PLANT RESISTANCE

Hypersensitive Response of Beans to *Apion godmani* (Coleoptera: Curculionidae)

RAMON GARZA,[1] JORGE VERA,[2] CESAR CARDONA,[3] NINA BARCENAS,[4] AND SHREE P. SINGH[5]

Centro Internacional de Agricultura Tropical, CIAT, Apartado Aéreo 6713, Cali, Colombia

J. Econ. Entomol. 94(4): 958–962 (2001)

ABSTRACT High levels of resistance to *Apion godmani* Wagner have been reported in bean, *Phaseolus vulgaris* L., landraces from Mexico. We report on the role of hypersensitivity to *A. godmani* in five resistant and three susceptible bean genotypes. In susceptible genotypes (cultivars 'Canario 107', 'Jamapa', and 'Zacatecas 45'), the eggs and first instars of *A. godmani* were embedded in the pod mesocarp and usually were surrounded by healthy tissue. In contrast, in resistant landraces ('Amarillo 154', 'Amarillo 155', 'J-117', 'Puebla 36', and 'Pinto 168'), necrotic tissues developed concentrically around the oviposition site, encapsulating eggs and dead larvae. An inverse relationship between percentage egg and larval encapsulation at the early immature pod stages and percentage of damaged seeds at harvest was found. Results indicate that hypersensitivity in developing pods plays an important role in antibiosis to *A. godmani* in beans. This information will facilitate future genetic and biochemical research and provide much needed information concerning the phenotypic basis of resistance to *A. godmani* in bean.

KEY WORDS *Apion godmani*, *Phaseolus vulgaris*, host plant resistance, hypersensitivity, antibiosis

Apion godmani WAGNER is a major pest of beans, *Phaseolus vulgaris* L., in Mexico, Guatemala, Honduras, El Salvador, and northern Nicaragua (McKelvey et al. 1947, Mancía 1973). *A. godmani* larvae feed on immature seeds inside developing pods of beans. Thus, seed yield, seed quality, seed germination, and market value are all adversely affected. The economic importance of this pest varies considerably (Cardona 1989) depending on the cultivar, sowing time, cropping system, agronomic practices used, prevailing environmental conditions, and infestation levels. Yield losses can be as high as 90% in some endemic areas in the highlands of Mexico and Guatemala and in mid-altitude valleys of El Salvador and Honduras (Cardona 1989, Garza 1998).

Cardona (1989) summarizes the biology of *A. godmani*. Adults usually appear soon before or at flowering and cause light feeding damage to leaves and flowers before mating. Eggs are laid in the mesocarp of developing pods (1–4 cm long). The adult female chews a small hole in the mesocarp of the pod, usually above the developing seed, and lays a white semitranslucent egg that hatches in 8–9 d. There are three larval instars that in total last 19 d. First instars burrow through the pod wall searching for the developing seed, and all three instars feed on the developing seeds. One larva per infested seed is normal, but three to five larvae per seed may be found when infestations are very high. The last instar forms a pupation chamber inside the pod. The pupal stage lasts an average of 10 d, and newly emerged adults disperse to forested areas until returning when the next bean crop is about to flower. There is one generation per cropping season.

As discussed by Cardona and Kornegay (1999), host plant resistance may be the only sustainable approach to the integrated management of this pest, especially in areas where beans are grown by resource-poor farmers. Repeated efforts have been made to screen for resistance to *A. godmani*. McKelvey et al. (1947) identified sources of resistance in bean accessions from Mexico. Some of these were later reconfirmed by Guevara (1961) in Mexico and by Mancía (1973) in El Salvador. Additional sources of resistance were identified in wild *P. vulgaris* germplasm (Acosta et al. 1992) and in cultigens (Beebe et al. 1993, Garza and Muruaga 1993). More recently, new accessions from Mexico with high levels of resistance were found by Garza et al. (1996). Considerable progress has been made in breeding for resistance to *A. godmani* in Central America (Beebe et al. 1993) and in Mexico (Garza et al. 1996).

