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**Note**

# Physiological Races of *Phytophthora infestans* in Korea

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**A total of the 261 *Phytophthora infestans* isolates collected from 2003 to 2005 in Korea were investigated for their physiological race composition. Among the isolates, we detected 18 physiological races and the dominant races were R0.1.3.5.6.10.11 and R0.1.3.5.6.7.10.11 with frequencies of 18.4% and 11.4%, respectively. All of the *P. infestans* races carried multiple virulence genes and showed virulence to the potato resistance genes R1, R3, R5, R6, R7, R10 and R11, but not to R8 and R9. Therefore, it is likely that the physiological races of *P. infestans* were diverse in Korea.**

***Keywords*** : *Phytophthora infestans*, physiological races, virulence

Potato late blight, caused by the heterothallic oomycete *Phytophthora infestans*, was most commonly associated with the Irish Potato Famine of the 1840s and is one of the most devastating potato diseases in many parts of the world. The disease is most destructive in the area with frequent cool and moist weather. Late blight may kill the foliage, stems and tubers or fruits of potato (*Solanum tuberosum*) or tomato (*Solanum lycopersicum*) plants at any time during the growing season (Fry et al., 1992; Goodwin et al., 1992b). Original inoculum of potato late blight pathogen is sporangia or zoospores produced through asexual reproduction. The diseased seed tuber is a source of primary infection of late blight. Though the heavily infected-tubers rot during storage, those infected lightly are used for planting and are stored until the following spring.

Physiological race is a major factor for the potato breeding (Anonymous et al., 1996). Several major genes (R-genes) for resistance to *P. infestans* have been identified in the potato. The resistance associated with the presence of these genes was not durable (Turkensteen 1993). Therefore, it has been suggested that only minor resistance genes are desired, providing a general resistance, independent of the virulence of the pathogen (Wastie 1991; Umaerus and Umaerus 1994). The most important problem of genetic variability in the pathogens is the difference of virulence toward the host. The variability of virulence in *P. infestans* populations is recognized as a major reason for the breakdown of disease resistance in the cultivated potato (Wastie 1991). According to the report of Swiezynski et al. (2000), isolates collected throughout the world represented the races with 3-11 of complex virulence spectra. Knapova and Gisi (2002) reported that potato isolates represented the races with highly complex virulence spectra than tomato isolates.

The objective of this study was to examine physiological race of *P. infestans* isolated in Korea. This result would be useful for disease resistance and selection program, and provide further information regarding to the origin of *P. infestans* in Korea.

From 2003 to 2005, potato leaflets with symptoms of late blight were collected from commercial fields of five provinces (including Jeju-do) and from agricultural research stations in Korea. For fungal isolation, the healthy potato tuber surface was sterilized using 70% of ethanol and were sliced by knife with 5-8 mm thickness. Potato tuber slices were put on the diseased leaf fragment in Petri dishes and incubated for 5-7 days at 22°C. The fungal mycelia grew actively on the surface of the tuber slices. Each isolate was obtained by transferring hyphal tips on a selective V-8 juice agar (200 ml V-8 juice, 4.5 g calcium carbonate, 20 g agar, 800 ml distilled water) containing 500 mg/l of ampicillin, 200 mg/l of vancomycin, 50 mg/l of rifampicin, 100 mg/l of pimaricin, 35 mg/l of PCNB, and 10mg/l of benomyl. Purified isolates were maintained at 22°C on V-8 juice rye agar medium.

To determine the race differentiation of the *P. infestans* isolates, they were grown on V-8 juice rye agar medium for 14 days at 22°C in darkness. The cultures were washed and rubbed with 5 ml of distilled water to prepare sporangial suspensions. The suspension concentration was adjusted to $5\times10^4$ sporangia per milliliter using a hemacytometer. The sporangium suspensions were placed in a refrigerator (4°C) for 1 hour until ready to be used.

*In vitro* plantlet of differential hosts carried a single specific gene for resistance (R1 to R11) against *P. infestans* and the cultivar "Atlantic" as a susceptible control contain-

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ed no known R gene. The plants were grown for 4 to 6 weeks in the greenhouse with natural sunlight. Detached leaf assay test was used to assess the virulence of each isolate. Leaves of similar age and size were detached from each test plant and leaflets were sprayed with sterile distilled water by a hand sprayer. The leaflets were placed abaxial side up on a 2% water agar medium. Each leaflet was inoculated with two drops (20 μl) of sporangia suspension adjusted to a concentration of about $5 \times 10^4$ sporangia per ml. A single leaflet was used as an experimental unit and three to four replications were conducted per test. Petri plates were placed on damp tissue in transparent boxes and covered with transparent plastic sheets. Leaflets were incubated at room temperature (about 23°C) and inverted at 24 h after inoculation. The reaction of each leaflet was examined with a light microscope for sporulation of *P. infestans* at 5 days after inoculation. If sporulation was observed, the interaction was rated as compatible; if sporulation was not observed, the interaction was rated as incompatible. The number of compatible interactions for each isolate was recorded.

