Fungal pathogenesis in plants and crops pdf

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for lasting resistance. Early stem rust epidemics initiated studies of pathogenic variability, epidemiology and genetics of host pathogens in Pgt (Loegering Review, 1984 and Roelfs, 1985). Pgt's specialization in different races has had a major impact on wheat farming and production. Numerous varieties protected by genes have become
susceptible to stem rust, often with devastating boom and bust effects (e.g. Jin, 2011; Kolmer et al., 2007; Martens and, 1989; Park, 2007; Pretorius et al., 2000), a widely used resistance gene, has resumed stem rust research (Singh et
al., 2011). Seven variants of the Ug99 line were reported, varying in virulence for Sr21, Sr24, Sr31 and Sr36 (Singh et al., 2011). As a result of its adaptive ability, fitness and virulence attributes (90% of the world's wheat are susceptible), the Ug99 racial group has been recognized as a serious threat to food security (Flood, 2010;
Mackintosh and Pretorius, 2011; Singh et al., 2010; Along with advances in detection, genetic mapping and management of genes and quantitative loci of traits (UG99) (Durable Rust Resistance in Wheat Project, ), significant progress has been made in understanding the molecular basis of pathogenicity in Pgt et al.,
2011). Continued surveillance and racial analysis studies, combined with pathogenic genomics, will enable characteristics and the use of sustainable varieties are essential for the future and effective control of rust worldwide (Lowe et al.,
2011; Mackintosh and Pretorius, 2011). Fusarium graminearum (teleomorph Gibberella zeae), which is found in the order of Hypocreales, is a very destructive pathogen of all types of grains (). Locally, F. graminearum co'exists and co'infects with other types of Fusarium. The greatest economic losses occur when floral fabrics become
infected (Figure 7). This disease basically reduces the quality of grain rather than reduces grain yield, and leads to mycotoxin contaminated grain. Worldwide, all major cereal growing regions have reported a recurrence of the Fuzarium epidemics (Leonard and Bushnell, 2003). In the post-harvest period, if the contaminated grain is stored
or transported with too high moisture content, the fungus increases after harvest and increases in mycotoxin levels (Magan et al., 2010). The floral tissues of hexaploid wheat are strongly contaminated with the Fusarium graminearum. This disease is often referred to as Fusarium Headache (FHB), Fusarium Ear Decline (FEB) or Head
Scabies. Mycotoxin-contaminated grain is often unsafe for human consumption, animal feed or malt. In Europe, the United States and other regions, strict upper limits were introduced on specific levels of mycotoxin in grain and food (EC) 1881/2006; ». Fusarium graminearum produces several trichotetsin mycotoxins, the most important of
which are deoxynivalinol (DON), acetylated derivatives of DON, nivalenol and phytoestrogen zeralene. DON binds to the protein peptiyl transferase in ribosome and inhibits the transfer of protein. Different natural isolates (term chemotypes) produce different types of mycotoxins (Alexander et al., 2011). Controlling flower fusarium
infections remains problematic. In most cereals, the sources of resistance identified are only partially effective, but spray coating and timing remain difficult. Minimizing sequential crops and plowing under any infected residues remain
the best way to reduce disease pressure at the local level. Graminearum infection of non-viral species in crop rotation, such as soybeans and sugar beets, is increasingly reported. Excellent genetic, biochemical, molecular-genetic, genomic, transcriptomic and isolate collection resources currently exist for F. graminearum ( , , ). The
genome has no repetitive sequences and contains low levels genes, but there is a high level of polymorphism Strains. When aligned with the genetic map, the two-speed gene is recognized with discrete areas of high recombination and high single-nucleotide polymorphisms (SNP) located near telomere and in the middle of
four large chromosomes. Comparisons with the sequenced genomes of F. verticillioides and F. oxysporum f. sp. lycopersici indicate that the high diversity of sites in the F. graminearum genome was the result of events synthesis of ancestral chromosomes (Cuomo et al., 2007; Ma et al., 2010). Direct gene replacement of interest, chosen
by marker using homologous recombination, is currently relatively simple. The production of mycotoxin DON contributes to the formation of diseases on wheat flower tissue (Proctor et al., 1995). In the absence of the DON, strong responses of the host defense are activated in rachis and hyphal colonization is limited to flower flowers.
