J. Ginseng Res.
Vol. 22, No. 4, 229~236(1998)

Seedborne Fungi and Fungicide Seed Treatment of Ginseng

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(Received April 7, 1998)

Abstract : The incidence of fungi and their possible contribution to low vigour were examined in a collection of ginseng (*Panax quinquefolius*) seed from Ontario. When examined after one winter of stratification in the field and storage at 4℃ for five months in the laboratory, the collection exhibited low vigour (plant stand 16.7% of seeding rate six weeks after seeding) and hight incidence (94%) of discoloured or soft seed. Fungi isolated (and incidence) from 1,304 endosperm halves recovered from surface-sterilized seed were, in order of abundance, *Fusarium roseum* (22.2%), *Chaetomium crispatum* (14.3%), *Fusarium oxysporum* (9.0%), *Fusarium solani* (9.0%), *Mucor* sp. (8.4%), *Alternaria* sp. (8.1%), *Zopfiella leucotricha* (7.8%), *Cylindrocarpon* sp. (0.9%), *Fusarium avenaceum* (0.9%), and *Volutella ciliata* (0.4%). Most of these fungi, including known and potential pathogens of ginseng (species of *Alternaria, Cylindrocarpon, Fusarium*, and *Trichoderma*), were associated with both healthy and diseased seed. Application of Benlate (benomyl), Thiram (thiram), or UBI-2584 (tebuconazole) to seed caused slight to pronounced reduction in emergence and did not significantly affect plant stand six weeks after seeding. The study demonstrated the high level of infection by fungi, including known and potential pathogens of the crp, in an arbitrary collection of ginseng seed from commercial sources, and the phytotoxicity of the fungicides tested when applied to moist stratified seed. The lack of efficacy of the fungicides precluded determination of the contribution of seedborne fungi to low vigour of the seed.

Key words : Ginseng, *Panax quinquefolius*, seed vigour, seedborne fungi, seed treatment, benomyl, thiram, tebuconazole.

Introduction

Seeds can be an important source of fungi pathogenic to seeds, roots or shoots.[1] Therefore, seed treatment with fungicides plays a critical role in controlling plant diseases, and has been the major approach to control of some historically devastating diseases such as loose smut of cereals.[2] The ginseng crop is propagated only from seed and diseases are major factors limiting production of the crop.[3-5] Thus it is important to determine whether ginseng seed harbour pathogenic fungi and whether seed treatments can reduce ginseng diseases.

There is little information on seed pathology of ginseng. However, two research groups have shown that numerous fungi can colonize ginseng seed. Fungi isolated from ginseng seed in Korea and China included *Cylindrocarpon* spp., *Alternaria* spp., *Phytophthora* spp., *Fusarium* spp., and *Rhizoctonia solani*.[6-8] These fungi are all potential pathogens of ginseng.[9,10] There is some evidence that seed can become colonized during stratification despite fungicidal treatment of seed. Tianyu and Weiqun (1992) reported that the incidence of fungal contamination of seed treated with Dithane M-45 was 66% after 90 days of stratification and 100% after 120 days.[7] This may explain

why treating seed with formalin before stratification did not affect seeding emergence or disease incidence.[3,11] In some cases, seed treatments reduce germination. Lee *et al.* (1981) showed that treating seed with captan before stratification delayer maturation of the embryo and suppressed seed dehiscence, possibly by eradicating fungi that normally soften the endocarp.[6]

In Canada, there are no fungicides registered for use as seed treatments on ginseng, and no published information regarding contaminatin of ginseng seed by pathogens, transmission of pathogens from seed to the crop, or effects of seed treatments on disease control or plant growth.[12] The objectives of this study, therefore, were to (1) identify and quantify fungi from ginseng seed in Ontario, and (2) determine plant growth and disease after applying fungicides to stratified seed.