A total of 261 isolates collected from 2003 to 2005 were used to investigate the physiological race of *P. infestans* in Korea. In 2003, five physiological races were detected and the race 0.1.3.5.6.10.11 was most common in potato field in Korea. The frequency of these races was accounted for 58.1%. Among the 190 isolates collected in 2004, 18 physiological races were detected, and the dominant races were R0.1.3.5.6, R0.1.3.5.6.10.11, and R0.1.2.3.4.5.6.10.11. The frequencies of the races were accounted for 12.1%, 14.7% and 15.3%, respectively. Other races were existed to lower frequency less than 10%. Among the total of 33 isolates of *P. infestans* collected from 2005, eight physiological races occurred, and R0.1.3.5.6.7.10.11 was the most common race. The frequency of race was accounted for 57.6% (Table 1). The physiological races from Gangneungshi and Pyongchanggun were composed to 16 and 15 races, respectively. The dominant race in Gangneungshi and Pyongchanggun were R0.1.3.5.6.7.10.11 and R0.1.3.5.6.10.11, respectively. However, the other locations were composed

**Table 1.** Prevalence of physiological race of *Phytophthora infestans* isolates collected from various locations in Korea from 2003 to 2005

| Location | No. of Isolates | Physiological race (%)[a] | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | A | B | C | D | E | F | G | H | I | J | K | L | M | N | O | P | Q | R |
| 2003 | | | | | | | | | | | | | | | | | | | |
| Gangneungshi | 11 | 0 | 3 | 5 | 0 | 2 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Jungsungun | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Pyongchanggun | 13 | 0 | 2 | 8 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 |
| Yangyanggun | 3 | 0 | 0 | 2 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Jejudo | 3 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 |
| Sub total number | 31 | 0 | 5 | 18 | 0 | 5 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 |
| % | | 0 | 16.1 | 58.1 | 0 | 16.1 | 0 | 3.2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 6.5 | 0 | 0 | 0 |
| 2004 | | | | | | | | | | | | | | | | | | | |
| Gangneungshi | 104 | 0 | 15 | 10 | 2 | 8 | 2 | 8 | 0 | 3 | 7 | 18 | 2 | 9 | 4 | 4 | 3 | 7 | 2 |
| Jungsungun | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 |
| Pyongchanggun | 81 | 1 | 8 | 18 | 6 | 3 | 0 | 2 | 1 | 5 | 6 | 11 | 0 | 6 | 1 | 4 | 1 | 8 | 0 |
| Yangyanggun | 3 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 |
| Sub total number | 190 | 1 | 23 | 28 | 8 | 11 | 2 | 11 | 1 | 8 | 13 | 29 | 2 | 17 | 5 | 8 | 4 | 17 | 2 |
| % | | 0.5 | 12.1 | 14.7 | 4.2 | 5.8 | 1.0 | 5.8 | 0.5 | 4.2 | 6.9 | 15.3 | 1.0 | 9.0 | 2.6 | 4.2 | 2.1 | 9.0 | 1.0 |
| 2005 | | | | | | | | | | | | | | | | | | | |
| Gangneungshi | 23 | 0 | 2 | 2 | 0 | 14 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 1 |
| Kimcheongun | 7 | 0 | 0 | 0 | 0 | 4 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 0 |
| Namwongun | 3 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| Boseonggun | 7 | 0 | 0 | 0 | 0 | 5 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 |
| Sub total number | 40 | 0 | 2 | 2 | 0 | 25 | 0 | 0 | 0 | 3 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 5 | 1 |
| % | | | 5 | 5 | | 62.5 | | | | 7.5 | | | | 2.5 | | 2.5 | | 12.5 | 2.5 |
| Total number | 261 | 1 | 30 | 48 | 8 | 41 | 2 | 12 | 1 | 11 | 13 | 29 | 2 | 18 | 5 | 11 | 4 | 22 | 3 |
| % | | 0.4 | 11.5 | 18.4 | 3.1 | 15.7 | 0.8 | 4.6 | 0.4 | 4.2 | 5.0 | 11.1 | 0.8 | 6.9 | 1.9 | 4.2 | 1.5 | 8.4 | 1.2 |

[a] A: race0.1.3; B: race0.1.3.5.6; C: race0.1.3.5.6.10.11; D: race0.1.3.5.6.7; E: race0.1.3.5.6.7.10.11; F: race0.1.2.3.5.6; G: race0.1.2.3.5.6.10.11; H: race0.1.2.3.5.6.7; I: race0.1.2.3.5.6.7.10.11; J: race0.1.2.3.4.5.6; K: race0.1.2.3.4.5.6.10.11; L: race0.1.2.3.4.5.6.7; M: race0.1.2.3.4.5.6.7.10.11; N: race0.1.3.4.5.6; O: race0.1.3.4.5.6.10.11; P: race0.1.3.4.5.6.7; Q: race0.1.3.4.5.6.7.10.11; R: race0.3.5.6.10.11.

