Disease Resistance, Virus Concentration, and Agronomic Performance of Soybean Infected with Cowpea Chlorotic Mottle Virus

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The research was supported by state and Hatch funds allocated to the Georgia Agricultural Experiment Stations and by the University of Georgia Biotechnology Program.
Accepted for publication 14 October 1986 (submitted for electronic processing).

ABSTRACT

Paguio, O. R., Boerma, H. R., and Kuhn, C. W. 1987. Disease resistance, virus concentration, and agronomic performance of soybean infected with cowpea chlorotic mottle virus. Phytopathology 77:703-707.

Virus concentration and agronomic performance of six soybean genotypes with different levels of susceptibility and resistance to the soybean strain of cowpea chlorotic mottle virus (CCMV-S) were studied under field conditions. In general, symptoms of mosaic and stunt were milder in a 1984 experiment than in two experiments in 1985. Virus concentration also was lower in 1984, particularly in the resistant genotypes. No seed yield loss was detected in 1984; however, Davis, a susceptible genotype, had a loss of 19% in one 1985 experiment and plant introduction (PI) 96983 had an average loss of 37% in the two 1985 experiments. Low seed quality was observed in five genotypes in one or more experiments. Plant height was the agronomic character that was affected most frequently by virus infection; reductions varied from 13 to 42%, with all genotypes affected in at least one experiment. Lodging, seed weight, and maturity date were affected to a limited extent by CCMV-S. Virus concentration was not always directly related to seed yield losses and plant height reductions. Only 3–15% as much virus was produced in PI 96983 as in Davis, yet seed yield losses and plant height reductions were consistently greater in PI 96983. PI 346304 and Bragg were similarly resistant to seed yield losses caused by CCMV-S under field conditions, and they have different types of resistance. Bragg reacted with necrotic lesions at the site of infection, and extremely low quantities of virus occurred in uninoculated leaves. Very mild or no symptoms were produced on PI 346304, which had a low concentration of CCMV-S.

Cowpea chlorotic mottle virus (CCMV), a member of the bromovirus group, is one of several soybean viruses that have economic importance (9). The virus can reduce soybean seed yield (5) and also cause minor alterations in the quality and quantity of oil and protein in seeds (4). CCMV was isolated from soybean, *Glycine max* (L.) Merr., in 1967 (7). The soybean isolate (strain S) is serologically identical to the type strain of CCMV, but it differs by causing a mild mottle in cowpea, *Vigna unguiculata* (L.) Walp., instead of bright chlorosis, and it differs in other biophysical properties. The tripartite RNA genome of CCMV is encapsidated in icosahedral particles. The virus has a narrow host range, mainly in legumes, and it is transmitted mechanically and by beetles.

Resistance in soybean to CCMV-S was first reported by Harris and Kuhn (5). The resistance, relatively large necrotic lesions on inoculated leaves, was shown to be conditioned by an allele at a single locus that exhibited complete dominance (2). When the disease reaction of 533 soybean genotypes was evaluated, three new types of resistance to CCMV-S were found (1). Plant introduction (PI) 346304 represented a resistance category with low virus concentration in uninoculated trifoliolate leaves (less than 10% compared with susceptible lines) and no or extremely mild symptoms. A second resistance category, represented by cultivar Williams, had small, local necrotic lesions and no systemic symptoms, although virus was detectable in the uninoculated leaves. An intermediate resistance category (cultivar Jackson) was characterized by reduced virus concentration (less than 50% of

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susceptible genotypes), delayed systemic reaction, and little stunting. A complex virus-host interaction was found in PI 96983; virus concentration and disease reaction characteristics appeared to be related to both susceptible and resistant reactions.

The purpose of this study was to relate concentration of CCMV-S to disease reactions and agronomic characteristics in genotypes with the various types of resistance in field environments.

MATERIALS AND METHODS

Experimental plots. Three experiments were conducted at the Plant Sciences Farm near Athens, GA: early planting in 1984, early planting in 1985, and late planting in 1985. The soils were an Appling coarse sandy loam in 1984 and a Cecil coarse sandy loam in 1985. Both soils are members of the clayey, kaolinitic, thermic family of the Typic Hapludults. The experimental areas were fertilized according to the Georgia Agricultural Cooperative Extension Service recommendation for soybean. In 1984, potassium at 70 kg/ha was applied; no fertilizer was required in 1985. Herbicides were applied for weed control, and the soil was fumigated to control plant parasitic nematodes.

