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REVIEW

Bacterial blight resistance in cotton: genetic basis and molecular mapping

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Abstract Bacterial blight (BB, caused by *Xanthomonas citri* pv. *malvacearum*, Xcm) is a worldwide disease of cotton (*Gossypium* spp.). The disease has been effectively controlled through the use of BB resistant cultivars and planting of acid-delinted seed. However, a resurgence of BB has been noted in the US in recent years due to the spread of Xcm race 18 and growing of susceptible transgenic cultivars, which calls for a renewed effort to develop new BB resistant cultivars. However, there has been a paucity of information in genetics and breeding for BB resistance since the 1990s due to the lack of research efforts. This review was prepared to fill this void with an objective to provide detailed results from past qualitative and quantitative genetic studies on BB resistance, including genetic designs and specific germplasm used for conducting research. More than 20 major resistance *B* genes (B_1 to B_8, B_{9K}, B_{9L}, B_{10K}, B_{10L}, B_{11}, B_{12}, B_{In}, B_n, B_s, and more than 4 unnamed genes), with at least two polygene complexes (B_{Sm} and B_{Dm}), have been identified. One *B* gene may be resistant to a single or multiple Xcm races, and pyramiding of several *B* genes can enhance resistance to a single or multiple Xcm races. Allelic relationships among some of the genes are currently unknown. Quantitative genetics has been employed to estimate heritability, gene effects, additive and dominance variances, and effective number of genes for BB resistance. The studies suggest that the additive effect and additive variance play a predominant role in BB resistance, while the dominant effect and variance play a reduced role in resistance. Heritability estimates are moderate to high depending on environmental errors, and 1–2 effective numbers of genes have been estimated, consistent with Mendelian genetic studies. Studies in molecular mapping of several BB resistance genes (B_2, B_3, b_6, and B_{12}) have been conducted with the focus on B_{12} as it is resistant to races 1 through 19. Portable DNA markers have been developed and used in marker-assisted selection for BB resistance. Finally, areas where there is a lack of information and controversies are identified and assessed. This review provides an updated comprehensive account of the genetic basis for BB resistance in cotton.

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Introduction

Bacterial blight (BB, caused by *Xanthomonas citri* pv. *malvacearum*, Xcm) has been historically one of the most devastating cotton (*Gossypium* spp.) diseases worldwide. The development of BB resistant cultivars in the 1970's and planting of acid-delinted seed has effectively controlled this disease. However, its resurgence has been noted in the US in recent years due to use of BB susceptible cultivars. Popular transgenic Bollguard (with a single Bt gene) cultivars were resistant to BB (including DP 555BR, DP 444BR, and ST 5599BR), but when the single gene Bt trait was replaced with Bollguard II (with two Bt genes), then the majority of U.S. cotton was planted to cultivars susceptible to BB (Phillips et al. 2017; Wheeler 2018). Seed companies have recently placed a higher emphasis on BB resistant cultivars, and as a result, BB has also declined (Wheeler 2020).

Evolution of Xcm races mirrored the development of cotton cultivars with new genes for resistance until the virulent race 18 evolved. Race 18 of Xcm is commonly found in most of the cotton producing regions of the world (Allen and West 1991; Akello and Hillocks 2002; de Sousa Braga et al. 2016; Hussain 1984; Saeedi Madani et al. 2010; Sampath Kumar et al. 2018; Shelke et al. 2012; Thaxton et al. 2001; Zachowski and Rudolph 1988). In a survey of cotton seed in northern Nigeria, only races 1, 12, 13, and 16 were found (Ajene et al. 2014). Race 19 was identified in Brazil (Ruano and Mohan 1982). Highly virulent strains of Xcm in central Africa in the 1970s and 1980s were identified that represent race 20 (Follin et al. 1988). The potential epidemic of this resurgent disease calls for a clear understanding of the genetic basis in resistance to BB in cotton. Although several reviews can be found in articles of Brinkerhorff (1970) and Innes (1983) and book chapters from Verma (1986) and Hillocks (1992), no review with updated knowledge has been published since then. Since most of these reviews and research papers were published in the 1930s to 1980s, it is difficult to obtain their full papers. It is important to understand the history and current affairs of breeding and genetics for BB resistance for identification of new resistance sources carrying different resistance genes and development of research and breeding strategies. This review is to provide a comprehensive summary on the genetic and molecular basis of BB resistance.

Qualitative genetics

In a series of ground-breaking work in Sudan between the 1930s and the 1950s, Knight transferred BB resistance from Upland (*G. hirsutum* L.) and diploid *G. arboreum* L., *G. herbaceum* L. and *G. anamalum* Waw. & Peyr. to Egyptian (*G. barbadense* L.) Sakel in Sudan and identified 10 major resistance genes. These genes include B_1, B_2, and B_7 from Upland, B_2, B_3 and B_{10} from *G. hirsutum* race *punctatum*, B_4 and B_6 from *G. arboreum*, B_5 from a perennial *G. barbadense*, recessive gene b_8 from *G. anomalum*, and B_9 from *G. herbaceum*. Several of these genes were also transferred to commercial Upland cultivars in Sudan. In the US, breeding and genetic studies in BB resistance were focused primarily on Upland cotton between the 1940s and 1960s. The efforts resulted in identification of several major *B* genes including B_7 (designated later by Knight), B_{12}, B_{In}, B_N and B_S, and several polygene modifiers or complexes. The following is a detailed summary of these research activities and major results (Table 1).

B_1 and B_2

Harland (1932) was perhaps first to study the segregation of BB resistance in cotton using crosses between Egyptian and Sea-Island cotton while working in a British cotton research station in Trinidad (West Indies). He noted that the resistance in F_1 was intermediate, while segregation in F_2 produced a series of gradations in resistance ranging from more susceptible than the susceptible Egyptian to more resistant than the resistant parent. However, most *G. barbadense* germplasm lines were susceptible, and the resistance levels in some lines were low. However, ground-breaking work on BB resistance did not begin until after Knight (Knight and Clouston 1939) at a British cotton research station in Sudan published results from a study on the genetic basis of BB resistance in a cross of resistant Upland Uganda B31 with susceptible *G. barbadense* X 1530 and N.T. 2,

Table 1 Major *B* genes and polygene complexes for bacterial blight resistance in cotton