**Table 2.** Distribution of physiological race of *P. infestans* isolated from various locations in Korea from 2003 to 2005

| Location | No. of Isolates | Physiological race (%)[a] | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | A | B | C | D | E | F | G | H | I | J | K | L | M | N | O | P | Q | R |
| Gangneungshi | 138 | 0 | 20 | 17 | 2 | 24 | 2 | 9 | 0 | 5 | 7 | 18 | 2 | 10 | 4 | 4 | 3 | 8 | 3 |
| Jungsungun | 3 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 |
| Pyongchanggun | 94 | 1 | 10 | 26 | 6 | 5 | 0 | 2 | 1 | 5 | 6 | 11 | 0 | 6 | 1 | 5 | 1 | 8 | 0 |
| Yangyanggun | 6 | 0 | 0 | 2 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 |
| Boseonggun | 7 | 0 | 0 | 0 | 0 | 5 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 |
| Kimcheongun | 7 | 0 | 0 | 0 | 0 | 4 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 0 |
| Namwongun | 3 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| Jejudo | 3 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 |
| Total | 261 | 1 | 30 | 48 | 8 | 41 | 2 | 12 | 1 | 11 | 13 | 29 | 2 | 18 | 5 | 11 | 4 | 22 | 3 |
| % | | 0.4 | 11.5 | 18.4 | 3.1 | 15.7 | 0.8 | 4.6 | 0.4 | 4.2 | 5.0 | 11.1 | 0.8 | 6.9 | 1.9 | 4.2 | 1.5 | 8.4 | 1.2 |

[a]A: race0.1.3; B: race0.1.3.5.6; C: race0.1.3.5.6.10.11; D: race0.1.3.5.6.7; E: race0.1.3.5.6.7.10.11; F: race0.1.2.3.5.6; G: race0.1.2.3.5.6.10.11; H: race0.1.2.3.5.6.7; I: race0.1.2.3.5.6.7.10.11; J: race0.1.2.3.4.5.6; K: race0.1.2.3.4.5.6.10.11; L: race0.1.2.3.4.5.6.7; M: race0.1.2.3.4.5.6.7.10.11; N: race0.1.3.4.5.6; O: race0.1.3.4.5.6.10.11; P: race0.1.3.4.5.6.7; Q: race0.1.3.4.5.6.7.10.11; R: race0.3.5.6.10.11.

