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RESEARCH PAPER

Bacterial blight of cotton

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Summary. Bacterial blight of cotton (*Gossypium* ssp.), caused by *Xanthomonas citri* pathovar *malvacearum*, is a severe disease occurring in all cotton-growing areas. The interactions between host plants and the bacteria are based on the gene-for-gene concept, representing a complex resistance gene/*avr* gene system. In light of the recent data, this review focuses on the understanding of these interactions with emphasis on (1) the genetic basis for plant resistance and bacterial virulence, (2) physiological mechanisms involved in the hypersensitive response to the pathogen, including hormonal signaling, the oxylipin pathway, synthesis of antimicrobial molecules and alteration of host cell structures, and (3) control of the disease.

Key words: *R* and *avr* genes, hypersensitive response, defense reactions, control.

Introduction

Cotton, which belongs to the genus *Gossypium* spp. L., is cultivated in hot and arid countries mainly for cellulose fibres, but also for livestock food, edible seed oil, paper, nets, and explosives manufacture. The oldest evidence for cultivated cotton dates from about 5000 BC, in Mexico and Pakistan (Brubacker *et al*., 1999). The most important cotton-growing areas are Central America, southern United States (US), West Africa, and Central and Eastern Asia, the US being the most important exporter along with India, Uzbekistan, Brazil and Australia. In 2011, the Food and Agriculture Organization of the United Nations indicated that China was the greatest producer of cotton producer, followed by India, the US and Pakistan (http://faostat.fao.org/ site/339/default.aspx). The use of new, environmentally adapted varieties allowed Australia and Syria to be among the best cotton producers in terms of yield per hectare (Source: FAOSTAT Database). This plant had a key strategic economical position during the colonial period in India, Egypt and West Africa, and during the American civil war. Today cotton remains one of the most important speculative annual crops, not only generating substantial financial resources, but also rough tensions and competition between least-developed countries and industrial ones. Cotton production provides income for approximately 100 million families, and approximately 150 countries are involved in cotton import and export. For example, in Mali, one of the poorest countries in Africa with low rainfall, life of almost 20% of the population was related to cotton culture in 2007. In Syria, economical sources indicated that more than 20% of the population partially or totally depend on cotton culture (Ministry of Agriculture and Agrarian Reform - MAAR, 2010).

Cotton plants are affected by many diseases and pests, causing this crop to rely heavily on chemical use (Hillocks, 2010). Insects are probably the most critical pests for cotton (such as *Pectinophora gossypiella, Scirtothrips dorsalis, Oxycarenus hyalinipennis, Lygus lineolaris* and *Spodoptera frugiperda*), along with phytonematodes to a less extent (*Meloidogyne incog-*

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nita, Rotylenchulus reniformis, and *Belonolaimus longicaudatus*) (Machado *et al.,* 2012), and microbe-induced infections also lead to significant losses. Cotton is attacked by fungal vascular pathogens such as *Fusarium oxysporum* f. sp. *vasinfectum* and *Verticillium dahliae.* Anthracnose (caused by *Colletotrichum gossypii*), rust (*Puccinia schedonardii,* and *P. cacabata*), stem canker (*Phoma exigua*), rots (*Sclerotium rolfsii,* and *Thievalopsis basicola*), mildew (*Leveillula taurica*) and leaf spots (*Alternaria macrospora, A. alternate, Cercospora gossypina,* and *Rhizoctonia solani*) are also observed worldwide. Virus diseases can be of economic importance throughout tropical Africa. They are generally associated with invasions of insect vectors which inject virus particles into cotton leaves. Begomo-, gemini- and mosaic viruses are commonly encountered in the field. (For an extensive review on cotton diseases see American Phytopathological Society, 2001).

The first detailed description of cotton bacterial blight (CBB) caused by *Xanthomonas citri* subsp. *malvacearum* (*ex* Smith, 1901) (*Xcm*) was made in the US (Atkinson, 1891). Today this disease occurs in all cotton-growing areas throughout the world (Hillocks, 1992; Zomorodian and Rudolph, 1993) and was shown to be a serious limiting factor of fibre production in the US (Senchina *et al*., 2003), India (Verma, 1986) and in Africa (Follin *et al*., 1988). Resistance genes (*R*) are deployed in fields to control CBB, but they can be overcome by the pathogen following mutation in the genome. Much knowledge has been gained during the last 35 years about how cotton modulates its defense strategy to *Xcm*. The present paper is an update of a previous review on CBB (Delannoy *et al*., 2005), with particular focus on (1) the genetic determination of the interaction between the pathogen and host plants, (2) mechanisms underlying cotton resistance to CBB, and (3) control methods for the disease.

The disease: occurrence, symptoms and infection cycle

CBB can be one of the most destructive infections that cotton encounters in fields. The magnitude of disease damage caused by *Xcm* varies according to climatic and environmental conditions and the host genotype. Fields have been found to show as much as 80% yield losses in certain areas. If anecdotal reports of CBB made in western Mediterranean countries (mainly affected by fungal wilts) are indicative of the whole Middle East, this disease occurrs irregularly and could induce severe losses. To prevent CBB extension in Egypt, which produced about a fifth of the African production in 2007, ten Egyptian cotton varieties from *G. barbadense* were tested for resistance to *Xcm* (Abdelrahman, personal communication). Turkey has become the most important cotton producer in the Middle East (about a fifth of the production in that region), including cotton produced in organic conditions. Old reports mentioned that yield reductions due to CBB may have reached 80% in the most critical years. Zachowski *et al.* (1990) indicated that the most important Turkish cotton cultivars were very susceptible to Turkish isolates of *Xcm*. Very little information is available, however, about susceptibility levels of Turkish cotton genotypes to *Xcm.* Reported in Iran (Amani, 1972), CBB has been observed in cotton fields in several provinces with outbreaks occurring in 2004 and 2005 (Arabsalmani *et al*., 2002). Yield losses due to *Xcm* were estimated to be 10 to 30% for Iranian cultivars (Razaghi *et al*., 2012). In Syria, the rate and severity of infestation by CBB under spring irrigation were significantly greater than when flood irrigation was used. During 2010, CBB caused more than 30% damage in some localities. Recent field surveys revealed the occurrence of a genetic diversity among Syrian *Xcm* isolates, which is currently under molecular investigation (Jalloul, personal communication). Although Syrian cultivars are of high quality and yield under the different agroclimatic conditions of the country, they are always very susceptible to CBB.

The impact of CBB on cotton yield depends on environmental conditions. High rainfall and humidity as well as warm temperatures favour disease development. The development of CBB requires an initial source of inoculum, relative humidity of 85%, high atmospheric temperature of 30–40°C, optimum soil temperature of 28°C, early sowing, delayed thinning, poor tillage, and late irrigation. *Xcm* is not able to survive in soil for long periods, although we know that the bacterium can survive on crop residues left fields from non-hosts. Rain followed by bright sunshine is highly favourable. *Xcm* is not able to spread long distances on wind currents; so far, dispersion of the bacterium occurred from wind-blown rain, heavy winds, or hail. If cotton has either never been planted in a particular field or the field has not grown cotton for several years, then the initial source of the bacterium is believed to have been from infested seed. In

the absence of host varieties with durable resistance, and treatments to eradicate the pathogen completely, it would be of interest to characterize environmental factors that may be useful to forecast the disease.

Several different symptoms of CBB (Figure 1), including angular leaf and cotyledon spots, water-soaked lesions, stem black arm lesions, leaf venial blight, boll rot and gummosis, and plantlet burning, have been identified in fields (Innes, 1983). Spots on infected leaves may spread along the major leaf veins to progress toward leaf petioles and stems, resulting in defoliation. Bolls may become infected, causing boll rot and resulting in rotten seed. As infection proceeds, boll lesions will be sunken and dark brown or black. Microscopic examination reveals that adhesion of *Xcm* to the cotyledons or leaf surfaces is a prerequisite for plant infection (Thiers and Blank, 1951). The pathogen then penetrates leaves through natural stomata or wounds caused by humans and/or insects. Systemic invasion of cotton plants occurs after leaf invasion followed by colonization of intercellular areas. A single infected plant in a cotton field has the ability to create widespread disease within that particular field under favorable environmental conditions.

The pathogen

The genus *Xanthomonas* is among the the top ten most important plant pathogenic bacteria, considering their hosts and their agronomic importance (Mansfield *et al.,* 2012). Initially, the responsible bacterium for CBB was called *Pseudomonas malvacearum*, then *Bacterium malvacearum*, *X. campestris* pv. *malvacearum,* and *X. axonopodis* pv. *malvacearum*. The pathogen is currently named *X. citri* pv. *malvacearum*, based on DNA analysis of the 16S-23S ribosomal intergeneric spacer sequences (Schaad *et al.,* 2006, 2007). The bacterium is a short motile rod, formed singly or in pairs and equipped with a single polar flagellum. It is gram negative, aerobic, non-acid producing, and non-spore forming, and the cells measure 1–1.2 × 0.7–0.9 μm in culture. *Xcm* produces yellow, convex, slimy colonies on nutrient agar.

Structure and function of *Xanthomonas citri* pv. *malvacearum vr* genes

Avirulence/pathogenicity of *Xanthomonas* pathovars is related to the occurrence of *avr* genes in their genomes (De Feyter and Gabriel, 1991; De Feyter *et al*., 1993; Gabriel, 1999), indicating that the interaction with their hosts is based upon the gene-for-gene concept (De Feyter *et al*., 1993, 1998). In an incompatible situation, the hypersensitive response (HR) phenotype is characterized by localized necrotic lesions resulting from host cell death (Goodman and Novacky, 1994). Due to variation in the genetic background of *Xcm* races in their hosts (Gabriel, 1999), however, strains are capable of overcoming the *R* genes that occur in elite cotton varieties (Schnathorst *et al*., 1960; Innes, 1983).

The idea that plasmids may have a consistent role in *Xcm* pathogenicity was not supported by recent work on African and American strains of the pathogen. This indicated that differences in virulence of *Xcm* races were not located in plasmids (Abdelrahim *et al*., 2013), although it was suggested that plasmids were closely associated with aggressiveness (Narra *et al.,* 2011). Along with molecular tools, differential screening of cotton genotypes with *Xcm* isolates also permitted identification of pathogen races in Middle-East regions including Turkey (Zachowski *et al*., 1990), Syria (Abdo-Hassan *et al.,* 2008), Iran (Saeedi Madani *et al.,* 2010; Razaghi *et al.,* 2012), and in the African countries Uganda (Akello and Hillocks, 2002), Nigeria (Ajene *et al.,* 2014), and Burkina Faso (Ouedraogo *et al.,* 2009). Evaluation of *Xcm* genetic diversity assessed using rep-PCR suggested that the virulence of *Xcm* was closely related to genotype and⁄or geoclimatic origin of the strains (Zhai *et al.,* 2010). Twenty-two different races of *Xcm* have been classified on the basis of their capacity to induce resistant or susceptible responses on an established panel of differential cotton hosts, with a number of highly virulent strains identified in Central Africa in the 1980s belonging to *Xcm* races 20, 21 and 22 (Follin, 1983; Follin *et al*., 1988; Akello and Hillocks, 2002; Delannoy *et al.,* 2005). *Xcm* race 18 from Africa and Asia, and probably also *Xcm* race 1, one of the most aggressive strains in the US and Australia, contained more than 100 effectors able to activate several signaling pathways (Zhai *et al.,* 2013). *Xcm* race 22 is known to overcome all *R* genes that have been used in the field.

