Supporting Information

Yu et al. 10.1073/pnas.1213755110

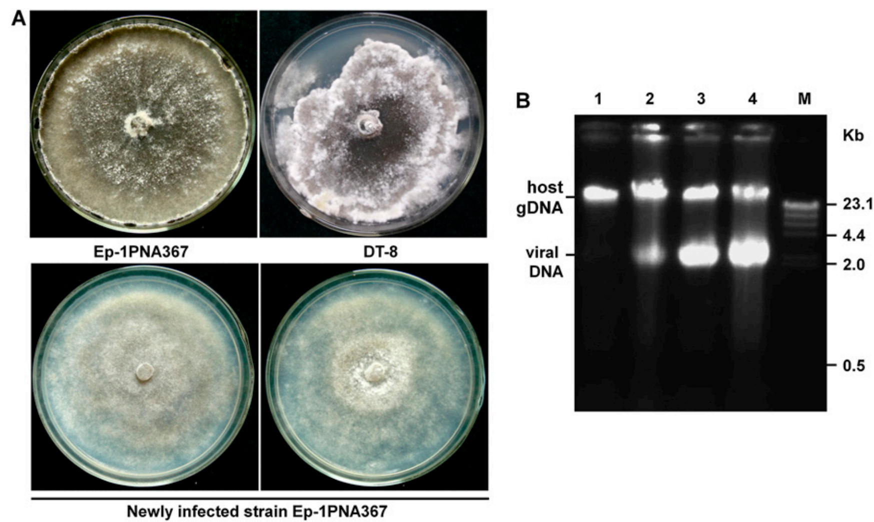

Fig. S1. Virus particles of SsHADV-1 infect the protoplasts of *Sclerotinia sclerotiorum* without being mediated by PEG. (*A*) Colony morphology of viral newly infected strain Ep-1PNA367. (*B*) Viral genome DNA extracted from newly infected strain Ep-1PNA367. Lane 1, strain Ep-1PNA367; lane 2, strain DT-8; lanes 3 and 4, newly infected strain Ep-1PNA367; lane M, weight marker. Protoplasts of *S. sclerotiorum* were directly mixed with virus particles.