(Jansen et al., 2005). DON synthesis is tightly regulated by at least three transcription factors: TRI6, TRI10 and TRI15. It is now recognized that another 160 pathogenic/virulence factors contribute to crop infection, most of which are post-infiltration (; Urban and Hammond-Cosak, 2012). A recent re-examination of the biology of the
process of flower wheat infection showed that there is a significant phase of guileless infection, in which the gif extracellularly pass between the living host cells. High expression of TRI genes is detected on the coming front and decreases after that (Brown et al., 2010, 2011). Host cells only die in intracellular gyphool intrusion, and
extensive degradation of plant cell walls is a relatively late process. These data show that F. graminearum uses a hidden approach to alleviate successful flower infectious process. These data show that F. graminearum uses a hidden approach to alleviate successful flower infectious process. These data show that F. graminearum uses a hidden approach to alleviate successful flower infections (Figure 8). Simplified three-article model of the infectious process.
(DON) inhibits the transfer of protein, which significantly suppresses the surge of reactions of plant protection (pictured in blue). Once the gif enters the plant cells, the presence of released proteins and sugars and the high density of fungal gifs lead to a strong activation of plant protection responses. Later, at the center of the lesion (10
days), the cellular contents of fungal cells living deep in the dead cortical tissue are moved into the gif just below the epidermis of the rickish and asexual spores then occurring. Fusarium oxysporum Schlecht. is an ubiquitous pathogen transmitted through the soil that causes vascular withering on a wide range of plants. Characteristic
symptoms of the disease include vascular browning, leaf epynasion, stunting, progressive withering, defoliation and plant death (Agrios, 2005). Complex F. oxysporum consists of different types of formae (f. sp.), which together total more than 100 different hosts, provoking serious losses in crops such as melon, tomatoes, cotton and
banana, among others (Michielse and Rep, 2009). Fusarium oxysporum is also a new human pathogen that can cause invasive infections in immunocompromised patients (Nucci and Anaissie, 2007; O'Donnell et al, 2004). Unlike the surprisingly wide range of housewives at the species level, individual F. oxysporum isolates cause
disease on only one or more plant species (Armstrong and Armstrong, 1981; Gordon and Martin, 1997). This dichotomy fascinated and puzzled generations of plant pathologists. Adding to the intrigue, phylogenetic studies show that various isolates of this form, infecting the same host plant, arose independently during evolution (O'Donnell
et al., 1998). Since F. oxysporum has no known sexual cycle, the mechanism by which these new pathogenic lines appeared otherwise different genetic backgrounds has long remained elusive. Recently an analysis of the complete sequence of the tomato pathogenic genome F. oxysporum f. sp. lycopersici (Fol) has identified the
presence of genomic regions of the line and LP, including four whole chromosomes that are absent in other types of Fusarium, such as grain pathogens F. graminearum and F. verticillioides (Ma et al., 2010). The transfer of two LS chromosomes from Fol to non-pathogenic isolate allowed it to cause disease in tomato plants. This suggests
that horizontal transmission of small chromosomes may be a factor in the emergence of new pathogenic lines (Ma et al., 2010). Genome sequences from additional F. oxysporum isolates will provide invaluable tools for further study of this hypothesis. Dominant plant resistance genes (R) to different races of F. oxysporum have been
identified in several cultures (Simons et al., 1998). The interaction between tomatoes and foul has been used to study the molecular basis of disease resistance and susceptibility (Houterman et al., 2008, 2009; Rep et al., 2004). These studies have led to the identification of a classical gene system with at least three genes of fungal