Structural and functional studies of several *avr* genes cloned from different races of *Xcm* (De Feyter *et al*., 1993; Chakrabarty *et al*., 1997) revealed that they belong to the *avrBs3/pthA* gene family, found widely and exclusively in *Xanthomonas* (Leach and White, 1996). Multiple functional and non-functional

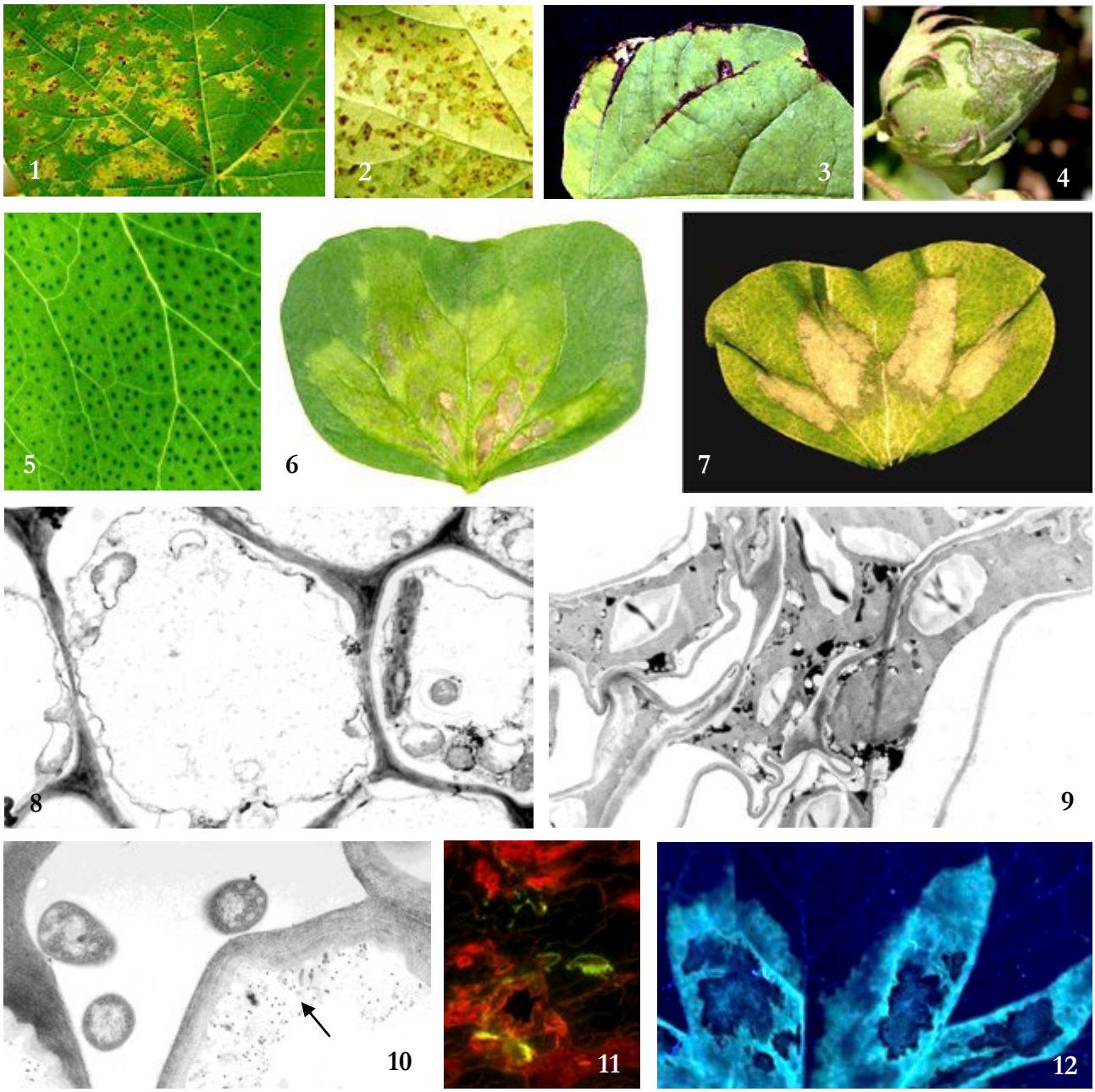

Figure 1. Symptoms of cotton bacterial blight on leaves showing angular leaf spots (1), water-soaked lesions (2), stem black arm lesions (3), fruit necrosis and gummosis (4). Susceptible cotyledons artificially infected displayed chlorosis and necrosis (6) as compared to healthy tissues in non-infected cotyledons (5). *Xanthomonas citri* pv. *malvacearum*-infiltrated areas of resistant cotyledon showed dry lesions typical of the HR cell death symptoms (7). Electron microscopic images of infected tissues in susceptible (8) and resistant leaves (9) 16 hours post-inoculation showing cell degradation and coagulation of the cytoplasm, respectively, bacterial cells in intercellular localization (10) close to a papilla immunolabelled for callose detection (arrow). Light microscope images showing yellow-green fluorescence of callose deposits stained with aniline blue in infected resistant leaves (11) and HR areas with blue bright fluorescence under UV illumination, indicating the presence of phenolic-like compounds (12). Photo credits. 1, 2 and 7, A. Jalloul; 3, J.F. Daniel; 4, 8, 9, 10, 11 and 12, M. Nicole; 5 and 6, M. Sayegh.

avrBs3 homologues in the *Xanthomonas* genome presumably arose by gene duplication and subsequent divergence (Boch and Bonas, 2010). In *Xcm*, the number of members of the *avrBs3* gene family varies from five to ten in the moderately virulent strain *Xcm*H (De Feyter and Gabriel, 1991; De Feyter *et al.*, 1998) and the highly virulent strain *Xcm*N (Chakrabarty *et al*., 1997). About twenty *avrBs3* family genes were cloned from races of *Xcm* (Gabriel, 1999), some of them being able to induce an HR when their products are delivered into resistant cotton plants *via* the type III secretion-system (TTSS). In *X. citri*, members of the *avrBs3* gene family provide some selective value for pathogenicity (Gabriel, 1999) associated with release of water-soaking, plant cell hyperplasia, and canker formation in citrus (Yang *et al*., 1994, 1996).

Xanthomonas genomics

The complete genomes of 18 species of *Xanthomonas* have been sequenced, encompassing 30 pathovars (Moreira *et al*., 2010; http://www.xanthomonas.org/genomes.html). Recently, the genomes of the two strains, *Xcm* race 18 and *Xcm* race 20 from Burkina Faso (Cunnac *et al.*, 2013), of a highly virulent *Xcm* strain from Sudan, and of a strain of race 18 from Nicaragua (Zhai *et al.*, 2013) were sequenced. They rose to a high quality draft with the objective of developing new molecular typing tools for epidemiological surveillance and guiding breeding programmes based on rapid and accurate identification of predominant lineages. Analysis of the *Xcm* race 18 genome revealed 98% homology with *X. c.* pv. *citri*. It also confirmed the presence of a TTSS and several type III effectors, including transcriptional activator-like effectors involved in symptom formation and avirulence reactions on cotton leaves.

Resistant cotton genotypes

The genus *Gossypium* is composed of some 45 diploid and five polyploid species, distributed throughout the world's arid and semi-arid tropics (Fryxell, 1992; Wendel and Cronn, 2003). The diploid cottons (n = 13) fall into seven cytogenetically distinct genome types designated A to G, and include two old world A-genome species, *G. arboreum* and *G. herbaceum*, which have probably been cultivated for fibre for more than 5,000 years (Brubaker *et al*., 1999). The new world allotetraploid cottons ($n = 2x = 26$) include the agronomically predominant species *G. hirsutum* and *G. barbadense*. The tetraploid cottons are believed to have descended from a cross that occurred naturally between an old world A-genome diploid and a new world D-genome diploid, following transoceanic migration of the A-genome diploid some 1.5 million years ago (Wendel and Crown, 2003).

Resistance to CBB varied considerably within the genus *Gossypium*, since annual varieties of the diploid *G. arboreum* and *G. herbaceum*, which have been cultivated for centuries on the Indian sub-continent, were highly resistant or immune to the disease (Hunter *et al.*, 1968; Wallace and El-Zik, 1989; Hillocks, 1992;). The tetraploid genotypes *G. hirsutum* provided the broadest spectrum of disease expression, varying from fully susceptible to resistant. At the opposite extreme, little resistance occurred naturally in *G. barbadense*. Breeding of cotton plants for *Xcm* resistance started after 1939, when the heritable nature of resistance to CBB in cotton was demonstrated (Knight and Clouston, 1939). The development of CBB resistant varieties of both tetraploid species, through transfer of *R* genes originally derived from other species of *Gossypium*, has since become widespread, principally due to recognition of the cost-effectiveness of blight-resistant cultivars in disease control. Extensive breeding programmes are still conducted in several countries to improve cotton resistance to CBB. Inheritance studies to transfer *R* genes to elite genotypes, from diploid indigenous cultivated cotton were actively developed, focusing on the most common virulent races 1 and 18 (Bayles and Verhalen, 2007; Haidar *et al.*, 2007; Shelke *et al.*, 2012). In China, the cultivar Zhongzhi Cotton 2, which is resistant to Verticillium wilt and cotton bollworm, also displayed immunity to CBB after a screening of 128 resistant progenies resulting from the highly resistant cultivar, Zhongzhi upland Cotton 372 (http://www.cotton247.com/cotton-production/planting/china-develops-verticillium-wilt-resistant-cotton/). Due to the complexity of the genetic background of the allotetraploid cultivated cotton varieties, the use of wild or poorly domesticated diploid genotypes would be a valuable pool for efficient *R* genes to CBB.

The cotton genes for resistance to cotton bacterial blight

To date, 18 major *R* genes (the so-called *B* genes) or polygene complexes in cotton have been identified

to be closely associated with resistance to CBB (Follin, 1986; Hillocks, 1992; Table 1). No single *R* gene confers durable resistance or immunity to CBB, due to evolutionary shifts in *Xcm* virulence in response to the selection pressures imposed by resistant varieties (Brinkerhoff *et al.*, 1984). However, pyramiding of *B* genes has been sometimes successfully used to produce lines with high resistance against multiple races of *Xcm*. For example, pyramiding B_2 with B_3 and other polygenic complexes (Bird, 1982; Brinkerhoff *et al.*, 1984) has provided substantial protection against all races of *Xcm* identified in the US (Essenberg *et al.*, 2002). In Africa, resistance to CBB was obtained by combining *B* genes (B_2B_3 and $B_9LB_{10}L$) from *G. hirsutum* which confer resistance to all *Xcm* races except races 20, 21 and 22 (Delannoy *et al.*, 2005). The line Im216 displayed exceptional resistance, often described as "immunity" (Brinkerhoff *et al.*, 1984; Bayles and Johnson, 1985). Although the parental lines from which Im216 was developed carried the major resistance genes B_2, B_3, b_7 and the polygenic complex B_{Sm} (Brinkerhoff *et al.*, 1984), the *R* genes in Im216 are not precisely known.