Advances have also been made in the understanding of the mechanisms of resistance to *A. godmani*. The general mechanism of resistance in most beans is antibiosis, but ovipositional antixenosis may also play a

[1] Programa de Entomología, CEVAMEX, INIFAP, Apartado Postal No. 10, Chapingo, Estado de Mexico 56230, Mexico.
[2] Instituto de Fitosanidad, Colegio de Postgraduados, 56230, Montecillo, Estado de Mexico, Mexico.
[3] Centro Internacional de Agricultura Tropical, CIAT, A. A. 6713, Cali, Colombia.
[4] Instituto de Recursos Genéticos y Productividad, 56230, Montecillo, Estado de Mexico, Mexico.
[5] University of Idaho, 3793 North 3600 East, Kimberly, ID, 83341–5076. Published as Idaho Agricultural Experiment Station Journal Article No. 00705.

0022-0493/01/0958–0962$02.00/0

major role in some accessions
case of antibiosis, preliminary
dona (unpublished data) in Hor
1992) in Mexico suggested that h
be an important mechanism to
of damage to the seeds in resist
observed in resistance screenin
tant accessions seemed to resp
forming a callus that could imp
of the pod wall.

According to Fernandes (199
few examples of hypersensitivity
anism to phytophagous insects.
examples include resistance to
beetles, adelgids, and wood was
the resistance of wheat to Hes
1992, Grover 1995), of mustard
and DeVay 1987), and of potato
beetle (Balbyshev and Lorenze
we present evidence of hyper
cessions resistant to *A. godmar*

Materials and M

Plant Materials and Resista
bean accessions previously ide
godmani (Garza 1992, Beebe
1996) were used in this study:
155', 'J-117', 'Puebla 36', and
landraces originating from di
Mexican highlands and differ
agronomic characteristics (G
168 possesses characteristics
rango, and all others belong t
al. 1991). Bean cultivars 'Jam
'Canario 107' were chosen
(Garza et al. 1996). 'Jamapa'
g/100 seeds) black bean; 'Zac
seeded (25–40 g/100 seeds) b
107' is a large-seeded (>45 g
These cultivars belong to be
Durango, and Nueva Granad
al. 1991).

The five resistant and three
were screened for *A. godma*
domized complete block de
Each test plot consisted of
with a spacing of 10 cm bet
and 85 cm between rows. Be
rainfed conditions in late Ma
October. No fertilizers were
trolled by hand. The field
ducted at the Santa Lucía de
the Mexican Instituto Naci
Forestales y Agropecuarias
during the 1994–1996 croppi
of the site is 2,250 m and me
670 mm, mostly distributed
ber. Mean annual temperatu
are 16°C and 70%, respecti
only major pest observed ca
plants during the experimer

major role in some accessions (Garza 1998). In the case of antibiosis, preliminary observations by Cardona (unpublished data) in Honduras and by (Garza 1992) in Mexico suggested that hypersensitivity might be an important mechanism to explain the low levels of damage to the seeds in resistant genotypes. It was observed in resistance screening nurseries that resistant accessions seemed to respond to oviposition by forming a callus that could impede larval penetration of the pod wall.

According to Fernandes (1990), there are relatively few examples of hypersensitivity as a resistance mechanism to phytophagous insects. The best-documented examples include resistance to galling insects, bark beetles, adelgids, and wood wasps. Other examples are the resistance of wheat to Hessian fly (Shukle et al. 1992, Grover 1995), of mustard to *Pieris* spp (Shapiro and DeVay 1987), and of potato to the Colorado potato beetle (Balbyshev and Lorenzen 1997). In this arcticle we present evidence of hypersensitivity in bean accessions resistant to *A. godmani.*

Materials and Methods

Plant Materials and Resistance Evaluations. Five bean accessions previously identified as resistant to *A. godmani* (Garza 1992, Beebe et al. 1993, Garza et al. 1996) were used in this study: 'Amarillo 154', 'Amarillo 155', 'J-117', 'Puebla 36', and 'Pinto 168'. All five are landraces originating from different locations in the Mexican highlands and differ in seed type and other agronomic characteristics (Garza et al. 1996). Pinto 168 possesses characteristics of the bean race Durango, and all others belong to race Jalisco (Singh et al. 1991). Bean cultivars 'Jamapa', 'Zacatecas 45', and 'Canario 107' were chosen as susceptible checks (Garza et al. 1996). 'Jamapa' is a small-seeded (<25 g/100 seeds) black bean; 'Zacatecas 45' is a medium-seeded (25–40 g/100 seeds) beige bean; and 'Canario 107' is a large-seeded (>45 g/100 seeds) beige bean. These cultivars belong to bean races Mesoamerica, Durango, and Nueva Granada, respectively (Singh et al. 1991).

