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Temperature increases associated with climate change have become a major challenge with respect to agricultural output. Despite the urgent need to improve crop thermotolerance, only a limited number of heat-tolerant varieties have been developed. Heat can affect the whole plant, but development of male gametophytes seems to be particularly sensitive. Only a few degrees above optimal growth temperature results in male sterility, which is a primary cause lower yields. To better understand the problem underlying heat sterility, we aimed to examine the genetic basis of pollen thermotolerance, using tomato as a model. Within the tomato clade (Solanum section Lycopersicon), we screened 61 accessions of 13 wild species by exposing them to continuous mild heat (CMH) and found high natural variation for pollen viability (PV), the number of pollen (PN) and reproductive organ sizes. Using the most thermotolerant wild plant and a thermosensitive tomato cultivar, an interspecific bi-parental F2 mapping population of 218 individuals was generated, phenotyped under CMH. Following genotyping of all individuals, composite interval mapping revealed Quantitative Trait Loci (QTLs) for all traits. A dominant and an additive QTL for PV with a LOD score of 4.4 and 7.3 explaining 8.9 and 13.4% of the phenotypic variance, respectively, showed the complexity of the PV trait. QTLs for organ size traits were co-localized and also identified in independent F2 mapping populations, indicating a potentially broad and conserved genetic effect. To downsize the number of candidate genes underlying the PV QTLs and study the physiological consequence of the QTLs, premature anthers of 20 F2 individuals with varying tolerance levels and QTL composition were used for RNA-seq. We hypothesize that a wide range of genes determines PV in tomato.

### COFFEE Break (3:00 – 3:20 pm), Conference Center lobby and patio

### Session X • RESISTANCE, PATHOGENS, PESTS and MICROBIOMES

Gitta Coaker and Kevin Babilonia, Chairs **3:20** — **4:50** pm, *Conference Center Ballroom* 

#### 3:20 pm

# PHYTOPHTHORA BLIGHT IN POTATO: TIPPING THE BALANCE BETWEEN RESISTANCE AND SUSCEPTIBILTY

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Plants are continuously challenged by pathogens but because of their effective multi-layered defence system plant diseases are an exception rather than a rule. The first layer of defence is governed by plasma membrane-associated receptors known as pattern recognition receptors (PPRs). The second layer is mediated by intracellular receptors, which are largely nucleotide-binding leucine-rich repeat (NLR) proteins also known as resistance (R) proteins. Our research focuses on late blight, a devastating disease on potato and tomato that is caused by the oomycete pathogen *Phytophthora infestans*, and infamous because of the Irish potato famine in the mid 19<sup>th</sup> century. To colonize host plants, pathogens secrete effectors that can modulate host defence. Well-known are the RXLR effectors that are produced by *Phytophthora* species and related oomycetes, and translocated into host cells. To counteract the pathogen, potato exploits R proteins, the intracellular NLR immune receptors that confer resistance to *P. infestans* upon recognition of a RXLR effector, with each R protein having its own

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matching RXLR effector (or AVR protein). In the absence of a matching R protein, RXLR effectors manipulate the cell machinery by targeting host proteins, the so-called effector targets, thereby paving the way for successful infection. As an example I will elaborate on the NLR R1 in potato, its matching effector AVR1 in *P. infestans*, and the AVR1 effector target Sec5, a subunit of the exocyst complex. In addition to R proteins there are also some PRRs known that confer resistance to *Phytophthora*. We identified a family of cell surface receptors classified as L-type lectin receptor kinases (LecRKs). Arabidopsis has 45 LecRK genes, of which several play a role in resistance to a variety of plant pathogens including *Phytophthora*. LecRKs are wide-spread in plants, and this justifies exploitation of LecRKs as novel sources for disease resistance. A further understanding of the mechanisms underlying R protein- and PPR-mediated resistance is crucial to design novel strategies for introducing resistance traits in crops.

#### 3:50 pm

## TOMATO RECEPTOR FLAGELLIN-SENSING 3 BINDS FLGII-28 AND ACTIVATES THE PLANT IMMUNE SYSTEM

Hind S.<sup>1</sup>, Strickler S.<sup>1</sup>, Boyle P.<sup>1</sup>, Dunham D.<sup>1</sup>, Bao Z.<sup>1</sup>, O'Doherty I.<sup>1,2</sup>, Baccile J.<sup>1,2</sup>, Hoki J.<sup>1,2</sup>, Viox E.<sup>1</sup>, Clarke C.<sup>3</sup>, Vinatzer B.<sup>3</sup>, Schroeder F.<sup>1,2</sup>, Martin G.<sup>1,4</sup>

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The recognition of conserved microbe-associated molecular patterns (MAMPs) by pattern recognition receptors (PRRs) is one of the initial events that activates pattern-triggered immunity (PTI) in both plants and animals. This immune response leads to the rapid generation of reactive oxygen species (ROS), activation of mitogen-associated protein kinases (MAPKs), and extensive changes in the transcriptome that together hinder the infection process. The first plant PRR-MAMP pair, consisting of FLS2 and its ligand the flagellin epitope flg22, works in concert with the co-receptor BAK1 to activate immune signaling.

Certain solanaceous plants, including tomato, potato and pepper, detect flgII-28, a region of bacterial flagellin that is distinct from that perceived by the well-characterized FLS2 receptor. The discovery that tomato recognizes a second flagellin MAMP, combined with extensive natural variation and recent availability of the genome sequence for this species, offered the opportunity to identify the flgII-28 receptor using a genetic approach. Here, we use natural variation in tomato heirloom varieties and a mapping-by-sequencing approach to identify a receptor-like kinase gene, named *FLAGELLIN-SENSING 3 (FLS3)*, which confers responsiveness to flgII-28. We demonstrate that FLS3 is the flgII-28 receptor and show that FLS3-mediated immunity enhances resistance to a bacterial pathogen. In addition, FLS3 signaling is BAK1-dependent and is suppressed by bacterial effectors AvrPto and AvrPtoB. The emerging awareness that pathogens have numerous ways to subvert or evade host immunity has highlighted the need for identifying new mechanisms to enhance the plant immune system. Introduction of novel immune receptors into crop plants offers the potential to improve food quality and yield.

#### 4:10 pm

## LOCAL SMRT RENSEQ ENABLES RAPID CLONING OF RPI-SMIRA1 AND R10 FROM POTATO CULTIVAR SARPO MIRA

<u>Witek K.</u><sup>1</sup>, Xing L.<sup>1,2</sup>, Jupe F.<sup>1,4</sup>, Bryan G.<sup>3</sup>, Hein I.<sup>3</sup>, Jones J.D.G.<sup>1</sup> <sup>7</sup>The Sainsbury Laboratory, Norwich Research Park, Norwich, United Kingdom, <sup>2</sup>Nanjing Agricultural