Tomato plant cell death induced by inhibition of HSP90 is alleviated by *Tomato yellow leaf curl virus* infection

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Summary

To ensure a successful long-term infection cycle, begomoviruses must restrain their destructive effect on the host cells and prevent drastic plant responses, at least in the early stages of infection. In contrast to many plant viruses, the monopartite begomovirus *Tomato yellow leaf curl virus* (TYLCV) does not induce a hypersensitive response and cell death upon whitefly-mediated infection of virus-susceptible tomato plants, until diseased plants decay. The way begomoviruses evade the plant defenses and interfere with the cell death pathways is still poorly known. We show here that the chaperone HSP90 and its co-chaperone and SGT1 play a cardinal role in the establishment of TYLCV infection. Inhibition of HSP90 as well as silencing of the *Hsp90* and *Sgt1* genes leads to accumulation of damaged ubiquitinated proteins, and to a cell death phenotype. TYLCV infection relieves the cell death symptoms. TYLCV suppresses the HSP90-dependent inactivation of 26S proteasome degradation and the transcriptional activation of the heat shock transcription factors *HsfA2* and *HsfB1* and that of the downstream genes *Hsp17*, *Apx1/2*. Following TYLCV-induced suppression of plant response to viral infection, TYLCV can accumulate and express its pathogenic functions in a permissive environment.