The five resistant and three susceptible bean entries were screened for *A. godmani* damage using a randomized complete block design with six replicates. Each test plot consisted of one row, 12 m in length, with a spacing of 10 cm between plants within rows and 85 cm between rows. Beans were planted under rainfed conditions in late May and harvested in early October. No fertilizers were added; weeds were controlled by hand. The field experiments were conducted at the Santa Lucía de Prías Research Station of the Mexican Instituto Nacional de Investigaciones Forestales y Agropecuarias (INIFAP) near Texcoco during the 1994–1996 cropping seasons. The elevation of the site is 2,250 m and mean annual precipitation is 670 mm, mostly distributed between May and October. Mean annual temperature and relative humidity are 16°C and 70%, respectively. *A. godmani* was the only major pest observed causing damage to the bean plants during the experiments. Occasional low populations of the Mexican bean beetle, *Epilachna varivestis* Mulsant (Coleoptera: Coccinellidae) were removed by hand, and no pesticides were applied.

To quantify the incidence of hypersensitive reactions to *A. godmani* oviposition, five pods (3 cm long) per replication were collected at random every 3 d for a period of 27 d to obtain a sample size of 50 pods per plot. Sampling was initiated soon after the onset of flowering in late June, which is when adults become active in the field. The pods were taken to the laboratory and examined carefully under a binocular stereoscope for the presence of oviposition punctures., the pods were dissected longitudinally with a scalpel to remove the pericarp and expose the eggs or first instars present in each oviposition site. Those surrounded by necrotic tissue were recorded as encapsulated. When no egg or larva was found at the oviposition site, the mesocarp was carefully dissected to detect first instars burrowing within the pod. Dead or living larvae surrounded by necrotic tissue were recorded as encapsulated.

To measure resistance levels, a random sample of 30 pods per plot was taken at crop maturity. Each pod was examined by carefully opening the pod along the ventral suture and removing each seed, which was then checked for *A. godmani* damage. The numbers of damaged seeds and total seeds were counted, and the percentage of damaged seeds calculated. Accessions were classified as susceptible (>50% damaged), intermediate (30–50% damaged), or resistant (<30% damaged) as described by Garza et al. (1996).

Statistical Analyses. All data were analyzed using SAS (SAS Institute 1988). Data were transformed to arcsine $\sqrt{\text{proportion}}$ and analyzed using the general linear model (GLM) procedure. Linear contrasts were used to test for differences in percentage encapsulation and percentage damaged seeds between susceptible and resistant groups. Untransformed means are presented.

Results

Careful examination of infested pods revealed striking differences for *A. godmani* damage among bean genotypes. In susceptible reactions, the eggs of *A. godmani* were embedded in the mesocarp of the pod and were usually surrounded by healthy tissue (Fig. 1). In hypersensitive reactions, eggs were surrounded (encapsulated) by necrotic tissue developed concentrically around the oviposition site. There was partial to complete autolysis of the cells surrounding the egg; this was accompanied by cellular hypertrophy and the development of masses of amorphic, granular tissue in the area surrounding the oviposition site. Eggs within this callus appeared normal, and no evidence was found of physical damage (crushing or shriveling) as a result of tissue proliferation. Hatching did not seem to be affected by pod tissue proliferation as evidenced by the high proportion (up to 70%) of first instars found encapsulated within the callus. This suggests that resistance may be caused by the extensive proliferation of tissue in infested pods, which trapped first