A 6 × 2 split-plot treatment design with four replications of a randomized complete block experimental design was used in each experiment. Main plots consisted of eight rows each of six soybean genotypes: Davis, Coker 237, Jackson, PI 96983, PI 346304, and Bragg. The rows were 6.1 m long and spaced 76 cm apart. Subplots consisted of four rows of each genotype that were either inoculated with CCMV-S or left uninoculated (control).

The experiments were planted on 28 May 1984, 17 May 1985, and 24 June 1985. The plots were seeded at a rate of 41 viable seeds per meter and thinned to 16 plants per meter by 21 days after planting. Supplemental soil moisture was provided by overhead irrigation. During pod development and seed fill, the experimental areas received at least 25 mm of water (rainfall + irrigation) every 7 days.

Virus inoculation. Inoculum was prepared by partial purification (one cycle of ultracentrifugation) of CCMV-S from cowpea cultivar Clay that had been infected 10 days (1). Inoculum concentration was 500 μg of virus per milliliter of 0.01 M potassium phosphate buffer (pH 7.0) for the 1984 experiment and 1,000 μg/ml for the two 1985 experiments. Celite (1%) was added to the inoculum, and plants were inoculated about 25 days after planting by mechanical inoculation with a cheesecloth pad moistened with inoculum. In 1984 the center leaflet only of the youngest expanded leaf was inoculated, whereas all three leaflets of a similar leaf were inoculated in 1985.

Insecticide treatment. Beginning 2–5 days after inoculation, the three experiments were sprayed weekly with a mixture of acephate (O,S-dimethyl acetylphosphoramidothioate; Orthene, 0.17 kg a.i./ha) and permethrin 3-(phenoxyphenyl) methyl (+)-*cis, trans*-3- (2,2-dichloroethenyl)-2,2-dimethyl cyclopropane carboxylate; Ambush, 0.03 kg a.i./ha). The insecticide applications continued until pod set, about 12 wk after planting.

Disease ratings. All plots were inspected for virus symptoms at 2, 6, and 10 wk after inoculation. The rating systems, which differed in 1984 and 1985, are given in Table 1. Plants with symptoms atypical of CCMV and plants in uninoculated plots with any viruslike symptoms were tested for virus infection by both mechanical inoculation and serology (immunodiffusion).

Virus quantification. Virus concentration in infected plants was determined by a method described previously (1). It involved partial purification by one cycle of ultracentrifugation and ultraviolet absorption analysis following density gradient centrifugation. Leaf tissue was selected from inoculated, nonharvest rows. Each sample consisted of the center leaflet from the five newest expanded leaves of 10 plants of Davis, Coker 237, and Jackson or 20 plants of PI 346304, PI 96983, and Bragg. In the 1984 experiment, concentration was determined at 4, 9, and 16 wk after planting. In 1985 the two experiments were sampled at the late vegetative growth stage, about 11 wk after inoculation.

Uninoculated control plots were also sampled periodically. They were tested serologically (immunodiffusion and enzyme-linked immunosorbent assay) for the presence of CCMV and tobacco ringspot virus and by infectivity for any other virus.

Agronomic characters. Data for agronomic characters were collected from a 3.6-m section of the two middle rows of each subplot after removing 0.5 m of plants from each end of a plot at maturity. Seed yield was measured by mechanical harvesting and weighing of seed that had been air-dried and adjusted to 13% moisture. Plant height was measured at maturity from the ground level to the tip of the main stem of four plants per plot. Lodging was rated on a scale of 1 (all plants upright) to 5 (all plants prostrate). Seed weight was determined on a sample of 100 seeds, and seed quality was judged as very good to very poor on the basis of amount and degree of wrinkling, defective seed coats, greenishness, and moldy or rotten seeds. Maturity was determined as the date on which 95% of the pods were their mature pod color.

Temperature study. After inoculation with CCMV-S (100 μg/ml), PI 96983 plants were maintained under three environmental conditions: 24 C with a 16-hr photoperiod, 30 C with a 16-hr photoperiod, and in the greenhouse with a temperature range of 22–33 C. For the controlled temperatures, illumination with both incandescent and fluorescent lights was about 10,000 lux. Virus concentration was determined in inoculated unifoliate leaves and in uninoculated trifoliolate leaves. There were four replications per treatment, each consisting of one 10-cm-diameter pot with three plants.

