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SOURCES OF SOYBEAN INFECTION WITH BACTERIAL BLIGHT AND THIRE CONTROL

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ABSTRACT

Seed transmission, over wintering and survival of *Pseudomonas syringae pv. glycinae* on processing soybean were evaluated as well as the spread of the bacteria on alternative host and non-host plants. The bacteria were transmitted at a low rate from soybean seeds to the seedlings. Bacterial density in seeds obtained from systematically infected plants ranged from 110 to 115 cfu/g seed. Surviving bacteria associated with infested soybean debris were found in a higher number when debris was on soil surface compared to the buried debris. Population of the bacteria on alternative host and non-host plants ranged from 0 to 108 cfu/g fresh weight on leguminous plants and from 0 to 100 cfu/g fresh weight on non-leguminous weeds. The bacteria were not detected in naturally infected soybean seeds when soaked in 0.5% Hcl for 5 hours, 0.25% or 0.05% acidified cupric acetate for 2 minutes or in hot water (50-55°C) for 20-30 minutes. Hot water treatment had significantly improved seed germination. Other treatments were ineffective in inhibiting the bacteria and have varied effects on germination.

Keywords: Bacterial blight of soybean, Seed transmission.

INTRODUCTION

Pseudomonas syringae pv. glycinae (Coerper 1919) Young *et al.*, (1978) , cause bacterial blight of soyabean (*Glycine max L. Merr*) . It's a common disease of soybean in the growing areas all over the world. The pathogen is prevalent in cool and humid regions (Bradbury, 1986). Several other plants, including some common weeds are known as hosts of *P. syringae pv. glycinae* (Bradbury, 1986) .The pathogen can be isolated from lima bean (*Phaseolus lunatus*), bush bean (*Phaseolus vulgaris*), tepary bean (*Phaseolus acutifolius*), cowpea (*Vigna uniguiculata*), azuki bean (*Vigna angularis*), (Bogatsevska, 1990). The bacterium is mainly located in the seed coat, but it is also found in the cotyledons. Most crop infections originate from contaminated seeds during harvest or through infection of healthy seedlings from infected debris of the previous crop (Fahy and Lioyd ; 1983, Basu and Butler, 1986, and Park and Lim, 1985). Pathogen spread during the growing season is probably due to splashing during rainy weather (McGee, 1992). The bacteria can exist on buds and leaves of symptomless plants, and needs only favorable conditions in order to invade leaf tissue and to cause the disease (May *et al.*, 1997). Inoculation of flowers with the pathogen resulted in both pod and seed infection in plants grown under greenhouse conditions (Kauffman and Leben, 1974). One report showed that the level of seed infection by *P. savastanoi pv. glycinea* was well-correlated with reduced seed germination in laboratory (r=-0.72) and field tests (r=-0.69) (Nicholson and Sinclair, 1971). Yield losses of 18% have been recorded due to bacterial blight (Williams and Nyvall, 1980).It is recommended to avoid the use of

susceptible varieties, the use of healthy seed has been considered as the best control means of the disease. Oxytetracycline hydrochloride reduced seed transmission of the pathogen from 50-90% to 0-2% in a laboratory grow-out test, but was phytotoxic to the seedlings (Leben,1975). A seed-borne bacteria

of soybeans, antagonistic towards *P. glycinea* in agar diffusion tests, reduced seedling infection from 46-98% to 7-17% (Leben, 1975). Ultrasonic radiation reduced cotyledon infection (Krasnova, 1963).

The objectives of this research was to evaluate the bacterial dissemination, sources of *P. syringae pv. glycinae* and the effect of seed treatment for eradicating *P. syringae pv. glycinae* from naturally infected soybean seeds.

MATERIALS AND METHODS

Seed sources :

Soybean seeds used during this study were collected from legumes of soybean plants grown in the Research Farm at Giza (ARC). Three cultivars namely, Crawford, Clark and Calland were used.