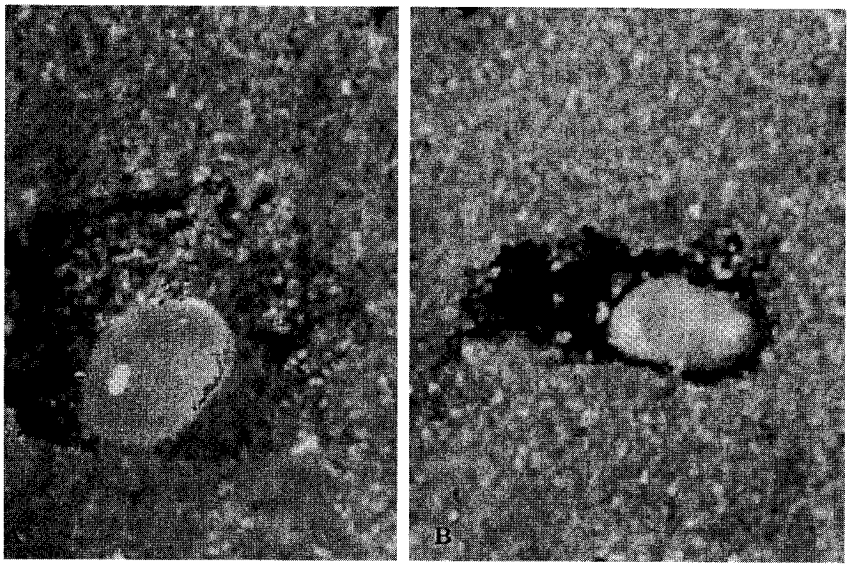

Fig. 1. *Apion godmani* eggs: embedded in mesocarp of a susceptible bean host (A); encapsulated by necrotic tissue in mesocarp of a highly resistant bean host (B).

instars so that death ensued. None of the encapsulated larvae showed signs of physical damage and appeared to have died by starvation, but we do not have evidence of this. Further examination of infested pods revealed that in highly resistant accessions, like 'J-117' and 'Amarillo 155', some first instars managed to enter the pod wall in search of developing seeds. Most of these larvae were dead and encapsulated by necrotic tissue.

Table 1. Percentage encapsulation of early immature stages (eggs and first instars) of *A. godmani* in the mesocarp of pods of selected bean accessions

Genotype	1994	1995	1996
	Resistant genotypes[a]		
Amarillo 154	29.2 ± 6.5	48.2 ± 6.0	—[ab]
Amarillo 155	33.4 ± 5.8	39.3 ± 5.8	30.8 ± 6.6
J-117	67.1 ± 7.9	52.5 ± 6.2	81.1 ± 8.4
Puebla 36	—	47.7 ± 3.8	8.0 ± 3.2
Pinto 168	—	30.7 ± 2.6	33.6 ± 4.1
Mean	43.2 ± 9.6[a]	43.7 ± 3.3[a]	38.4 ± 10.9[a]
	Susceptible genotypes		
Jamapa	5.0 ± 3.3	8.2 ± 0.7	4.9 ± 1.7[a]
Zacatecas 45	2.1 ± 0.8	3.1 ± 0.8	2.1 ± 1.0
Canario 107	2.6 ± 1.6	1.0 ± 0.8	1.1 ± 0.4
Mean	3.2 ± 0.7[b]	4.1 ± 1.7[b]	2.7 ± 0.9[b]

Means ± SEM of 6 replications per genotype, 50 pods per replication. Means within a column followed by different letters are significantly different. Mean separation by Scheffe's F method of significance testing for arbitrary linear contrasts ($P < 0.05$). ANOVA on transformed data testing for differences among genotypes: 1994: $F = 22.9$; df = 5, 25; $P < 0.01$; 1995; $F = 8.7$; df = 7, 35; $P < 0.01$; and 1996: $F = 26.4$; df = 6, 30; $P < 0.01$. ANOVA results for linear contrasts of resistant vs. susceptible genotypes: 1994 - $F = 17.6$; df = 1, 25; $P < 0.01$. 1995 - $F = 4.6$; df = 1.35; $P < 0.05$. 1996 - $F = 4.2$; df = 1, 30; $P < 0.05$.

[a] Based on previous studies (Beebe et al. 1993; Garza et al. 1996; Garza, unpublished data).

[b] Not included in trial because of lack of seed.

Analysis of percentage encapsulation of eggs and/or first instars for each year of testing (Table 1) revealed significant differences among genotypes, and results of linear contrasts showed that the role of encapsulation was greater in resistant genotypes than in susceptible ones. The trend between susceptible and resistant groups was consistent among years. Mean percentage encapsulation ranged from 1.0% in 1995 for 'Canario 107', a highly susceptible check, to 81.1% in 1996 for 'J-117'. 'J-117' is the most resistant accession tested to date for resistance to *A. godmani* (Garza et al. 1996).