TABLE 1. Disease ratings of field-grown genotypes infected with cowpea chlorotic mottle virus[a]

Genotype	1984[b] test	1985 early planting		1985 late planting	
		Mosaic[c]	Stunt[d]	Mosaic	Stunt
Davis (S)[e]	2.2	3.0	1.9	3.0	1.0
Coker 237 (MR)	2.0	3.0	2.2	2.2	1.4
Jackson (MR)	1.0	2.2	1.8	2.0	1.1
PI 96983 (R)	3.8	1.9	3.0	0.5	3.0
PI 346304 (R)	0.0	0.6	0.5	0.4	0.0
Bragg (HR)	0.0	0.0	0.5	0.0	0.0
LSD (0.05)	0.3	0.2	0.4	0.2	0.2

[a] Ratings were made about 10 wk after planting.
[b] 1984 rating system: 0 = no symptoms; 1 = mosaic, no stunt; 2 = mosaic, mild stunt; 3 = mosaic, moderate stunt; and 4 = mosaic, severe stunt.
[c] Mosaic rating: 0 = no symptoms, 1 = mild mosaic, 2 = mosaic, and 3 = severe mosaic.
[d] Stunt rating: 0 = no stunt, 1 = stunting less than 10%, 2 = stunting between 10 and 25%, and 3 = stunting more than 25%.
[e] Reaction to cowpea chlorotic mottle virus based on virus accumulation: S = susceptible, MR = moderately resistant, R = resistant, and HR = highly resistant.

RESULTS

Disease development. Mosaic and stunt symptoms caused by CCMV-S were generally milder in the 1984 experiment than in the two 1985 experiments. For example, the mosaic on Davis, Coker 237, and Jackson was more severe in 1985 than in 1984, and PI 346304 had no symptoms in 1984 and a mild mosaic in 1985 (Table 1). The inoculum caused fewer infection centers (number of necrotic lesions) on the inoculated leaves of Bragg in 1984 than in 1985. Five to 10 lesions per leaflet were observed in 1984, and 50–200 per leaflet in 1985. Regardless of the cause, symptoms were mild throughout the growing season in 1984 and much more pronounced in 1985. Furthermore, relative symptom severity was distinctive for each of the soybean genotypes and similar to the greenhouse tests reported earlier by Bijaisoradat and Kuhn (1).

Four days after inoculation, chlorotic spots were observed on the inoculated leaves of Davis, Coker 237, Jackson, and PI 346304; necrotic lesions in Bragg; and diffuse necrosis on PI 96983. Shortly thereafter, mosaic and/or chlorotic stippling developed on the uninoculated newer leaves on all genotypes except Bragg. Furthermore, at 2–3 wk after inoculation, stunting was noted on PI 96983 plants in 1984 and on all six genotypes in both experiments in 1985.

From 4 to 10 wk after inoculation, leaf discoloration symptoms

were relatively similar within genotypes. Mosaic was strongest in Davis, slightly milder on Coker 237 and Jackson, mild on PI 96983, very mild or absent on PI 346304, and absent on Bragg (Table 1). Severe stunt was observed on PI 96983 in all experiments (Table 1). Leaves on this genotype exhibited veinal necrosis and epinasty. Moderate stunting was noted on Davis, Coker 237, and Jackson and mild stunting on PI 346304 and Bragg in one or two experiments in 1985 (Table 1).

Virus concentration. In 1984, the relative virus concentration was similar in the uninoculated leaves sampled at 27, 46, and 56 days after inoculation within genotypes (Table 2). Virus concentration was highest in Davis and was substantially reduced in Coker 237 (38%), Jackson (10%), PI 96983 (5%), and PI 346304 (1%). No virus was detectable in Bragg.

In the two 1985 experiments, all soybean genotypes had more virus than they did in 1984 (Table 2). The increase in Davis was about twofold; it was 5–47 times greater in Coker 237, Jackson, PI 96983, and PI 346304. CCMV-S was clearly detectable in uninoculated, symptomless leaves of Bragg in both experiments in 1985. Davis and Coker 237 had nearly seven times more virus than PI 96983 and PI 346304, and Jackson was intermediate between the two extremes.