Materials and Methods

1. Seed

Samples of ginseng(*Panax quinquefolius* L.) seed that had been stratified in sand over winter in the the field were collected from five farms in southern Ontario in September 1994. The samples were combined then divided into three lots. Each lot was considered a replicate subsample of the composite sample. Each replication was buried in a 2:1 mixture of damp sand and seed and stored at 4℃ for five months, at which time the seed were starting to germinate. The containers of seed and sand were weighed before being placed in cold storage. At regular intervals during storage, water was added to maintain constant weight of the containers and their contents. Each replication of seed subjected to one winter of stratificatin in the field followed by five months of cold storage in the laboratory was examined for fungal infection and efficacy of fungicidal seed treatments.

2. Seed infection

To determine infection of seed by fungi, about 220 seeds (12 g) were weighed out from each replication, surface sterilized by immersion for one minute in 95% ethanol and for three minutes in a 10% solution of Javex® in water (0.6% sodium hypochlorite), rinsed in sterile deionized water, and blotted dry on autoclaved Whatman® filter paper. The seed coat was removed and the endosperm was split into two halves. Each endosperm half was assigned to one five categories based on its visual appearance, as described in Table 1. Category A was considered healthy and categories B-E were considered diseased.

The endosperm halves in categories A, B and C were surface sterilized as above and blotted dry. The location of the embryo was not recorded. Numerous detached embryos were observed in the rinse water, but portions of the embryo may have remained attached to one or both endosperm halves. Endosperm halves from categories D and E were ont surface sterilized.

Endosperm halves in categories A, B, and C, and portions of rotted material from endosperm halves in categories D and E, were transferred to 9-cm-diameter petri dishes containing potato dextrose agar (PDA) amended with 100 mg/L of novobiocin at the rate of four per dish. The numbers of endosperm halves sample from each re-

Table 1. Categories of endosperm halves of ginseng seed

Category	Endosperm appearance	
	Colour	Texture
A	White to cream, no orange brown or red-brown lesions	Firm
B	White to cream, a few scattered orange-brown to red-brown lesions covering less than 10% of the exterior surface	Firm
C	White to cream, more than 10% of the exterior surface covered with orange-brown to red-brown lesions, mostly 1~2 mm long	Firm
D	White to cream	Soft (creamy or cheesy)
E	Mix of white, cream, black, gree, yellow or pink	Soft (creamy or cheesy)

Table 2. Number of endosperm halves obtained from three replicate samples of ginseng seed

Data category	Endosperm category*				
	A	B	C	D	E
Sample 1	27	178	66	94	73
Sample 2	20	156	97	102	55
Sample 3	32	180	86	70	68
Total	79	514	249	266	196
%	6	39	19	20	15

* Endosperm categories described in Table 1.

plication in each disease category are shown in Table 2. The dishes were covered with black cloth and incubated under laboratory conditions (20℃). The cloth was removed after two days and fungi growing from the endosperm were transferred to PDA and grown under laboratory conditions for identification. Fungi were identified according to descriptions provided in the literature.[13-18]

3. Seed treatment

Three fungicides were tested on each replication for efficacy as seed treatments. The required amount of fungicide and 1 mL of water were added to a 500-mL mason jar and swirled to coat the side of the jar.[19] Seed to be treated with fungicide was selected from categories A, B, and C. The endosperm had split the seed coat and the seed was considered to be in the process of germinating. After addition of 25 g of seed, the jar was rotated on a 45° angle, clockwise and counterclockwise, until the sides of the jar were clear and the seeds were evenly coated. The fungicides used were (1) Benlate 50WP (Du Pont de Nemours, Streetsville, Ontario), containing benomyl 50 g a.i./100 g product, applied at the rate of 50 mg product/25 g seed; (2) Thiram 75WP (Uniroyal Chemical Ltd., Elmira, Ontario), containing thiram at 75 g a.i./100 g product, applied at the rate of 90 mg product/25 g seed; and (3) UBI-2584 (Uniroyal Chemical Ltd.), containing tebuconazole at 8.33 mg a.i./L, applied at the rate of of 0.06 ml product/25 g seed. These products were chosen because they were moderately to highly toxic to *C. destructans* in vitro.[20] Benlate was formulated for use as a foliar spray or soil drench. The other two products were formulated as seed treatments and contained dye to colour the seed. Insufficient seed was available to test more than one fungicide rate. Controls consisted of untreated seed agitated in a mason jar and non-agitated untreated seed. The seeds were not allowed to dry to avoid loss in viability. From each replication, 96 seeds were planted individually in PGX potting mix (Plant Products, Mississauga, Ontario) in 12.5-cm-diameter pots (Kord Products, Bramalea, Ontario). Plants were maintained in a greenhouse at 20℃ under polypropylene shade that transmitted 30% of the incident light. Seedling were watered as required and plants were monitored for emergence and damping-off over a period of six weeks. Plants were removed as they became diseased, surface sterilized in 70% ethanol for one minute, and plated onto 1.5% water agar containing 0.5 mL Tergitol NP-10, 50 mg streptomycin sulphate, 50 mg chlortetracycline, 50 mg chloramphenicol, and 1 mL 95% ethanol per litre of medium.