Promising near-isogenic lines homozygous for a single *B* gene were built in the susceptible Acala 44E background in order to investigate the contribution of individual *R* genes to the high resistance of Im216 (Essenberg *et al.*, 2002). The near-isolines possessing single *B* genes were predicted to be useful for studying *avr* gene activity and for mapping *B* genes, for comparing the physiology of the various *B*-gene regulated responses, and for identifying defense genes that are regulated by all *B* genes, as well as any that are *B*-gene specific (Patil *et al.*, 2005). In a recent study, the individual genes *B4, BIn,* and *b7* were used in the susceptible 'Acala 44E' background. The dominant *B4* and *BIn* and the recessive *b7 R* genes were pyramided (Essenberg *et al.*, 2014).

Table 1. The main cotton resistance genes.

R genes	Name	Origin	Date of discovery	Genotype	Ploïdy	Variety	Resistance level to BB
Major genes	B_2/B_3 (both dominant)	Uganda	1934	*Gossypium hirsutum*	4n	UgandaB31/ punctatum	Partial/high
	B_4 (partially dominant)	Tanzania ?	1948	*Gossypium arboreum*	2n	Multani	Partial
	B_5 (partially dominant)	?	1950	*Gossypium barbadense*	4n	Grendine White pollen	Partial
	B_6 (recessive)	Tanzania ?	1953	*Gossypium arboreum*	2n	Multani	Weak
	B_7 (sometimes dominant)	United States	1939	*Gossypium hirsutum*	4n	Upland Stoneville 20	High
	B_{9L}/B_{10L} (both dominant)	Central Africa/ Nigeria	1959	*Gossypium hirsutum*	4n	Allen Zaria 51-96	High/weak
	B_{10K} (dominant)	Lybia	1957	*Gossypium hirsutum*	4n	punctatum	Weak
	B_{11}	Nigeria	1965?	*Gossypium hirsutum*	4n	Allen	Weak
	$B_{In}/B_N/B_S$	United States	1952	*Gossypium hirsutum*	4n	?/Northern/ stormproof	High
	U_{9K} (dominant)	India	1963?	*Gossypium herbaceum*	2n	Wagad 8	High
Minor genes	B_{sm}	United States	1958	*Gossypium arboreum*	2n	?	Partial
	D_{sm}	United States	1958	*Gossypium hirsutum*	4n	Empire/ Deltapine	Partial
No R gene	-	-	-	*Gossypium hirsutum*	4n	Acala44	No resistance

Xcm race 1 carried the corresponding genes *avrB4, avrBIn,* or *avr b7*. In the field, the relative resistance of the Ac*b7* line and the Ac*B4* line depended upon the year, suggesting that their levels of resistance were weather-dependent. The resistance level of the Ac*B4, BInb7* pyramided line was not greatly enhanced over that of the single-*B*-gene lines, and was lower than that observed in the polygenic resistant background of the Im216 variety. This may be explained by the fact that the Im216 line also possesses other *R* genes which may activate additional transduction pathways or may modify the responses conditioned by *B4*, *BIn*, and *b7*, making them more rapid (Essenberg *et al.*, 2014). Results of this project reinforced the idea that the successful cotton resistance to *Xcm* is somehow more difficult to achieve than just pyramiding *R* genes.

The cotton resistance genes analogues

A high level of DNA polymorphism was detected within the domesticated cotton species (Paterson *et al*., 1996; Brubaker *et al.,* 1999; Paterson and Smith, 1999). Although genetic linkage maps are known to play fundamental roles in understanding the cotton genome structure (Yu *et al.,* 2012), little is yet understood about the organization of *R* genes in the cotton genome. The resistance gene analogues (RGAs) provide useful tools for the identification of full-length *R* genes from bacterial artificial chromosome and cDNA libraries. Genetic mapping revealed that many of the RGAs either co-segregated with, or are closely linked to, known disease *R* loci. Effort was made during the past 20 years to map F_2 progenies derived from inter-specific crosses between *G. hirsutum* and *G. barbadense* (Lacape *et al*., 2003; Rong *et al.,* 2004). RFLP or AFLP maps, as well as comparisons of genetic and physical maps, were used to delineate the chromosomal locations of cotton genes and quantitative trait loci (QTLs) that confer resistance to CBB (Reinisch *et al*., 1994; Wright *et al*., 1998). Several *R* gene loci have been assigned to different chromosomes (Rungis *et al.*, 2002). Based on a Simple Sequence Repeat/Single nucleotide polymorphism (SSR/SNP) methodology, molecular markers were developed for localizing *R* loci on cotton chromosomes to *Xcm* races, with the objective to improve the marker-assisted selection against this disease in American lines (Talercio *et al.*, 2006; Xiao *et al.*, 2010) and in Brazilian genotypes (Marangoni *et al*., 2013; Silva *et al.*, 2014).

The distribution of RGAs between the two subgenomes A and D of cotton is uneven, with RGAs being more abundant in the A than in the D subgenomes (He *et al.,* 2004). The majority of the RGAs identified were homologous within the three species, but their diversity was greater than expected at both the nucleotide and amino acid levels (Tan *et al.,* 2003). Four NBS-LRR type RGAs amplified from *G. barbadense* have been placed on the latest cotton genetic map (Rong *et al*., 2004). They are located on linkage groups to which the *R* genes B_2/B_3, and B_{6b} (chromosome 05) were previously mapped (Wright *et al.,* 1998). Furthermore, RFLP analysis of the Im216 line and seven *B*-gene containing Ac near-isogenic lines detected polymorphism in the B_{In3} gene with one of four RGA probes (Chakrabarty and Gabriel, 2004). Comparisons of an extensive collection of NBS-LRR type RGAs from *G. hirsutum* (He *et al*., 2004), *G. barbadense* (Gao *et al*., 2006), and *G. arboreaum* (Azhar *et al*., 2011) revealed that RGAs reside on a limited number of the cotton chromosomes, with those from a single subfamily tending to cluster and two of the RGA loci being co-localized with the CBB *R* genes. The occurrence of RGAs in endemic natural Brazilian populations of the tetraploid *G. mustelinum* was also assessed to evaluate resistance levels of the populations to different pathogens, including *Xcm*, in order to promote preventive strategies for *ex situ* conservation (Pinto de Menezes *et al.,* 2014).

Advances in the *G. raimondii* sequencing (Wang *et al.,* 2012) will most likely accelerate the knowledge of *R* gene localization in the genomes of tetraploid *G. hirsutum* and *G. barbadense* (Zhang *et al*., 2008; Paterson, 2009; Wang *et al.,* 2012; Yu *et al.,* 2013). Wei *et al.* (2013) annotated the NBS *R* genes at a genome-wide level in *G. raimondii*, offering information about the disease *R* genes to *Fusarium*, *Verticillium* and nematodes, but not to *Xcm*. This approach will also strengthen the exploration of genetic mechanisms underlying biosynthesis of cotton sesquiterpene phytoalexins and resistance against other pathogens and pests.

The cotton defense strategy

Plant defenses are assumed to play critical roles in directing the evolution between plants and their pathogens. The gene-for-gene concept of plant disease resistance predicts that pathogen avirulence and plant resistance result only if the pathogen

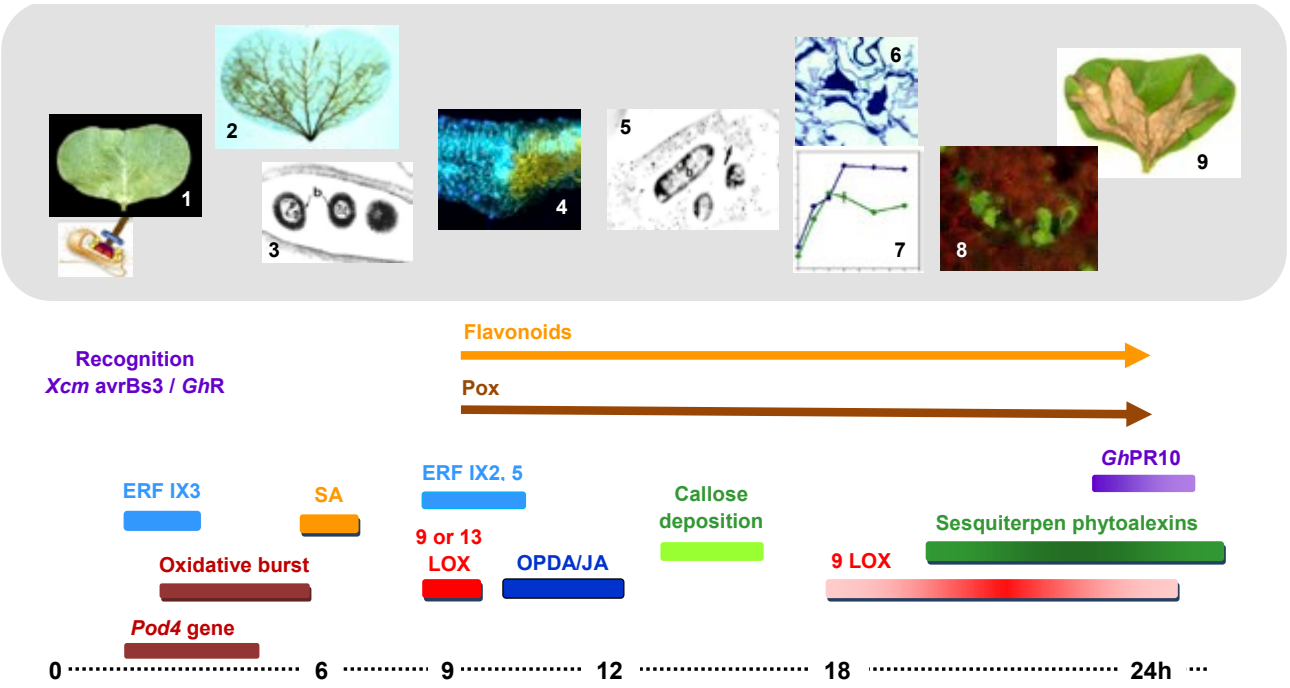

Figure 2. Time sequence of physiological events involved in the cotton HR to *Xanthomonas citri* pv. *malvacearum*. Following penetration of leaves by the pathogen (1 and 3), bacterial cells injected the effectors within the host nuclei. When recognized by host R proteins, several specific mechanisms were activated through signaling pathways. Genes of the ERF IX3 group are transcribed in parallel to the production of ROS (t = 3h; 2: localization of H_2O_2 in resistant leaves). Accumulation of SA culminated after the burst (t = 6h) and before activation of a *9- or 13-Lox* gene (t = 9h), transcription of *ERF* genes and synthesis of OPDA/JA (t = 12h). Production of flavonoids (orange stained in infected areas; 4) and total peroxidase activity (5: electron microscopic immunolocalization of peroxidase close to the bacteria) were also detected at the same time. Callose deposition contributed to stop the bacterial growth (7: green line) as compared to growth in susceptible plants (7: blue line), and preceded collapse of cells (6: condensation of the cytoplasm of HR cells). Appearance of HR symptoms in infected areas from t = 24h (9) occurred following dramatic increase in 9-Lox activity and strong accumulation of sesquiterpene phytoalexins (8: green fluorescence). Photo credits. 1, 2 and 7, A. Jalloul; 3, 5 and 6, M. Nicole; 4, G. Dai; 8, M. Essenberg; 9, M. Sayegh.

possesses an *avr* gene for which a corresponding *R* gene exists in the host plant (Flor, 1971). The *Xcm*-cotton interactions corresponded to the gene-for-gene model for host plant resistance (Gabriel *et al.*, 1986; Delannoy *et al.*, 2005), indicating the existence of specific molecular dialogue between the paired plant-bacterial gene participants. Microscope observations of HR tissues revealed more rapidly collapsed cells at infection sites with retracted plasmalemma, condensed cytoplasm, disorganized organelles including chloroplasts and nuclei, and accumulations of electron-dense material (Cason *et al.*, 1977; Al-Mousawi *et al.*, 1982a, 1982b). Electrophysiology also revealed transient depolarization of the plasma membranes at 7–12 h post-inoculation (hpi) which could be associated with major events closely related to the HR (Goodman and Novacky, 1994). The main mechanisms operating during the cotton HR to *Xcm* are summarized in Figure 2.