Virus in uninoculated plots. During the visual inspections, a few plants suspected of being infected with a virus were noted; however, none of the plants had CCMV-like symptoms. Sap extracts from the suspect plants were used to inoculate Bragg and Davis soybeans, California Blackeye and PI 186465 cowpeas, and *Phaseolus vulgaris* L. 'Bountiful' and 'Topcrop'. No plants were detected with CCMV-S. Sap from one plant caused symptoms on all six test hosts, and tobacco ringspot virus was identified by an immunodiffusion serology test. We conclude that CCMV-S did not spread from inoculated to uninoculated plants and that viruses other than CCMV-S did not affect any of the results of these field studies.

Agronomic performance. Eighteen comparisons (six genotypes × three experiments) between uninoculated (control) and inoculated plants were made for each of six agronomic characters (Table 3). The CCMV-S infection of the six genotypes had a significant ($P < 0.05$) effect on plant height (14 of 18 comparisons) more frequently than on the other characters. Seed quality and maturity date were different in 7 of 18 comparisons, lodging and seed weight in 6 of 18, and seed yield in 3 of 18. In the 1984 experiment, the virus infection caused a difference in about 20% of the 36 total comparisons. The effect of the virus was greater in both experiments in 1985, with about 50% of the comparisons being different.

TABLE 2. Concentration of cowpea chlorotic mottle virus in soybean genotypes grown in the field (three experiments)[a]

Genotype	1984 experiment[b]			1985 experiments[c]	
	27 days	46 days	56 days	Early planting	Late planting
Davis (S)[d]	152	148	175	380	204
Coker 237 (MR)	77	21	83	365	217
Jackson (MR)	30	3	13	301	59
PI 96983 (R)	4	10	...[e]	49	30
PI 346304 (R)	1	1	...	67	27
Bragg (HR)	ND[f]	ND	ND	<1	<1
LSD (0.05)	...	...	...	75	52

[a] Given as micrograms of purified virus per gram fresh weight of leaf tissue.
[b] Plots were seeded on 28 May 1984; virus concentration was determined at three dates after inoculation on 21 June.
[c] Early-planting experiment seeded on 17 May 1985 and late-planting experiment on 24 June 1985; virus concentration was determined at 48 and 55 days after inoculation, respectively.
[d] Reaction to cowpea chlorotic mottle virus (see Table 1).
[e] No evaluation.
[f] Not detectable.

TABLE 3. Agronomic performance of six soybean genotypes infected with cowpea chlorotic mottle virus and grown in three field experiments

Genotype	Seed yield (kg/ha)[a]		Plant height (cm)		Lodging (rating)[c]		Seed weight (g/100 seeds)		Seed quality (rating)[d]		Maturity (date)[e]	
	C[b]	I	C	I	C	I	C	I	C	I	C	I
1984 experiment												
Davis (S)[f]	2,742	2,930	112*[g]	102	2.9*	2.1	15.5	16.1	1.5	1.5	10-14	10-15*
Coker 237 (MR)	3,098	3,125	104*	89	2.0*	1.6	15.0	14.5	1.6	1.6	10-15	10-16*
Jackson (MR)	2,822	2,802	109	109	2.1	1.8	16.4	16.8	1.6	1.8	10-19	10-19
PI 96983 (R)	2,325	2,164	81*	66	1.8	1.5	20.4	19.3	2.4	2.4	9-25	9-26
PI 346304 (R)	1,962	1,929	132	140	4.0	3.9	10.7	11.0	1.6	1.5	10-19	10-19
Bragg (HR)	3,353	3,286	119	119	2.1	2.1	16.2	16.0	1.5	1.5	10-18	10-18
LSD (0.05)[h]	462		12		0.47		1.2		0.29		2.1	
1985 early planting												
Davis	2,789*	2,251	122*	89	3.2*	2.5	16.4*	14.3	2.6	2.6	10-14	10-15
Coker 237	2,567	2,762	117*	76	1.8	1.5	16.3*	14.9	2.2	2.4	10-16	10-20*
Jackson	2,446	2,103	131*	95	1.6	1.6	16.9*	14.1	2.2	2.5*	10-29	10-31*
PI 96983	2,406*	1,438	89*	52	2.1	2.1	20.5*	15.2	3.8	4.5*	9-25	9-28*
PI 346304	1,693	1,371	142	138	4.0*	3.5	10.6	9.8	1.8	1.8	10-16	10-16
Bragg	2,507	2,177	131*	115	2.4	2.1	16.6	16.5	2.0	2.0	10-20	10-20
LSD (0.05)	403		11		0.41		1.4		0.29		2.4	
1985 late planting												
Davis	2,406	2,298	112*	73	3.9	3.5	13.1	12.8	2.0	2.4*	10-18	10-18
Coker 237	2,675	2,453	96*	66	2.4*	1.6	14.1*	13.3	2.0	2.6*	10-16	10-17
Jackson	2,681	2,547	105*	79	2.5	2.1	15.4	14.8	2.1	2.9*	10-29	11-2*
PI 96983	2,130*	1,404	86*	50	3.6*	2.5	19.0*	13.6	2.8	4.0*	10-6	10-9*
PI 346304	2,003	2,137	137*	119	4.5	4.1	11.4	10.9	1.5	2.1*	10-29	10-29
Bragg	2,547	2,675	114*	94	3.5	3.8	15.3	15.8	2.1	2.1	10-22	10-23
LSD (0.05)	343		10		0.53		0.9		0.30		2.2	

