DOTTORATO DI RICERCA IN BIOLOGIA CELLULARE E DELLO SVILUPPO

Proposta di progetto di Dottorato

Titolo della ricerca: "Unravelling the role of cell wall in plant immunity"

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DESCRIZIONE DELLA RICERCA

Obiettivi della ricerca (max 4000 car.)

The cell wall (CW) is the first line of defense of plants against fungal pathogens. Several evidences indicate that components of the CW are directly involved in the plant resistance against pathogens. CW is a complex structure mainly composed of a cellulose-hemicellulose network embedded in a cohesive pectin matrix. Pectin is synthesized in a highly methylesterified form and is demethylesterified in muro by pectin methylesterases (PMEs). In addition to the transcriptional control, a mechanism of regulation of PME activity is played by specific endogenous inhibitor proteins (PMEIs) belonging to a large multigene family. Evidences indicate that plant PMEs and PMEIs participate in the remodeling of cell wall structure with consequences in plant growth and resistance to pathogens. Despite these evidences, the physiological role of these genes in plant immunity remains unclear. Botrytis cinerea, the causal agent of grey mold disease, is a broad-spectrum fungal necrotroph that causes serious pre and post-harvest rot in more than 200 species worldwide including important fruit crops. The aim of the project is to study the role of PMEs in pectin remodeling in response to B. cinerea to identify new pectin-related traits associated with quantitative resistance to the pathogen. Cross-disciplinary and innovative approaches of cell biology, glycomics and functional genomics will be exploited on a wide range of genotypes of Arabidopsis thaliana, altered in PME activity. Our research is also focused on tomato (Solanum lycopersicum) a model species for studying fruit development and ripening as well as for investigating fruit-pathogen interactions. Tomato is one of the widespread vegetable crops but the commercial varieties of S. lycopersicum are highly susceptible to B. cinerea. By contrast, the wild species S. pennellii is reported to exhibit a high level of resistance. A set of introgression lines (ILs) (S. pennellii in a S. lycopersicum background) provided by University of Naples Federico II will be used for the identification cell wall/pectin determinants involved in resistance to Botrytis. Our findings will aid the selection of cultivars improved in resistance to pathogens and fruit quality.
Phytopathogenic fungi negatively affect the agricultural production by reducing the plant yield and worsening the nutritional and qualitative characteristics of the harvest. Some of the most devastating crop diseases are caused by necrotrophic fungal pathogens. Among these Botrytis cinerea is considered the second most important fungal pathogen. Its broad host range and ability to cause disease both pre- and postharvest lead to large economic effects (both in terms of yield loss and cost of control). The plant cell wall (CW) is the foremost interface at which interactions between plants and fungi take place. Fungal pathogens use CW degrading enzymes (CWDEs) to degrade the different polysaccharides of the cell wall, gaining access to host tissues and causing extensive maceration. One clever mechanism that plants employ to evade disease is the biochemical modification of the CW components to protect themselves from digestion. Primary cell walls of dicot plants mainly consist of cellulose, hemicellulose and pectins. Pectins account for approximately 50% of dicot plant walls and homogalacturonan (HG) is the most abundant polysaccharide making about 65% of the pectin. In the CW, pectin methylesterases (PMEs), remove the methylesters from HG by releasing methanol and protons. Pectin demethylesterification affects the CW plasticity and porosity and makes HG susceptible to the degradation by polygalacturonases (PGs). PMEs participate in CW remodeling during physiological processes such as plant growth and fruit development and ripening. PMEs are also critical during plant-pathogen interactions and influence plant resistance to diseases. PME activity is induced during pathogen infection as part of the plant immune response. Specific PME isoforms are induced or repressed by necrotrophs as immune response although the role of the single PME isoforms need to be clarified. In addition to the transcriptional control, PME activity is also regulated by endogenous protein inhibitors (PMEIs) belonging to a large multigene family. PMEIs have been identified in many dicots including Arabidopsis and tomato. The control of PME activity can be also an important strategy to favor the action of PGs on demethylesterified HG backbone for the accumulation of pectin fragments (e.g. oligogalacturonides; OGs). OGs function as elicitors of plant defenses and are classical CW-derived damage-associated molecular patterns (DAMPs) that activate signaling cascades to reinforce the plant immunity. The demethylsterification of pectin by PME also represents the main mechanism to generate plant-derived methanol (MeOH), a DAMP-like alarm signal. The exposure to MeOH may result in a “priming” effect on intact tissue setting the stage for the intra and inter-plant immunity. The molecular mechanisms that control PMEs-mediated CW remodeling and production of DAMPs during defense are largely unknown and their discovery will help to develop plants with durable resistance to pathogens and improved yield and quality.

References


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