The analysis of percentage seed damage at harvest for each year of testing (Table 2) also revealed significant differences among bean accessions in all years tested. The trend between susceptible and resistant groups was consistent among years, and percentage seed damage was always greater in susceptible genotypes. Damage scores ranged from 4.3% in 1994 for 'J-117' to 84.7% in 1996 for 'Canario 107', the most susceptible check. These damage ratings were consistent with previously published results (Beebe et al. 1993, Garza et al. 1996). An inverse relationship between percentage encapsulation at the early immature pod stages and percentage dry seeds damaged at harvest was detected (Fig. 2).

Discussion

Hypersensitivity is a term primarily used by plant pathologists. The hypersensitivity reaction encompasses all morphological and histological changes in the host that, when infected/infested by an injurious agent, elicit the premature necrosis of the infected tissue, as well as inactivation and restriction of the infectious agent (Agrios 1988, Fernandes 1990). The

Table 2. Percentage seed damage by *A. godmani* in selected bean accessions

Genotype	1994	1995	1996
	Resistant genotypes[a]		
Amarillo 154	8.6 ± 1.3	9.6 ± 2.0	—[b]
Amarillo 155	11.0 ± 1.6	16.0 ± 2.3	10.9 ± 1.6
J-117	4.3 ± 0.4	6.8 ± 1.9	5.7 ± 2.3
Puebla 36	—	8.3 ± 1.8	12.1 ± 2.1
Pinto 168	—	7.5 ± 2.2	10.4 ± 1.6
Mean	8.0 ± 1.5[b]	9.6 ± 1.5[b]	9.8 ± 1.0[b]
	Susceptible genotypes		
Jamapa	59.7 ± 6.2	72.5 ± 1.8	63.2 ± 2.3
Zacatecas 45	46.1 ± 4.7	47.7 ± 4.1	32.4 ± 2.7
Canario 107	81.6 ± 1.9	81.0 ± 1.1	84.7 ± 4.0
Mean	62.5 ± 7.9[a]	67.1 ± 8.0[a]	60.1 ± 11.9[a]

Means ± SEM of 6 replications per genotype, 30 pods per replication. Means within a column followed by different letters are significantly different separation by Scheffe's F method of significance testing for arbitrary linear contrasts ($P < 0.05$). ANOVA on transformed data testing for differences among genotypes: 1994; $F = 56.4$; df = 5, 25; $P < 0.01$; 1995; $F = 320.7$; df = 7, 35; $P < 0.01$; and 1996: $F = 95.7$; df = 6, 30; $P < 0.01$). ANOVA results for linear contrasts of resistant vs. susceptible genotypes: 1994 - $F = 49.7$; df = 1, 25; $P < 0.01$. 1995 - $F = 30.3$; df = 1, 35; $P < 0.01$. 1996 - $F = 2.5$; df = 1, 30; $P < 0.05$.

[a] Based on previous studies (Beebe et al. 1993; Garza et al. 1996; Garza, unpublished data).

[b] Not included in trial because of lack of seed.

existence of hypersensitive responses to sessile stages of insects (e.g., eggs) in several crop species has been postulated by Fernandes (1990) as an efficient defense mechanism because it is usually fatal to the intruder. Examination of the gross histological reaction to *A. godmani* infestation in young, developing bean pods suggests that the reaction of resistant accessions to oviposition by the insect is, in most cases, one of hypersensitive necrosis of the tissues surrounding oviposition sites. More importantly, the necrosis restricts and inactivates neonates, preventing their dispersal to developing seeds and, thus acting as an important mortality factor (antibiosis). To a lesser extent, larval feeding can also elicit hypersensitive reactions. Thus, in highly resistant accessions like J-117 and Amarillo 155, the hypersensitive reaction to infestation can be so strong that first instars burrowing in the pod wall can also be encapsulated.