Detection and Identification of the bacterium :

1- 1 Kg of soybean seeds were ground in a blender, (Fieldhouse, *et al.* 1984) the coarse flour was collected in 2 L. of sterile tap water in a plastic bag, mix thoroughly and allow to stand for 1-2 hours at room temperature (22-25°C). 4x 1:10 serial dilutions were prepared using (Nutrient broth as diluent 9 ml). 0.05 ml seed extract and the serial dilution were transfered onto 3 plats of SNA medium (5.0g Pepton, 3.og Beef extract, 50g Sucrose, 900 ml distilled water, 1.5 Boric acide, 100 ml distilled water amended with 10 µg/ml cephalexin) and King's B medium BBD (Bacterial blight differential medium) (250.0g, Na-Deoxycholate, 15.og Proteose peptone No.3, 0.5g Yeast extract, 2.5g Glycerol, 7.5g Agar, 250 ml Distilled water, 10.0g Casein, 250 ml Distilled water amended with 10 µg/ml cephalexin) Streaking using L-shaped glass rod. Reference culture of *P. syringae pv. glycinae* was used. The plates were incubated at 25°C for 3-4 days. King's B medium BBD plates were examined for fluorescent colonies and SNA plates for levan-positive colonies. Whit colonies were transfered to King's B medium plates and incubated 2-3 days at 25°C.

2- A turbid suspension of the bacterial isolates 10^{7-8} cfu/ml was used for artificial incculation and ELISA method. Soybean leaves were inoculated, inoculum was applied to the underside of first or second trifoliolate leaves of the tested susceptible cultivar. The leaves were sprayed with sterile water using a camel hair brush. Plants were grown in the greenhouse at 20-30°C for 7-10 days, then checked for symptom development (Fett, 1979).

3- Eonzyme-linked Imunosorbent assay (ELISA):

Wells of a 96-well plate were filled with 100µl of anti- *P. syringae pv. glycinae* is polyclonal antiserum diluted to 10 µg of IgG. ml in 0.1 M

carbonate buffer. Incubated for 2 hr. at 37°C or overnight at 4°C. Then washed out the plate. Sample were prepar in sample buffer (phosphat buffer saline (pH 7.2)(PBS), 0.2 % (w/v) skim milk powder, 2% (w/v). Polyvinylpyrolidone, and 0.05% (v/v) Tween 20) or dilute sample was prepared with another diluent 1:1 in sample buffer. Pipet samples into wells at 100 µl/well and incubated for 1 hr. at 37°C and or overnight at 4°C. Then wash out the plate. Unreacted protein binding sites were blocked by incubating 200 µl/well of 0.2% skim milk powder in PBS for 30 min at 37°C and washed out the plate. Monoclonal antibody was diluted to a predetermined level (e.g. 1:2000 of ascitic fluid) in PBS containing 0.1% skimed milk powder and pipet into wells at 100 µl/well and incubated for 1 hr. at 37°C then wash out the plate. Secondary antibody was diluted, specific to the primary antibody, conjugated with alkaline phosphatase in the same buffer as above. The optimum dilution will vary with each source of conjugate and therefore, must be determined by trying a range of dilutions (usually 1:1000-1:5000 of commercial preparations) on a Known positive sample. Pipet the diluted enzyme conjugate into wells at 100 µl/well and incubated for 1 hr. at 37°C then washed out the plate. Dissolve p-nitrophenyl phosphate at 0.5 mg/ml in 1 M diethanolamine buffer, pH 9.8 and add to wells at 100 µl/well. Incubate at 37°C for 30-60 min and absorbance was measured at 405 nm with a plate reader.

Seed transmission:

Seed were harvested from several naturally infected soybean plants collected from commercial fields during August 2000 and from symptomatic inoculated Crawford plants. Seeds were soaked in a cell suspension of *P. syringae pv.glycinae* 10^9 cfu/ml for 5 min and then air dried overnight. 100 seeds of Crawford were planted in 49-cell plastic trays at the rate of 25/plastic tray containing a commercial potting mix and arranged in a randomized complete block design. The trays were placed on larger trays and watered from below to avoid splashing and dispersal of the pathogen. Diseased seedlings were removed at the onset of symptoms, and randomaly selected seedlings were sampled to confirm the prescence of the causal organism by isolation on King's B medium BBD. The experiment was terminated 4 wk after planting. Seeds collected from three inoculated asymptomatic Crawford plants were planted in the greenhouse and observed for 3 weeks for development of symptoms.

