***axonopodis* pv. *citri*, causing citrus bacterial canker disease.**

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Relative degree of resistance of citrus to *Xanthomonas axonopodis* pv. *citri*, the causal bacterium of canker, was investigated. Growth rate of a bacterium in leaf tissues after infiltration, disease incidence, and percent of lesion area were compared. By using growth rate[$(GR=(A_t - A_{t-1})/A_{t-1}$] host plants were differentiated into susceptible and resistant. Growth rates reached to peak at 40 hrs after inoculation and then declined. The growth rate in leaf tissues of a moderately susceptible cultivar, *Citrus sinensis* var. Lane late(sweet orange), was the highest, and those of *C. unshiu* × *C. sinensis*(kiyomi), *C. junos*(yuzu), [(*Citrus. unshiu* × *C. sinensis*) × *C. reticulata*](shiranuhi), and *C. unshiu*(satuma mandarin) were similar. This result indicates that the growth rate of the bacterium in leaf tissues can be effectively used for evaluation of disease resistance for citrus plants to *X. axonopodis* pv. *citri*. The disease on sweet orange occurred earlier than relatively resistant citrus plants tested. The percent of lesion area on leaf was also higher in sweet orange than those of satsuma mandarin, shiranuhi and kiyomi, and yuzu. The disease severity was highest on sweet orange and followed by kiyomi, shiranuhi, satsuma mandarin, and yuzu.

3-21. Dispersal of *Xanthomonas axonopodis* pv. *citri*, the Causal Bacterium of Citrus Canker, on Unshiu Orange.

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Dispersal of *Xanthomonas axonopodis* pv. *citri*, causing citrus bacterial canker disease on Unshiu orange was investigated at previously infested plots at Seogwipo in Jeju island of Korea. The bacterial pathogen overwintered in lesions started to multiply at late May, and disease firstly observed one month after detection of phage from lesions. The disease gradually increased, however, it dispersed non-directionally to nearby plants from inoculum sources. Diseased plants were aggregated to form a cluster throughout the experiment. Population dynamics of phage on symtomless leaf surface and the disease severity were compared in the nursery. Increase of phage population on symptomless leaf surface preceded one month to that of the disease severity. Population of phage increased constantly from late July to October, however, the disease severity decreased from late August to late October. It was assumed that the decrease of disease severity might be due to disease-induced defoliation.

3-22. Characterization of disease outbreak pattern of transgenic potato plants with the coat protein gene of Potato leaf roll virus.

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Since the demonstration that the transgenic plants expressing tobacco mosaic virus(TMV) coat protein(CP) gene showed resistance to TMV infection, there have been numerous attempts to produce virus-resistant plant by introducing of a part of or modified viral genome. This study was conducted to investigate the characterization and variability of disease outbreak of transgenic potato(T-potato) with the CP gene of potato leaf roll virus(PLRV) in an isolated field from 2000 to 2002. In the field inspection, incidence of PLRV on T-potato showed only 3.5%, while non-transgenic potato(N-potato) revealed 13.4%. Infection rate of PLRV was considerably low on T-potato with 4.2% compared to 15.4% of N-potato in ELISA tests. Those of potato virus M, potato virus Y and potato virus X on both potatoes were not statistically different. Infection of potato virus A was not observed on both potatoes. Incidence of potato late blight caused by *Phytophthora infestans* on T-potato and N-potato did not differ each other with 52.7%, and 50.8%, respectively. Mating type of the causal fungus isolated from both potatoes was all A1 types. Results indicates that the CP gene of PLRV affects specifically to the virus in the transgenic potato.

3-23. Molecular pathological interactions between Apple stem grooving virus (ASGV) and its fungi.

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Apple stem grooving virus (ASGV) belongs to *Capillovirus* and infects pome fruits. Transmission mode of ASGV is known by grafting and mechanical inoculation into susceptible hosts, not by any other natural vectors. But we have observed the spread of ASGV in the field without mechanical inoculation or grafting. Transmission seems to be occurred from tree-to-tree and tree-to-susceptible herbaceous plants along but not across ditches in the field. In order to ascertain this possibility, various fungi were isolated and cultured from ASGV-infected plants and 69 isolates were characterized. By means of RNA dot-blot hybridization and PCR analysis, 3 isolates were sorted out for further studies. The isolates were identified to *Talaromyces sp.* and belonged to *Phenicillium* by morphological characteristics and molecular markers. As an experimental host, 10 kidney beans (*Phaseolus vulgaris*) were screened and Kyunggi-5 was selected for virus amplification and symptom development. Kyunggi-5 infected by fungi which seemed to carry ASGV showed the typical disease symptoms and viral coat protein genes were detected from