During the disease development of bacterial rot, mitochondrial dysfunction might have a role in induction of non-apoptotic cell death and the disease development, but not in apoptotic cell death in lettuce leaves. These results suggested that the development of the bacterial rot may be mediated by at least two independent cell death pathways, including apoptotic cell death and non-apoptotic cell death.

P10–12

DspA/E, a type III effector essential for pathogenicity of *Erwinia amylovora*, induces cell death both on host and non host plants and delays *PR1* expression.

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Erwinia amylovora is responsible for Fire blight, a necrotic disease of apple and pear tree. It relies on a type III secretion system (TTSS) to induce disease on hosts and HR on non-host plants. The DspA/E protein is secreted in vitro through the TTSS and is essential for E. amylovora pathogenicity. This protein was implicated in the generation of an oxidative stress during disease as well as suppression of Salicylic Acid- dependent callose deposition (Venisse, et al., 2002; DebRoy, et al., 2004). We investigated the fate of DspA/E in planta. Using a specific polyclonal antiserum we detected DspA/E secreted following infection of apple seedlings with the wild-type strain CFBP1430. However, when dspA/E was artificially delivered into apple or tobacco plant cells via agroinfection, it induces necrotic symptoms, indicating that DspA/E is also likely to be injected through the TTSS. We confirmed that DspA/E acts as a major cell death inducer during disease and HR since a dspA/E mutant is severely impaired in its ability to induce electrolyte leakage. When monitoring the response of tobacco to dspA/E transient expression, isolated cDNA fragments corresponded to genes involved in programmed cell death. Expression of the SA-responsive gene PR1 was inhibited during dspA/E transient expression on tobacco. Therefore, DspA/E plays a dual role by promoting disease necrosis and suppressing SA-dependent basal defense response.

Litterature cited:

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P10–13

Susceptibility of *Theobroma cacao* to *Crinipellis perniciosa*: a programmed cell death triggered by calcium oxalate degradation.

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Since 1989, the witches' broom disease due to the pathogenic fungus *Crinipellis perniciosa* increased in Bahia, Brazil, destroying the cultivation of cacao trees and leading to important economical, ecological and social changes in the concerned areas. The aim of the research developed in the laboratory is to acquire a good knowledge of the determinism of the interaction between the cacao tree and the pathogen *Crinipellis*.

Cacao infected by *C. perniciosa* exhibits symptoms such as hypertrophic growth, broom formation, tissue degeneration and necrosis. Tissue degradation in cacao triggered by *C. perniciosa* was characterized: DNA fragmentation, a typical symptom of cells suffering apoptosis, was observed by agarose gel electrophoresis and TUNEL analysis through witches' broom disease. Calcium oxalate crystals were observed in healthy and diseased susceptible cacao, but not in resistant variety. The involvement of calcium oxalate in apoptosis has been previously observed in some animal cells, and we showed that it was related to plants involved in PCD. We demonstrated that *C. perniciosa* was capable to survive *in vitro* in highly oxidizing environments, suggesting its resistance to reactive oxygen species. This result was coherent with the high accumulation of H_20_2 observed *in planta* using the DAB method. We demonstrated that switch between green to dry brooms, with a subsequently change of the fungus phase from parasitic to necrotrophic in a compatible cacao-*C. perniciosa* interaction, involves a PCD triggered by the pathogen, probably *via* calcium oxalate crystal degradation and H_2O_2 production.

P10–14

Sweet pathogenesis - sucrose synthase expression in the phloem of phytoplasma infected maize (*Zea mays* L.)

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The phloem, conceivably the largest and the most intricate cell continuum, hosts the smallest and the simplest cells – phytoplasmas. These uncultivable, wall-less bacteria inhabit the sieve tubes and cruise between plants by insect vectors that feed on phloem and by vegetative propagation. Phytoplasmas cause several hundred incurable plant diseases with considerable economic consequences. The reduced translocation of the phloem sap in infected plants has been recognized already in the pioneering studies on phytoplasma diseases but its root cause remains a black box. The recently sequenced genome of a phytoplasma revealed the smallest set of metabolic pathways identified in an organism to date and no known pathogenesis related genes. Phytoplasmas do, however, have several copies of glycolytic enzymes, which suggests that they may interfere with the host sugar metabolism. Indeed, in the cultivable relative of phytoplasmas, *Spiroplasma citri*, a correlation between the utilization of fructose and pathogenicity was found. In our study, the interaction of maize (*Zea mays* L.), a model plant for sugar metabolism, and maize bushy stunt phytoplasma was studied. Hexoses, the primary cell fuel, are normally kept at very low concentrations in the phloem of plants and are provided by sucrose synthase in companion cells that cleaves sucrose into UDP-glucose and fructose. In maize, the isozyme sucrose synthase 1 (SS1) is known to be induced by low cell energy status brought about either by oxygen or hexose deprivation. The competition of phytoplasma for hexoses and their impact on the host cell energy status was therefore tested by analyzing SS1 expression in the phloem using western blot and immunofluorescence. The several fold induction of SS1 found in companion cells of infected plants implies low energy status of the phloem, while sucrose metabolism is increased. Based on these results and literature data we propose a novel mechanism of pathogenesis, in which competition of phytoplasmas for fructose short circuits the host cell sugar metabolism, driving the phloem into hypoxia.

P10–15

Induction of protein secretory pathway is required for systemic acquired resistance.