.f. = 27). No increased compensation occurred with increasing damage. Because the net loss and expected loss were both based on predictive equations, the variance from individual predictions should be added to variance based on the twenty-eight differences observed. When this was done, the mean difference of +39.3 g was just significant ($t = 2.047$, $s_{\bar{d}} = 19.219$, $P \approx 0.052$, d.f. = 25). Therefore, slight partial compensation of dry weight did occur following simulated bird damage at the milk stage of maturity. An average of 39.3 g was recovered of the 171.9 g average kernel weight destroyed, or 22.9%. This partial compensation amounts to 28.3% of the amount available at milk stage for translocation. At the dough stage of maturity, the average compensation of 5.6 g (3.3%) was not significant.

DISCUSSION

We conclude that slight compensation of kernel weight that partially reduced net loss occurred following simulated bird damage to tip kernels. This compensation was probably accomplished through translocation into undamaged kernels of a portion of the biomass that would have been deposited in the damaged kernels; stimulation of a plant growth response is not necessary to account for the amount of weight regained. Compensation occurred only following damage in the milk stage, 22 days after silking. The average amount of compensation was calculated to be 23% of the average potential weight of the destroyed kernels. This was equivalent to 0.86% of the total kernel weight of damaged ears, a rather insignificant portion.

Although we found that compensation did occur, certainly it was small. This contradicts the earlier reports of Dyer (1975; 1976) where increased yield followed bird damage and is more in agreement with Duncan & Hatfield (1964). A statement of these latter

authors that 'kernels above and below partially developed kernels often appear to be abnormally large but later study showed that the actual weight difference was insignificant' was for the most part supported by our experiment.

Simulated bird damage, especially heavy damage early in kernel development, changed the incidence of fungus, sprouting, and insect damage that occurred before harvest. Quantification of secondary loss became necessary to separate its effect on yield from that directly due to bird damage. The magnitude of secondary loss, which in this study ranged up to 2.1% and averaged 0.78% of the kernel weight in plots damaged early in development, can certainly be as important as compensation which averaged 0.86% of the kernel weight. We now question the conclusions drawn from our analysis of the 1975 data as reported by Woronecki, Ingram & Dolbeer (1976). Fungus incidence in 1975 was lower whereas sprout and insect damage were higher in damaged ears. Unfortunately, although the incidence of secondary damage was recorded in that study, no estimate of the extent of secondary damage was made.

An important result shown clearly in the present study was that corn kernels contain only 20 to 40% of their final biomass at the time they are usually consumed by blackbirds (Fig. 1 and Table 3). Thus, a blackbird consuming 5 g (dry weight) of corn per day during the milk and dough stages would actually reduce final yield of corn by 12 to 25 g a day plus possible additional secondary loss. This important fact has been overlooked in bioenergetics studies that use feeding rates and numbers of birds to estimate the impact of blackbird populations on corn crops (Wiens & Dyer 1975; Williams 1975).

By way of summary, using the visual assessments of bird damage for the thirty damaged ears in each of these plots and other related data gathered in this study, we present the following example of bird damage estimation in corn. Our simulated damage field had 85.7% of the ears damaged, an average primary loss to birds of 23.6 kernels per ear. The birds would have consumed from 0.73 to 1.55% (milk and dough stage damage, respectively) of the final kernel weight which caused a loss averaging 3.82% of the final total kernel weight (Table 3). Because some compensation through translocation of dry matter occurred, 0.86 and 0.12%, respectively, damage was somewhat reduced to 2.96 and 3.70% of the total kernel weight. Visual assessment of damage at harvest estimated 75.0 and 79.0% of the ears damaged with an average kernel surface area loss of 2.91 and 3.40% for milk- and dough-stage damage, respectively (Table 4). Therefore for the distribution of bird damage and compensation observed in this study, the estimated weight loss based on visual estimation of kernel surface area damaged would underestimate actual loss only by 0.05 and 0.30% of the total kernel weight; the actual loss was 1.4 and 8.1% greater than visually estimated for early and late damage, respectively. Secondary loss, due to fungus and sprouting, increased the actual loss from 3.82% to 4.60% and 4.14% of the total dry weight for milk and dough stage damage respectively, and unless percentage surface area affected by secondary loss is also visually estimated, this would increase the difference between actual and estimated loss.

This example illustrates that primary bird damage estimates based on surface area of kernels destroyed are correlated with actual loss but the relationship can be affected by any environmental factors which influence the severity of secondary loss.

ACKNOWLEDGMENTS

We thank Gibbs Aero Sprayers, Fremont, Ohio; Schlessman Seed Co., Milan, Ohio; and Lepley Bros., Bellevue, Ohio, for cooperation and the services they provided. K. M. Cote,

B. F. Dotson, P. C. Kleinhenz, and K. L. Marquess helped collect and tabulate the data. D. Haugen and G. R. Rost provided field assistance.

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(Received 2 January 1980; revision received 10 June 1980)