[a] Adjusted to 13% moisture.
[b] C = uninoculated control, I = infected.
[c] 1 (all plants upright) to 5 (all plants prostrate).
[d] 1 (very good) to 5 (very poor).
[e] Month and day of month.
[f] Reaction to cowpea chlorotic mottle virus (see Table 1).
[g] Asterisks indicate significant difference between inoculated and control plots within genotype based on LSD (0.05).
[h] For comparison of all means in both columns within each of three experiments.

TABLE 4. Effect of environmental conditions on symptoms and virus concentration in soybean plant introduction 96983 infected with cowpea chlorotic mottle virus

Growth conditions	Symptoms[a]		Virus concentration (μg/g)[b]	
	Unifoliate[c]	Trifoliate[c]	Unifoliate	Trifoliate
24 C[d]	C, DN, VN	None	37 ± 5[e]	<1
30 C[d]	C, NLL, VN	E, M, SN, SS	175 ± 3	22 ± 1
Greenhouse	C, VN	E, M, SS	406 ± 91	24 ± 2
Field	C, VN	E, M, SS	...	23[f]

[a] C = chlorosis, DN = diffuse necrosis, E = epinasty, M = mottle, NLL = necrotic local lesions (expanding type), SN = systemic necrosis (sometimes death), SS = severe stunt, and V = vein necrosis.
[b] Micrograms of virus per gram (fresh weight) of leaf tissue.
[c] Inoculated unifoliate leaves; systemically infected trifoliate leaves.
[d] Plants grown in growth chambers.
[e] $\bar{x} \pm$ SE.
[f] Average of four shown for PI 96983 in Table 2.

Seed yield was reduced by CCMV-S infection in only two of the six genotypes (Table 3). Yield of PI 96983 was reduced an average of 37% in two of the three experiments, and Davis yield was reduced 19% in one experiment. Plant height of Davis (average 24% reduction), Coker 237 (27%), and PI 96983 (34%) was reduced in all experiments; Jackson (26%) and Bragg (15%) in two experiments; and PI 346304 (13%) in one experiment. Virus infection reduced lodging in four soybean genotypes in one experiment. Seed weight was reduced about 27% in PI 96983 in two experiments, and lesser weight reductions were noted in Davis, Coker 237, and Jackson in one experiment. No seed weight reduction was detected in PI 346304 and Bragg. All soybean genotypes except Bragg had reduced seed quality when the seed came from infected plants in the 1985 late-planting experiment. Low seed quality was noted also in Jackson and PI 96983 in one other experiment. Maturity date was delayed 1–4 days in four of the six genotypes, with no delay being observed with PI 346304 and Bragg.