The oxidative burst

The oxidative burst produces reactive oxygen species (ROS) in a range of physiological events during plant life. In cotton, ROS are known to be produced during fiber elongation (Mei *et al.*, 2009). In HR-like resistance, the burst is considered as a key event, which was investigated during the cot-

ton defense to *Xcm*. In cotyledons of the Réba B50 cultivar (containing the B_2B_3 genes) challenged by *Xcm* race 18, the burst dramatically peaked 3 hpi. Several lines of microscopic, biochemical and molecular evidence have suggested that a cationic wall-bound peroxidase (Pod) could be involved in the production of superoxide anions (O_2^-) (Martinez *et al*., 1998; Delannoy *et al*., 2003). Consequent accumulation of H_2O_2 results from dismutation of O_2^- by a Mn superoxide dismutase (SOD), the transcription of which precedes enzymatic activity and production of O_2^- (Voloudakis *et al*., 2006). The plasmalemma NADPH-oxidase responsible for generation of O_2^- in other plants responding hypersensitively to avirulent pathogens remained silent during this burst. However, molecular approaches showed high transcription activity of a *GhNADPH-oxydase* gene between 12 and 18 hpi (Voloudakis, personal communication), the role of which has not yet been investigated.

Systemic resistance

Correlated with the HR is the systemic acquired resistance response (SAR), with salicylic acid (SA) as one of the specific markers. The *Xcm* avirulent race 18 interacting with the B_2B_3 Réba B50 genotype induced accumulation of SA 6 hpi, after the oxidative burst at infection sites, and systemically in whole plants 24 hpi (Martinez *et al.*, 2000). Although the role of H_2O_2 in cotton defense was not investigated, a relation with the accumulation of SA was proposed, but not yet verified. The *Xcm*-induced SAR showed an increase in Pod activity in non-infected leaves of the infected plants and a significant reduction of bacterial growth in post-infected leaves 24 hpi (Martinez *et al.*, 2000). Treatment of cotton leaves by exogenous SA triggers the SAR and its correlative effects, including increase in the expression of the *GhLox1* gene associated with membrane lipid degradation, and stimulation of the Lox pI 4.6 isoform activity (Jalloul *et al*., 2002; Marmey *et al*., 2007; Keshkiah *et al*., personal communication). Indirect evidence of induced systemic resistance (ISR) in cotton challenged by *Xcm* was obtained with possible production of ethylene (ET) (Champion, personal communication), but this result needs to be confirmed. The production of jasmonic acid (JA) specifically associated with the *Xcm*-triggered resistance is discussed below.

The oxylipin pathway

Enzymatic lipid peroxidation generated oxylipins result from the degradation by lipoxygenases (Lox) of linoleic and linolenic acids, the two major poly-unsaturated fatty acids of plant membranes (Liavonchanka and Feussner, 2006). This is a signature of plant cells reacting hypersensitively (Montillet *et al*., 2002), widely documented in the last past 10 years (Farmer and Mueller, 2013). Lox activity in infected susceptible cotton plants was weak, occurred late and did not correlate with water loss, but was concomitant with leaf chlorosis. During the HR, Lox activity was detected, associated with an increase in activity of Lox anionic isoforms, massive accumulation of 9S-fatty acid hydroperoxides, drastic water loss of reacting cells, the appearance of HR lesions at the leaf surfaces (Jalloul *et al*., 2002), and was preceded by accumulation of *GhLox1* transcripts (Jalloul *et al*., 2002; Marmey *et al*., 2007). Correlatively, a cotton patatin-like gene was also expressed during the HR to *Xcm,* most likely to set free the unsaturated fatty acids from membranes before peroxidation (Cacas *et al*., 2008). Screening different *B*/*avr* gene combinations revealed that Lox response was concomitantly triggered during various cotton/*Xcm* incompatible interactions (Sayegh-Alhamdia *et al*., 2008), strengthening the previous evidence that this reaction is a key event of HR cell death (Jalloul *et al*., 2002; Montillet *et al*., 2002). The expression of the *GhLox1* gene occurred sooner after inoculation with race 1 in the immune Im216 and highly resistant AcHR lines than in near-isogenic lines AcB4 and AcBIn (Essenberg *et al*., 2014).

A branch of oxilipin metabolism involved in plant defense to pathogens is the octadecanoid pathway leading to the synthesis of OPDA (12-oxo phytodienoic acid) and JA (Wastermack and Hause, 2013). Production of JA during the cotton HR was suspected several years ago (Jalloul, personal communication). Pharmacological approaches on cotton infected by *Xcm* race 18 revealed that exogenous JA and diethyldithiocarbamic acid (DIECA), an inhibitor of the JA signalling pathway, modulated the activity of Lox isoforms (Jalloul, personal communication). Methyl JA vapours also induced accumulation of H_2O_2, but not that of SA, and stimulated the *GhLox1* gene expression, *GhLox1* activity, and reduced multiplication of a *Xcm* virulent race (Marmey *et al*., 2007). Recent accurate investigation of the JA pathway in the cotton challenged by *Xcm* showed that this hormone was

specifically synthesized *de novo* during the triggered HR and correlated with induced expression of the JA biosynthesis genes [allene oxide synthase (*GhAOS*), allene oxide cyclase (*GhAOC*), acyl-coA oxidases (*GhACX*)] (Cacas *et al.*, personal communication).

Ethylene-response transcription factors (ERF) are believed to play crucial roles in the activation of plant defense responses. Analysis of the transcript profiles of the cotton ERF group IXa in the regulation of specific resistance to *Xcm* showed that expression of four members of the group IXa were induced on challenge with *Xcm*, and were induced synergistically by JA in combination with ET. This suggested that the encoded ERF proteins may play key roles in the integration of both signals to activate JA- and ET-dependent responses (Champion *et al.*, 2009). Recently, the *ORA47*-like transcription factor, an ERF of the group II, was shown to be responsible for JA accumulation during *Xcm*-induced cotton HR by positively regulating octadecanoic pathway genes (Cacas *et al.*, personal communication).

Taken together, these data show that the oxylipin pathway may have a central position in the cotton defense strategy, and indicate that JA mediated the HR cell death to *Xcm* by regulating up- and downstream several defense responses, including transcription of *GhLox1*.

The accumulation of sesquiterpenoid phytoalexins

In the previous review on CBB (Delannoy *et al.*, 2005), the accumulation of sesquiterpenoid phytoalexins during interactions of cotton cultivars with *Xcm* was summarized. Two groups of cotton phytoalexins were identified; those with oxygen functions on C-7 (DHC, HMC, LC, and LCME) and those with oxygen on C-8 (dHG and HG) (Wang *et al.*, 2003). Derivatives of dHG and HG are produced in healthy plants, into the lysigenous glands. Both HG and dHG are important phytoalexins in the response to the vascular wilt fungi (Mace *et al.*, 1985; Zhang *et al.*, 1993). The cadinene sesquiterpene cyclase enzyme (Davila *et al.*, 1995; Davis and Essenberg, 1995) was purified from *Xcm*-inoculated cotton cotyledons (Davis *et al.*, 1996). The cDNA of this enzyme has been cloned and expressed in *Escherichia coli* to produce enzymically active (+)-δ-cadinene synthase (Davis *et al.*, 1998; Luo *et al.*, 2001). Six sesquiterpenoid phytoalexins were identified in resistant cotton plants to restrict bacterial multiplication (Essenberg *et al.*, 1982, 1990; Abraham *et al.*, 1999). They accumulated during the HR but did not appear to be associated with particular *R* genes, as cotton lines carrying single genes B_2, b_7, or B_{In} synthesized phytoalexins during interactions with incompatible races. Furthermore, the susceptible *R* genes-free line Ac44 produced almost no phytoalexins in response to *Xcm*, but accumulated significant amounts during its non-host response to *X. c. campestris* (Essenberg *et al.*, 1990).

The yellow-green fluorescence detected in cells undergoing the HR close to *Xcm* microcolonies (Essenberg *et al.*, 1979, 1992b) was spectrally similar to that of the phytoalexins LC and LCME (Pierce and Essenberg, 1987; Essenberg *et al.*, 1992b). Fluorescent HR cells isolated from inoculated, resistant cotyledons contained 40 times more DHC than as symptomless cells, together with higher amounts of other phytoalexins (Pierce *et al.*, 1996). The bacteria in the intercellular spaces adjacent to those phytoalexin-rich HR cells were exposed to the phytoalexins which diffused outside cells to stop the bacterial growth (Pierce *et al.*, 1996). In leaves of highly resistant lines possessing several *B* genes, the bacteria stop multiplying 3 to 4 d after inoculation with phytoalexins, being detectable during the second day (Essenberg *et al.*, 1990, 1992a; Pierce *et al.*, 1996). Three different cotton lines, evaluated for their production of phytoalexins to account for their resistance to CBB, displayed much than more than adequate concentrations of phytoalexins at the right times and places *in planta*. Evidence suggested that the biosynthetic sesquiterpene pathway was transcriptionally regulated; microarray analysis of transcripts in *Xcm*-inoculated leaves of the highly resistant line Im216 demonstrated that inoculation led to higher transcript levels of two genes for (+)-δ-cadinene synthase *cdn1-C* and *cdn1-*, of a gene for (+)-δ-cadinene-8-hydroxylase, *CYP706B1*, and also of *hmg-1* and *hmg-2* genes encoding HMG-CoA reductase, the rate-limiting enzyme of the central mevalonate pathway (Patil *et al.*, 2005). Immunohistochemical staining of (+)-δ-cadinene synthase revealed more intense staining in the HR cells than in symptomless neighbouring cells (Park, 1997). Results of a pulse-labeling study were consistent with these observations: [$^{13}C_2$]acetate was incorporated into DHC and HMC most rapidly during the period of rapid appearance of green-fluorescent HR cells (24 to 72 hpi), consistent with the hypothesis that these cells achieve a burst of phytoalexin biosynthesis before they die (Górsky *et al.*, 1995). However, ^{13}C incorporation

into phytoalexins continued 1 and 2 d after HR cell numbers reached their maximum, suggesting that the surrounding healthy tissues also synthesized some phytoalexins.