4. Data analysis

To compare means statistically, data were subjected to analysis of variance (ANOVA). When ANOVA indicated significant treatment effects, means were compared by the least significant difference (LSD) test at P=0.05. Percentage data on incidence of fungi in endosperms were analyzed as untransformed data (x), as $\sqrt{x}$, and as $\sqrt{(x+0.5)}$. The latter two transformations were recommended by Steel and Torrie (1980, p. 235) for percentage values in the range 0-20.[21] Data were analyzed using SAS/STAT User's Guide, Release 6. 03 Edition, SAS Institute Inc., Cary, NC.

Results

Most (94%) of the endosperm halves were diseased (categories B-E) and most diseased halves occurred in category B (39%, Table 2). Because each seed contains two endosperm halves, the incidence of infected seed could range from one to two times the incidence of infected endosperm halves. For example, a subset of 10 endosperm

Table 3. Frequency of infection (%) of ginseng endosperm halves by fungi

Fungus	Endosperm category†					
	A	B	C	D	E	All
Fusarium roseum†	42.0a§	19.3bc	34.7ab	5.3c	32.7ab	22.2
Chaetomium crispatum†	51.0a	14.7b	21.7b	6.7b	0.0b	14.3
*Fusarium oxysporum**	14.0	4.0	9.0	14.7	10.7	9.0
*Fusarium solani**	0.0	13.0	10.7	7.3	5.7	9.0
Mucor sp.*	19.7	3.7	8.7	7.3	13.7	8.4
Alternaria sp.*	10.3	10.0	6.7	3.0	9.7	8.1
Zopfiella leucotricha†	0.0b	12.0a	15.0a	0.0b	0.0b	7.8
*Cylindrocarpon destructans**	7.6	5.7	5.6	1.9	8.4	5.4
*Fusarium merismoides**	14.3	4.0	0.0	2.3	6.7	3.8
Trichoderma sp.†	0.0b	3.3a	0.0b	0.0b	0.0b	1.2
Cylindrocarpon sp.*	0.0	0.0	2.3	1.9	0.0	0.9
Fusarium avenaceum†	17.7a	0.0b	0.0b	0.0b	0.0b	0.9
*Volutella ciliata**	0.0	0.0	0.0	2.0	0.0	0.4

† Endosperm categories described in Table 1.
† F test for effect of endosperm category significant at p=0.05.
§ Within a row, means followed by the same letter are not significantly different according to a protected LSD test at p=0.05.
* F test for effect of endosperm category not significant at p=0.05, LSD tests not conducted.

halves could have come from five to 10 seed. Therefore, five infected endosperm halves out of 10 halves (50% infected endosperm halves) could represent 50-100% infected seed. The incidence of seed infection must therefore be equal to, or greater than, the incidence of infected endosperm halves.

The results of analyses on untransformed and transformed values for incidence of fungal infection in endosperm halves were essentially identical. Therefore, results are presented for untransformed values (Table 3).