Gene symbol	Action of the gene	Resistance source	References
B_1	Weak, dominant	Uganda B31 (*Gossypium hirsutum*)	Knight and Clouston (1939)
B_2	Strong, dominant	Uganda B31 (*G. hirsutum*)	Knight and Clouston (1939)
		Albar (West Africa), UKBR (Tanzania) (*G. hirsutum*)	Innes (1965a, b, c, d, 1969)
		Certain US Upland cotton	Brinkerhoff et al. (1979)
B_3	Partially dominant	Schroeder 1306 (*G. hirsutum* var. *punctatum*)	Knight (1944)
B_4	Partially dominant	Multani strain NT 12/30 (diploid *G. arboreum*)	Knight (1948)
B_5	Partially dominant	Grenadine White Pollen (a perennial *G. barbadense*)	Knight (1950)
B_6/b_6	Recessive	Multani strain NT 12/30 (*G. arboreum*)	Knight (1953a, b)
		Mwanza local UKBR61/12 (Tanzania) (*G. hirsutum*)	Saunders and Innes (1963) and Innes (1969)
B_7/b_7	Recessive	Stoneville 20 (*G. hirsutum*)	Blank (1949) and Knight (1953a, b)
	Dominant		Green and Brinkerhoff (1956)
			Innes and Brown (1969)
B_8/b_8	Recessive	A wild diploid *G. anomalum*	Knight (1954)
B_{9K}	Strong, dominant	Wagad 8 (cultivated diploid *G. herbaceum*)	Knight (1963) and Innes (1965a, b, c, d)
B_{9L}	Strong, dominant	Allen 51-296 (west Africa, *G. hirsutum*)	Lagiere (1960) and Innes (1965a, b, c, d)
B_{10K}	Weak, partially dominant	Kufra Oasis (Libya, *G. hirsutum*)	Knight (1963) and Innes (1965a, b, c, d)
B_{10L}	Weak, dominant	Allen 51-296 (west Affrica, *G. hirsutum*)	Lagiere (1960) and Innes (1965a, b, c, d)
B_{11}	Weak	Wagad 8 (cultivated diploid *G. herbaceum*)	Innes (1966)
B_{12}	Strong, dominant	S295 (Africa, *G. hirsutum*)	Follin et al. (1988) and Wallace and El-Zik (1989)
B_{In}	Dominant	Unknown cultivar (*G. hirsutum*)	Green and Brinkerhoff (1956)
B_N	Dominant	A resistant selection from Northern Star (*G. hirsutum*)	Green and Brinkerhoff (1956)
B_S	Dominant	A resistant selection from Stormproof 1 (*G. hirsutum*)	Green and Brinkerhoff (1956)
B_{SM}	Polygene complex	Stoneville 2B and Empire (*G. hirsutum*)	Bird and Hadley (1959)
B_{DM}	Polygene complex	Deltapine (*G. hirsutum*)	Bird and Hadley (1959)
$B_?$	Dominant	A Chinese Indigenous diploid variety (*G.herbaceum*)	Innes (1965a)
$B_?$	Dominant	An irradiated mutant in Westburn 70 (*G. hirsutum*)	Brinkerhoff et al. (1979)
$B_?$	Partially dominant	Indian *G. hirsutum*	Singh et al. (1987)
$B_?$	Dominant	C2 (67) 577 and C2 (69) 1455 in Pakistan (*G. hirsutum*)	Sajjad et al. (2007)

both selected from Sudan Sakel. They used artificial inoculation and rated > 25,000 plants in F_1, F_2, BC_1F_1, and many families from BC_2F_1, BC_3F_1, BC_1F_2, BC_2F_2, BC_3F_2, and BC_2F_3 using a rating scale of 0 for immunity to 12 for the most susceptible plants, similar to the susceptibility of *G. barbadense* Sakel (11 was omitted in the rating system). This comprehensive genetic study was similarly employed in his many follow-up studies leading to the identification of 10 resistant *B* genes. A typical 15R (resistant):1S

(susceptible) ratio in F_2 and many families in backcrosses were observed; so was a typical 3R:1S ratio in BC_1F_1 and many families in BC_2F_1 and BC_3F_1, and a 1R:1S ratio in backcrossing families from plants selected for backcrossing to the susceptible parent. Thus, two major resistance genes B_1 and B_2 were identified and assigned gene designations. The two susceptible Sakel lines (*G. barbadense*) had the highest grade 12 and were assigned the $b_1b_1b_2b_2$ genotype, while the resistant Uganda B31 (*G. hirsutum*) was assigned the $B_1B_1B_2B_2$ genotype. Both B_1 and B_2 were dominant, but B_1 was weak in its resistance and conferred a grade 10.1 resistance, while B_2 was strong in resistance with a grade 6 to 7 resistance. However, B_2 itself did not provide adequate protection against BB. The combination of B_1 and B_2 in either the heterozygous or homozygous condition produced a resistance grade of 5 to 6 in the Sakel background. In addition, they noted a close correlation between leaf and stem resistance (r = 0.50–0.63). As a result of backcrossing, the BB resistance genes were successfully transferred from Upland to Sakel, both separately and in single and two-gene combinations while maintaining the spinning quality (Knight 1944). The two genes were accumulative, but in the Sakel background, the two genes did not give the same level of resistance as in Upland B31, suggesting that a modifier complex was lost during the transfer.

Knight (1947) further showed that B_1 was closely linked to (or possibly identical to) a recessive dwarf gene, designated d_a, which produced a 'dwarf-bunched' phenotype in conjunction with another duplicate recessive gene d_b. The B_2 gene in Uganda B31 was also detected in crossing with susceptible Upland (Knight and Clouston 1941). A number of other Uplands also possessed B_2, such as a Punjab Upland selection "513" (Knight and Clouston 1941), Nigerian Allen selections- Albar 49 and its selection Bar 17/1, Albar 51 and its selection Albar 3 MB (giving BA 195/61), Bar 11/5, Bar 11/7 (Innes 1963a), Mwanza Local selections (59/567) and UKBR61/12 from Tanzania (Innes 1965a, 1969), and certain cultivars from the US (Brinkerhoff 1970). Innes (1963b) first suspected that the superior resistance of the Albar derivatives- Bar 17/1 and BA 195/61, compared with that of Bar 11/5 (with B_2B_2) and Bar 11/7 (with $B_2B_2B_3B_3$), was attributed to the presence of additional modifier and minor genes in combination with B_2. Although the presence of B_3 was not ruled out, the effect of B_3 can be difficult to detect in the presence of B_2. However, Innes (1965a) later suggested that the Albar derivatives carried the major gene B_2, and possibly B_3, as widely assumed due to the result of introgression from *G. hirsutum* var. *punctatum*. Interestingly, several *G. barbadense* also possessed B_2 including a Sea-Island origin BAR 898, B 181, NT14 and RU 4 derived from U 4, and SP 84 R heterozygous for B_2, and the resistance in some of the genotypes might have been derived from Upland cotton (Knight 1944; Knight and Hutchinson 1950).