Other defense responses

Callose-containing papillae were sometimes seen in periplasmic areas close to bacterial colonies (Daï *et al*., 1996), probably contributing to physical limiting of mesophyll colonization by the bacteria. Histochemical detection of flavonoids by fluorescence showed strong staining in living cells at the margins of lesions of infected resistant cotyledons, and the main part of necrotic areas, before HR cell death was completed (Daï *et al.,* 1996). A flush of the anthocyanin chrysanthemin vacuolar red pigment is an indicator of a resistance response in cotton leaves and cotyledons (Kangatharalingam *et al*., 2002). In bright sunlight in the field, the red epidermal cells occurred in a continuous ring surrounding each site and contributed to the ink-spot appearance of those lesions. There was some evidence that flavonoids in infected cotton cells provided protection to the mesophyll cells from light-dependent phytoalexin toxicity (Edwards *et al.,* 2008). The antioxidant status of phenol compounds was often correlated with Pod activity, known to be involved in plant defense to stress. The increase of total Pod activity during cotton HR to *Xcm* was associated with increase in anionic isoforms and in transcriptional activities of class III Pod genes (Delannoy *et al*., 2003, 2004). Although having no explicitly known functions (Delannoy *et al.,* 2006), the *pod 10* gene was specifically induced by *Xcm* race 18, but rapidly inhibited during the HR. By contrast, the *pod 4* gene was strongly activated at 2 hpi, and its transcript lasted at least 12 h (Delannoy *et al.,* 2003). *Pod 4* could be a potent candidate gene for the generation of superoxide anions associated with the oxidative burst. Recent research on cotton MAP kinases (Shi *et al.,* 2011), proposed that *GhMPK16* has a role in two signaling pathways, one that responds to pathogens and another involved in drought stress. *GhMPK16* may thus serve as a point for crosstalk between biotic and abiotic stress response signaling.

Control of the cotton bacterial blight

Methods to control CBB are available with more or less efficacy, including chemical treatments, agronomical techniques, extensive breeding programmes, biological and biotechnology approaches. Several of these have been combined to control this disease (Brinkerhoff, 1970; Brinkerhoff *et al*., 1984; Hillocks, 2010), leading to satisfactory levels of cotton protection, but with negative ecological impacts. In several countries, control has been achieved through the use of resistant genotypes associated with cultural methods. To prevent initial infection of cotton genotypes by CBB, it is advocated to:

- plant high-quality, blight-resistant varieties, if available,
- identify infected plants and varieties, and remove them if possible,
- shred stalks, incorporate cotton debris into soil, and harvest infested fields as soon as possible,
- not cultivate or move equipment through fields, when foliage is wet,
- use growth regulators to prevent host rank growth,
- use an alternative crop in fields that had CBB the previous year,
- control irrigation,
- manage seed sanitation to overcome the diseases, using acids, copper compounds or chlorine derivatives and heat treatments.

Initiated in the 20th century (Putcha *et al.,* 1998), research on biocontrol agents to protect cotton from CBB is currently active, since these agents are known to be eco-friendly. *Trichoderma harzianum, Pseudomonas fluorescens* and *Bacillus subtilis* were isolated from cotton rhizosphere soil and tested individually for their effectiveness in controlling cotton diseases (Medeiros *et al*., 2011), including CBB (Fallahzadeh-Mamaghaniet *et al.,* 2009; Fallahzadeh and Ahmadzadeh, 2010; Salaheddin *et al*., 2010; Jagtap *et al.,* 2012). These bioagents triggered the defense related enzymes involved in synthesis of phenols. Higher activities of Pod, phenylalanine ammonia-lyase, polyphenol oxidase and ß-1,3-glucanase were observed in *P. fluorescens*- and *T. harzianum*-treated cotton plants after challenge inoculation with *Xcm*. Seed treatment with these bioagents enhanced seed germination, limited growth of *Xcm,* and also induced systemic resistance in plants (Raghavendra *et al.,* 2013). Medicinal plant extracts (Babu *et al*., 2007; Satyaa *et al.,* 2007) and a white crystalline solid

from the red alga *Portieria hornemannii* (Sivakumar, 2014) were also tested for their anti-bacterial effects on *Xcm*. Extracts from *Allium* sp., *Origanum vulgare*, and *Althea officinalis*, and the crude crystalline compounds of *P. hornemannii* had inhibitory effects on *Xanthomonas* growth *in vitro* and these materials or plants could be potential candidates for the management of CBB. Similarly, plant extracts from *Datura alba*, *Moringa olifera*, *Azadirachta indica* and *Syzgyium cumini* and homeopathic products (Aviara, Influenzium and Hepatitus) have showed significant biological control of CBB in greenhouses and in fields (Javed *et al.*, 2013); they were advised as preferred to the use of synthetic chemicals. A promising complementary way to control CBB in cotton would be to use natural elicitors to potentialize cotton defense responses. Thus, triggering the different signaling pathways, such as the oxilipin pathway, may have positive effects on cotton defense (Cacas *et al.*, personal communication).

Introduction of new traits through genetic engineering and/or biotechnology could be a way to improve cotton resistance to pests. Genetically modified cotton, also known as *Bt*-cotton for *Bacillus thuringiensis* which encodes an insecticidal protein, has become widespread, covering a total of 6.8 million hectares in 2002, and 25 million hectares in 2012 around the world, mostly in India, China, Pakistan, Brazil, the US and some African countries. Cotton was only modified to resist herbicides and the bollworm, but the insect has developed resistance to the *Bt*-cotton. Experimental genetically modified cotton was investigated for its responses to pathogenic microbes, but not yet in large field tests. To our knowledge, such research has not stringently considered resistance to *Xcm*. Genetic engineering of transgenic cotton for resistance to leaf curl disease, using an antisense RNA approach, was described as a potential technique to managing this disease (Jagannathan and Balasubramani, 2010). The transcriptome profile in leaves and roots of the transgenic cotton line T-34 expressing *hpa*1*Xoo*, the encoding harpin gene from *X. oryzae* pv. *oryzae*, was analysed using a cotton cDNA microarray (Miao *et al.*, 2010). This transgenic cotton line up-regulated defense genes and signaling pathways more than its non-transgenic parental line in response to the vascular fungi *V. dahliae* and *F. oxysporum* f. sp. *vasinfectum*. It would be of interest to unravel these defense responses when this line is infected by *Xcm*.

Concluding remarks

In addition to cotton fibre quality, durable disease resistance to pathogens and pests is a key objective for plant breeders developing new cotton varieties. Although specific to a particular *Xcm* race, resistance conferred by a single dominant *R* gene may be overcome by the pathogen. So far, the creation of pyramided *R* gene-containing lines effective against all races of *Xcm* has not been achieved. Hence, cloning of *R* genes is desirable to facilitate development of agronomic cultivars possessing stable, multigenic resistance. Comparison of an elite tetraploid cotton cultivar to its wild diploid ancestors will provide new insights into how polyploidy may provide better understanding of the cotton genome (Page *et al*., 2012). This will could utilize the cotton genomic resources available online (http://www.cottongen.org/; http://www.cottondb.org/). Sequencing of the *Xcm* genome (Cunnac *et al.*, 2013; Zhai *et al.*, 2013) and comparison with genomes of other Xanthomonads (http://www.xanthomonas.org/genomes.html), with the aim of assessing the close relationships between *Xcm* effectors and cotton R proteins, may shed light on the evolution of host-specific features. Demonstration of the roles of effectors in pathogenicity and disease is crucial for understanding the regulation of cotton genes activated, or repressed, during the HR and the downstream modulation of the cotton defense strategy. Presently, we do not know how the *Xcm* effectors interact with the *R* genes, nor how the *R* genes lead to all the effects shown in Figure 2.

Agroecology and ecology engineering for cotton pest and disease management should open promising alternative avenues for phytosanitary protection, mainly in landscape farming, in close association with integrated pest management strategies. Organic cotton (http://www.organiccotton.org/oc/index.php), although covering less than 3% of the world's cultivated lands, is considered as an exciting challenge in accordance with the principles of sustainable development to prevent the effects of pests and diseases, and to preserve biodiversity.

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Literature cited

Abdelrahim K.A.A., A.A. Al-Arfaj, A.M.A. Mashaly and K. Rudolph 2013. Plasmids in races of *Xanthomonas axonopodis* pv. *malvacearum* (*Xam*), the causal agent of bacterial blight of cotton. *African Journal of Microbiology Research* 7, 807–813.

Abdo-Hasan M., H. Khalil, B. Debis and N. MirAli 2008. Molecular characterization of Syrian races of *Xanthomonas axonopodis pv. malvacearum*. *Journal of Plant Pathology* 90, 431–439.

Abraham K.J., M.L. Pierce and M. Essenberg 1999. The phytoalexins desoxyhemigossypol and hemigossypol are elicited by *Xanthomonas* in *Gossypium* cotyledons. *Phytochemistry* 52, 829–836.

Ajene I.J., K.C. Shenge and A.D. Akpa 2014. Races of *Xanthomonas citri* subsp. *malvacearum*, the causal organism of bacterial blight of cotton in northern Nigeria. *Archives of Phytopathology and Plant Protection* http//dx.doi.org/10.1080/03235408.2013.873561.

Akello B. and R.J. Hillocks 2002. Distribution and races of *Xanthomonas campestris* pv. *malvacearum* on cotton (*Gossypium hirsutum*) in Uganda. *Journal of Phytopathology* 150, 65–69.

Al-Mousawi A.H., P.E. Richardson, M. Essenberg and W.M. Johnson 1982a. Cotyledon and leaf ultrastructure of bacterial blight-immune cotton line inoculated with a low level of *Xanthomonas campestris* pv. *malvacearum*. *Phytopathology* 72, 1231–1234.

Al-Mousawi A.H., P.E. Richardson, M. Essenberg and W.M. Johnson 1982b. Ultrastructural studies of a compatible interaction between *Xanthomonas campestris* pv. *malvacearum* and cotton. *Phytopathology* 72, 1222–1230.

Amani B. 1972. Black arm disease of cotton. *Iranian Phytopathology Journal* 8, 112–121.

American Phytopathological Society. 2001. *Compendium of Cotton Diseases*, Second Edition (T.L. Kirkpatrick and C. S. Rothrock, ed.), St. Paul, MN, 100 pp.

Arabsalmani M., H. Rahimian, F. Azad and A. Qhasemi 2002. Occurrence of bacterial blight of cotton caused by *Xanthomonas axonopodis* pv. *malvacearum* in Khorasan province. In: *Proceeding of the 15th Iranian Plant Protection Congress*, Razi University, Kermanshah, Iran, 68.

Atkinson G.F. 1891. The black rust of cotton. *The Alabama Agricultural Experimental Station Bulletin* 27, 1–16.

Azhar M.T, I. Amin, A. Bashir and S. Mansoor 2011. Characterization of resistance gene analogs from *Gossypium arboreum* and their evolutionary relationships with homologs from tetraploid cottons. *Euphytica* 178 351–362. doi, 10.1007/s10681-010-0310-0.

Babu S., S. Satish, D.C. Mohana, M.P. Raghavendra and K.A. Raveesha 2007. Iranian medicinal plants against plant pathogenic *Xanthomonas* pathovars. *Journal of Agricultural Technology* 3, 307–316.

Bayles M.B. and W.M. Johnson 1985. Growth of homologous and heterologous phytopathogenic bacteria in cotton lines susceptible, resistant, or immune to blight. *Phytopathology* 75, 515–519.

Bayles M.B. and L.M. Verhalen 2007. Bacterial blight reactions of sixty-one upland cotton cultivars. *Journal of Cotton Science* 11, 40–51.

L.S. Bird 1982. The MAR (multi-adversity resistance) system for genetic improvement of cotton. *Plant Disease* 66, 172–176

Boch J. and U. Bonas 2010. *Xanthomonas* AvrBs3 family-type III effectors, discovery and function**.** *Annual Review of Phytopathology* 48, 419–436.

Brinkerhoff L.A. 1970. Variation in *Xanthomonas malvacearum* and its relation to control. *Annual Review of Phytopathology* 8, 85–110.