The most abundant genus was *Fusarium* Link : Fr., including *F. oxysporum* Schlechtend. : Fr., *F. solani* (Mart.) Sacc., *F. avenaceum* (Fr. : Fr.) Sacc., *F. merismoides* Corda, and *F. roseum* Link : Fr. The incidence of infection of endosperm halves by species of *Fusarium* ranged from 0.9% (*F. avenaceum*) to 22.2% (*F. roseum*). *Fusarium oxysporum* and *F. roseum* occurred in all endosperm categories. *Fusarium roseum* was the most abundant species and was most common in endosperm categories A, C, and E. *Fusarium oxysporum* was the third most common species, and was equally abundant in all endosperm categories. *Fusarium solani* was the fourth most abundant species, occurring with equal frequency in all disease categories but not detected in healthy endosperms. Of the two least abundant species of *Fusarium, F merismoides* was recovered from all categories except C, and *F. avenaceum* was obtained only from healthy endosperms.

Cylindrocarpon destructans (Zinssmeister) Scholten was the eighth most commonly isolated fungus, and infected 5.4% of the endosperm halves. It was isolated from healthy endosperms and all categories of diseased endosperms at frequencies (1.9~8.4%) that were not significantly different. In addition, a few isolates of unidentified species of *Cylindrocarpon* were recovered from categories C and D.

Other fungi isolated from endosperm included, in descending order of abundance, *Chaetomium crispatum* (Fuckel) Fuckel (categories A-D), *Mucor* P. Mich. : Fr. sp. (all categories), *Alternaria* Nees sp. (all categories), *Zopfiella leucotricha* (Speg.) Malloch and Cain (categories B and C), *Trichoderma* Pers.:Fr. sp. (category B only), and *Volutella cilicata* Alb. & Schw.:Fr. (category D only). Incidence of infection of endosperm halves by this group of fungi ranged from 0.4%(*V. ciliata*) tp 14.3% (*C. crispatum*).

In controls in the seed treatment experiment, about 80 seedlings developed from the 96 seeds sown per replication (Table 4). All emergence in

Table 4. Effects of benoimyl, thiram, and tebuconazole seed treatments on number of emerged plants[§], diseased plants[†], and surviving plants[‡]

Plant category	Treatment				
	Untreated, unshaken	Untreated, shaken	Benomyl	Thiram	Tebuconazole
Emerged	81.0a[¶¶]	79.7a	62.7ab	34.0c	36.7bc
Diseased	67.3a	63.7a	29.7b	16.3b	25.0b
Surviving*	13.7	16.0	33.0	17.7	12.0

[¶] 96 seeds were sown in each of three replications.
[§] Plant emergence was monitored for six weeks after seeding.
[†] Diseased plants were removed when symptoms were observed.
[‡] Surviving plants were those remaining when the experiment was terminated six weeks after seeding.
* F test effect of seed treatment not significant at p=0.05, LSD tests not conducted.

controls occurred within five days of seeding. Six weeks after seedling, 64~67 (80~83%) of the plants had become diseased and had been removed from the pots. ANOVA indicated significant effects of treatment on numbers of emerged and diseased plants but not on final stand. There were no significant differences between the two controls in any plant category (Table 4). Treatment of seed with fungicide reduced emergence compared to the controls, significantly and by more than 50% in the case of thiram and tebuconazole (Table 4). New seedlings continued to emerge in treatment pots for up to four weeks after seedling. Fewer diseased plants developed from treated seed than from untreated seed, but final stand was not affected by ssed treatment. In controls and treatments, ungerminated seeds were not rotted but neither they nor diseased seedling yielded fungal colonies when plated on isolation medium.

Discussion

Most (94%) endosperm halves were diseased, 13 taxa of fungi were recovered from seed, and eight of these taxa included forms pathogenic to ginseng or other plants (*Alternaria, Cylindrocarpon destructans, Cylindrocarpon* sp., *F. avenaceum, F. oxysporum, F. roseum, F. solani*, and *Trichoderma*). The taxa isolated were similar to those reported by Lee *et al.* (1981), Tianyu and Weiqun (1992) and Tianyu *et al.* (1989).[6-8] Isolated taxa known to be pathogenic to ginseng included *C. destructans, F. oxysporum*, and *F. solani*.[4-23] It is likely that some of these fungi contributed to the disease symptoms observed on the seed, but this remains to be determined. Endosperm category significantly affected incidence of only five fungi: *Fusarium roseum, Chaetomium crispatum, Zopfiella leucotricha, Trichoderma* sp., and *Fusarium avenaceum*, and only two species, *Trichoderma* sp. and *Z, leucotricha*, were significantly more abundant in one or more categories of diseased seed than in healthy seed.