B_3

Knight (1944) discovered that BAR3, a strain of *G. hirsutum* var. *punctatum* exhibiting a grade 1–2 resistance (0 = immunity; 12 = full susceptibility) possessed two linked resistance genes, B_2 and B_3. Gene B_3 was a partially dominant gene which conferred a grade 7–8.1 resistance when heterozygous and a grade 4.1–7.1 resistance when homozygous in the *G. barbadense* Sakel background. B_2 and B_3 were additive, in that $B_2b_2B_3b_3$ and $B_2B_2B_3b_3$ plants exihibited a resistance grade of about 4, and $B_2B_2B_3B_3$ and $B_2b_2B_3B_3$ showed a grade 3 resistance. B_2 and B_3 were linked at a recombination frequency of 0.324 (Knight 1944). The B_2B_3 genotype is common in *punctatum*, as three other *punctatum* strains-Gambia Native, Hindi Weed and Darfur Local also possessed B_2B_3. Through backcrossing and selections, B_1, B_2, and B_3 were transferred to the Sakel background individually or in multi-gene combinations, lending the ability to detect individual or accumulated effects of the three *B* genes, and to use them in allelic tests with other sources of BB resistance. Upland BAR 7/8 and BAR NT 96 possessed B_2 and B_3 and were commercialized in Sudan. Many selections from Nigerian Allen also carried B_2B_3 (Innes 1963b, 1965a, 1969).

B_4

Since complete immunity to BB was found in the two Old World cultivated A genome diploid species *G. arboreum* and *G. herbaceum*, genetic studies and transfer of this immunity to New World tetraploid cotton species was undertaken by Knight (1948). Through repeated backcrossing up to BC_5, the immune (grade 0) Multani (Sangttineum) strain NT

12/30 (belonging to race *bengalense* of *G. arboreum*) with colchicine doubled chromosomes was crossed to susceptible Sakel (*G. barbadense*). Resistance in the F_1 (with a synthesized AAAD genome) ranged from 1 to 5 with an average of 2.6, indicating incomplete dominance for resistance. BC_4F_2 and BC_5F_3 from fertility restored heterozygous resistant BC_4F_1 or BC_4F_2 (grade 6–9) gave a 3R:1S ratio, and the BC_5F_1 from backcrossing the heterozygous resistant BC_4F_1 or BC_4F_2 to Sakel segregated in a 1R:1S ratio, as expected for a major gene, designated B_4. B_4 is non-allelic to B_1, B_2, or B_3 but additive to B_2 and B_3. However, no immune plants were observed, indicating that other resistance genes in the immune diploid parent were lost during the interspecific backcrossing process. In diploid F_2 and backcross progenies of a cross between NTI2/30 and a susceptible semi-wild Sudan type (race *soudanense* of *G. arboreum*) Nuba Red, Knight (1948) confirmed that the immunity in Multani cotton depends on the major B_4 gene, accompanied by a strong complex of minor genes.

B_5

Knight (1950) showed that two forms of BB resistance occurred in *G. barbadense*: weak resistance as represented by cultivated Sea Island lines BA 1–1, BA 1–5, and BA 1–14 from St Vincent, Montserrat, and Barbados, respectively, with resistance grades of 7–10; and strong resistance as represented by perennial Grenadines White Pollen (BP 1–1) with a resistance grade of 5–7. The F_2 derived from the most resistant F_1 from crossing the above three resistant Sea Island lines with the susceptible Sakel were all as susceptible as Sakel, and resistance was not recovered, indicating that no major gene was present in the three lines. However, the resistance in Grenadines White Pollen was due to the presence of B_5 fortified by minor genes. B_5 was found to be variable in expression but, in general, the homozygotes (5–6 grade) expressed stronger resistance than the heterozygotes (6–8 grade). B_5 was independent of B_1 (8–9 grade), B_2 (5–6 grade), B_3 (5–6 grade), and B_4 (6–7) in crosses between Grenadines White Pollen (possessing B_5) and four different Sakel lines, each with one of the other four *B* genes. B_5 was additive in its effects when in combination with these other four *B* genes, respectively, but non-allelic, as 15R:1S ratios were observed in F_2 populations.

B_6

Knight (1953a) initially discovered B_{6m} in plants from a BC_2 progeny with a resistance grade 3 (better than other progeny) when he was transferring B_4 from Multani strain NTI2/30 (*G. arboreum*) to Sakel (*G. barbadense*). However, the progeny was found to be an outcross with an unknown line possessing B_2. Further backcrossing of these resistant plants to susceptible Sakel and X1730A produced 1 (B_2B_{6m}):1 (B_2b_{6m}):2 (b_2B_{6m} and b_2b_{6m}). To verify B_{6m} was indeed from the *G. arboreum* parent, the diploid parent with chromosome doubling was again crossed and backcrossed with Sakel. Since B_{6m} did not exact any effect on BB resistance in the absence of other *B* genes, individual BC_2 plants with grade 12 were crossed with a Sakel line BLR 14/16 possessing B_2. The individual F_1 plants were then crossed with the susceptible Sakel, which gave a ratio of 1 (B_2B_{6m}) (3–5 grade):1 (B_2b_{6m}) (5–7 grade):2 (b_2B_{6m} and b_2b_{6m}) (grade 12). B_{6m} increased the resistance of B_2 by approximately two grades. B_{6m} in combination with B_2 and B_3 conferred a resistance closely approaching immunity in the Sudan Sakel background (*G. barbadense*).

In Sudan, however, Innes (1962) later isolated B_{6m} in the Sakel background alone and showed that the resistance of Sakel with B_{6m}, was similar to that of B_2 when inoculated with Xcm by foliar spraying and vein inoculation, suggesting similar mechanisms for the two genes. Saunders and Innes (1963) further showed that B_{6m} was in fact a recessive resistance gene of moderate effect when homozygous (b_6b_6, 7–9 grade), as the progeny (F_2 and F_3) of heterozygotes produced a ratio of 1R (b_6b_6): 3S (B_6B_6 and B_6b_6, 10–12 grade). They then simplified the gene symbol to B_6, but the resistance allele should be named as b_6 due to its recessive nature. B_6 was additive in its effects when combined with B_2, B_3, B_4 or B_5 but not with B_1 or B_7. B_6 with B_2 was the most effective combination, giving 3–6 grades, while B_2B_2/B_2b_2 or b_6b_6 alone gave 6–9 grades. Innes (1969) further showed that the B_6 gene was present in Mwanza Local UKBR61/12 and that the high resistance of subsequent selections 59/567 resulted from the interaction of B_2 with a gene, or a complex of genes occupying the same locus as or closely linked to B_6. The B_6-type gene was obtained by steady selection pressure to gradually increase BB resistance over years.