auriulation, some of which can function as both projectors and R-based plant immunity suppressors (considered in Takken and Rep, 2010). The unusual ability of a single foal to isolate the disease in both tomato plants and immunosuppressive mice provides a unique model for studying the trans-kingdom pathogenicity in fungi (Ortoneda
et al., 2004). Genetic analysis using targeted mutants has shown that the signaling components needed to infect tomato plants, such as mitogeneactive protein, may be indispensable for virulence in mice (Di Pietro et al., 2001; Martinez-Rocha, et al., Ortoneda et al., 2004). Others, such as pH pH
transcription factor PacC, necessary for virulence in the mouse model, but not in plants (Caracuel et al., 2003; Ortoneda et al., 2004). These results show that F. oxysporum uses fundamentally different infection strategies in plants and mammals. Further research relates to the common virulence mechanisms that are necessary for both
types of host. Recent advances in understanding the genetic basis of pathogenicity in F. oxysporum include screens inserting into the width of the mutagenez genome (Lopez-Berges et al., 2009a), leading, among other things, to the discovery of the main regulator of pathogenic development (Michielse et al., 2009b),
the identification of the preserved pathway of nitrogen reaction, regulating invasive growth functions (Lopez Berges et al., 2010) and the characteristic of a new transembraneic sensor type of mucins (Perez-Zelion and DiPitro), 2011). Future research in the F. oxysporum system will continue to provide new knowledge about the molecular
mechanisms of fungal pathogenicity (Figure 9). (A) Fusarium oxysporum microcolidia (C) sprouting on the surface of the tomato root. Penetration occurs due to the directional growth of the infectious gif (IH) to the natural discovery between epidermal root cells (the place of penetration indicated by the arrow). (B) Fusarium oxysporum
hypha grows in tomato root xylem vessel (from Di Pietro et al., 2001). Blumeria graminis is an erisifale (Takamatsu, 2004). This causes powdered mold herbs (Figure 10), including wheat and barley, which are among the leading crops worldwide (FaoStat, 2011). Grain mold infections reduce grain yields and need to be controlled to ensure
an economically viable product. Control of Blumeria is achieved through the deployment of fungicides and disease-resistant plant varieties. These controls need to be constantly reviewed, updated and developed in response to fungicide resistance, changing regulatory limitations and the evolution of mold strains that can overcome host
resistance. Barley leaves infected blumeria graminis f. sp hordei. Typical powdered pustules produced by mold colonies that grow on the outer surface of thousands of highly infectious asexual conids blown up by air currents to spread the disease. Epiphytic colonies feed on intracellular haustoria,
which develop inside epidermal cells (see Figure 11). Erisifals have a wide range of host specificity, ranging from the broad polyphagia observed in many wild tendons (Ing. 1990) to the extremely narrow B. graminis ranges, where 'formae speciales' and hordei can only infect wheat or barley, respectively (Wyand and Brown, 2003). Other
erythpoliss cause affect many other high-quality fruits and vegetables, including tomatoes, cucumbers, grapes and strawberries. Their biology, mold (Glawe, 2008). All powdered molds strictly oblige biotrophic pathogens: they are absolutely dependent on a living host plant. Epidemics
are caused by a fast series of asexual cycles, which begin with the landing of air-drop conidia on the surface of the host. After a few minutes, the coneia sprouts. Blumenia, unlike other molds, produces the first short primary germ tube, designed for surface sensing (Wright et al., 2000; Yamaoka, et al., 2006). After a few hours from the
conidium comes a secondary germ tube, a septum and differentiates an elongated, hooked uppressory, from which the peg develops a complex multi-hygitate haustoria, surrounded by a perigaustoric matrix and a specialized host membrane (Figure 11)
(Hukelhoven and Panstruga, 2011). Like other mandatory biotrophic pathogens, the guztoria is dedicated to feeding and controlling host immunity and metabolism (Panstruga and Dodds, 2009). Plant-derived nutrients are transported to a gif growing on the surface of the plant. Within 3 days after vaccination condiidides develop from
