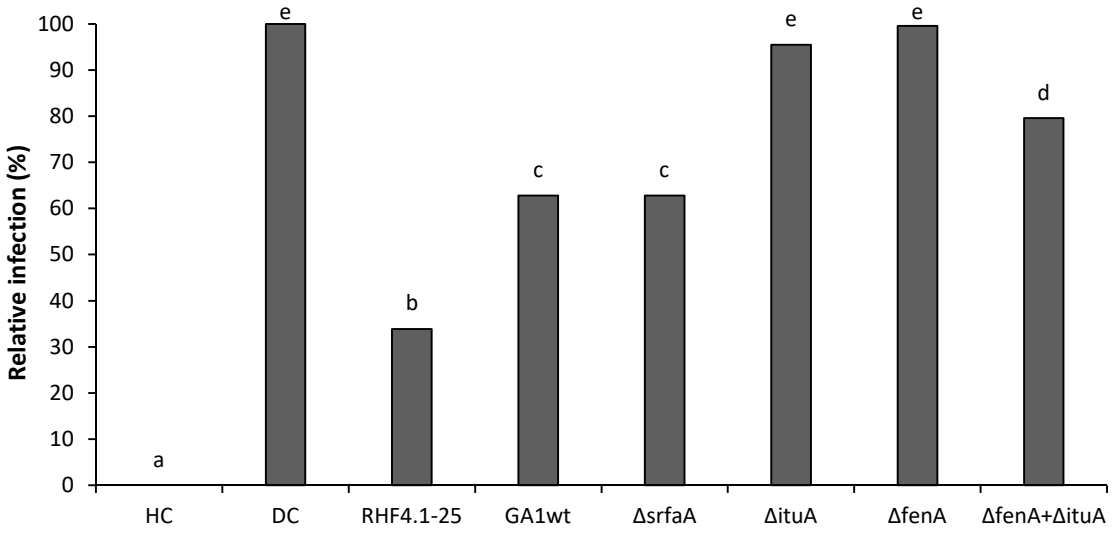

Figure S1. Potential of *B. velezensis* isolates RHF4.1-25 and GA1 and GA1 mutants impaired in CLiP production to induce systemic resistance against *P. oryzae* in rice (variety CO39) grown in sterile potting soil. Data from Figure 4 are here expressed as % relative infection in comparison with the diseased control (n=27). Univariate ANOVA followed by Duncan's post hoc tests were used and different letters among these treatments indicate statistically significant differences ($P < 0.05$). HC: healthy control; DC: diseased control; RHF4.1-25 and GA1: wild type *B. velezensis*; Δ*srfaA*: surfactin mutant of GA1; Δ*ituA*: iturin mutant of GA1; Δ*fenA*: fengycin mutant of GA1; Δ*fenA* + Δ*ituA*: combined inoculation with fengycin and iturin mutant of GA1.

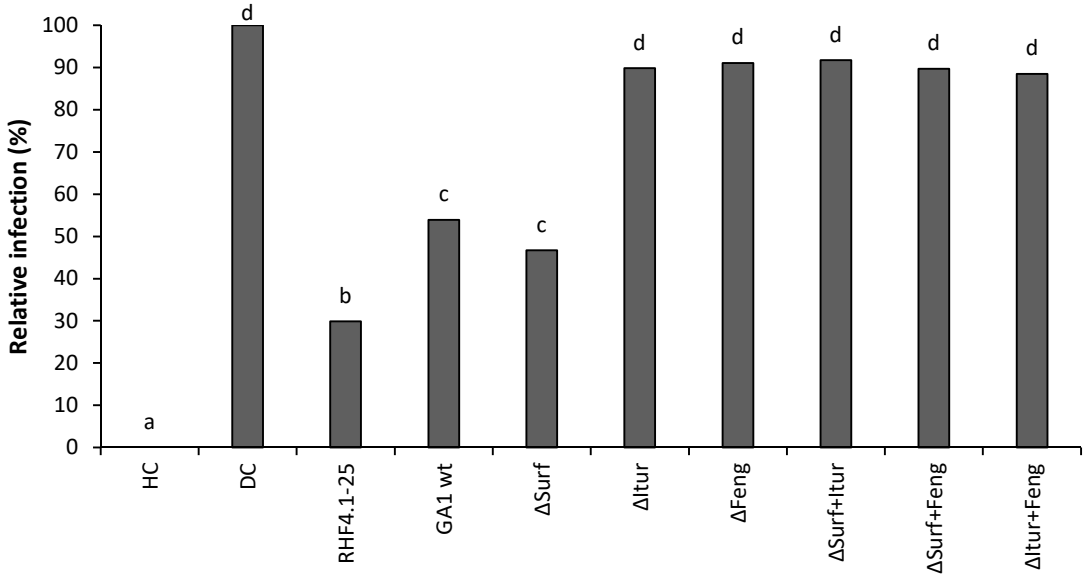

Figure S2. Potential of *B. velezensis* isolates RHF4.1-25 and GA1, and GA1 mutants impaired in CLiP production to induce systemic resistance against *P. oryzae* in rice (variety CO39) grown in sterile **potting soil**. Data from Figure 5 are here expressed as % relative infection in comparison with the diseased control (n=27). Univariate ANOVA followed by Duncan's post hoc tests were used and different letters among these treatments indicate statistically significant differences ($P < 0.05$). HC: healthy control; DC: diseased control: RHF4.1-25 and GA1: wild type *B. velezensis*; Δ*srfaA*: surfactin mutant; Δ*ituA*: iturin mutant; Δ*fenA*: fengycin mutant. Δ*srfaA-ituA*: double mutant impaired in surfactin and iturin production; Δ*srfaA-ituA*: double mutant impaired in surfactin and fengycin production; Δ*fenA-ituA*: double mutant impaired in iturin and fengycin production.

