Effector-triggered immunity (ETI) of *Brassica oleracea* to the infection of *Xanthomonas campestris* pv. *campestris*

M. Tortosa, P. Velasco, M.E. Cartea and V. M. Rodríguez.

Black rot, considered one of the most destructive diseases of *Brassica* plants, is caused by *Xanthomonas campestris* pv. *campestris* (Xcc). Despite of its economic repercussion, little is known about the infection development and *Brassica* defense mechanisms against this bacteria. During a well established bacterial infection, pathogen effectors are recognized by specific plant proteins that triggers the effector triggered immunity (ETI), which results in an accelerated disease resistance. With the aim of understanding the physiological processes triggered during late Xcc infection, we analyzed the metabolome of *Brassica oleracea* plants 6 and 12 days post-infection (dpi) by using an LC-ESI-Q-TOF. Fifty-seven and 223 features are accumulated differentially between control and inoculated plants 6 and 12 dpi, respectively. Surprisingly, the response at 6 dpi is characterized by a significant decrease of phenolic compounds such as glycosylated or glycosylated-acylated flavonoids, compounds that use to act as precursors of molecules involved in the plant defense systems. Besides, there is an increase of glycoprophospholipids which may suggest an activation of lipid signaling. In contrast, the 12 dpi response is characterized by a high increase of phenolic compounds including salicylic acid, a well-known key phytohormone involved in both local and systemic resistance. Likewise, high levels of Tryptophan were observed; the Tryptophan biosynthetic pathway provides precursors for plant defense-related secondary metabolic compounds and is known to be induced by pathogens. This work reports some of processes that are carried out during a late Xcc infection in *B. oleracea* plants and provides new insights into aspects of plant-pathogen interaction.