Brinkerhoff L.A, L.M. Verhalen, W.M. Johnson, M. Essenberg and P.E. Richardson 1984. Development of immunity to bacterial blight of cotton and its implications for other diseases. *Plant Disease* 68, 168–173.

Brubaker C.L., Bourland F.M. and Wendel J.F. 1999. The origin and domestication of cotton. In: *Cotton, Origin, History, Technology, and Production* (W.C. Smith, J.T. Cothren, ed.), New York, John Wiley & Sons Inc., 3–31.

Cacas J.L., P. Marmey, M. Alhamdia, A. Jalloul, A. Rojas-Mendoza, A. Clérivet, M. Nicole and J.L. Montillet 2008. Patatin-like genes are specifically expressed in cotton during *Xanthomonas campestris*-induced hypersensitive cell death. *Plant Cell Reports* 28, 155–164.

Cason E.T., P.E. Richardson, L.A. Brinkerhoff and R.K. Gholson 1977. Histopathology of immune and susceptible cotton cultivars inoculated with *Xanthomonas malvacearum. Phytopathology* 67, 195–198.

Chakrabarty P.K. and D.W. Gabriel 2004. *Initiatives in cloning bacterial blight resistance genes from cotton*. In: *International Cotton Genome Initiative ICGI–2004 Workshop.* Hyderabad, Andhra Pradesh, India, 52.

Chakrabarty P.K., Y.P. Duan and D.W. Gabriel 1997. Cloning and characterization of a member of the *Xanthomonas avr/pth* gene family that evades all commercially utilized cotton *R* genes in the United States. *Phytopathology* 87, 1160–1167.

Champion A., E. Hébrard, B. Parra, C. Bournaud, M. Marmey, C. Tranchant and M. Nicole 2009. Molecular diversity and gene expression of cotton ERF transcription factors reveal that ERF group IXa members are responsive to jasmonate, ethylene and *Xanthomonas*. *Molecular Plant Pathology* 10, 471–485.

Cunnac S., N. Forero Serna, E. Ortiz, B. Szurek, L. Noel, M. Arlat, M.A. Jacques, L. Gagnevin, S. Bolot, S. Carrere, M. Nicole, and R. Koebnik 2013. High-Quality Draft Genome Sequences of two *Xanthomonas citri* pv. *malvacearum* strains. *Genome Announcements* 1, 1–2.

Dai G.H., M. Nicole, C. Martinez, E. Bresson, J.P. Agnel, J.F. Daniel, C. Andary and J.P. Geiger 1996. Flavonoids accumulate in cell walls, middle lamellae and callose-rich papillae during an incompatible interaction between *Xanthomonas campestris* pv. *malvacearum* (race 18) and cotton. *Physiological and Molecular Plant Pathology* 49, 285–206.

Davila Huerta G., H. Hamada, G.D. Davis, R.D. Stipanovic, C.M. Adams and M. Essenberg 1995. Cadinane-type sesquiterpenes induced in *Gossypium* cotyledons by bacterial inoculation. *Phytochemistry* 39, 531-536.

Davis G.D. and M. Essenberg 1995. (+)-*d* -Cadinene is a product of sesquiterpene cyclase activity in cotton. *Phytochem-*

istry 39, 553–567.
Davis E.M., J. Tsuji, G.D. Davis, M.L. Pierce and M. Essenberg 1996. Purification of (+)-*d*-cadinene synthase, a sesquiterpene cyclase from bacteria-inoculated cotton foliar tissue. *Phytochemistry* 41, 1047–1055.
Davis E.M., Y.S. Chen, M. Essenberg and M.L. Pierce 1998. cDNA sequence of a (+)-*d*-cadinene synthase gene (Accession No. U88318) induced in *Gossypium hirsutum* L. by bacterial infection. *Plant Physiology* 116, 1192.
De Feyter R. and D.W. Gabriel 1991. At least six avirulence genes are clustered on a 90-kilobase plasmid in *Xanthomonas campestris* pv *malvacearum*. *Molecular Plant-Microbe interactions* 4, 423–432.
De Feyter R., Y. Yang and D.W. Gabriel 1993. Gene-for-gene interactions between cotton *R* genes and *Xanthomonas campestris* pv. *malvacearum avr* genes. *Molecular Plant-Microbe Interactions* 6, 225–237.
De Feyter R., H. McFadden and L. Dennis 1998. Five avirulence genes from *Xanthomonas campestris* pv. *malvacearum* cause genotype-specific cell death when expressed transiently in cotton. *Molecular Plant-Microbe Interactions* 11, 698–701.
Delannoy E., A. Jalloul, K. Assigbetsé, P. Marmey, J.P. Geiger, J.F. Lherminier, J.F. Daniel, C. Martinez and M. Nicole 2003. Activity of class III peroxidases in the defense of cotton to bacterial blight. *Molecular Plant-Microbe Interactions* 16, 1030–1038.
Delannoy E., P. Marmey, C. Penel and M. Nicole 2004. Les peroxydases végétales de classe III. *Acta Botanica Gallica* 151, 353–380.
Delannoy E., B. Lyon, P. Marmey, A. Jalloul, J.L. Montillet, J.F. Daniel, M. Essenberg and M. Nicole 2005. Resistance of cotton to *Xanthomonas campestris* pv. *malvacearum*. *Annual Review of Phytopathology* 43, 62–82.
Delannoy E., P. Marmey, A. Jalloul and M. Nicole 2006. Molecular analysis of cotton peroxidases. *Journal of Cotton Science* 10, 53–60.
Edwards W.R., J.A. Hall, A.R. Rowlan, T. Schneider-Barfield, T.J. Sun, M.A. Patil, M.L. Pierce, R.G. Fulcher, A.A. Bell and M. Essenberg 2008. Light filtering by epidermal flavonoids during the resistant response of cotton to *Xanthomonas* protects leaf tissue from light-dependent phytoalexin toxicity. *Phytochemistry* 69, 2320–2328.
Essenberg M., E.T. Jr. Cason, B. Hamilton, L.A. Brinkerhoff, R.K. Gholson and P.E. Richardson 1979. Single cell colonies of *Xanthomonas malvacearum* in susceptible and immune cotton leaves and the local resistant response to colonies in immune leaves. *Physiological Plant Pathology* 15, 53–68.
Essenberg M., M.A. Doherty, B.K. Hamilton, V.T. Henning, E.C. Cover, S.J. McFaul and W.M. Johnson 1982. Identification and effects on *Xanthomonas campestris* pv *malvacearum* of two phytoalexins from leaves and cotyledons of resistant cotton. *Phytopathology* 72, 1349–1356.
Essenberg M., P.B.J. Grover and E.C. Cover 1990. Accumulation of antibacterial sesquiterpenoids in bacterially inoculated *Gossypium* leaves and cotyledons. *Phytochemistry* 29, 3107–3113.
Essenberg M., M.L. Pierce, E.C. Cover, B. Hamilton, P.E. Richardson and V.E. Scholes 1992a. A method for determining phytoalexin concentrations in fluorescent, hypersensitively necrotic cells in cotton leaves. *Physiological and Molecular Plant Pathology* 41, 101–109.
Essenberg M., M.L. Pierce, B. Hamilton, E.C. Cover, V.E. Scholes and P.E. Richardson 1992b. Development of fluorescent, hypersensitively necrotic cells containing phytoalexins adjacent to colonies of *Xanthomonas campestris* pv *malvacearum*. *Physiological and Molecular Plant Pathology* 41, 85–99.
Essenberg M., M.B. Bayles, R.A. Samad, J.A. Hall, L.A. Brinkerhoff and L.M. Verhalen 2002. Four near-isogenic lines of cotton with different genes for bacterial blight resistance. *Phytopathology* 92, 1323–1328.
Essenberg M., M.B. Bayles, M.L. Pierce and L.M. Verhalen 2014. Pyramiding *B* genes in cotton achieves broader, but not always higher, resistance to bacterial blight. *Phytopathology* 104, 1088-1097.
Fallahzadeh V. and M. Ahmadzadeh 2010. Induction of systemic resistance to bacterial blight caused by *Xanthomonas axonopodis* pv. *malvacearum* in cotton by fluorescent pseudomonads of cotton rhizosphere. *Journal of Agricultural Technology* 6, 341–348.
Fallahzadeh-Mamaghani V., M. Ahmadzadeh and R. Sharifi 2009. Screening systemic resistance-inducing fluorescent Pseudomonads for control of bacterial blight of cotton caused by *Xanthomonas campestris* pv. *malvacearum*. *Journal of Plant Pathology* 91, 663–670.
Farmer E. and M.J. Mueller 2013. ROS-Mediated Lipid Peroxidation and RES-Activated Signaling. *Annual Review in Plant Biology* 64, 429–450.
Flor H.H. 1971. Current status of the gene-for-gene concept. *Annual Review of Phytopathology* 9, 275–296.
Follin J.C. 1983. Races of *Xanthomonas campestris* pv. *malvacearum* (Smith) Dye in Western and Central Africa. *Coton et Fibres Tropicales* 38, 277–280.
Follin J.C. 1986. La sélection du cotonnier (*Gossypium hirsutum L.*) pour la résistance aux maladies présentes en Afrique au sud du Sahara. *Coton et Fibres Tropicales*, Supplément 7, 19–22.
Follin J.C., B. Girardot, V. Mangano and R. Benitez 1988. Nouveaux résultats sur le déterminisme génétique de la résistance foliaire totale du cotonnier (*Gossypium hirsutum* L.) à la bactériose *Xanthomonas campestris* pv. *malvacearum* (Smith) Dye, race 18 et 20. *Coton et Fibres Tropicales* 43, 167–175.
Fryxell P.A. 1992. A revised taxonomic interpretation of *Gossypium* L. (Malvaceae). *Rheedea* 2, 108–165.
Gabriel D.W. 1999. The *Xanthomonas avr/pth* gene family. In: *Plant-Microbe Interactions*, vol. 4 (G. Stacey, N.T. Keen, ed.), APS Press, St. Paul, MN, 39–55.
Gabriel D.W, A. Burges and G.R. Lazo 1986. Gene-for-gene interactions of five cloned avirulence genes from *Xanthomonas campestris* pathovar *malvacearum* with specific resistance genes in cotton. In: *Proceedings of the National Academy of Science of the USA*, 83, 6415–6419.
Gao Y., W. Guo, L. Wang and T. Zhang 2006. Isolation and characterization of resistance and defense gene analogs in cotton (*Gossypium barbadense* L.). *Science in China Serie C, Life Sciences* 49, 530–542.

Goodman R.N. and A.J. Novacky 1994. *The hypersensitive reaction in plants to pathogens, a resistance phenomenon*. American Phytopathological Society, St. Paul, MN, 256 pp.

Górski P.M., T.E. Vickstrom, M.L. Pierce and M. Essenberg 1995. A ^{13}C-pulse-labeling study of phytoalexin biosynthesis in hypersensitively responding cotton cotyledons. *Physiological and Molecular Plant Pathology* 47, 339–356.

Haidar S., I.A. Khan, S. Mansoor and Y. Zafar 2007. Inheritance studies of bacterial blight disease resistance genes in cotton (*Gossypium hirsutum L.). Pakistan Journal of Botany* 39, 603–608.

He L., C. Du, L. Covaleda, Z. Xu, A.F. Robinson, J.Z. Yu, R.J. Kohel and H.B. Zhang 2004. Cloning, characterization, and evolution of the NBS-LRR-encoding resistance gene analogue family in polyploid cotton (*Gossypium hirsutum* L.). *Molecular Plant-Microbe Interactions* 17, 1234–1241.