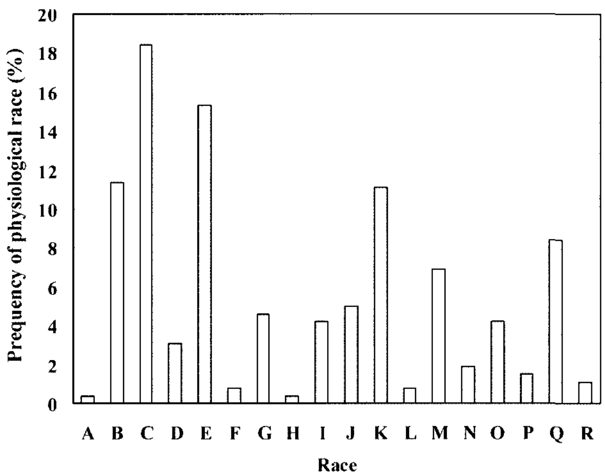


**Fig. 1.** The percentages of physiological race in *P. infestnans*. Each physiological race was same as in Table 1.

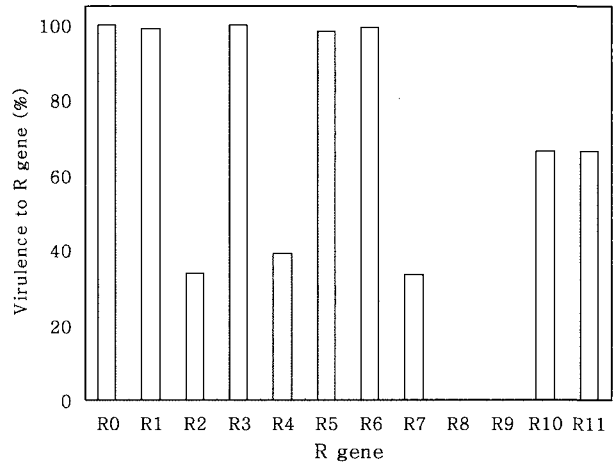


**Fig. 2.** The percentages of virulence genes in *P. infestnans*.

of 2 to 4 races (Table 2). All of the isolates carried two or more complex virulence genes and they could breakdown the host containing several resistance genes. Overall, race 0.1.3.5.6.10.11 out of the 18 physiological races was detected with the highest frequency (Fig. 1). Most isolates of *P. infestans* collected from 2003 to 2005 were virulent to the potato carrying the resistance genes R1, R3, R5 and R6. However, all of the isolates were not virulent to the R8 or R9 plants (Fig. 2). Therefore, the breeding program against potato late blight in Korea needs to be modified according to these changes of *P. infestans* in potato fields. The analysis of physiological race in the *P. infestans* population should be necessary to breed resistant potato as well as to study the pathogen migration. The physiological races of the pathogen would gradually change according to host and climatic condition around the potato fields.

First study for physiological race of *P. infestans* was carried out from 1968 to 1981 by Hahm and Kang (1974) using four virulence factors available since 1950s (Black et al., 1953) in Korea. However, differential plant varieties for the identification of 11 virulence factors were available from 1968 (Malcolmson, 1969). Hahm and Kang (1974) reported that 11 physiological races occurred in Korea, of which race0, race3 and race4 were most common. According to our results using 11 virulence factors, race0.1.3.5.6.10.11 and race0.1.3.5.6.7.10.11 out of the isolates investigated in this study were predominant. Swiezynski et al. (2000) reported that isolates collected throughout the world represented races carrying 3-11 of complex virulence spectra. Vanderplank (1968) stated that when a vertical resistance gene was present in the host, the pathogen must be able to develop virulence genes to match resistance genes of host plant. However, most commercial potato cultivars growing in Korea did not contain any resistance gene. Therefore, occurrence of diverse physiological races in Korea could not support the Vanderplank's theory. Instead, it has been thought that zoospores produced through asexual reproduction were differentiated to diverse race to survive in very bad environment as described by Abu-El Samen (2003). Another possibility is the pathogen influx from foreign countries with potatoes.

## References

Abu-El Samen, F. M., Secor, G. A. and Gudmestad, N. C. 2003. Variability in virulence among asexual progenies of *Phytophthora infestans*. *Phytopathology* 93:293-304.

Anonymous, 1996. CIP Circular 22(1). International Potato Center, Lima, Peru.

Black, W., Mastenbroek, C., Mills, W. R. and Peterson, L. C. 1953. A proposal for an international nomenclature of races of *Phytophthora infestans* and of genes controlling immunity in Solanum demissum derivatives. *Euphytica.* 2:173-179.

Fry, W. E., Goodwin, S. B., Matuszak, J. M., Spielman, L. J., Milgroom, M. G. and Drenth, A. 1992. Population genetics and intercontinental migration of *Phytophthora infestans*. *Annu. Rev. Phytopathol.* 30:107-129.

Goodwin, S. B., Spielman, L. J., Matuszak, J. M., Bergeron, S. N. and Fry, W. E. 1992. Clonal diversity and genetic differentiation of *Phytophthora infestans* populations in northern and central Mexico. *Phytopathology* 82:955-961.

Hahm, Y. I. and Kang, E. H. 1974. Physiologic race of *Phytophthora infestans* (Mont.) de Bary at alpine area, Korea during the period from 1971 to 1973. *Korean J. Plant Protec.* 13: 227-231.

Knapova, G. and Gisi, U. 2002. Phenotypic and genotypic structure of *Phytophthora infestans* populations on potato and tomato in France and Switzerland. *Plant Pathol.* 51:641-653.

Swiezynski, K. M., Domanski, L., Zaraycka, H. and Zimnoch-Guzowska, E. 2000. The reation of potato differentials to *Phytophthora infestans* isolates collected in nature. *Plant Breed.* 119:119-126.

Turkensteen, L. J. 1993. Durable resistance of potatoes against *Phytophthora infestans.* In: Durability of Disease Resistance, ed. by T. Jacobs and J. E. Parlevliet, pp. 115-124. Kluwer Academic Publishing., Dordrecht, Holland.

Wastie, R. L. 1991. Breeding for resistance. *Adv. Plant Pathol.* 7: 193-224.

Umaerus, V. and Umaerus, M. 1994. Inheritance of resistance to late blight. In: Potato Genetics, ed. by J. E. Bradshaw and G. R. Mackay, pp. 365-401. CAB International, Wallingford, UK.

Vanderplank, J. E. 1968. Disease resistance in plants. New York, Academic. 349pp.