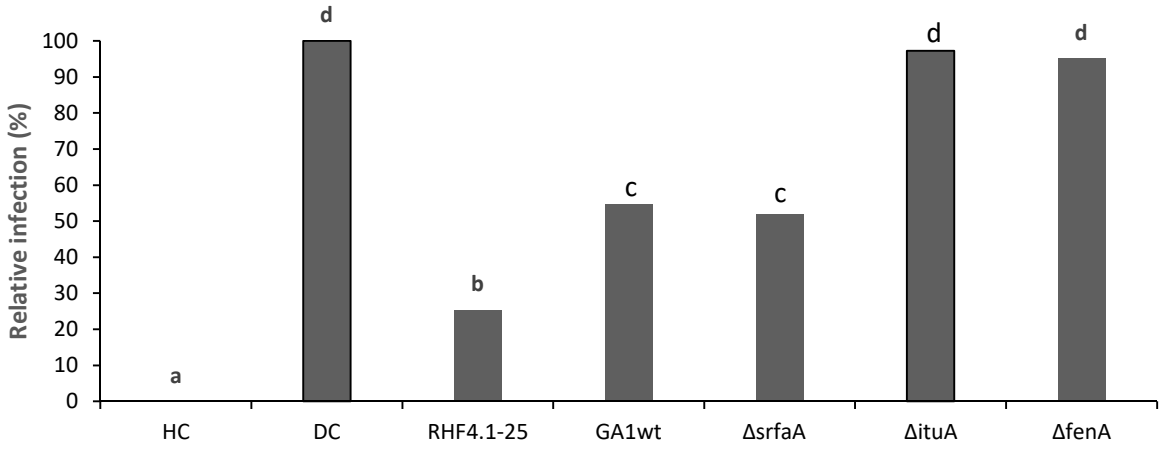

Figure S3. Potential of *B. velezensis* isolates RHF4.1-25 and GA1, and GA1 mutants impaired in CLiP production to induce systemic resistance against *P. oryzae* in rice (variety Jasmine 85) grown in sterile acid sulfate soil. Data from Figure 5 are here expressed as % relative infection in comparison with the diseased control (n=15). Univariate ANOVA followed by Duncan's post hoc tests were used and different letters among these treatments indicate statistically significant differences ($P < 0.05$). HC: healthy control; DC: diseased control; RHF4.1-25 and GA1: wild type *B. velezensis*; Δ*srfaA*: surfactin mutant of GA1; Δ*ituA*: iturin mutant of GA1; Δ*fenA*: fengycin mutant of GA1.

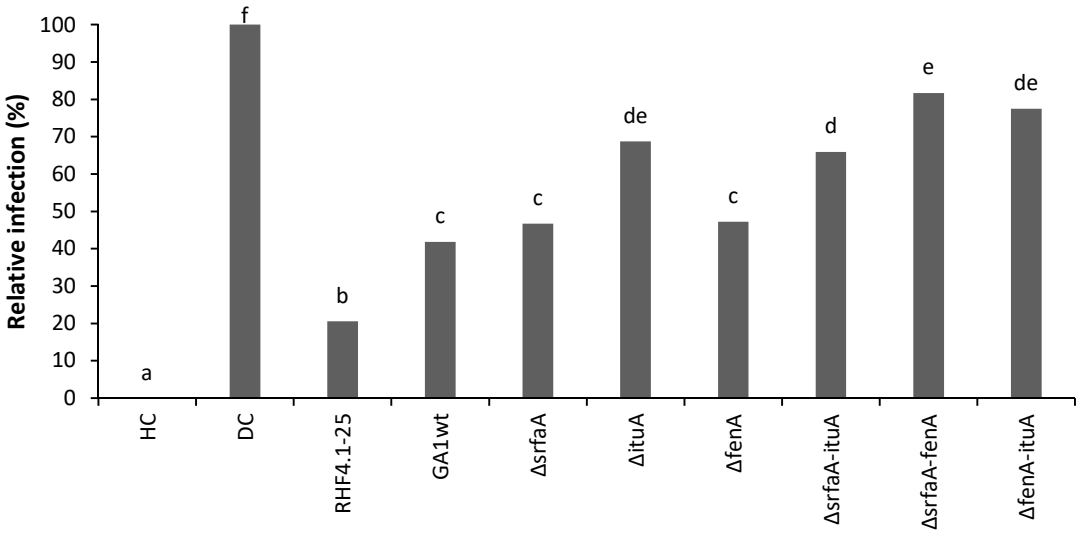

Figure S4. Direct antagonistic activity of cell-free supernatants obtained from CLiPs-producing *B. velezensis* RHF4.1-25, *B. velezensis* GA1 wild type and its mutants against *P. oryzae* VT5M1. Data from Figure 9 are here expressed as % relative infection in comparison with the diseased control (n=21). One-way ANOVA followed by Duncan's post hoc tests were used and different letters among these treatments indicate statistically significant differences ($P < 0.05$). HC: healthy control; DC: diseased control: RHF4.1-25 and GA1: wild type *B. velezensis*; Δ*srfaA*: surfactin mutant; Δ*ituA*: iturin mutant; Δ*fenA*: fengycin mutant. Δ*srfaA-ituA*: double mutant impaired in surfactin and iturin production; Δ*srfaA-ituA*: double mutant impaired in surfactin and fengycin production; Δ*fenA-ituA*: double mutant impaired in iturin and fengycin production.