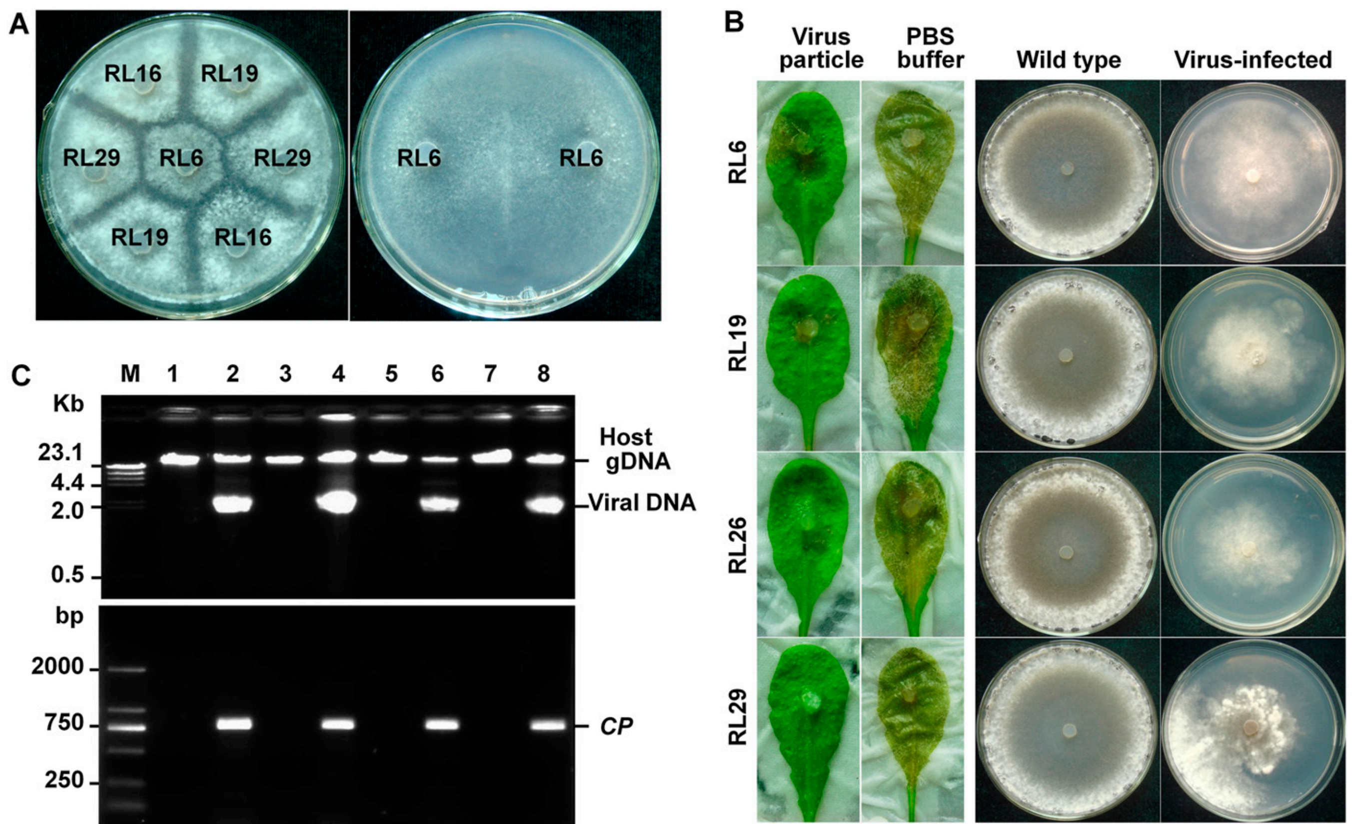

Fig. S2. Extracellular transmission of SsHADV-1 particles to *S. sclerotiorum* isolates belonging to different vegetative compatibility groups (VCGs). (*A*) Four isolates cultured on the same potato dextrose agar (PDA) plate containing red food coloring to show the incompatibility reaction zones. One randomly selected isolate (RL6) was dual-cultured in one PDA plate to show compatible reaction. (*B*) Suppression of *S. sclerotiorum* (strain Ep-1PNA367) lesion development on detached *Arabidopsis thaliana* leaves previously treated with virus particles and isolation of virus-infected colonies from such abnormal lesions. The wild-type strain and virus-infected isolates were incubated on PDA plates for 7 d at 20 °C. (*C*) The virus-infected cultures were confirmed both by total genomic DNA analysis and PCR amplification. PCR amplification was performed using specific primers, which were designed based on the *CP* sequence of SsHADV-1. DNA samples (*Upper*) and PCR products (*Lower*) were fractionated on 1.0% agarose gel. Lane M: λ-Hind III-digested DNA marker (*Upper*) and DL2000 DNA marker (*Lower*). Lanes 1 and 2; 3 and 4; 5 and 6; and 7 and 8 indicate the wild-type and virus-infected isolates of RL6, RL19, RL26, and RL29, respectively.

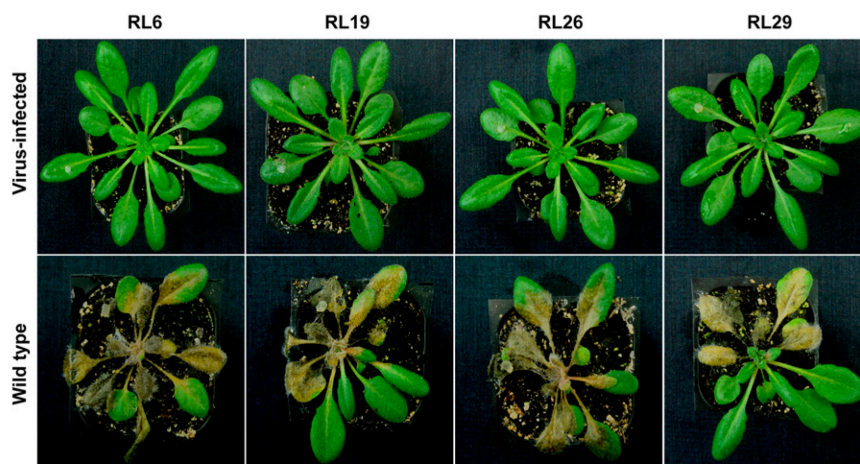

Fig. S3. Virulence of four *S. sclerotiorum* strains (RL6, RL19, RL26, and RL29) belonging to different vegetative compatibility groups on plants of *A. thaliana* before or after infection with SsHADV-1. Inoculated plants were incubated under high relative humidity (100%) at 20 °C for 4 d.