B_7

The American Upland Stoneville 20 cultivar, which was resistant to BB in leaves, stems and bolls, was selected from the susceptible cultivar Stoneville 2A (Simpson and Weindling 1946). This resistance was transferred to other susceptible Upland cultivars in the US through backcrossing, and it became a major resistance source used in many breeding programs across the country (Simpson and Weindling 1946). In 1946, Simpson indicated that the resistance in Stoneville 20 was inherited as a simple recessive character but its full expression required modifying or minor genes. Through crosses between Stoneville 20 and several susceptible Uplands, Blank (1949), in Texas, confirmed that the resistance was inherited as a single recessive gene, when the expected 1R:3S ratio was observed in F_2 populations and the progeny of segregating BC_1F_1 and BC_1F_2 plants. Green and Brinkerhoff (1956) in Oklahoma confirmed that resistance in Stoneville 20 was indeed controlled by a major recessive gene named b_7, but that segregation was obscured by other genes of lesser individual effect. In the US, Stoneville 20 was highly resistant to race 1 but only slightly resistant to race 2. Bird and Hadley (1959), in Texas, further studied four parents including Stoneville 20 and their F_1, F_2, F_3 and backcross progenies and demonstred that the dominance of BB resistance to race 1 and 2 in Stoneville 20 depended on the other parent used in a cross. In a cross of Stoneville 20 × highly resistant Deltapine, the resistance was dominant, while the resistance in a cross of Stoneville 20 × susceptible Acala was inherited as a recessive trait. In a cross of Stoneville 20 × Stoneville 2B with a low level of resistance, no dominance was observed. In the F_2 population, some plants were resistant to race 1 but susceptible to race 2 or vice versa, while other plants were resistant to both races. Based on a half diallel crossing involving six Upland parents in Africa, Innes et al. (1974) indicated that the behavior of the B_7 gene was not consistent with that of a simple Mendelian locus with incomplete dominance.

However, in an interspecific cross and backcrosses between Stoneville 20 (grade 6–7) and *G. barbadense* Sakel (grade 12) in Sudan, Knight (1953b) observed that resistance in the progeny was dominant with a 1R (grade 8–10):1S (grade 12) ratio in BC_1F_1 and a 3R:1S ratio in F_2 of BC_2, BC_3 and BC_4. Therefore he assigned B_7 for the resistance gene in Stoneville 20. He further showed that B_7 was non-allelic to B_1-B_6 because susceptible plants were observed in F_2 populations from crosses between Stoneville 20 and *G. barbadense* BAR2/11 (B_1B_1), BLR14/16 (B_2B_2), BAR14/9 (B_3B_3), BAR14/19 (B_4B_4) and BAR14/20 (B_5B_5). The testcross between F_1(Stoneville 20 × B_2-carrying BLR14/16) and Sakel showed 2 (4–7 grade):1 (8–9 grade):1 (12 grade) ratio and 15 (3–7 grade):1 (8–10 grade) ratio in F_2 of Stoneville 20 × B_2-carrying BAR 7/1. Based on the observation that the level of resistance in Stoneville 20 was reduced during the backcross process, Knight (1953b) suggested that the resistance in Stoneville 20 was due to the presence of the major gene B_7 accompanied by minor resistance genes.

b_8

Resistant wild diploid B genome *G. anomalum* (grade 2–4) was crossed with susceptible the A genome *G. arboreum* cultivar Java (grade 8–9), and then backcrossed to Java to transfer BB resistance to the cultivated diploid species (Knight 1954). Resistance was recessive, but the typical 1R:3S segregation ratio for BB responses was not observed in BC_1F_2, BC_1F_3, BC_1F_4, BC_2F_2 and BC_3F_2. Nevertheless, Knight (1954) assigned b_8 to the gene transferred from *G. anomalum* to *G. arboreum*, and further, he showed that b_8 was closely linked to R_2 (a petal spot gene on chromosome A07) at a recombination frequency of 1.4%. Because the behavior of b_8 was distinctively different from that of the other seven *B* genes, Knight suggested that b_8 was not allelic to any of them. It is unknown if this resistance was ever transferred into cultivated tetraploid cotton.

B_{9K} and B_{9L}

Through repeated backcrossing up to BC_7, Knight (1963) successfully transferred two partially dominant resistance genes from the Indian *G. herbaceum* cultivar Wagad 8 (after chromosome doubling) to Sakel (*G. barbadense*). The gene with a stronger resistance effect, designated B_9, conferred resistance ranging from grade 5–6 when homozygous, to grade 7–8 when heterozygous, and segregated in the typical 3R:1S ratio in the F_2. B_9 was non-allelic to other *B* genes, because when homozygous backcross

plants possessing B_9 were crossed with Sakel lines possessing B_2, B_3, B_4, B_5, or B_7, a typical 15R:1S ratio was observed in the F_2. The gene with a weaker level of resistance, when homozygous, gave a range of 7 to 8 grade (when heterozygous, it gave 9–12 grade). However, no gene symbol was assigned to this gene with the low level of resistance, because gene homology tests were not performed. Innes (1965a) later confirmed the presence of B_9 and minor genes in Wagad 8 following a cross to susceptible *G. herbaceum* var. *africanum* 'ET's.

Perhaps without knowing Knight's work on B_9 and B_{10}, Lagiere (1960), while working in French-speaking Africa, named B_9 and B_{10} to the two major BB resistance genes identified in resistant Upland Allen 51-296. Innes (1965a, b) confirmed that the high resistance in Reba W 296 (derived from Allen 51-296 and purported to be homozygous for B_9B_{10}) was due to the major gene B_9, and a number of minor genes with additive effects. However, the Lagière's B_9 was not homologous to Knight's B_9 and was also independent of other *B* genes, because susceptible plants (in a 15R:1S ratio, except B_6) were observed in F_2 populations of crosses between Bar 11/13 (a resistant line from the F_3 of Reba W296 × susceptible Sudan Upland XA 129) and Upland Bar lines each possessing B_1, B_3, B_5, B_6, or B_7. F_2 populations of crosses between Bar 11/13 and Sakel type Bar 14 lines (*G. barbadense*) each possessing a different *B* gene (B_1 to B_7, B_9 and B_{herb}) also produced a 15R:1S ratio. Thus, the symbols B_{9L} and B_{9K} were assigned to the two different resistance genes from the Lagiere and Knight sources, respectively (Innes 1965b). Innes (1966) observed that, while the leaf resistance of B_{9K} Sakel was high, its stem resistance was of a low level and suggested that leaf, stem and boll resistance may be under the same genetic control in some crosses but not in others.

