

behavior, the effect of different XfDSF was tested *in vitro*. In total, 611 genes differentially expressed ( $P < 0.05$ , false discovery rate) were identified. Among them, 334 known genes were downregulated, whereas 276 genes were over-expressed. Moreover, 256 genes were differentially expressed in the Temecula - *ΔrpfF* mutant- XfDSF treatment combination. A large number of differentially expressed genes with unknown functions were identified.

### TRANSCRIPTOME ANALYSIS OF TWO OLIVE CULTIVARS IN RESPONSE TO *XYLELLA FASTIDIOSA* INFECTION.

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The CoDiRO strain of *Xylella fastidiosa* subsp. *pauca* (*Xfp*) is ravaging olive (*Olea europaea*) groves in southern Italy, causing a destructive disease denoted Olive Quick Decline Syndrome (OQDS). Field observations show that the *Xfp*-infected plants of the cv. Ogliarola salentina develop more severe symptoms than that of cv. Leccino. A global transcriptome profiling comparing the two olive cultivars, infected or not by *Xfp*, was performed to ascertain whether a tolerant condition of cv. Leccino exists, which could be exploited for lessening the economic impact of the disease on the local olive industry. The study revealed that 659 and 447 genes were differentially regulated upon *Xfp* infection, in cvs Leccino and Ogliarola salentina, respectively, whereas 512 genes resulted altered between the two infected cultivars. The analysis showed that plants of both cultivars perceive the presence of *Xfp*, mainly involving cell wall-associated proteins. The predominant response of cv. Leccino, which is missing in cv. Ogliarola salentina, consists on the up-regulation of genes encoding receptor-like kinases and receptor-like proteins. This different transcriptome response determines a lower pathogen concentration in the cv. Leccino, suggesting that it may harbor genetic constituents and/or regulatory elements which counteract *Xfp* infection. These findings suggest that cv. Leccino is endowed with an intrinsic tolerance to *Xfp*, which makes it eligible for further studies aimed at investigating molecular pathways underlying its different defense response.

**POTATO SPINDLE TUBER VIROID UP-REGULATES DNA METHYLATION-RELATED GENES AND ANTAGONIZES THE INFECTIVITY AND THE ACCUMULATION OF A GEMINIVIRUS.** E.M. Torchetti<sup>1</sup>, M. Pegoraro<sup>1</sup>, B. Navarro<sup>1</sup>, M. Catoni<sup>3</sup>, E. Noris<sup>2</sup>, F. Di Serio<sup>1</sup>. <sup>1</sup>Consiglio Nazionale delle Ricerche, Istituto per la Protezione Sostenibile delle Piante, Bari, Italy. <sup>2</sup>University of Cambridge, The Sainsbury Laboratory, Cambridge, United Kingdom. <sup>3</sup>Consiglio Nazionale delle Ricerche, Istituto per la Protezione Sostenibile delle Piante, Torino, Italy. E-mail: francesco.diserio@ipspp.cnr.it

Tomato is a natural host of *Potato spindle tuber viroid* (PSTVd) and *Tomato yellow leaf curl Sardinia virus* (TYLCSV), which are representative members of pospoviroids (infectious non-coding circular RNAs) and geminiviruses (single-stranded DNA viruses), respectively. While molecular events during infection have been explored separately for each one of these two nuclear replicating pathogens, plant responses during mixed infections are unknown. In this context, dissection of DNA methylation pathway is particularly interesting because it is well known that plants may methylate viral DNA to impair geminivirus infection, while whether viroids interfere with host DNA methylation pathways is unknown. Exploiting an experimental system based on PSTVd and TYLCSV co-infecting the same tomato plant, and applying qRT-PCR, methylation-sensitive restriction and bisulfite conversion assays, we found that: i) when plants were co-infected, TYLCSV infectivity and accumulation were strongly impaired, indicating an antagonistic action of PSTVd; ii) PSTVd alone or in double infection with TYLCSV significantly upregulated the expression of key genes governing DNA methylation in plants; iii) PSTVd promoted a strong hypermethylation of TYLCSV DNA in tomato plants co-infected by both pathogens, thus supporting a mechanistic link with the antagonism of the viroid on the virus during co-infection. Besides providing the first solid evidence that a viroid may interfere with host regulatory networks involved in DNA methylation, these data open new perspectives on the possible involvement of viroid-induced epigenetic changes in plant responses against other biotic and abiotic stresses.

**DISTINCT EFFECTS OF TOMBUSVIRAL p19 RNA SILENCING SUPPRESSOR ON SMALL RNA MEDIATED PATHWAYS IN PLANTS.** L. Kontra<sup>1,2</sup>, T.