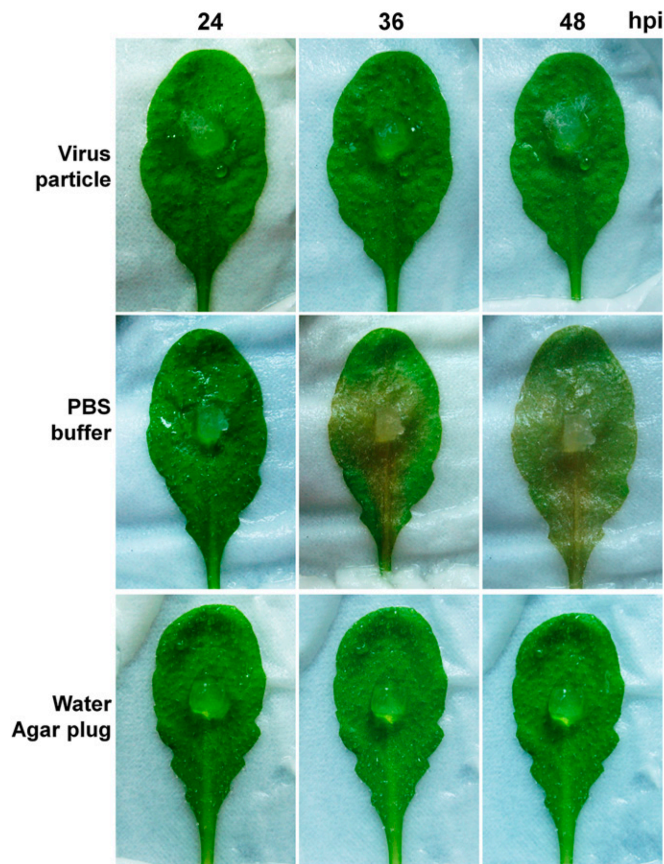

Fig. S4. Infection of *S. sclerotiorum* strain Ep-1PNA367 with purified particles of SsHADV-1 on detached leaves of *A. thaliana*. Lesion development on virus-treated leaves was delayed and the lesions were much smaller than those on PBS buffer-treated leaves. Agar plugs were used as control.

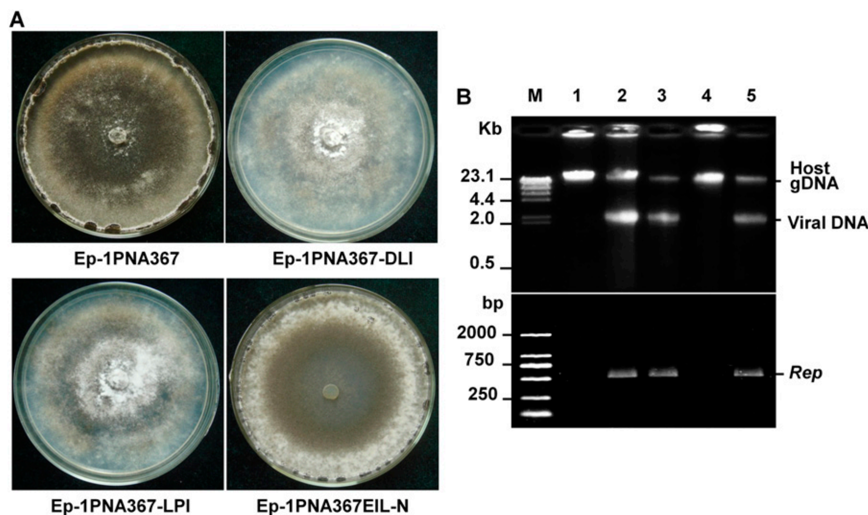

Fig. S5. SsHADV-1–infected *S. sclerotiorum* strains isolated from virus-treated lesions. (*A*) Colony morphology of cultures that were reisolated from atypical lesions caused by strain Ep-1PNA367 on virus-inoculated leaves. Cultures isolated from atypical lesions on detached leaves or intact plants were designated Ep-1PNA367-DLI and Ep-1PNA367LPI, respectively; typical lesions on nontreated leaves were designated Ep-1PNA367EIL-N. The virus-free strain Ep-1PNA367 was used as control. All cultures were incubated on PDA plates for 7 d at 20 °C. (*B*) Total DNA samples extracted from cultures, and PCR amplification to verify infection of strain Ep-1PNA367 by SsHADV-1. A specific primer pair used for PCR amplification was designed based on the Rep sequence. DNA samples (*Upper*) and PCR products (*Lower*) were analyzed on 1.0% agarose gel. Line M: λ-Hind III-digested DNA marker (*Upper*) and DL2000 DNA marker (*Lower*). Lanes 1–5, DNA samples from isolates Ep-1PNA367, Ep-1PNA367-DLI, Ep-1PNA367-LPI, Ep-1PNA367EIL-N, and DT-8. The positions of host genomic DNA and viral DNA are indicated.

Fig. S6. SsHADV-1 particles inhibited infection of rapeseed (*Brassica napus*) leaves with *S. sclerotiorum* via contact with infected petals. Ten microliters of viral particles (2 mg/mL) or 0.01 M phosphate buffer (pH 7.0) were added as droplets on leaf surface, and then petals were placed on droplets of viral suspension or PBS buffer. Twenty microliters of ascospores suspension of *S. sclerotiorum* (1 × 106 ascospores/mL) was inoculated onto the upper side of petals. Leaves were kept in an incubator with high humidity (100%) at 20 °C for 4 d.