B_{10K} and B_{10L}

In a plant protection conference held in London in 1956, Knight reported B_{10}, a partially dominant resistance gene identified in *G. hirsutum* var. *punctatum* Kufra Oasis in Libya (Knight 1957). However, no details could be found in the literature. Due to different sources of Knight's and Lagiere's B_{10}, Innes (1965b) proposed symbols B_{10L} and B_{10K} for the two B_{10} genes of the same sources as B_{9K} and B_{9L}, respectively, although allelic tests were not made.

B_{11}

Innes (1966) assigned the previously unnamed gene with a weaker resistance (designated B_{herb} by Innes 1965c) transferred by Knight (1963) from *G. herbaceum* Wagad 8 to Sakel as B_{11}, because it was non-allelic to other *B* genes based on gene homology tests between B_{11} and other *B* genes. He also confirmed the presence of strong environmental effect on the expression of B_{11} and noted that B_{11} was much more effective in the Upland background than in Sakel (*G. barbadense*), but the opposite was true of B_{9K}.

B_{12}

Crosses with Upland S295 in Paraguay and France resulted in segregations for BB resistance to race 18 and 20 (Follin et al. 1988). They concluded that resistance to each race was dominant and controlled by one gene and that Upland S295 (resistant to both races) possessed a major gene for race 18 and a minor gene for race 20, but the two genes were tightly linked. Wallace and El-Zik (1989) confirmed that the resistance in S295 to a mixture of US races (1, 2, 7, and 18) and African HV1 (race 20) was dominant and controlled by the same resistance gene, designated B_{12}, or two closely linked genes, from crosses with Tamcot CAMD-E (resistant to race 1, 2, 7 and 18 but susceptible to race 20) and Stoneville 825 (susceptible to all the above races). A 3R (1–3 grades):1S (4–10 grades) ratio in F_2 progenies and 1R:1S ratio in BC_1F_1 progenies were observed. The responses in cotyledons and true leaves were correlated, indicating that resistance in cotyledons and true leaves was controlled by the same genetic mechanism. This was further confirmed by Wright et al. (1998) based on an F_2 population derived from S295 × highly susceptible Pima S-7 (*G. barbadense*) and Xiao et al. (2010) using an intraspecific Upland population of resistant Delta Opal × susceptible DP388 infected with race 18.

B_{In}, B_n, and B_s

Green and Brinkerhoff (1956) in Oklahoma reported that the resistance in Upland breeding lines 1-10-B-4-B, 20-8-1-3-1, and 6-77-5-8 was controlled by single dominant genes, B_I, B_N, and B_s, respectively. Line 1-10-B-4-B was derived from a resistant plant of an unknown cultivar found in a farmer's field near

Indiahoma, OK, and the F_2 resulting from a cross with Stoneville 20 gave a 13R:3S ratio, indicating segregation for 1 dominant (designated B_1 from line 1-10-B-4-B) and 1 recessive gene (b_7 from Stoneville 20) for resistance. Line 20-8-1-3-1 was selected from Northern Star, and the F_2 resulting from a cross with 1-10-B-4-B (B_1) segregated in a 15R:1S ratio, indicating a different dominant gene (designated B_N) in 20-8-1-3-1. Line 6-77-5-8 was selected from Upland Stormproof No. 1, and its F_2 with 1-10-B-4-B (B_1) gave a 15R:1S ratio, indicating another different dominant gene, designated B_S. However, the F_2 of 6-77-5-8 (B_S) × 20-8-1-3-1 (B_N) did not give a 15:1 ratio but produced more resistant plants (595R:14S) than expected, and all F_3 plants were resistant. Results indicated that B_1 was independent of b_7, B_N and B_s, but B_N and B_S could be the same or closely linked gene. The authors stated that studies were in progress to clarify the relationship of the above genes; however, no follow-up reports were published. Therefore, their allelic relationships with one another and with other *B* genes are currently unknown. To avoid confusion with B_1, B_1 was later renamed as B_{1n}.

B_{Sm} and B_{Dm}

Bird and Blank (1951) showed that the Upland Deltapine cultivar had a higher degree of tolerance than two other Upland cultivars- Stoneville 2B and Acala. The F_2 progenies from a cross of Stoneville 20 with Deltapine displayed a higher degree of BB resistance than the F_2 offspring from a cross of Stoneville 20 with Acala, although both crosses inherited the same major gene (B_7) for resistance from Stoneville 20. This led them to suggest that the susceptible parents may possess different numbers of minor genes for resistance which influenced the degree of resistance produced by the major gene. In a follow-up study, Bird and Hadley (1959) showed that Stoneville 20 contained two effective genetic components determining its BB resistance: one is the major gene B_7 and the other a constellation of minor genes designated B_{Sm}, which was also found in Stoneville 2B. Deltapine, however, possessed another composite component B_{Dm}, while Acala had no effective resistant component. Therefore, the cultivars used in the study were assigned the following genotypes: Stoneville 20, $B_7B_7B_{Sm}B_{Sm}b_{Dm}b_{Dm}$; Deltapine, $b_7b_7b_{Sm}b_{Sm}B_{Dm}B_{Dm}$; Stoneville 2B, $b_7b_7B_{Sm}B_{Sm}b_{Dm}b_{Dm}$, and Acala, $b_7b_7b_{Sm}b_{Sm}b_{Dm}b_{Dm}$. However, the composite components in Stoneville 20 and Stoneville 2B may not be identical. The genes were additive and the effect of the major gene (B_7b_7) was greater and less influenced by environment in the presence of the two B_{-m} genes of the composite components than in the presence of one. There was no evidence of linkage between genetic factors controlling Stoneville 20 resistance.

Other unnamed BB resistance genes

In addition to B_9 and B_{11} in Wagad 8 (*G. herbaceum*), Innes (1965a) detected another major resistance gene in a Chinese indigenous diploid variety (*G. herbaceum*), and the gene was not homologous with B_9 from the same species. However, no symbol was given to this gene.