specialized cells of the foot and produce a mass of condiy: the same powder of these molds. At the end of the host growth season compatible strains mate and produce chasmothecia (Braun et al., 2002), which also act as recreational structures to survive adverse conditions. Bloomeria graminis f. sp. hordei haustorium. This haustorium
was isolated by manually dissecting the epidermis of an infected sheet of barley and assimilation from the host cell wall with a protoplasting cocktail; it was spotted with a lectin (wheat agglutinin) associated with Alexa-288. The multi-digit structure is surrounded by the perigaustoric membrane of the host origin. Like other molds, this
specialized membrane is continuous with the host plasma membrane, but has very different biochemical properties (Micali et al., 2011). In mold, the perihousetorial membrane is considered a gateway for nutrients and effectors. Bar, 10 microns. The genomes of barley and other powdered moulds are much larger than those of the related
Ascomycota (Spanu et al., 2010). This is due to the extraordinary spread of retrotransposons. The expansion of the genome is accompanied, on the one hand, by the mass distribution of genes that are
projected to encode protein effectors. Currently, two classes of effector are recognized: ECA paralogical effectors for the Avrk1 and Avra10 genes, связанных с ретротранспонами (Ridout et al., al., Sakriston et al., 2009); and more conventional small protein secrets, which are further characterized as highly pedigree specific (Spanu et
al., 2010). The importance of B. graminis, therefore, is the result of its constant and central role as a causal agent of disease of well-known cereals (Murray and Brennan, 2010), and because it is a model for the study of other mold and other mandatory biotrophic pathogens. Ascomycete Mycosphaerella graminicola (anamorph septoria
tritici) is in the order of Dothideales and causes Septoria tritici blotch (STB) wheat disease (Figure 12). This is one of the major economic constraints on wheat infection begins with a hypnonic enlargement on the surface of the leaf
and penetration through the stomat without differentiation of the apppressoria (Figure 13). A long period of non-cellless colonization (7 days) then follows, leading to the formation of the apppressoria (Figure 13). A long period of non-cellless colonization (7 days) then follows, leading to the formation of the apppressoria (Figure 13). A long period of non-cellless colonization (7 days) then follows, leading to the formation of the apppressoria (Figure 13). A long period of non-cellless colonization (7 days) then follows, leading to the formation of the apppressoria (Figure 13). A long period of non-cellless colonization (7 days) then follows, leading to the formation of the apppressoria (Figure 13). A long period of non-cellless colonization (7 days) then follows, leading to the formation of the apppressoria (Figure 13). A long period of non-cellless colonization (7 days) then follows, leading to the formation of the apppressoria (Figure 13).
necrotrophic nutrition (Figure 13). Septoria tritici blotch (STB) is a wheat disease caused by the gramicolon Mikosfaerella. Model for wheat leaf vaccination, DPI) involves the release of protective and suppressive apoplastic effectors of plants (red
and blue triangles) from slowly growing intercellular hyphais. After 7 days there is a death of the cells of the leaves and loss of permeability of the membrane, which allows the release of nutrients in the apoplast. Protective effects are off, although other potentially toxic effects (blue diamonds) can be produced. Extensive hypchal growth
and asexual spores (pycnidia and pycnidiospore formation) are now supported in leafy lesions. Mikospheerella gramicol is an established model of the body for studying the dynamics and evolution of pathogen populations. Long-term data sets (from 1843 to 2003) have shown that STBs are affected by climate change, including those
associated with the industrial revolution (Bearchell et al., 2005). It has also been reported that up to 90% of global genetic variations of this fungus may be present within a single contaminated wheat field (zhan et al., 2003). This level of genetic diversity arises from the heterothalyal reproductive system and the formation of ascosporas.