The species of *Alternaria* isolated were not identified, but may have included *A. panax* Whetzel in Whetzel & Rosenb. This species causes Alternaria blight of ginseng, characterized by red to brown lesions on stems and leaves.[24] Parke and Shotwell(1989) reported that *A. panax* can infect ginseng berries.[23] It is therefore possible that *A. panax* infected the seed examined in this study and contributed to disease on seed and seedings. Future studies should examine the pathogenicity of *Alternaria* from ginseng seed, and the possibility that *A. panax* is seedborne.

The most abundant group of fungi recovered were rapidly growing isolates of *Fusarium* producing pink, yellow or red pigmentation. This group may have included species such as *F. graminearum, F. culmorum* and *F. avenaceum*. A few isolates were identified as *F. avenaceum*, the remainder were labelled *F. roseum*. Further work on the pathogenicity of *Fusarium* species to ginseng is warranted, given their abundance in ginseng seed and their pathogenicity to a wide range

of plants.[25]

It is not known when the seed became infected with fungi. It is likely that some, possibly most, infection occurred during stratification, as suggested by the results of Tianyu and Weiqun (1992).[7] During stratification, the endosperm matures and the endocarp cracks, exposing the endosperm to microorganisms in the stratification medium.

Several of the fungal taxa recovered from seed, especially species of *Cylindrocarpon* and *Fusarium*, are known to cause root rot. Root pathogens might be transmitted from seed to the developing root, to the soil, and to other roots. Seed-borne pathogens would be strategically located to be early colonists of the developing root, and therefore to have a competitive advantage over other root colonists. *Cylindrocarpon destructans* infected 6.6% (35/593) of endosperm halves in categories A and B, those most likely to produce plants, and 5.4% of endosperm halves over all categories. Ziezold *et al.* (199xb) observed that, during one growing season, one infected root caused disease in as many as 1.6 neighbouring roots within a radius of 5 cm.[26] If all surviving plants from seed infected with *C. destructans* produced roots affected by disappearing root rot, a rate of seed contamination of 6% could produce about 10% diseased roots within a year, and possibly more in older gardens. Seed may therefore be a significant source of inoculum causing root rot.

In view of the occurrence of potentially pathogenic fungi on the seed, the effects of fungicidal seed treatments on plant stand and disease were determined. The fungicides were applied at rates consistent with those recommended for seed of other crops. but two (thiram and tebuconazole) significantly reduced emergence compared to untreated controls. Phytotoxicity of fungicides to ginseng seed reported previously was attributed to suppression of fungi that normally soften the endocarp.[6] In the present study, the fungicides might have been phytotoxic because the seeds were wet and the endocarp was ruptured. These features would provide the fungicides with greater access to the embryo than occurs when they are applied to the intact and dry surface of most agricultural seeds. Bateman *et al.* (1986) also considered that injury to the seed coat increases toxicity of fungicdal seed treatments.[2] Ginseng seed must be kept moist to remain viable. Therefore it was not possible to follow published seed treatment protocols, which require the chemical to dry on the seed surface for a minimum of 24 hours.[19~29] In addition, liquid formulations tend to be more phytotoxic than others, possibly contributing to the phytotoxicity of tebuconazole.[28] Overcoming the phytotoxicity of fungicides to ginseng seeds remains a major challenge, and will be essential to the development of registered products.

The most encouraging effects of seed treatments were provided by benomyl, which did not significantly reduce emergence. in addition, the number of healthy plants from benomyl-treated seed was greater at the end of the trial, although not significantly, so than in the controls. Benomyl seems worthy of further evaluation as a seed treatment for ginseng. The availabilty of effective, non-phytotoxic seed treatments will permit basic studies on the contribution of seed-borne fungi to diseases and low seed vigour of ginseng, in addition to providing a practical and economic approach to managing these fungi.

