Abstract

Fruits and vegetables are highly perishable and become very sensitive to diseases during storage. Synthetic fungicides are generally used to reduce postharvest losses due to diseases. In recent years, the use of synthetic fungicides is being questioned and several promising alternatives are being developed, including the treatment of postharvest crops with hormic doses of UV-C. The physiological basis of the UV-induced disease resistance is not well understood. This study was conducted to gain insight into the defense mechanisms involved in UV-induced disease resistance. The interaction between *B. cinerea* and tomato fruits treated with the hormic dose of 3,7 kJ/m ^{2} was used as a model system.

One of the early consequences of UV stress was an increase in the susceptibility of treated fruits to gray mold. However, resistance developed gradually and appeared to involve multicomponent, defenses. The phytoalexin, rishitin, was induced and accumulated in treated tissues. The prophylactic effect of UV-C was in part related to its ability to induce inhibitory levels of rishitin before inoculation with *B. cinerea* .

UV-C treatment affected the protein profile of tomato fruits by inhibiting the synthesis of some proteins which are probably senescence-related, by enhancing some constitutive proteins, and by inducing new ones. Of the induced proteins, one basic β-1,3-glucanase and two acidic chitinases were pathogenesis-related (PR) proteins, since they were also induced in non-irradiated fruits inoculated with *B. cinerea* .

UV-C treatment modified the topography and fine structure of the tomato fruit surface. The formation of a flaky epicuticular wax which was observed in control fruits stored for 35 days was prevented or delayed in UV-treated fruits. The topographical and biochemical modifications may have impaired the ability of *B. cinerea* to adhere to the surface and infect the fruit. At the ultrastructural level, the UV-C treatment caused the collapse of the 3 to 5 first cells layers leading to the formation of a physical barrier, the CWSZ (cell wall stacking zone). The CWSZ acted as a buttress preventing ingress of *B. cinerea* towards the inner tissue. Furthermore, all the defense mechanisms induced by UV were enhanced in response to *B. cinerea* inoculation. (**Abstract** shortened by UMI.)