which initiate epidemics every year (Chen and McDonald, 1996; Linde et al., 2002; Waalwijk et al., 2002). Most of the economic expenses incurred by My. graminicola arises indirectly from its rapid evolution in response to electoral pressures, including to wheat diseases and the sustainable use of fungicides. Examples of this the speed at
which my. Graminicol populations evolved and spread a genetic mutation that was resistant to the strobilurine fungicide class (Fraaije et al., 2005), as well as the rapid evolution of the target cyp51 protein in response to the widespread use of specific fungical azoles (Cools et al., 2011). Mikosfeerella graminicola was also the subject of
intensive genetics and genomics analyses. Genome sequences of isolate models, together with relatives collected from wild herbs, were published recently (Goodwin et al., 2011; Stukenbrock et al., 2011). These studies have identified the existence of 21 chromosomes, eight of which are all indispensable for plant infection and vary in
number and structure between isolates (Goodwin et al., 2011; Stukenbrock et al., 2011; Wittenberg et al., 2009). Functional genomic analysis of plant infection has identified a number of genes important for signaling the
transition to hygal growth and stomatal penetration (Orton et al., 2011). Subsequent post-penetration of stealth pathogenesis is supported by a reduced set of genes for cellular sten grading enzymes (Goodwin et al., 2011) and expression of high-level secret proteins, which inhibit the activation of plant defenses (Marshall et al., 2011). This
initial evasion of plant protection reflects a mechanism more typical of mandatory biotrophs. However, what follows is the acute activation (Keon et al., 2007; Rudd et al., 2008). This is an intriguing mechanism for future analysis (Figure
13), involving an initial ploy, followed by the subsequent hijacking of plant protection alarms (Deller et al., 2011), which can be separated by other members of the most common and important genera of plant-based pathogenic
fungi. Virtually every crop grown around the world is subject to one or more types of colletotrichum. These fungi cause anthraconous spots and malformations of parts of air plants and rot after harvest. Members of this genus cause great losses to economically important crops, especially fruits, vegetables and ornamental crops. The
damage caused by Colletotrichum spp., extends to important staple food crops, including bananas, cassava and sorghum, grown by natural farmers in developing countries in the tropics and subtropics. It is particularly successful as a post-harvest pathogen because the hidden infections that started before harvest do not become active
until the fruit is preserved or Shelf. Up to 100% of stored fruit can be lost as a result of Colletotrichum disease (Prusky, 1996). Colletotrichum is an asexual genus classified in imperfect mushrooms. It belongs to the Coelomycetes, producing its conidia in acervuli. Despite significant changes, Colletotrichum's taxonom remains in a state of
constant change. There are many uncertainties regarding the systematic fungal pathogens of this genus, and, depending on the criteria, the number of species can range from 29 to more than 700 (von Arx, 1957; Sutton, 1992). One of the most confusing species is C. gloeosporioides. For example, 594 types of colletorium have been
reclassified by von Arks as synonyms of C. gloeosporioides (table 2). Table 2. The main view of Colletotrichum. Colletotrichum species host Lifestyle C. gloeosporioides and papaya/citrus/many other hosts of hemibiotrophy C. acutatum Strawberry/other Necrotrophy C. coccodes tomato gemibiotrophy C. graminicola Corn hemibiotrophy C.