Growers routinely place stratified seed in water and discard those that float. Although the assumption was not tested, it is likely that floating seed are in categories D and E, and that growers plant seeds in categories A-C. The incidence of healthy seeds (category A) in a collection of seeds drawn randomly from categories A-C would likely have been about 9% (79/842, Table 2), whereas 83-84% of the seed in untreated controls produced emerged seedings. It is therefore clear that diseased ginseng seed in categories B and C can germinate and produce seedings. However, the percentage of plants alive after six weeks (14~17%) approached the percentage of healthy seeds sown (9%). This suggests that poor quality seed is a major cause of early declines in plant stand, and that plant stand within a few weeks after seding will

approximate the incidence of healthy seed. This conclusion, which remains to be tested, emphasizes the importance of seed quality in producing satisfactory early stands of ginseng. The effect of pathogens on seed quality remains to be determined.

Although fungi were readily isolated from seed after stratification, they could not be isolated from seed or seedings in pots, possibly because of high contamination by mites, bacteria and nematodes. Thus, it was not possible to determine pathogens present in seeds and diseased plants at the end of the seed treatment experiment.

Given the high incidence of diseased seed in this arbitrary collection from five commercial sources, and the broad range of pathogens carried on seed, it is likely that pathogens infecting ginseng seed infest fumigated soils, reduce emergence of seedlings, lower plant stands, and cause diseases of roots and shoots. Seed treatments may help to manage some of these problems. Further work should be done to decrease the phytotoxicity of fungicides to ginseng seeds and to improve their efficacy in disease control. Future trials could test fungicides toxic to *C. destructans* in vitro, powder formulations, and reduced quantities of fungicide and water.

Acknowledgement

This research was supported by funding from Agriculture and Agri-Food Canada (AAFC) and the Ginseng Growers Association of Canada via the AAFC Matching Investment Initiative programme, and was carried out under contract 01684-3-0465/01-LON. Field trials were carried out with facilities provided by the Southern Crop Protection and Food Research Centre of AAFC at Delhi, Ontario.

References

1. Jeffs, K. A. ed. : *Seed Treatment*. 2nd Edition. BCPC Publications Thornton Heath, Surrey, England. 332 pp. (1986).
2. Bateman, G. L., Ehle, H., and Wallace, H. A. H. : Fungicidal treatment of cereal seeds. Pages 83-111 in K. A. Jeffs, ed., Seed Treatment. 2nd edition. BCPC Publications, Thornton Heath, Surrey, England (1986).
3. Proctor, J. T. A. and Louttit, D. : Stratification of American ginseng seed : Embryo growth and temperature. *Korean J. Ginseng Sci.* **19**, 171-174 (1995).
4. Reeleder, R. D. and Brammall, R. A. : Pathogenicity of *Pythium* species, *Cylindrocarpon destructans*, and *Rhizoctonia solani* to ginseng seedings in Ontario. *Can. J. Plant Pathol.* **16**, 311-316 (1994).
5. Yu, Y. H. and Ohh, S. H. : Problems and present status of research on ginseng diseases in Korea. Pages 120-130 in W. G. Bailey, C. Whitehead, J. T. A. Proctor, and J. T. Kyle. eds. The Challenges of the 21st Century, Proceedings of the International Ginseng Conference, 17-22 July, 1994, Vancouver, BC. W. G. Bailey, Department of Geography, Simon Fraser University, Burnaby, BC. (1995).
6. Lee, J. C., Chung, Y. R. and Ohh, H. P. S. H. : Influence of seed dressing with Captan WP on the dehiscence of *Panax ginseng* seeds. *Korean J. Ginseng Sci.* **28**, 262-266 (1981).
7. Tianyu, Z. and Weiqun, C. : A preliminary study of seed pathology of *Panax quinquefolium*. *Korean J. Ginseng Sci.* **16**, 244-245 (1992).
8. Tianyu, Z., Yong, Z., Weiqun, C. and Changwei, Z. : An analysis of seedborne fungi of imported seeds of *Panax quinquefolium*. *Acta Phytopathol. Sin.* **19**, 134 (1989).
9. Howard, R. J., Garland, J. A. and Seaman, W. L., eds. : Diseases and Pests of Vegetable Crops in Canada. The Canadian Phytopathological Society and Entomological Society of Canada, Ottawa, Ontario. 554 pp. (1994).
10. Reeleder, R. D. and Fisher, P. : Diseases of Ginseng. Ontario Ministry of Agriculture, Food and Rural Affairs Factsheet 260/630. 4 pp (1995).
11. Hildebrand, A. A. : Root rot of ginseng in Ontario caused by members of the genus *Ramularia*. *Can. J. Res.* **12**, 82-114 (1935).
12. Anonymous : Ginseng Pest Control Recommendations. Ontario Ministry of Agriculture, Food and Rural Affairs, Toronto, Ontario. Publication