In his Ph.D. study, Owen (1967) in the US performed a genetic study of BB resistance in five resistant lines of American Upland cotton based on F_1, F_2, backcross and F_3 generations in the field and in growth chambers. Resistance was incompletely dominant in all five lines, and segregation results indicated one resistance gene in three lines, two genes in another line and several genes in the other line. There was also evidence for additional genes providing a greater degree of resistance. However, no follow-up studies were published. Brinkerhoff et al. (1979) in Oklahoma reported that the BB resistance in a moderately resistant mutation, induced by irradiating seed of a susceptible Westburn 70 with fission neutrons, was due to a single dominant gene, different from B_3, B_4, B_5, b_7, or B_N.

Based on the observations of seven resistant, moderately resistant and susceptible Upland parents and their F_1 and F_2 populations evaluated in the field, Singh et al. (1987) in India reported that resistance to BB was incompletely dominant and that the cultivars studied differed for two resistance loci. Sajjad et al. (2007) in Pakistan showed that the BB resistance to race 18 in two breeding lines—C2 (67) 577 and C2 (69) 1455, in crosses with susceptible DPL-7340-424, was dominant and controlled by a single resistance gene at both seedling and adult stages. The resistance gene in the two resistant lines was the same, as no susceptible segregants were observed in their F_2 and backcross progenies. However, it is unknown if the

gene was the same as B_{12}. Based on a testcross and F_2 population, Sajjad et al. (2003) showed that the gene for resistance to BB and the gene for resistance to cotton leaf curl virus were linked with a recombination frequency of 25.9–32.8%.

B gene combinations

Due to the fact that a single *B* gene was either ineffective in conferring BB resistance to a predominant Xcm race, or that new Xcm races developed rapidly to defeat single *B* genes, pyramiding of two or more B genes in one cultivar or line has been vital to breeding for BB resistance (Table 2). Knight developed different B gene combinations in his breeding and genetic work for BB resistance in Sudan. He showed that, in the *G. barbadense* Sakel background, B_2 gave a resistance of grade 5–7, and B_3 gave a 4–7 grade, but the B_2B_3 combination gave grade 3. Interestingly, the genes B_2B_3 were more effective in conferring BB resistance when transferred to an Upland genetic background than in the Sakel (*G. barbadense*) background. His work was continued by Innes in the 1960s. Innes (1964) compared BB resistance in F_2 families from diallel crosses involving Sakel homozygous for each of the B genes, B_1 to B_7, and showed that the best resistance was conferred by B_2B_6, closely followed by B_1B_4, B_2B_4, B_3B_6, and B_4B_6. Also exhibiting additivity, but conferring intermediate resistance was B_1B_3, B_2B_3, B_3B_4, B_3B_5, B_4B_5, B_4B_7, B_5B_6, and B_5B_7. B_1 and B_4 were transferred to the Upland Wilds Sus 16/1, which was found not to be absent of the d_b gene for dwarfing. B_7 was more effective when transferred to Acala 4-42 than in Wilds Sus 16/1. B_1 and B_5 were each able to increase the resistance of the Upland Wilds Sus 16/1 when transferred, but B_1 was more effective, demonstrating the importance of genetic background. In a set of diallel crosses between seven Bar lines of Sakel, each homozygous for one *B* gene in the series conferring BB resistance, Innes (1965d) showed that the

Table 2 *B* gene combinations and associated germplasm for bacterial blight resistance in cotton

Gene combination	Cultivar/line	Resistance to races	References
B_2B_3	Bar 14/25 and Barakat (Gb), Sudan	Field immunity in Sudan	Knight (1957)
	Bar 11/7 (Gh), Sudan	Widely grown in Sudan	
	Uganda Allen, BJA-592, BTK-12, MK-73	Race 1 to 19, susceptible to Race 20	
$B_2B_3B_{Sm}$	101-102B (Gh), US	All races in the US	Bird (1960)
B_2B_6	Barac (67) B, Bar 12/16, Bar 14/40, UK 77 (Gh)		Innes (1974)
B_2B_{9K}	Bar 14/60	As effective as B_2B_6 and $B_2B_3B_6$	Innes (1974)
$B_2B_3B_4$	(Gh), US	Immune to all known US races	Bird (1966)
$B_2B_3B_6$	Bar 14/48, VSI (Gb), Sudan	Complete immunity in Sudan	Knight (1957)
	Cascot L-7(Gh), US	US	
$B_2B_3B_{9L}$	Reba B50 from Allen x Stoneville (Gh)		
B_2B_7	Acala 1517BR2 (Gh), US	Race 1 and 2	
B_2B_{Sm}	Rex, Rex SL, Rex SL66 (Gh), US	Race 1	
$B_2B_3b_7$	Im-216 (Gh), US	Race 1 to 18 (all races in the US)	Brinkerhoff et al. (1984)
$B_2B_3B_7B_?$	Tamcot lines (Gh), US	Race 1 to 18 (all races in the US)	Bird (1976, 1979a, b)
$B_2B_3B_7B_4$	Cascot B-2 (Gh), US	US	
$B_2 + poly$	Albar 673		
$B_{9L}B_{10L}$	Allen Zaria (Gh)		
	Reba W-296, B-50, P-279 (Gh)		

Gh *Gossypium hirsutum*, Gb *G. barbadense*

combination B_2B_9 produced resistance as high as that given by B_2B_6. B_4B_6 and B_1B_9 also conferred high resistance. A cross between a line carrying B_6 and one having a weak resistance gene derived from *G. herbaceum*, designated B_{herb} (B_{11} later on) also showed high resistance. The $B_6 \times B_{herb}$ cross gave a high resistance rating but its F_2 generation showed two types of families with differing resistance, indicating that a third gene was probably present.

While breeding for BB resistance at Shambat, Sudan, Innes (1961) demonstrated that needle inoculation of the main vein of the leaf under greenhouse conditions was a useful supplementary procedure in transferring the modifier B_{6m}, both alone and in combination with B_2 (B_2B_{6m}). The mean lesion length from this inoculation technique was used to distinguish between homozygotes for B_2 and B_2B_3; however it could not differentiate between B_2 and B_2B_3 types in segregating progenies. Environmental changes also influenced the difference in mean lesion length between B_2 and B_2B_3 resistance, such that differences were detected at Shambat, but not at Wad Medani. In the field, B_2B_3 resistance is more effective than B_2 alone. Innes (1963b) observed the greatest loss of field BB resistance occurred in Bar 14/25 (B_2B_3-Sakel). The breeding lines B_2Sakel, Bar 14/16, and B_2B_3 Upland, and Bar 11/7 also showed decreased field resistance, but not the corresponding B_2 type Bar 1 and Knight's B_2B_3 *punctatum*; and Bar 3/5 also maintained its resistance. Bar 3/5 and Bar 11/7 differed only in modifier genes. Innes (1974) summarized his work in Sudan on inoculation experiments for a wide range of Egyptian cotton (*G. barbadense*) lines homozygous for single genes and for digenic and trigenic combinations of Knight's *B* genes for BB resistance. Although leaf inoculation was successful, stem inoculation was only partially so, and boll inoculation, using two different techniques, failed to produce measurable disease symptoms. There was a good general relationship between leaf and stem resistance, and a close association between resistance to natural attack in the field and leaf resistance to artificial inoculation. The strong resistance conferred by B_2B_{9K}, which was as effective as B_2B_6 or $B_2B_3B_6$, was confirmed. No other combination was as effective when inoculated artificially. Nevertheless, in a natural field infestation, only mild symptoms were found in lines homozygous for B_1B_{9K} and for B_4B_6. Under the same conditions, lines with B_2B_6 showed no symptoms, but those with B_2B_3 were severely attacked.