Hillocks R.J. 1992. Bacterial blight. In: *Cotton Diseases* (RJ Hillocks, ed.), Wallingford, UK, CAB International Redwood press, Melksham, 39–85.

Hillocks R.J. 2010. Cotton diseases and their control in the 21st century. In: *Cotton: Technology for the 21st Century* (Ph.J. Wakelyn, M.R. Chaudry, ed.), International Cotton Advisory Council, Washington DC, USA, 155–180.

Hunter R.E., L.A. Brinkerhoff and L.S. Bird 1968. Development of a set of Upland cotton lines for differentiating races of *Xanthomonas malvacearum. Phytopathology* 58, 830–832

Innes N.L. 1983. Bacterial blight of cotton. *Biological Reviews* 58, 157–176.

Jagannathan A. and G. Balasubramani 2010. *Biotechnological approaches to combat cotton leafcurl virus disease, Genetic Engineering and molecular marker techniques for cotton leaf curl virus disease*. Lambert Academic Publishing, 88 pp.

Jagtap G.P., A.M. Jangam and Deya U. 2012. Management of bacterial blight of cotton caused by *Xanthomonas axonopodis* pv. *malvacearum. Scientific Journal of Microbiology* 1, 10–18.

Jalloul A., J.L. Montillet, K. Assigbetsé, J.P. Agnel, E. Delannoy, C. Triantaphylides, J.F. Daniel, P. Marmey, J.P. Geiger and M. Nicole 2002. Lipid peroxidation in cotton, *Xanthomonas* interactions and the role of lipoxygenases during the hypersensitive reaction. *The Plant Journal* 32, 1–12.

Javed M.T., M.A. Khan, M. Ehetisham-ul-Haq and Atiq 2013. Biological management of bacteria blight of cotton caused by *Xanthomonas campestris* pv. *malvacearum* through plant extracts and homeopathic products. *Research Journal of Plant Disease and Pathology* 1, 1–10.

Kangatharalingam N., M.L. Pierce, M.B. Bayles and M. Essenberg 2002. Epidermal anthocyanin production as an indicator of bacterial blight resistance in cotton. *Physiological and Molecular Plant Pathology* 61, 189–195.

Knight R.L. and T.W. Clouston 1939. The genetics of black-arm resistance. 1. Factors B_2 and B_3. *Journal of Genetics* 38, 133–159.

Lacape J.M., T.B. Nguyen, S. Thibivilliers, B. Bojinov, B. Courtois, R.G. Cantrell, B. Burr and B. Hau 2003. A combined RFLP-SSR-AFLP map of tetraploid cotton based on a *Gossypium hirsutum* x *Gossypium barbadense* backcross population. *Genome* 46, 612–626.

Leach J.E. and F.F. White 1996. Bacterial avirulence genes. *Annual Review of Phytopathology* 34, 153–179.

Liavonchanka A. and Y. Feussner 2006. Lipoxygenases, occurrence, functions and catalysis. *Journal of Plant Physiology* 163, 348–357.

Luo P., Y.H. Wang, G.D. Wang, M. Essenberg and X.Y. Chen 2001. Molecular cloning and functional identification of (+)-*d* -cadinene-8-hydroxylase, a cytochrome P450 monooxygenase (CYP706B1) of cotton sesquiterpene biosynthesis. *The Plant Journal* 28, 95–104.

Mace M.E., R.D. Stipanovic and A.A. Bell 1985. Toxicity and role of terpenoid phytoalexins in verticillium wilt resistance in cotton. *Physiological Plant Pathology* 26, 209–218.

Machado A.C.Z., L.C.C.B. Ferraz and M.M. Inomoto 2012. Pathogenicity of *Pratylenchus brachyurus* on cotton plants. *Journal of Cotton Science* 16, 268–271.

Mansfield J., S. Genin, S. Magori, S. Citovski, M.S. Anum, P. Ronald, M. Dow, V. Verdier, S.V. Beer, M.A. Machado, I. Toth, G. Salmond and G. Foster 2012. Top 10 plant pathogenic bacteria in molecular pathology. *Molecular Plant Pathology* 13, 614–629.

Marangoni M., L. Girotto, M.P. Nunes, W.P. Almeida, R. Galbieri, Y. Schuster and Y.R. Mehta 2013. Search for a Microsatellite Marker Linked with Resistance Gene to *Xanthomonas axonopodis* pv. *malvacearum* in Brazilian Cotton. *American Journal of Plant Sciences* 4, 2039–2042.

Marmey P., A. Jalloul, M. Alhamdia, K. Assigbetsé, J.L. Cacas, A. Voloudakis, A., Clérivet A. Champion, J.L. Montillet and M. Nicole 2007. Cloning of a 9-lipoxygenase involved in the signalling pathway of the hypersensitive reaction of cotton *Gossypium hirsutum* to *Xanthomonas campestris* pv. *malvacearum*. *Plant Physiology and Biochemistry* 45, 596–606.

Martinez C., J.L. Montillet, E. Bresson, J.P. Agnel, G.H. Dai, J.F. Daniel, J.P. Geiger and and M. Nicole 1998. Apoplastic peroxidase generates superoxide anions in cells of cotton cotyledons undergoing the hypersensitive reaction to *Xanthomonas campestris* pv. *malvacearum* race 18. *Molecular Plant-Microbe interactions* 11, 1038–1047.

Martinez C., J.C. Baccou, E. Bresson, Y. Baissac, J.F. Daniel, A. Jalloul, J.L. Montillet, J.P. Geiger, K. Assigbetsé and M. Nicole 2000. Salicylic acid mediated by the oxidative burst is a key molecule in local and systemic responses of cotton challenged by an avirulent race of *Xanthomonas campestris* pv. *malvacearum. Plant Physiology* 122, 757–766.

Medeiros F.H.V., R.M. Souza, F.C.L. Medeiros, H. Zhang, T. Wheeler, P. Payton, H.M. Ferro and P.W. Paré 2011. Transcriptional profiling in cotton associated with *Bacillus subtilis* (UFLA285) induced biotic-stress tolerance. *Plant Soil* 347, 327–337.

Mei W., Y.S. Qin W. and Y. Zhu 2009. Cotton GhPOX1 encoding plant class III peroxidase may be responsible for the high level of reactive oxygen species production that is related to cotton fiber elongation. *Journal of Genetics and Genomics* 36, 141–150.

Miao W., X. Wang, C. Song, Y. Wang, Ren Y. and J. Wang 2010. Transcriptome analysis of *Hpa1Xoo* transformed cotton revealed constitutive expression of genes in multiple signalling pathways related to disease resistance. *Journal of Experimental Botany* 61, 4263–4275.

Ministry of Agriculture and Agrarian Reform (MAAR), 2010.

Cotton Conference - Directorate of Bureau of Cotton. Aleppo, Syria.

Montillet J.L., J.P. Agnel, M. Ponchet, F. Vailleau, D. Roby and C. Triantaphylides 2002. Lipoxygenase-mediated production of fatty acid hydroperoxides is a specific signature of the hypersensitive reaction in plants. *Plant Physiology and Biochemistry* 40, 633–639.

Moreira L.M., N.F. Jr. Almeida, N. Potnis, L.A. Digiampietri, S.S. Adi, J.C. Bortolossi, *et al*. 2010. Novel insights into the genomic basis of citrus canker based on the genome sequences of two strains of *Xanthomonas fuscans* subsp. *aurantifolii*. *BMC Genomics* 11, 238.

Narra P.H., C.S. Saripalli and J. Gopalakrishnan 2011. Host induced changes in plasmid profile of *Xanthomonas axonopodis* pv. *malvacearum* races. *African Journal of Biotechnology* 10, 2451–2454.

Ouédraogo S.L., D. Sanfo, I. Somda and B.C. Tiemtore 2009. Analyse de l'influence du fonds génétique, des conditions climatiques et du mode de protection phytosanitaire sur l'expression de la bactériose chez différentes variétés de cotonnier au Burkina Faso. *Tropicultura* 27, 31–34.

Page J.T., M.D. Huynh, Z.S. Liechty, K. Grupp, D. Stelly, A.M. Hulse, H. Ashrafi, A. Van Deynze, J.F. Wendel and JA. Udall 2012. Insights into the evolution of cotton diploids and polyploids from whole-genome re-sequencing. *G3* (Bethesda) 3, 1809–1818.

Park C.U. 1997. *Temporal and spatial patterns of a phytoalexin biosynthetic enzyme and autofluorescence in bacteria-inoculated cotton*. PhD thesis. Oklahoma State Univ. Stillwater, 91 pp.

Paterson A.H. 2009. *Genetics and Genomic in cotton. Plant Genetics/Genomics*, vol. 3. Springer Ed, 510 pp.

Paterson A.H. and R.H. Smith 1999. Future horizons, biotechnology for cotton improvement. In: *Cotton, Origin, History, Technology, and Production* (W.C. Smith, J.T. Cothren, ed.), John Wiley & Sons Inc., New York, 415–432.

Paterson A.H., D.M. Stelly, J.F. Wendel and X. Zhao 1996. Status of genome mapping in the *Malvaceae* (cottons). In *Genome Mapping of Plants* (A.H. Paterson, ed.), RG Landes Company, Austin, TX, 229–242.

Patil M., M.E. Pierce, A. Phillips, B. Venters and M. Essenberg 2005. Identification of genes up-regulated in bacterial-blight-resistant upland cotton in response to inoculation with *Xanthomonas campestris* pv. *malvacearum*. *Physiological and Molecular Plant Pathology* 67, 319–335.

Pierce M. and M. Essenberg 1987. Localization of phytoalexins in fluorescent mesophyll cells isolated from bacterial blight-infected cotton cotyledons and separated from other cells by fluorescence-activated cell sorting. *Physiological and Molecular Plant Pathology* 31, 273–290.

Pierce M.L., E.C. Cover, P.E. Richardson, V.E. Scholes and M. Essenberg 1996. Adequacy of cellular phytoalexin concentrations in hypersensitively responding cotton leaves. *Physiological and Molecular Plant Pathology* 48, 305–324.

Pinto de Menezes I.P., F.A. Gaiotto, N.D. Suassuna, L.V. Hoffmann and P.A.V. Barroso 2014. Susceptibility of *Gossypium mustelinum* populations to the main cotton diseases in Brazil. *Journal of Agricultural Science* 6, 39–47.

Putcha S., K. Barker and S. Allen 1998. *Biocontrol of cotton diseases*. In: *Proceedings* of the *9th Australian Cotton Conference*. Broadbeach, Queensland, Australia, 555–559.

Raghavendra V.B., L. Siddalingaiah, N.K. Sugunachar, C. Nayak and N.S. Ramachandrappa 2013. Induction of systemic resistance by biocontrol agents against bacterial blight of cotton caused by *Xanthomonas campestris* pv. *malvacearum*. *ESci Journal of Plant Pathology* 2, 59–69.

Razaghi A., N. Hasanzadeh and A. Ghasemi 2012. Characterization of *Xanthomonas citri* subsp. *malvacearum* strains in Iran. *African Journal of Microbiology Research* 6, 1165–1170.

Reinisch A.J., J.M. Dong, C.L. Brubaker, D.M. Stelly, J.F. Wendel and A.H. Paterson 1994. A detailed RFLP map of cotton, *Gossypium hirsutum* x *Gossypium barbadense*, chromosome organization and evolution in a disomic polyploid genome. *Genetics* 138, 829–847.