610. 31 pp.(1997, 1997-1998).
13. Booth, C. : The Genus *Cylindrocarpon*. Commonwealth Mycological Institute, Mycological papers 104. 56 pp. (1966).
14. Cain, R. F. : Studies of coprophilous Ascomycetes. *Can. J. Bot.* **34**, 699-710 (1956).
15. Domsch, K. H., Gams, W., and Anderson, T. H. : Compendium of Soil Fungi. Vol. 1. Academic Press, New York. 859 pp. (1980).
16. Guarro, J., Cannon, P. E. and van der AA, H. A. : A synopsis of the genus *Zopfiella* (Ascomycetes, Lasiosphaeriaceae). *Systema Ascomycetum* **10**, 79-112 (1992).
17. Nelson, P. E., Toussoun, T. A. and Marasas, W. F. O. : Fusarium species : An Illustrated Manual for Identification. The Pennsylvania State University Press, University Park, PA. 198 pp. (1983).
18. von Arx, J. A. : Plant Pathogenic Fungi. Cramer, Berlin. 288 pp. (1987).
19. Mathre, D. E. and Hansing, E. D. : Evaluating seed-treatment fungicides. Pages 248-451 in K. D. Hickey, ed. Methods for Evaluating Pesticides for Control of Plant Pathogens. APS Press, St. Paul., MN.
20. Ziezold, A. M., Hall, R., Reeleder, R. D. and Proctor, J. T. A. : Toxicity of fungicides in vitro to *Cylindrocarpon destructans*. *Korean J. Ginseng Sci.* (submitted).
21. Steel, R. G. D. and Torrie, J. H. : Principles and Procedures of Statistics : A Biometrical Approach. McGraw-Hill, New York, NY. 633 pp. (1980).
22. Cheon, S. R. and Kim, H. J. : Comparison of resistance to root rot caused by *Fusarium solani* in ginseng breeding lines. *Korean J. Ginseng Sci.* **14**, 50-56. (1990).
23. Parke, J. L. and Shotwell, K. M. : Diseases of Cultivated Ginseng. Univ. Wis. Madison Coll. *Agric. Life Sci. Res. Div. Res. Bull.* 3465. 16 pp. (1989).
24. Brammall, R. A. : Disappearing root rot. Pages 296-297 in R. J. Howard, J. A. Garland, and W. L. Seaman, eds. Diseases and Pests of Vegetable Crops in Canada. The Canadian Phytopathological Society and Entomological Society of Canada, Ottawa, Ontario. (1994).
25. Nelson, P. E., Toussoun, T. A. and Cook, R. J., eds. : Fusarium : Diseases, Biology, and Taxonomy. The Pennsylvania State University Press, University Park, PA. 457 pp. (1981).
26. Ziezold, A. M., Hall, R., Reeleder, R. D. and Proctor, J. T. A. : Effect of drenching soil with benomyl, propiconazole, and fluazinam on incidence of disappearing root rot of ginseng, *Korean J. Genseng Sci.* (submitted).
27. Dhingra, O. D. and Sinclair, J. B. : Basic Plant Pathology Methods. CRC Press Inc., Boca Raton, FL. 355 pp. (1985).
28. Hansing, E. D : Evaluation of seed treatment fungicides. Fungicide and Nematicide Tests 30, 1-3 (1975) *American Phytopathological Society*, St. Paul, MN.
29. Kraft, J. M. : Field and greenhouse studies on pea seed treatments. *Plant Dis.* **66**, 798-800 (1982).