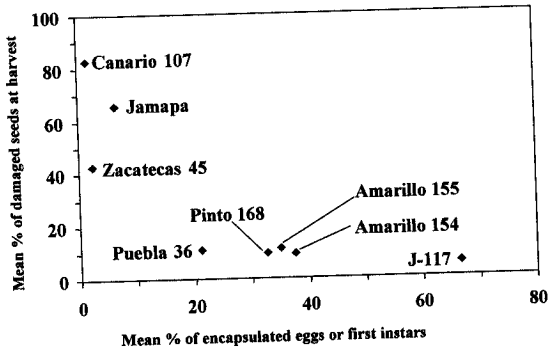

Fig. 2. Relationship between percentage encapsulation of first instars and percentage damaged seeds at harvest in eight bean accessions evaluated for resistance to *Apion godmani*.

The inverse relationship between percentage encapsulation and percentage damaged dry seeds at harvest suggests that high larval mortality from encapsulation in resistant accessions is an important antibiotic factor. We therefore postulate that antibiosis caused by hypersensitivity is the main mechanism of resistance to *A. godmani* in at least some beans. Nonetheless, ovipositional antixenosis (Garza 1998) can also play a major role in some resistant accessions.

Hypersensitivity as a plant defense mechanism can be effective only against organisms that are unable to move readily to new feeding sites when defensive responses are elicited (Grover 1995). This certainly is the case with *A. godmani* larvae, which are embedded in host tissue and have strongly limited movement. The host plant–insect relationship in this case is specific, a condition that, according to Fernandes (1990), is conducive to the development of hypersensitivity, hence resistance. *A. godmani* directly affects the reproductive capacity of the host plant and, as evidenced in this report, has an intimate relationship with its host. As discussed by Beebe et al. (1993), *A. godmani* is native to Mexico, a primary center of diversity and domestication of *Phaseolus* species, including *P. vulgaris* (Gepts et al. 1986, Debouck 1999). *P. vulgaris* therefore had a long time to coevolve with and develop resistance to *A. godmani* in Mexico. This may explain why high levels of resistance are found in some Mexican bean landraces, whereas no adequate resistance has been found in genotypes originating in other regions (Beebe et al. 1993).

In most of the resistant bean landraces used in this study, two genes segregating independently control resistance to *A. godmani* (Garza et al. 1996). One gene, *Agm*, has no effect when present alone, whereas the other gene, *Agr*, alone confers intermediate resistance. However, when both genes are present, resistance is higher. In some bean landraces such as Amarillo 169, a single dominant gene conferred high levels of resistance to *A. godmani*, irrespective of the alleles at the other locus, whereas Pinto Texcoco and Pinto 168 possessed two different genes imparting intermediate resistance. The fact that resistance to this insect is either dominant or intermediate is in agreement with the model suggested by Fernandes (1990), who postulates that hypersensitivity is usually controlled by an individual gene, or more rarely, by a few genes with major effects. It remains to be elucidated whether the genes controlling hypersensitivity in resistant bean landraces—Amarillo 154, Amarillo 155, J-117, Puebla 36, and Pinto 168—included in this study are the same as those controlling ovipositional antixenosis detected by Garza (1998) in these genotypes. Polymorphic, well-characterized recombinant inbred lines developed from highly resistant × susceptible bean populations would be needed to find direct or tightly linked flanking polymerase chain reaction-based DNA markers for the *A. godmani* resistance genes. These findings would facilitate future genetic and biochemical research and provide much needed information con-

cerning the phenotypic basis of resistance to *A. godmani* in bean. Furthermore, results of this research would facilitate and expedite pyramiding of different genes and development of highly resistant bean germplasm and cultivars.

Acknowledgments

We thank John Miles (Centro Internacional de Agricultura Tropical) for critical review of the manuscript; Eduardo Mondragón and Lucio Anides for technical assistance in the field; and the Swiss Development Corporation and Profrijol (Programa de Frijol para Centroamérica) for financial support. This article is published as Idaho Agricultural Experiment Station Journal Article No. 00705.

References Cited

Acosta, J., R. Rosales, and R. Garza. 1992. Resistance to the pod weevil in wild *Phaseolus vulgaris.* Ann. Rep. Bean Improvement Coop. 35: 103–104.

Agrios, G. N. 1988. Plant Pathology, 3rd ed. Academic, New York.

Balbyshev, N. F., and J. H. Lorenzen. 1997. Hypersensitivity and egg drop: a novel mechanism of host plant resistance to Colorado potato beetle (Coleoptera: Chrysomelidae). J. Econ. Entomol. 90: 652–657.

