Fusarium oxysporum mediated underpinning of cell type-specific modulation in multiple host interaction

Rendicontazione

Informazioni relative al progetto

FOUNDATION

ID dell'accordo di sovvenzione: 750669

Sito web del progetto

DOI
10.3030/750669

Progetto chiuso

Data della firma CE
17 Marzo 2017

Data di avvio
1 Luglio 2018

Data di completamento
30 Giugno 2020

Finanziato da
EXCELLENT SCIENCE - Marie Skłodowska-Curie Actions

Costo totale
€ 170 121,60

Contributo UE
€ 170 121,60

Coordinato da
UNIVERSIDAD DE CORDOBA
Spain

Questo progetto è apparso in...

18 Maggio 2021

4 Febbraio 2020

Periodic Reporting for period 1 - FOUNDATION (Fusarium oxysporum mediated underpinning of cell type-specific modulation in multiple host interaction)

Periodo di rendicontazione: 2018-07-01 al 2020-06-30

Sintesi del contesto e degli obiettivi generali del progetto

Fungal pathogens have a dramatic impact on human nutrition by reducing crop productivity. Many fungal-plant interactions occur in the soil and crucially affect plant health. Very little is known on how soil-borne pathogens infect roots and deal with host immunity. A particularly destructive group of root-infecting fungi cause vascular-wilts, which attack many crops, colonizing the roots and cause progressive wilting. These diseases are amongst the most difficult to control, as the pathogens are usually inaccessible to chemicals being inside roots. Therefore, they are controlled with unspecific compounds for fumigation that have a detrimental effect. To generate a sustainable solution against these fungi, a comprehensive understanding of how these pathogens establish compatibility across diverse hosts and the plant processes they target is crucially needed.

The fungal genus Fusarium contains many soil-borne pathogens causing wilting diseases in numerous crops. Currently, there is little information on the crucial biotrophic infection stage (living phase) of Fusarium. The fungus penetrates the roots and ultimately colonizes the xylem, leading to

death/no death and even endophytic lifestyles. Therefore, Fusarium is an excellent model to investigate multi-host compatibility and immune suppression. Controlling soilborne phytopathogens is very difficult, due to their high persistence and striking capacity to adapt to the host plant and to the environment. Because most soil fungicides are now banned by EU legislation, there is an urgent need to develop new control strategies, which need to be efficient, durable and environmentally friendly. Identifying the molecular events that underpin disease development across diverse hosts resolves the longstanding question on how these fungi sense the host vascular system. This is of fundamental interest in microbial interactions. The overall aim of my proposal is to understand the mechanisms underlying plant colonization by F. oxysporum to conquer a broad host range. My hypothesis is that alteration of the intercellular space (apoplast) where the pathogen mainly resides during the biotrophic stages on host/non-host will serve as a site for action of ´core´ pathogenicity determinants to colonize a broad spectrum of agronomically important crops.

Lavoro eseguito dall'inizio del progetto fino alla fine del periodo coperto dalla relazione e principali risultati finora ottenuti

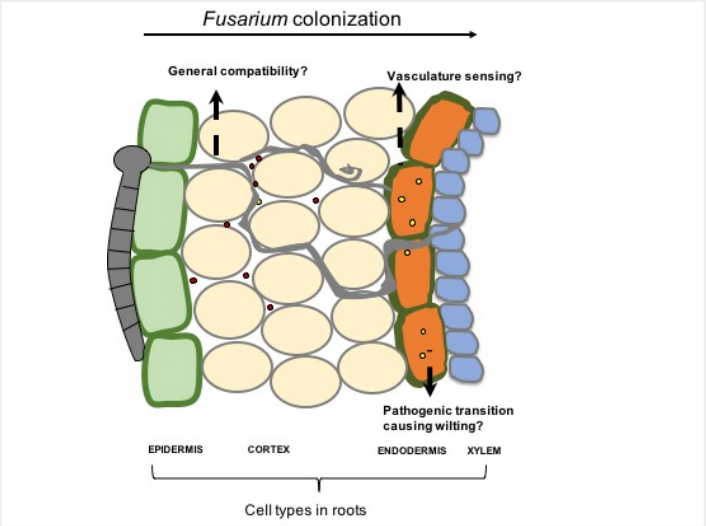
As F. oxysporum mainly grows intercellularly during early stages, we hypothesized that the pathogen may secrete pathogenicity proteins, known as effectors, in this intercellular space for determining general compatibility and to deal with host immunity. As a part of my MSCA fellowship, I mainly established the isolation of apoplastic fluid from infected tomato roots and used discovery proteomics to identify effectors.
These identified ´core apoplastic effectors´ encode for small, secreted, cysteine rich hypothetical proteins, which are conserved across the Fusarium species complex (Redkar et al., in prep). These core effectors termed (ERCs: Effector for Root Compatibility ERC1-3), are induced in planta upon colonization of host/non-host and contribute in virulence. I also performed a comprehensive RNA-Seq analysis during the early stages of colonization by F. oxysporum and found that a major proportion of the secretome primarily encodes for effectors that are conserved across the Fusarium specific complex. This suggests that Fusarium rely on a conserved effector repertoire for establishing compatibility on hosts and non-host and then host-specific determinants may primarily play a role in determining wilting which is host-specific.