boninense Wide range host Hemibiotrophy C. trifolii Alfalfa Hemibiotrophy C. capsici Pepper/other hosts Necrotrophy C. truncatum Legumes/tobacco Hemibiotrophy C. orbiculare melon / cucumber Gemibiotrophy C. sublineolum Sortom Gemibiotrophy C. truncatum
Legumes Hemibiotrophy C. musae Banana Hemiciotrophy C. lindemuthianum Common As a result of its importance as a pathogen, its unique intracellular hemibiotrophy C. lindemuthianum Common As a result of its importance as a pathogen, its unique intracellular hemibiotrophy C. lindemuthianum Common As a result of its importance as a pathogen, its unique intracellular hemibiotrophy C. lindemuthianum Common As a result of its importance as a pathogen, its unique intracellular hemibiotrophy C. lindemuthianum Common As a result of its importance as a pathogen, its unique intracellular hemibiotrophy C. lindemuthianum Common As a result of its importance as a pathogen, its unique intracellular hemibiotrophy C. lindemuthianum Common As a result of its importance as a pathogen, its unique intracellular hemibiotrophy C. lindemuthianum Common As a result of its importance as a pathogen, its unique intracellular hemibiotrophy C. lindemuthianum Common As a result of its importance as a pathogen, its unique intracellular hemibiotrophy C. lindemuthianum Common As a result of its importance as a pathogen, its unique intracellular hemibiotrophy C. lindemuthianum Common As a result of its importance as a pathogen, its unique intracellular hemibiotrophy C. lindemuthianum Common As a result of its importance as a pathogen, its unique intracellular hemibiotrophy C. lindemuthianum Common As a result of its importance as a pathogen and its importance as a pathogen and its importance as a pathogen as a 
and outstanding history as an exemplary pathogen for the fundamental biochemical, physiological and genetic research. For example, the phenomenon of pathogenic variations (race/variety specificity) was first recognized in C. lindemuthianum (Barrus, 1911). The colletotrychum on beans was a model for many early studies on
phytoalexins (review by Kuc, 1972). In the 1970s and 1990s, much of the work that established and characterized the phenomenon of systemic acquired/induced resistance (SAR) was done using the Colletotrichum-cucumber pathosystem (Durrant and Dong, 2004). Key genes of cyclic adenosine phate (CAMP), MAPK and RAS/small G-
protein and calcium were cloned. The function of these genes, especially during conydia germination and the development of apppressoria, was characterized in several types of colletorium (Chen and Diekman review, 2004; Chen et al., 2006; Dickman and Yarden, 1999; Dickman et al., 1995; Takano et al., 2000). Most types of
colletotrichums establish infection through a brief biotrophic phase associated with a large intracellular primary gif, although some species are described as subcutaneous, such as C. capsici. The fungus later switches to a destructive, necrotrophic phase associated with narrower secondary hyfams that ram throughout the host tissue
(Figure 14). Molecular details that regulate the hemibiotrophic lifestyle (also known to occur in other fungal species, such as Magnaporthe) have long been of interest to phytopathologists. In particular, factors regulating the transition from biotrophy to necrotrophy are waiting to be identified. The recently completed sequence of C.
graminicola (Vaillancourt et al., Department of Plant Pathology, University of Kentucky, Lexington, Kentucky), along with several other types of colletorichum in the pipeline, promises to increase our understanding of this important fungal phytopathogen. Transmission of an electronic micrograph showing hemiotrophic growth of
colletothrichum destructivum during caupea infection. Notice the thick biotrophic vesicles (IV) infections after the penetration of apressoria. The host cell is still alive, and its plasma membrane can be seen around the gif. Subsequently, thinner necrophemic penetrating gif (PH) degrades tissue while growing inside the cell. A, apressory; C,
conidium. Photo courtesy of Dr. Richard O'Connell (Department of Plant Microbe Interaction, Max Planck Plant Research Institute, Cologne. Corn smut is not an economically important or destructive disease. Thus, Ustilago Maydis did not get into the top 10 for this reason. Although its symptoms on corn can be quite dramatic (Figure 15,
center), in most cases infections remain local, do not spread and therefore are not associated with serious losses in maize yield. On the contrary, farmers in Mexico infect corn artificially to collect infected cobs for cooking Huitlacoche, a traditional dish popular in pre-Hispanic times. The stages of development of Ustilago maydis, tools and
symptoms of the disease. (A) Haploid cells show promising growth. (B) Compatible haploid strains U. maydis, express cytoplasmic red fluorescent protein (GFP) (green) under the composite mat protector and produce filament dicarion (yellow). (C) The solo-pathogenic strain, which