Seed treatments:

Untreated seeds were (control) or soaked (immersed) in 600 ml aqueous solutions of 0.03 M Hcl for 10 min, 0.6 M Hcl for 5 hr. (Shoemaker and Echandi,1976), 0.5% Naocl for 20 min, 1.05 Naocl for 40 min, 0.5% $Ca(ocl)_2$ for 20 min, 0.1% formalin for 20 min, 0.25% Cupric acetate acidified in 0.005 N acetic acid (ACA) for 20 min, 0.5% ACA (0.5 cupric acetate) for 20 min, water at 52°C for 20 min, or water at 56°C for 30 min. All treatments except the hot water treatments were done first at room temperature and then kept in solutions heated to 52°C. After treatment, the seeds were rinsed twice in sterile distilled water and placed on paper towels to dry overnight at room

temperature in a laminar flow hood. Three tested subsamples of 24 g from each treatment were removed and assayed for *P. syringae pv.glycinae* by the seed-extract agar plating method

(Fatmi,and Schaad,1989). 300 seeds from each treatment were tested for germination using the wet blotter test as recommended by the Association of official Seed Analysis. In addition, four replicates of 50 seeds each were planted in a stream sterilized soil mix. Greenhouse temperature was varied from 13°C (at night) to 22°C (during the day). After 10 days, seedlings will fully expanded cotyledons were recorded.

RESULTS

Extraction of the bacterium:

The bacterial colonies are recognized by fluorescence on KB agar, and by the LOPAT tests (levan production, oxidase reaction, potato rot, arginine dihydrolase production and tobacco hypersensitivity; reactions: +,-,-,-,+). After 2-3 days *of* inoculation at 25°C. marked suspected colonies were developed. Colonies are Gram-negative rods with rounded ends, 1.2-1.5 x 2.3-3.0 µm long, motile with one to several polar flagella. On nutrient agar circular, smooth, and glistening, with an entire margin. White and raised, but not viscid, producing levan, but does not hydrolyse gelatin and sorbitol. Colonies appearance was typical with *P. syringae pv. glycinea* (Fieldhouse *et al.* 1984)

Pathogenicity test:

Symptoms are particularly conspicuous on leaves and consisted of small angular lesions, usually water-soaked at the centre and surrounded by a yellow-green halo. Later, the spots coalesce to form dark-brown necrotic areas with yellow margins. The spots are usually scattered or grouped over the leaf lamina, resulting in irregular lesions or several large necrotic areas. Later, the spots became dry and fall out giving the leaf a ragged appearance. Lesions are frequently found on stems, petioles and pods. Seedlings were stunted and died if the growing point was infected.

Serological tests:

Measuring optical density at 490 nm. absorbance of ELISA reader was more twice as high as absorbance of healthy so the reaction give positive result.

Isolation from plant debrise mixed with soybean seed inaddition to leguminous weeds showed the presence of *P. syringae pv. glycinea.*

Seed transmission:

Diseased seedlings developed from all the tested seed samples whether obtained from naturally infected plants or artificially inoculated seeds (Table 1). Infection ranged from 6% for seedlings grown from seed collected from naturally infected clark cv. during August 2000 to nearly 100% Crawford cv. that was inoculated with *P. syringae pv. glycinea* . Symptom development on

seedlings developed from infested Calland cv. seeds was similar to seedlings grown from seed from symptomatic plants. Whitish flaky spots are observed on cotyledons of soybean seedlings. The center of individual spots often remain green for a few days and later becoming necrotic. Several infected cotyledons wither and die premature followed by wilting and death of the seedlings.

Table 1. Effect of the mode of seed transmission of soybean bacterial blight on the percentage emergence and diseased plants of the tested cultivars.

Seed source / Period		Emergence %	Diseased plants %
clark cv	July 2000	96.0	6.0
	August 2000	85.0	9.0
Crawford cv inoculated with	July 2000	90.0	30.0
P. syringae pv. glycinea	August 2000	99.0	55.0
Calland cv inoculated with	July 2000	93.0	60.0
P. syringae pv. glycinea	August 2000	97.0	75.0

Seed treatment:

P. syringae pv. glycinea could not be detected in extracts of seed soaked in 0.5% Hcl, 0.25% or 0.5% ACA, hot water, or the heated solution of several chemicals (Table 2). Moreover, the pathogen was not detected when samples from these treatments were directly placed on King's B medium BBD. Most of the tested treatment reduced saprophytic bacteria associated (Table 3). However, seed treatment with 0.5 M Hcl or 0.25% ACA at 52°C eradicated all saprophytes. On the other hand some treatments adversely affected seed germination (Table 3). The treatment 0.5 M Hcl and ACA significantly (P=0.05) reduced germination in the blotter test. In contrast, when seeds were planted in soil, the germination of seeds treated with ACA did not differ significantly from that of the control.

Results were similar with seeds treated with water at 52°C, germination was significantly less than the control in the blotter test but significantly greater than the control in the soil test. In further tests treatment with hot water or with ACA either at room temperature or at 52°C did not reduce the germination of seeds of three tested cultivars in soil.

