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▶ To cite this version:

Richard O'Connell, Stéphane Hacquard, Pamela Gan, Emiel Ver Loren van Themaat, Jochen Kleemann, et al.. Insights into Colletotrichum hemibiotrophy from genome and transcriptome sequencing. XI International Fungal Biology Conference, Sep 2013, Karlsruhe, Germany. hal-02808987

HAL Id: hal-02808987 https://hal.inrae.fr/hal-02808987

Submitted on 6 Jun 2020

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Insights into Colletotrichum hemibiotrophy from genome and transcriptome sequencing

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Species of *Colletotrichum* cause devastating diseases on numerous crop plants worldwide. These pathogens use a hemibiotrophic infection strategy that involves the formation of specialized cell-types for initial penetration (appressoria), growth inside living plant cells (biotrophic hyphae) and tissue destruction (necrotrophic hyphae). The genomes of 4 species were sequenced to date: C. higginsianum (Ch), C. graminicola (Cq), C. orbiculare (Co) and C. fructicola, which respectively infect Arabidopsis, maize, cucurbits and strawberry. All 4 genomes encode extraordinarily large inventories of plant cell wall-degrading enzymes, with the three dicot pathogens having twice as many pectinases as the monocot pathogen C. graminicola, reflecting adaptation to host cell wall composition. Genes encoding secondary metabolism (SM) enzymes, especially those involved in the synthesis of polyketides, terpenes and alkaloids, are also expanded compared to other sequenced fungal pathogens, suggesting Colletotrichum species are capable of producing great chemical diversity. Similar to biotrophic pathogens, all 4 species encode large, lineage-specific repertoires of putative effector proteins, including an expanded family of chitin-binding LysM proteins that may function in evasion of PAMP-triggered immunity. The transcriptional dynamics underlying hemibiotrophy were examined using RNA-Seq (*Ch*, *Cq*) and microarrays (*Co*). Although appressoria formed *in vitro* resemble those *in planta*, comparison of their transcriptomes showed >1,500 *Ch* genes were induced only upon host contact, suggesting that plant signals sensed by appressoria reprogram fungal gene expression in preparation for host invasion. SM enzymes and secreted effectors were among the most highly upregulated genes in appressoria, suggesting a role for these infection structures in host manipulation. During the biotrophic phase, transcripts encoding effectors and SM enzymes were also abundant, but there was no specific induction of nutrient uptake transporters, suggesting biotrophic hyphae function primarily as organs for delivering small molecule and protein effectors rather than nutrient acquisition. At the switch to necrotrophy, genes encoding a vast array of wall-degrading enzymes, proteases and membrane transporters were up-regulated, enabling the pathogen to mobilize nutrients from dead and dying cells to fuel its rapid growth and sporulation.