We also investigated on the nature of the active intercellular root proteome during Fusarium colonization and how its activity triggers defense signaling. Infected apoplastic fluid was used for Activity Based Protein Profiling (ABPP), which indicated that Fusarium is able to suppress different classes of proteases. Based on these findings and our interests in the apoplastic immunity, I lead a research spotlight article in Trends in Plant Science (doi.org/10.1016/j.tplants.2019.06.009). The paper highlights work on effector from Ustilago maydis Pit2, that has a protease inhibitory domain, and which mimics the protease substrate. Additionally, I also co-authored a review on the current knowledge about Molecular Interactions of the smut fungi and their hosts in Annual Reviews (doi: 10.1146/annurev-phyto-082718- 100139). This has impacted ongoing research in the plant-pathogen interaction community.
Moreover, I have used Arabidopsis- Fusarium patho-system to understand the host derived signals that the fungus senses in reaching xylem. The root endodermis is a protective cell layer which

separates cortical zone and vasculature. The space between endodermal cells becomes impregnated with a lignin-based hydrophobic polymer, termed the Casparian strip (CS), and acts as a selective barrier. Cell wall-associated membrane proteins, (CASPs) fuse to form a continuous band that is guided by the well-defined Schengen (SGN) pathway. I hypothesize that vascular-wilts likely interfere with different components of the SGN pathway to sense the vasculature. We used a collection of Arabidopsis mutants across the SGN pathway, that show varying degrees of pathogenicity phenotype with F. oxysporum Fo5176. Hence, The CS pathway seems to be involved in guiding Fusarium towards the vasculature. Results of my project were presented at research seminars at CEPLAS Fellows Conference at University of Dusseldorf, Germany and IISER, Pune, India.
Moving forward, I will continue in my host lab through the Juan de la Cierva Fellowship. I am working to functionally characterise the identified CORE effectors in Fusarium. I will also explore the interesting leads of the interference of Fusarium with the Casparian Strip pathway in sensing vasculature. Upon completion, I aim to publish two research papers detailing my findings.
In addition to the proposed work, I was also the corresponding author of two commentary articles: one which describes how root pathogen have evolved to target master transcriptional regulators of the Salicylic acid pathway (doi:10.1098/rstb.2015.0459) and another which provides an overview of how pathogens have evolved to use carrier proteins for virulence functions (doi: 10.1111/nph.14137).

Progressi oltre lo stato dell'arte e potenziale impatto previsto (incluso l'impatto socioeconomico e le implicazioni sociali più ampie del progetto fino ad ora)

Findings from my MSCA project, have resulted in new knowledge about the colonization strategies of root pathogens. The identification of conserved effectors in Fusarium that likely mediate compatibility, will enable us to improve our understanding of the molecular mechanisms underlying the need of a short biotrophic phase in the pathogenic life cycle. In addition, identifying the apoplastic plant processes that pathogens like Fusarium manipulate will provide new avenues to understand the molecular cross-talk and help in developing resistance. Overall, I believe results from my project will have broad implications, as they provide first glimpses of the crucial asymptomatic infection phases of these important fungal pathogens.
Furthermore, by promoting my research via general articles, interaction with the School pupils by Skype a Scientist, I have been able to reach a general audience. This has provided me with a great platform to promote my work and general awareness of my research to the public. Moreover, my participation in conferences has allowed me to expand my professional network increasing my opportunities to become a successful Principal Investigator.



Graphical abstract of the objectives of MSCA-IF FOUNDATION

Ultimo aggiornamento: 14 Ottobre 2020

Permalink: https://cordis.europa.eu/project/id/750669/reporting/it