Table 2. Effect of soybean seed treatments on the development of *P. syringae pv. glycinea* and the associated saprophytes[a].

Treatments	*P. syringae pv. glycinea*	Associated saprophytes
Untreated	1.85×10^4	3.8×10^5
0.25% Hcl, 10min.	2.1×10^4	2.3×10^3
0.5% Hcl, 5 hr.	0.0	1.1×10^2
0.5% Naocl, 20min.	6.5×10^4	1.9×10^4
0.5% Ca $(ocl)_2$ 20 min.	2.5×10^3	3.5×10^4
0.1% Formalin,20 min.	3.6×10^3	4.1×10^3
0.25% ACA, * 20 min.	0.0	1.1×10^3
0.5% ACA, 20 min.	0.0	2.6×10^4
Water at 52°C, 20 min.	0.0	1.5×10^3
Water at 65°C, 30 min.	0.0	3.5×10^3
0.25% Hcl at 52°C,10min.	0.0	2.1×10^3
0.5% Hcl at 52°C, 5 hr.	0.0	o.o
0.5% Naocl at 52°C,20min.	0.0	3.8×10^4
0.5% $Ca(ocl)_2$at52°C,20 min.	0.0	2.1×10^3
0.1% formalin at 52°C, 20 min	0.0	1.9×10^3
0.25% ACA at 52°C, 20 min.	0.0	0.0
0.5% ACA at 52°C, 20 min.	0.0	3.5×10^3

a= Data are colony-forming units and the mean of three replicates of 10,000 seeds.,. Seeds were assayed by plating liquid extracts onto BBD medium.
* Acidified cupric acetate

Table 3. Effect of physical and chemical seed treatments on germination percentage of the three soybean tested cultivars.

Treatments	germination (%)[w]	
	Blotter	Soil[y]
Untreated	9.20 abc	80.0 bcde
0.25% Hcl, 10min.	85.0 de	85.0 b
0.5% Hcl, 5 hr.	0.3 l	4.0 h
0.5% Naocl, 20min.	94.0 ab	75.0 cdef
0.5% Ca $(ocl)_2$ 20 min.	94.3 abc	52.0 g
0.1% Formalin,20 min.	90.0 cd	85.0 ab
0.25% ACA, * 20 min.	13.0 g	82.0 bcd
0.5% ACA, 20 min.	8.0 h	70.0 ef
Water at 52°C, 20 min.	80.0 e	94.0 a
Water at 65°C, 30 min.	90.0 bcd	85.2 ab
0.25% Hcl at 52°C,10min.	2.0 l	75.0 cdef
0.5% Hcl at 52°C, 5 hr.	90.5abcd	4.0 h
0.5% Naocl at 52°C,20min.	91.0 abcd	85.6 b
0.5% $Ca(ocl)_2$at52°C,20 min.	91.0 abcd	85.0 ab
0.1% formalin at 52°C, 20 min	10.5 gh	84.0 bc
0.25% ACA at 52°C, 20 min.	12.0 gh	75.0 def
0.5% ACA at 52°C, 20 min.	10.5 bh	74.0def

w=Mean follwed by the same letter do not differe significantly (p=0.05) according to Duncan's multiple range test.
y=Three replicates of 100 seeds each were tested following rules of the association of afficial seed Analysis.