During the 1950s in Texas, Bird transferred *B* genes to different Upland cottons including Empire and Deltapine from Knight's *B* gene containing Sakel (*G. barbadense*) strains by backcrossing and inbreeding. In 1960, he developed an immune Upland line 101-102B (carrying $B_2B_3B_{Sm}$) through interspecific introgression (Bird 1960). He used Upland Empire WR (containing B_{Sm}) as the recurrent parent to cross and backcross for five generations with B_2B_3-containing Bar 4/16 (*G. barbadense*), followed by crossing to a possible b_7-containing Upland MVW. In each backcross, the most resistant plants were selected for further backcrossing after screening with a mixture of Xcm races. Immune plants were only observed after several backcrosses. Through this process, Bird developed many Tamcot lines with high levels of BB resistance possessing different *B* gene combinations, including $B_2B_3B_4$ and $B_2B_3B_4b_7$ conferring immunity against all known BB races in the US (e.g. Bird 1976, 1979a, b). In Oklahoma, an immune line, Im 216, was developed by Brinkerhoff (Brinkerhoff et al. 1984), as a selection from a segregating population of Bird's B_2B_3 Empire (one of the parental populations used to develop 101-102B), after several generations of inbreeding and selection for resistance to a mixture of races 1, 2, 4, and 10. The immunity of Im 216 was completely dominant and thought to be due to $B_2B_3B_7$, because the F_2 data from a cross between Im 216 and fully susceptible Acala 44 fitted a segregating ratio for two dominant and one recessive independently inherited genes. Several fully susceptible F_2 plants also showed segregation of resistance, indicating the existence of the recessive resistance gene b_7. However, the F_2 progeny also fitted a segregating ratio for two independent dominant genes.

El-Zik and Bird (1967) reported that the B_4 gene was the most effective factor, followed by B_2B_3, $B_2B_3B_7$, B_2B_{6m}, and B_7 when Upland cotton was inoculated with race 1, race 2, or their mixture. El-Zik and Bird (1970) confirmed that B_4 gave a higher level of BB resistance than did B_2, B_3, or B_7 against five BB races and was as effective as $B_2B_3B_6$. In the Empire background, B_4 was more effective than B_2B_6, B_2B_3, or $B_2B_3B_7$. However, B_4 may be not as effective in other genetic backgrounds or testing conditions. Essenberg et al. (2002) in Oklahoma reported the development and genetic characterization of four near-isogenic

lines (NILs) of Upland, each carrying one of the single homozygous BB resistance genes, B_2, B_4, B_{In}, or b_7. The NILs were derived from at least six backcrosses to the susceptible recurrent parent Acala 44, followed by single plant-progeny row selection for uniformity. In the Acala 44 background, B_2, B_4, and B_{In} are partially dominant genes, and b_7 is partially recessive. Resistance to race 1 was ranked as $B_4 \sim b_7 > B_{In} \sim B_2$. Essenberg et al. (2014) used these NILs to develop gene-pyramid lines with all possible combinations of two and three *B* genes. Isogenic Xcm strains carrying single avirulence (*avr*) genes were used to identify plants carrying specific resistance *B* genes. Under field conditions in north-central Oklahoma, pyramid lines exhibited broader resistance to individual races and, consequently, higher resistance to a race mixture. It was predicted that lines carrying two or three *B* genes would also exhibit higher resistance to race 1, which possesses many *avr* genes. However, they did not approach the level of resistance of Im 216. In a growth chamber evaluation, Im 216 (carrying $B_2B_3B_7$) exhibited considerably lower bacterial populations than any of the one- (such as B_4), two-, or three-*B*-gene (such as combinations with B_4) lines.

Genes conferring BB resistance may be associated with resistance to other diseases in cotton. Brinkerhoff and Hunter (1961) in Oklahoma first noted that BB resistant lines contained a much higher proportion of Fusarium wilt resistant plants than susceptible populations. However, the association of resistances to different diseases may be breeding population specific. Cauquil and Follin (1970) in Africa studied boll rot resistance in three American Empire WR genotypes and seven Central African Upland lines differing in BB resistance. Results suggested that lines possessing major genes for BB resistance (B_2B_3 and $B_2B_3B_{6m}$) exhibited greater resistance to fungal boll rot than lines lacking these genes. Resistance mechanisms associated with the pericarp were more effective in the $B_2B_3B_{6m}$ genotype than in the B_2B_3 genotype, indicating that the presence of the modifier gene B_{6m} in association with B_2B_3 further improved resistance to boll rots. However, the B_{6m} gene had less effect on resistance mechanisms located within the boll. Results showed that boll rot resistance located in the pericarp was greater when two major genes (B_2B_3 or B_9B_{10}) were present, while a single resistance *B* gene was ineffective. Bird (1972) confirmed that resistance to five diseases, including BB resistance, was interrelated with common resistance genes. Bird (1982) further reported that BB resistance had the strongest association with resistance to Fusarium wilt/root-knot nematode complex and a lower association with resistance to Verticillium wilt, Phymatotricum root rot and seedcoat resistance to mold. These studies led to the development of the multi-adversity resistance (MAR) program at Texas A&M University (Bird 1982, 1986), and the release of numerous MAR germplasm and cultivars (e.g. Bird 1979a, b; El-Zik and Thaxton 1996, 1997; Thaxton and El-Zik 2004). However, the relationship between resistance to BB and Fusarium wilt in MAR lines could not always be verified by independent studies. For example, Tamcot Sphinx, a MAR line with resistance to BB, was listed as moderately resistant to Fusarium wilt on its plant variety protection certificate (#009600134). In a Fusarium wilt infested field in Gaines county, TX, with moderate Fusarium wilt and high root-knot nematode pressure, this cultivar had Fusarium wilt symptoms that were more severe than any other cultivar tested (Wheeler and Gannaway 1998). Major resistance genes for BB and quantitative resistance genes for other pathogens, were presumably pyramided in some of the MAR germplasm lines. However, the concept currently lacks evidence to suggest that resistance genes for different pathogens, including Xcm and Fusarium wilt, are the same, or even linked on the same chromosomes.