expresses the cytoplasmic RFP from the composite promoter and the appressorial marker gene fused into triple GFP, effectively forms an apressory on the hydroxy fatty acids. (D) Macroscopic symptoms of U. Maydis disease on a corn leaf 12 days after infection (left) and fungal gifs in tumor
tissues 10 days after infection (right) are visualized with confocal fungal gifs painted WGA-AF488 (green); the walls of plant cells are colored with iodide propidium (red). (E) I'm not an example, an example, an example, an example, an example, an example in beginner
cells. (G) Highly effective homologous recombination is used to replace genes using hygromycin resistance (CbxR) or phleomycin resistance (PhleoR) as the dominant markers chosen. (H) The Phylogenetic tree of W. Maidis and his next of kin. The figures were kindly
provided by Patrick Berndt and Rolf Russer (U. Maydis Symptoms of disease in the infected corn cob, center) as at the Max Planck Institute for Ground Microbiology. Please note that their permission to use these numbers has been obtained. What are the attractions of this fungus and why it became a model for biotrophic, plant-
pathogenic basidiomycetes? Ustilago maydis can be grown in culture in certain media, haploid fungus and growing budding (Figure 15A) and forms compact colonies on plates that can be replica coated. These were actually some of the reasons for choosing this fungus for seeded early studies on homologous recombination (Holliday,
2004). For host colonization and the development of symptoms, corn seedlings can be infected. Symptoms can development, and mating
and formation of a dikaryotic filament is absolutely necessary. These steps can be replicated in the laboratory (Figure 15B) depending on the formation of apressor on the corresponding hydrophobic surfaces (Figure 15C). The proteins that control these events are known: pheromones and pheromone receptors, as well as the
hetonodimerical transcription factor formed after cell merging, which causes a regulatory cascade (Brefort et al., 2009; Heimel et al., 2010, b). This knowledge has allowed the development of haploid solopathogenic strains that cause diseases without the need for partner mating (B'lker et al., 1995; Kuemper, et al., 2006). Such strains
allow forward genetic screens and significantly reduce the work in reverse approaches of genetics. Homologated recombination is surprisingly effective (Kuemper, 2004). Tools include adjustable promoters to study basic genes and various coloring
techniques, to visualize fungus during biotrophic growth (Figure 15D), and fluorescent proteins for live cell imaging of gene expression and visualization of subcellular structures (Figure 15D), and fluorescent proteins for live cell imaging of gene expression and visualize fungus during biotrophic growth (Figure 15D), and fluorescent proteins for live cell imaging of gene expression and visualization of subcellular structures (Figure 15D), and fluorescent proteins for live cell imaging of gene expression and visualize fungus during biotrophic growth (Figure 15D), and fluorescent proteins for live cell imaging of gene expression and visualize fungus during biotrophic growth (Figure 15D), and fluorescent proteins for live cell imaging of gene expression and visualize fungus during biotrophic growth (Figure 15D), and fluorescent proteins for live cell imaging of gene expression and visualize fungus during biotrophic growth (Figure 15D), and fluorescent proteins for live cell imaging of gene expression and visualize fungus during biotrophic growth (Figure 15D), and fluorescent proteins for live cell imaging of gene expression and visualize fungus during biotrophic growth (Figure 15D), and fluorescent proteins for live cell imaging of gene expression and visualize fungus during biotrophic growth (Figure 15D), and fluorescent proteins for live cell imaging of gene expression and visualize fungus during biotrophic growth (Figure 15D), and fluorescent fungus during fungus during biotrophic growth (Figure 15D), and fluorescent fungus during fun
annotation and curatorial is available (K'mmper et al., 2006; 2006; The genome is skinny and contains little repetitive DNA. Transcription profiles (Figure 15E) were created for the most important stages of fungal and plant reactions (available through GeneExpressionOmnibus: . This spurred back-to-back approaches to genetics and
allowed us to identify cluster genes encoding the secret effects that play a crucial role during host colonization, and determine which tissues might be infected (Doehlemann et al., 2009, 2011; Koemper, et al., 2010). It also became apparent from genome analysis that U. maydis is more closely related to humans than
novice yeast, and numerous proteins are shared only by U. maydis and Homo sapiens. These include proteins involved in the basic principles of long-distance transport, mitosis, motor organization of microtubules and homologous recombination. Thus, Ustilago Maidis is the ideal place to study such processes (Banuett et al., 2008;
