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 longi-caudatus*) (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 $1–1.2 \times 0.7–0.9$ 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
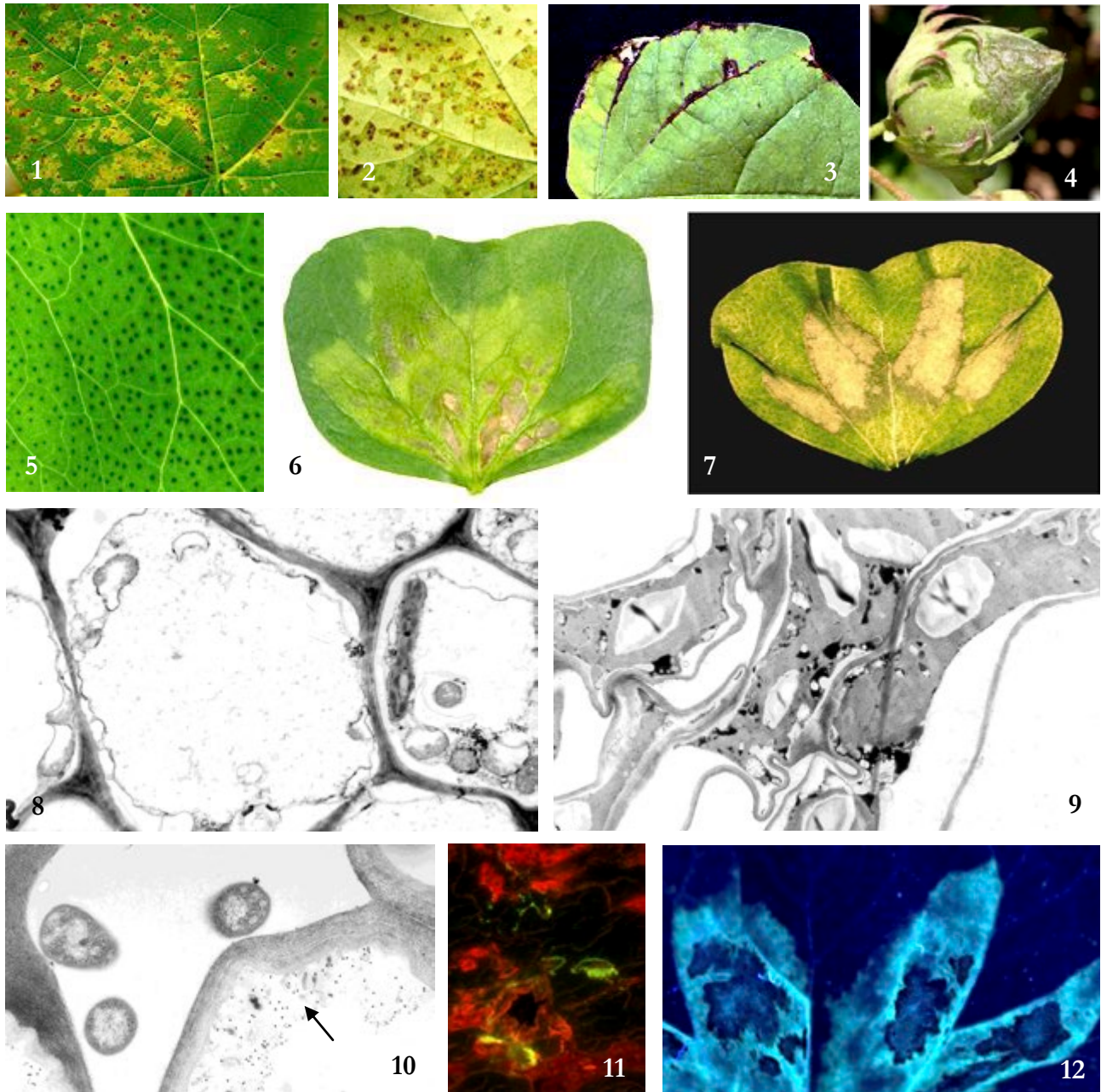
Figure 1. Symptoms of cotton bacterial blight on leaves showing angular leaf spots (1), water-soaked lesions (2), stem black arm lesions (3), fruit necrosis and gummosis (4). Susceptible cotyledons artificially infected displayed chlorosis and necrosis (6) as compared to healthy tissues in non-infected 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
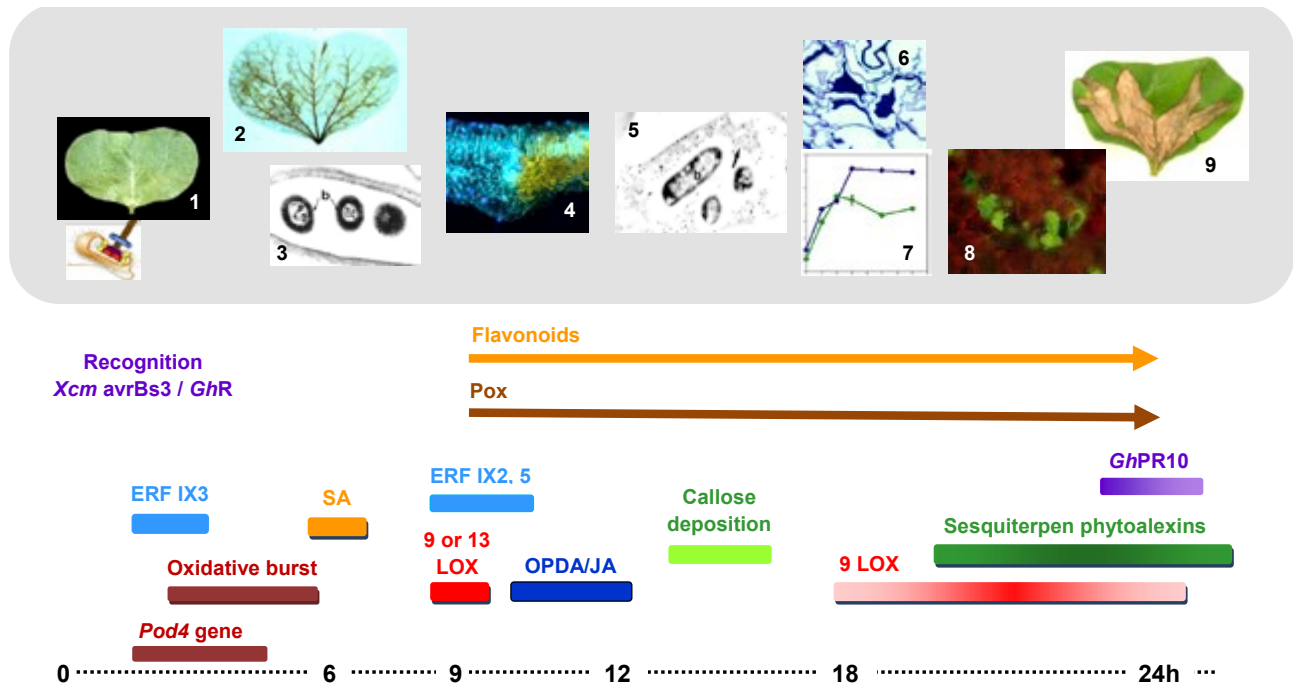
Figure 2. Time sequence of physiological events involved in the cotton HR to *Xanthomonas citri* pv. *malvacearum*. Following penetration of leaves by the pathogen (1 and 3), bacterial cells injected the effectors within the host nuclei. When recognized by host R proteins, several specific mechanisms 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-inocula-

tion (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 bacte-

rial 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, agro-nomical 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.

Acknowledgements

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