Rong J., C. Abbey, J.E. Bowers, C.L. Brubaker, C. Chang, P.W. Chee, T.A. Delmonte, X. Ding, J.J. Garza, B.S. Marler, C. Park, G.J. Pierce, K.M. Rainey, V.K. Rastogi, S.R. Schulze, N.L. Trolinder, J.F. Wendel, T.A. Wilkins, T.D. Williams-Coplin, R.A. Wing, R.J. Wright, X. Zhao, L. Zhu and A.H. Paterson 2004. A 3347-locus genetic recombination map of sequence-tagged sites reveals features of genome organization, transmission and evolution of cotton (*Gossypium*). *Genetics* 166, 389–417.

Rungis D., D. Llewellyn, E.S. Dennis and B.R. Lyon 2002. Investigation of the chromosomal location of the bacterial blight resistance gene present in an Australian cotton (*Gossypium hirsutum* L.) cultivar. *Australian Journal of Agricultural Research* 53, 551–560.

Saeedi Madani A., A. Marefat, K. Behboudi and A. Ghasemi 2010. Phenotypic and genetic characteristics of *Xanthomonas citri* subsp. *malvacearum*, causal agent of cotton blight, and identification of races in Iran. *Australasian Plant Pathology* 39, 440–445.

Salaheddin K., V. Valluvaparidasan, D. Ladhalakshmi and R. Velazahan 2010. Management of bacterial blight of cotton using a mixture of *Pseudomonas fluorescens* and *Bacillus subtilis*. *Plant Protection Science* 46, 41–50.

Satyaa V.K., S. Gayathiria, R. Bhaskarana, V. Paranidharan and R. Velazhahan. 2007. Induction of systemic resistance to bacterial blight caused by *Xanthomonas campestris* pv. *malvacearum* in cotton by leaf extract from a medicinal plant zimmu (*Allium sativum L.* × *Allium cepa* L.). *Archives of Phytopathology and Plant Protection* 40, 309–322.

Sayegh-Alhamdia M., P. Marmey, A. Jalloul, A. Champion, A.S. Petitot, A. Clérivet and M. Nicole 2008. The lipoxygenase response is associated with resistance of various cotton genotypes to the bacterial blight disease. *Journal of Phytopathology* 156, 542–549.

Schaad N.W., E. Postnikova, G. Lacy, A. Sechler, I. Agarkova, P.E. Stromberg, V.K. Stromberg and A.K. Vidaver 2006. Emended classification of xanthomonad pathogens on citrus. *Systematic and Applied Microbiology* 29, 690–695.

Schaad N.W., E. Postnikova, G. Lacy, A. Sechler, I. Agarkova, P.E. Stromberg, V.K. Stromberg and A.K. Vidaver 2007. List of new names and new combinations previously effectively, but not validly, published. Validation list no. 115. *International Journal of Systematic and Evolutionary Microbiology* 57, 893–897.

Schnathorst W.C., P.M. Halisky and R.D. Martin 1960. History,

distribution, races and disease cycle of *Xanthomomas malvacearum* in California. *Plant Disease Reporter* 44, 603–608.

Senchina D.S., I. Alvarez, R.C. Cronn, B. Liu, J. Rong, R.D. Noyes, A.H. Paterson, R.A. Wing, T.A. Wilkins and J.F. Wendel 2003. Rate variation among nuclear genes and the age of polyploidy in *Gossypium*. *Molecular Biology and Evolution* 20, 633–643.

Shelke G.V., L.P. Aurangabadkar, A.R. Kashikar, S.R. Wadyalkar, M.S. Phalk, H.H. Kharkar and G.V. Umalkar 2012. Identification of resistance source for bacterial blight disease caused by *Xanthomonas axonopodis* pv. *malvacearum* and it's genetic inheritance in upland cotton. *Cotton Research Journal* 6, 167–173.

Shi J., L. Zhang, H. An, C. Wu and X. Guo 2011. *GhMPK16*, a novel stress-responsive group D MAPK gene from cotton, is involved in disease resistance and drought sensitivity. *BMC Molecular Biology* 12, 22.

Silva R.A., P.A.V. Barroso, L.V. Hoffmann, M. Giband and W.C. Coutinho 2014. A SSR marker linked to the B 12 gene that confers resistance to race 18 of *Xanthomonas axonopodis* pv. *malvacearum* in cotton is also associated with other bacterial blight resistance gene complexes. *Australasian Plant Pathology* 43, 89–91.

Sivakumar, S.R. 2014. Antibacterial potential of white crystalline solid from red algae *Porteiria hornemanii* against the plant pathogenic bacteria. *African Journal of Agricultural Research* 9, 1353–1357.

Taliercio E., R.D. Allen, M. Essenberg, N. Klueva, H. Nguyen, M. Patil, P. Payton, A.C.M. Millena, A.L. Phillips, M.L. Pierce, B. Scheffler, R. Turley, J. Wang, D. Zhang, J. Scheffler 2006. Analysis of ESTs from multiple *Gossypium hirsutum* tissues and identification of SSRs. *Genome* 49, 306–319.

Tan H., F.E. Callahan, X.D. Zhang, M. Karaca, S. Saha, J.N. Jenkins, R.G. Creech and D.P. Ma 2003. Identification of resistance gene analogs in cotton (*Gossypium hirsutum* L.). *Euphytica* 134, 1–7.

Thiers H.D. and L.M. Blank 1951. A histological study of bacterial blight of cotton. *Phytopathology* 1, 499–510.

Verma J.P. 1986. *Bacterial Blight of Cotton. Boca Raton*. CRC Press, Inc., FL, USA.

Voloudakis A., P. Marmey, A. Jalloul, E. Delannoy, C. Martinez and M. Nicole 2006. Expression of superoxide dismutase genes during interactions of cotton with *Xanthomonas campestris* pv. *malvacearum*. *Physiological and Molecular Plant Pathology* 68, 119–127.

Wallace T.P. and K.M. El-Zik 1989. Inheritance of resistance in three cotton cultivars to the HV1 isolate of bacterial blight. *Crop Science* 29, 1114–1119.

Wang Y.H., G. Davila-Huerta and M. Essenberg 2003. 8-Hydroxy-(+)-*d*-cadinene is a precursor to hemigossypol in *Gossypium hirsutum*. *Phytochemistry* 63, 219–225.

Wang K., Z. Wang, F. Li, W. Ye, J. Wang, G. Song, Z. Yue, L. Cong, H. Shang, S. Zhu, S. Zou, Q. Li, Y. Yuan, C. Lu, H. Wei, C. Gou, Z. Zheng, Y. Yin, Z. Zhang, K. Liu, B. Wang, S. Song, N. Shi, R.J. Kohel, R.G. Percy, J.Z. Yu, Y. Zhu, J. Wang and Y. Shuxun 2012. The draft genome of a diploid cotton *Gossypium raimondii*. *Nature Genetics* 44, 1098–1103.

Wasternack C. and B. Hause 2013. Jasmonates, biosynthesis, perception, signal transduction and action in plant stress response, growth and development. An update to the 2007 review in Annals of Botany. *Annals of Botany* 111, 1021–1058.

Wei H., W. Li, X. Sun, S. Zhu and J. Zhu 2013. Systematic analysis and comparison of nucleotide-binding site disease resistance genes in a diploid cotton *Gossypium raimondii*. *Plos One* doi, 10.1371/journal.pone.0068435.

Wendel J.F. and R.C. Cronn 2003. Polyploidy and the evolutionary history of cotton. *Advances in Agronomy* 78, 139–186.

Wright R.J., P.M. Thaxton, K.M. El-Zik and A.H. Paterson 1998. D-subgenome bias of *Xcm* resistance genes in tetraploid *Gossypium* (cotton) suggests that polyploid formation has created novel avenues for evolution. *Genetics* 149, 1987–1996

Xiao J., D.D. Fang, M. Bhatti, B. Hendrix and R. Cantrell 2010. A SNP haplotype associated with a gene resistant to *Xanthomonas axonopodis* pv. *malvacearum* in upland cotton (*Gossypium hirsutum* L.). *Molecular breeding* 25, 593–602.

Yang Y., R. De Feyter and D.W. Gabriel 1994. Host-specific symptoms and increased release of *Xanthomonas citri* and *X. campestris* pv. *malvacearum* from leaves are determined by the 102-bp tandem repeats of *pthA* and *avrb6*, respectively. *Molecular Plant-Microbe Interactions* 7, 345–355.

Yang Y., Q. Yuan and D.W. Gabriel 1996. Watersoaking function(s) of *Xcm*H1005 are redundantly encoded by members of the *Xanthomonas avr/pth* gene family. *Molecular Plant-Microbe Interactions* 9, 105–113.

Yu J.Z., R.J. Kohel, D.D. Fang, J. Cho, A. Van Deynze, M. Ulloa, S.M. Hoffman, A.E. Pepper, D.M. Stelly, J.N. Jenkins, S. Saha, S.P. Kumpatla, M.R. Shah, W.V. Hugie and R.G. Percy 2012. A high-density simple sequence repeat and single nucleotide polymorphism genetic map of the tetraploid cotton genome. *G3 (Bethesda)* 2, 43–58.

Yu J.S., S. Jung, C.H. Cheng, P.S. Ficklin, T. Lee, P. Zheng, D. Jones, R.G. Percy and D. Main 2013. CottonGen, a genomics, genetics and breeding database for cotton research. *Nucleic Acid Research* 42, 1229–1236.

Zachowski M.A. and K. Rudolph 1989. Characterization of *Xanthomonas campestris* pv. *malvacearum* (angular leaf spot of Cotton) isolates from Turkey and resistance tests of Turkish cotton cultivars. *Phytopathologia Mediterranea* 28, 10–15.

Zachowski M.A., Ö. Cınar and K. Rudolph 1990. Race identification of *Xanthomonas campestris* pv. *malvacearum* isolates in Turkey and reaction of Turkish cotton cultivars against several races. *Journal of Turkish Phytopathology* 19, 53–61.

Zhai J., Y. Luo, D. Zheng and X. Huang 2010. Evaluation of genetic diversity of highly virulent strains of *Xanthomonas campestris pv. malvacearum* by rep-PCR Fingerprinting. 2010. *Journal of Phytopathology* 158, 764–768.

Zhai J., Z. Xia, W. Liu, X. Jiang and X. Huang 2013. Genomic sequencing globally identifies functional genes and potential virulence-related effectors of *Xanthomonas axonopodis* pv. *malvacearum*. *European Journal of Plant Pathology* 136, 657–663.

Zhang J., M.E. Mace, R.D. Stipanovic and A.A. Bell 1993. Production and fungitoxicity of the terpenoid phytoalexins in cotton inoculated with *Fusarium oxysporum* f. sp. *vasinfec-*

tum. *Journal of Phytopathology* 139, 247–252.
Zhang H.B., Y. Yaning Li, B. Wang and P.W. Peng Chee 2008. Recent Advances in Cotton Genomics. *International Journal of Plant Genomics* ID 742304, 20 pp. doi, 10.1155/2008/742304.
Zomorodian A. and K. Rudolph 1993. *Xanthomonas campestris* pv. *malvacearum*, cause of bacterial blight of cotton. In: *Xanthomonas* (J.G. Swings, E.L. Civerolo, ed.), Chapman and Hall, London, UK, 25–30.

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