Holloman, 2011; Steinberg and Perez Martin, 2008). Where is this system going? This will create a hierarchy of effector actions during plant colonization (Eichhorn et al., 2006; Wahl et al., 2010) and serve as a plan of comparative approaches (Schirawski et al., 2010), which are
likely to reveal an understanding of the evolution of the genome and the specialization of the host. Unlike most other pathogen affecting the linen and flax industry, its main influence on food and fiber production was (and
comes) from its role as an exemplary system, providing insight into the molecular basis of plant immunity. Благодаря новаторским генетических рас патогена и устойчивости и восприимчивости к ржавчине в лене, H.H. Flor заметил, что существует
определенная связь между каждым геном, определяющим патогенность в ржавчине (Avr) генов и соответствующими генами (R) в принимающей определяющим патогенность в ржавчине (Avr) генов и соответствующими генами (R) в принимающей определяющим патогенность в ржавчине (Avr) генов и соответствующими генами (R) в принимающей определяющим патогенность в ржавчине (Avr) генов и соответствующими генами (R) в принимающей определяющим патогенность в ржавчине (Avr) генов и соответствующими генами (R) в принимающей определяющей резистентности (Flor, 1956; Лоуренс, 1988; 
basis for harmonizing the breeding process of other crop species for resistance to pathogens. The observations also provided insights into the breeders' experience that individual resistance genes deployed in large areas are often broken by mutations of one relevant auriulation gene or genetic re-range during sexual reproduction of
previously hidden recessive sexually active alleles for the production of homogo offspring In practical terms, this classical genetic knowledge also underpins the approach to stronger resistance by pyramiding several resistance genes in a single genotype of crop species. The gene gene hypothesis has also given rise to the assumption
that resistance genes encode receptor proteins that detect the presence of specific auriulation proteins in the pathogen, and the resulting recognition event, which generates host resistance, the basis for the development of modern knowledge of the basis of plant immunity. The cycle of life out of flax rust, Melampsora lini. Genetic analysis
of flax rust depends on the ability to replicate all stages of the life cycle under controlled conditions for the production of independent and oververted offspring from flax rust isolates, and is complicated by rust, which is a mandatory biotrophic requiring all manipulations to be undertaken on a live plant-host. The flax rust system was among
the first three systems to provide cloned resistance genes and the identification of a new class of immune receptors, cytoplasmic nucleotide binding leukine-rich proteins (NB-LRR), whose specific members ensured plant resistance to fungi, oomycetes, bacteria, viruses, nematodes, sucking insects and parasitic plants(Ellis). The same
protein group was subsequently identified in animals as part of the congenital immune system. The study of alleles from L and chimeric genes between alleles expressed in transgenic flax gave the first indication that the specificity of the interactions of genes and genes was determined by the sequence of change in the LRR domain of
receptor proteins, and that the likely origin of new specific features of resistance in nature is the result of point mutations, re-assortment of flax resistance proteins implied that Avr (effector) proteins were detected by R proteins in host cells.
This was subsequently confirmed by the cloning of several Avr genes from flax rust, which encode various proteins, is released from rust and were taken into the plant's cell through the mediation of absorption signals located near the N'terminus protein Avr (Rafiqi et al., 2010). It has been shown that the molecular basis for gene
recognition relies on the specific and direct interaction of flax R proteins and the corresponding Avr proteins that interaction of plant resistance when jointly expressed in plants (Wang et al., 2007). The study of the flax rust fungus and its interaction with
the host is now set for further progress after Agrobacterium conversion systems for this mandatory biotrophy (Lawrence et al., (Figure 16). The authors would like to thank Dr. Diana Hurd for disseminating information on the vote and comparing the results of the vote. JALVK recognizes Marcela Estegio (University of Chile, Santiago,
Chile), Erzjubet Sundor (University of Debrecen, Hungary), Jan Sukhoi (CSIRO, Urrbrae, Australia), Dirk van Eden (Terason Ltd, Wellington, South Africa), Peter Schreier (Cologne, Germany), Ernst Waltering (University of Wageningen) KECC and JJR received grants from the Research Council Agrios, G.N. (2005) Plant Pathology. St.
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