O-7. Antiviral *R* Genes of Plants May Have Experienced Different "Selections" in Nature and in Crop Fields

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Plant genomes carry more than 100 copies of *R* (*Resistance*) genes that encode receptor proteins. Each of the gene products directly or indirectly recognizes a pathogen-derived protein to induce resistance against the pathogen. An *R* gene of *Arabidopsis thaliana*, *RCY1*, recognizes the capsid protein (CP) of cucumber mosaic virus (CMV) and induces hypersensitive reaction (HR), which is characterized by programmed-cell death of the infected cells and inclusion of the virus in the inoculated leaves. Our recent study showed that the average number of CMV genomes that established cell infection (i.e., MOI, multiplicity of infection) after cell-to-cell movement decreased from 3.60±0.26 to 2.76±0.28 upon resistance induction. A CMV mutant that shows smaller decrease of MOI against *R*-gene recognition caused systemic HR (SHR) of the plants, which results in systemic death of the plant individuals. This result suggests that inefficient recognition allow expansion of the virus and cause death of the plant individual. A simulation suggested that such an individual death can function as a suicide strategy to protect neighboring plants, that are often "kin" of an infected plant, by diminishing the source of infection. Thus, systemic death caused by inefficient virus recognition by *R* genes can be positively selected in nature, serving as a starting point for further adaptation toward more efficient recognition. On the other hand, systemic death may have been negatively selected in crop field during the history of crop-plant domestication. Based on this idea, I will discuss possible new strategies for hunting *R* genes.