DISCUSSION

The causal agent of soybean bacterial blight is *Pseudomonas syringae pv. glycinea* . Various seed health assays have been used during this research to detect bacterial blight pathogen: liquid assays with planting on semiselective media, plant inoculations, and ELISA test . All these methods for detection of bacterial blight pathogen reported by (Fett, 1979, Leben, 1972; Burbage and Sasser, 1983; Fieldhouse *et al.*, 1984 , Leary et al., 1988). The existence of improved semiselective media has made possible large-scale screening of seed lots for *P. syringae pv. glycinea* as well as for other seed-borne bacterial pathogens,(Alvarez *et al.* 1995). The commercial availability of an antiserum to *P. syringae pv. glycinea* (Agida, Inc., Elkhart, IN) has created new opportunities to use ELISA techniques in detecting the pathogen (Leary *et al.*, 1988). Pathoginicity bioassays are indispensable for confirming the identify of *P. syringae pv. glycinea* by using soybean plants (Ovchinnikova and Potlaichuk, 1980 and Kennedy 1969). Contaminated seeds, infected debris, alternative hosts species and volunteer soybean seedlings are important source of inoculum (Fahy and Lioyd, 1983, Basu and Butler 1986, and Park and Lim, 1985). The bacterial blight pathogen has been reported to be seed-transmitted (Varvaro, 1983) . the obtained results demonstrate seed transmission from both naturally diseased and artificially inoculated, symptomatic plants . The recovery of the pathogen from both seed coats and embryos of seeds from symptomatic plants indicates that seeds are both internally and externally contaminated 1% transmission of the pathogen in seeds can be significance led to 100% seedlings of infection (Gasperini *et al.*, 1982). Although several seed treatments are available for eradicating seed-borne bacteria (McGee, 1992), non has been effective for eradicating *P. syringae pv. glycinea* . An effective seed treatment must destroy the bacteria beneath as well as on the seed coat (Leben, 1975). Results obtained showed that seed soaking in Naocl destroyed surface population of the pathogen but apparently did not eliminate internal population. Although Naocl treatments have been recommended in the past (Fatmi, and Schaad,1989), these treatment are not effective in this research with Naocl, Shocmaker and Echandi (1976) inoculated seed in the laboratory and used a grow-out assay to evaluate the effectiveness of the treatment. They also recommended treating seed with about 0.5 M Hcl for 5-10 hr. to eradicate the pathogen. Acid treatments eradicate the pathogen, but they may severely reduce germination. (Bogatsevska and Lalova, 1987) used certain herbicides (trifluralin, metolachlor, alachlor, metabromuron) and unsaturated 1,3,5-triazine derivatives (Konstantinova and Bogatsevska, 1989) have a bactericidal effect against *P. syringae pv. glycinea.* The results were confirmed by field experiments (Bogatsevska and Lalova, (1987). This is not considered to be an acceptable method of control. Our study confirms that soaking seed in water at 56°C is an effective treatment (Shocmaker and Echandi (1976). In this case, germination is not adversely effected. However a temperature of 56°C may be difficult to maintain accurately under commercial condition, and some seed lots could be injured. Treatment with

0.25 or 0.5% ACA at room temperature eradicated *P. syringae pv. glycinea* from the seed and soaking seed in heated solutions of 0.25 or 0.5% ACA virtually eliminated all external and internal microbes. The effects of these treatments on germination varied. Germination of the treated seeds did not differ significantly from that of the untreated control but was significantly reduced in the blotter test . The reason for this discrepancy is unknown. However the sever reduction in germination in the blotter test may be resulted from the failure of cupric acetate residues to disperse . In soil test , the cupric acetate could react with or bound to anionic components within soil and therefore not available . the authors recommended the assaying of all soybean seed lots for *P. syringae pv. glycinea* by the seed-extract, agar plating assay, (Fieldhouse *et al.*, 1984 , Leary *et al.*, 1988). Any positive test the seeds then treated with water at 52°C or 0.25% ACA at 52°C for 20 min. sample should be tested first to ensure that germination is not adversely affected and all treated seed lots should be reassayed for *P. syringae pv. glycinea* to verify the effectiveness of the treatment.

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مصادر أصابة نباتات فول الصويا ببكتيرة اللفحة وكيفية مكافحتها

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تم دراسة معدل انتقال بكتيريا *Pseudomonas syringae pv. glycinea* خلال بذرة فول الصويا وحيويتها وانتشارها من خلال العوائل النباتية القريبة الصلة لنباتات فول الصويا. تم تقدير كثافة النمو البكتيرى فى البذرة لنباتات أصيبت جهازيا وتراوح هذا المعدل من ١١٠ الى ١١٥ خلية بكتيرية لكل جرام بذرة. وجدت البكتيرة المسببة للفحة بنسبة مرتفعة مصاحبة لبقايا نباتات فول الصويا. وتراوح معدل تجمعات البكتيرة على العوائل القريبة أو البعيدة عن فول الصويا بين صفر الى ١٠٨ خلية بكتيرية فى النباتات التابعة للعائلة البقولية . لم يتم عزل البكتيرة من نباتات مصابة طبيعيا تم نقعها فى ٥و. ٪ حمض هيدروكلوريك لمدة خمس ساعات أو ٢٥و. ٪ أو ٥و. ٪ كبريتات نحاس (ACA) لمدة دقيقتين أو فى الماء الساخن (٥٠° م) أو ٥٥° م لمدة ٢٠ – ٣٠ دقيقة بالتتابع. وأظهرت النتائج أن معاملة البذرة بالماء الساخن لها تأثير مشجع للإنبات . بينما لم يكن للمعاملات الأخرى دور فعال فى تثبيط البكتيرة كما كان لها تأثيرا سلبيا على نسبة الإنبات .