Beebe, S., C. Cardona, O. Díaz, F. Rodríguez, E. Mancía, and S. Ajquejay. 1993. Development of common bean (*Phaseolus vulgaris* L.) lines resistant to the bean pod weevil, *Apion godmani* Wagner, in Central America. Euphytica 69: 83–88.

Cardona, C. 1989. Insects and other invertebrate bean pests in Latin America, pp. 505–570. *In* H. F. Schwartz and M. A. Pastor-Corrales [eds.], Bean production problems in the Tropics, 2nd ed. Centro Internacional de Agricultura Tropical, CIAT, Cali, Colombia.

Cardona, C., and J. Kornegay. 1999. Bean germplasm resources for insect resistance, pp. 85–99. *In* S. L. Clement and S. S. Quisenberry [eds.], Global Plant Genetic Resources for Insect-resistant Crops. CRC, Boca Raton, FL.

Debouck, D. G. 1999. Diversity in *Phaseolus* species in relation to the common bean, pp. 25–52. *In* S. P. Singh [ed.], Common Bean Improvement in the Twenty-first Century. Kluwer, Dordrecht, Netherlands.

Fernandes, G. W. 1990. Hypersensitivity: a neglected plant resistance mechanism against insect herbivores. Environ. Entomol. 19: 1173–1182.

Garza, R. 1992. Identificación de los componentes de la resistencia al ataque del picudo del ejote, *Apion godmani,* que poseen los materiales de frijol seleccionados, pp. 13–22. *In* Informe de investigación 1991 del proyecto Apion en Mexico. SARH-INIFAP CEVAMEX, Chapingo, Mexico.

Garza, R. 1998. Mecanismos de resistencia del frijol, *Phaseolus vulgaris* L., al picudo del ejote *Apion godmani* Wagner (Coleoptera: Curculionidae) y búsqueda de marcadores moleculares como indicadores de esta resistencia. Ph.D. dissertation, Colegio de Postgraduados, Montecillo, Texcoco, Edo. de Mexico, Mexico.

Garza, R., and J. S. Muruaga. 1993. Resistencia al ataque del picudo del ejote *Apion* spp. en frijol *Phaseolus* spp. Agron. Mesoam. 4: 77–80.

Garza, R., C. Cardona, and S. P. Singh. 1996. Inheritance of resistance to the bean pod weevil (*Apion godmani* Wagner) in common beans from Mexico. Theor. Appl. Genet. 92: 357–362.

Gepts, P., T. C. Osborn, K. Rashka, and F. A. Bliss. 1986. Phaseolin protein variability in wild forms and landraces of the common bean (*Phaseolus vulgaris* L.): evidence for multiple centers of domestication. Econ. Bot. 40: 451–468.

Grover, P. B. 1995. Hypersensitive response of wheat to the Hessian fly. Entomol. Exp. Appl. 74: 283–294.

Guevara, C. J. 1961. El combate del picudo del ejote mediante la combinación de variedades resistentes e insecticidas. Agric. Tec. Mex. 1(12): 17–19.

Mancía, J. E. 1973. Evaluación de variedades de frijol tolerantes al picudo de la vaina *Apion godmani* Wagner. SIADES (El Salvador) 2: 15–20.

McKelvey, J. J., J. Guevara, and A. Cortés. 1947. Apion pod weevil: a pest of beans in Mexico. J. Econ. Entomol. 40: 476–479.

SAS Institute. 1988. SAS/STAT user's guide, release 6.03 ed. SAS Institute, Cary, NC.

Shapiro, A. M., and J. E. DeVay. 1987. Hypersensitivity reaction of *Brassica nigra* L. (Cruciferae) kills eggs of *Pieris* butterflies (Lepidoptera: Pieridae). Oecologia (Berl.) 71: 631–632.

Shukle, R. H., P. B. Grover, Jr., and G. Mocelin. 1992. Responses of susceptible and resistant wheat associated with Hessian fly (Diptera: Cecidomyiidae) infestation. Environ. Entomol. 21: 845–853.

Singh, S. P., P. Gepts, and D. Debouck. 1991. Races of common bean (*Phaseolus vulgaris,* Fabaceae). Econ. Bot. 45: 379–396.

Received for publication 4 October 2000; accepted 17 March 2001.