Effect of environment on PI 96983. Inoculated leaves of PI 96983 had some degree of necrosis but no discrete necrotic local lesions, regardless of temperature conditions after inoculation (Table 4). Uninoculated trifoliolate leaves had no symptoms at 24 C but strong symptoms at 30 C, in the greenhouse, and in the field. Virus concentration in PI 96983 was low, medium, and high in inoculated leaves at 24 C, 30 C, and in the greenhouse, respectively (Table 4). Virus was barely detectable (less than 1 μg/g of tissue) in systemically infected leaves at 24 C, and the concentration was similarly low at 30 C, in the greenhouse, and in the field (Table 4).

DISCUSSION

Davis was selected as a susceptible control for these studies with soybean genotypes resistant to CCMV-S because it is highly susceptible to the virus (1), and a study in 1968–69 showed an average seed yield reduction of 27% (5). In the current study, seed yield in Davis was reduced (19%) in only one of three experiments. It appears that the host is tolerant to seed yield reduction under some growing conditions. Tolerance, as used here, is defined as a negligible response (yield loss) in a host with relatively high virus-concentration levels and relatively unrestricted movement of the virus (10). Irrigation was provided in the 1984–85 tests, and seed yield and plant height of uninoculated plants were 40 and 30% greater, respectively, than in the 1968–69 tests. We speculate that more favorable environmental conditions existed for the current study and that soybeans are able to compensate for the virus infection.

On the basis of virus concentration and movement, cultivars Coker 237 (Table 2) and Jackson showed some resistance to CCMV-S (1). The moderate level of resistance appeared to be sufficient to prevent seed yield reduction under favorable growing conditions.

In PI 96983, disease severity was not directly related to virus concentration, particularly when compared with the reactions in susceptible and moderately resistant genotypes. At the relatively low temperature of 24 C, the virus concentration levels in PI 96983 were similar to those in the resistant, necrotization reaction in Bragg (low virus concentration in inoculated leaves and barely detectable levels in symptomless uninoculated leaves). At 30 C, however, the systemic disease reaction was very severe, similar to the reaction in the field, and virus concentration was low. Although PI 96983 may be resistant to CCMV-S under some environmental conditions, it is an undesirable genotype in geographic areas where temperatures frequently exceed 30 C. The nature of the CCMV-S/PI 96983 interaction is complex and may be related to other virus/host interactions in which local necrotization is followed by severe symptoms, usually necrosis in systemically infected leaves (3,6).

Resistance to CCMV-S in genotypes PI 346304 and Bragg appeared to be highly effective. It is significant that the type of resistance differed in the two genotypes. Local necrotization and very low virus concentration levels occurred in Bragg, whereas low virus concentration levels without necrotization occurred in PI 346304. It seems highly probable that these two types of resistance are controlled by different host genes, and it may be desirable to incorporate both types into new soybean breeding lines. The importance of multiple resistance genes is emphasized by the recent discovery of a new strain (N) of CCMV that causes a susceptible reaction (no necrosis, high virus concentration, and systemic mosaic symptoms) in Bragg (8). On the other hand, PI 346304 remains resistant, with a low concentration of CCMV-N.

In general, the relative concentration of CCMV-S in susceptible and resistant soybean genotypes was similar in these field studies to the concentration found in greenhouse studies (1). With the exception of Coker 237, genotypes with resistance consistently had less virus than Davis. Similar to the greenhouse studies (1), virus was not restricted to necrotic local lesions in Bragg in the field. In the two 1985 experiments, CCMV-S was detectable in uninoculated, symptomless infected leaves of Bragg. Virus concentration was similar from the early to late vegetative growth stages of soybeans (27–56 days after inoculation; Table 2). Furthermore, the relative position of the susceptible and resistant genotypes remained the same during that period. Virus concentration in the field plants differed somewhat among our three experiments. Virus concentrations in all genotypes were higher in 1985 than in 1984, and differences were more noticeable in the resistant genotypes than in susceptible Davis. We speculate that the number of infection centers (number of necrotic lesions on Bragg) may be responsible for the differences between 1984 and 1985.

Although seed yield was not consistently reduced in the susceptible genotype, plant height was routinely reduced in the moderately resistant genotypes and sometimes in the resistant ones (Table 3). It appears that plant size (amount of foliage), within certain limits, does not affect seed yield in CCMV-S infected soybeans. Seed quality, seed weight, and maturity date were also affected negatively by the virus infection. These factors should not be ignored when the effects of CCMV-S on soybean are considered.

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