Quantitative genetics

Since the reactions of cotton plants to Xcm infections can be quantified in the field or greenhouse based on a rating scale such as 0 to 12 in Sudan, and 1 to 10 in the US, quantitative genetic techniques, such as F_2 and parents, generation-mean analysis, and diallel analysis, have been used to investigate the genetic basis of BB resistance (see Table 3 for a summary). Bird and Hadley (1959) used a generation-mean analysis to evaluate parents and their F_1, F_2, F_3, BC_1P_1 (i.e., $F_1 \times P_1$) and BC_1P_2 (i.e., $F_1 \times P_2$) between the resistant parent Stoneville 20 (known to carry B_7), and three susceptible parents for resistance to race 1 and 2. Only additive variance in each cross was detected with a moderate heritability (0.45–0.50), and the resistance in Stoneville 20 was conferred by two effective genetic components determining resistance,

Table 3 Quantitative genetics of bacterial blight resistance in cotton

References	Design	Location	Gene action	Heritability
Bird and Hadley (1959)	Generation means	US	Only additive variance detected	0.45–0.50
Innes and Brown (1969)	5 × 5 diallels F_1 and F_2	Sudan/Uganda	Additive variance predominant	Na
Innes et al (1974)	6 × 6 diallels F_1 and F_2	Sudan/Uganda	Additive variance predominant	Na
Mahill and Davis (1978)	4 × 7 NCII	US	Both GCA and SCA detected	Na
Wallace and El-Zik (1990)	Generation means	US	Additive variance predominant	0.59–0.68
Luckett (1989)	10 × 10 diallels F_1	Australia	Additive and dominance effects	0.76–0.81
Singh et al (1989)	10 × 10 diallel F_2	India		
Bachelier et al. (1992)	8 × 8 full diallels	Chad	SCA detected	0.24
Wright et al (1998)	4 F_2 and parents	US	na	0.79–0.98

na not available

i.e., major gene B_7 and minor gene B_{Sm}. Wallace and El-Zik (1990) employed the generation-mean analysis to investigate resistance to three new isolates (highly virulent race 20 of Xcm from central Africa) in three resistant Upland lines (Tamcot CAMD-E, LEBO-CAS3-80 and S295), susceptible Stoneville 825, and their F_1, F_2 and backcross progenies based on their responses in cotyledons and/or true leaves. The results showed that resistance was dominant with a duplicate type of digenic interaction, but additive effects were predominant as the narrow-sense heritability estimates ranged from 0.59 to 0.68. In four F_2 crosses, Wright et al. (1998) estimated a higher broad-sense heritability for BB resistance including: 0.91 for resistance to races 2 and 4 in Empire B2 × Pima S-7; 0.79 for resistance races 2 and 4 and 0.58 for resistance to race 18 in Empire B2 × Pima S-7; 0.91 for resistance to races 2 and 4 and 0.95 for resistance to races 7 and 18 in Empire B2b6 × Pima S-7; and 0.97 for resistance to races 2, 4, 7 and 18 in S295 × Pima S-7. It is worthwhile pointing out that the above three studies, in a span of 40 years, were all conducted by the same research program at Texas A&M University.

Most quantitative genetic studies in BB resistance have been based on diallel crossing schemes. In the same Texas A&M research program, El-Zik and Bird (1967) conducted an 8-Upland parent (each carrying a different *B* gene or combination) diallel study for resistance to BB races 1, 2 and a mixture of both races over 2 years. The diallel was comprised of four breeding lines—146-25 (B_4), 34G (B_2B_3), 91-92A ($B_2B_3B_7$) and 14G (B_2B_{6m}), highly resistant to both races 1 and 2, and four other parents- Austin 7 (B_7, resistant to race 1), Empire WR (B_{Sm}), Deltapine TPSA (B_{Dm}), and Texacala (with no known major B genes, susceptible to both races). The Hayman–Jinks diallel genetic analysis showed that both additive and dominant variance components were significant with a mean dominance of 0.84–0.93, suggesting that BB resistance was partially dominant but approaching complete dominance. The minimum number of genes for BB resistance was estimated to be one. Innes and Brown (1969) used Uplands Reba W296 (with B_{9L}), Bar 7/1 (B_2), Bar 24/5 (B_7), Bar 11/11 (B_6) and Acala 4-42 (b) to make a 5 × 5 diallel, and used the Hayman–Jinks genetic model to analyze data of the parents and both F_1 and F_2 evaluated in Sudan for leaf resistance, and parents and F_2 for leaf and boll resistance in Uganda. Results showed that only the additive variance component was significant, and the authors concluded that additivity, together with partial dominance, accounted for most of the genetic variation in BB resistance. While epistasis was not detected, there was a strong interaction in the $B_2 \times B_6$ cross. Among the five parents, Reba W296 had the highest number of dominant alleles for BB resistance. In a follow-up experiment on both F_1 and F_2, in a half diallel from six Upland parents, including three different parents inoculated with two isolates in Sudan (two locations) and Uganda, Innes et al. (1974) studied the genetic variation of BB resistance based on Hayman–Jinks and Allard models. These parents possessed different *B* genes or combinations, including three Sudanese Uplands—Bar 7/1 2 (B_2), Bar 24/5 3 (B_7) and Bar 12/16 (B_2B_6) and three US Uplands—101-102B ($B_2B_3B_{Sm}$), Acala 1517BR (B_7) and Acala

4-42 (no resistance *B* gene). Additive effects were most important, and non-additive effects were due to the dominance effect. Evidence suggested that minor genes affected the behavior of B_7 and enhanced the resistance conferred by B_2B_3. The behavior of the B_7 gene was not consistent with its being a simple Mendelian locus with incomplete dominance. Genetic variances varied depending on inoculum, being lower with one Xcm culture than with another. Since 101-102B possessed a polygenic complex, in addition to genes B_2 and B_3, progeny of 101-102B showed that resistance built up through selection of minor genes could be effectively transferred. In studying 10 Upland parents and their 45 F_2 progenies for BB resistance, Singh et al. (1989) in India showed that parent 101-102B had the greatest number of dominant alleles for resistance to BB. The Hayman–Jinks genetic model was used by Luckett (1989) in Australia in a half diallel of 10 Upland parents, which included three resistant lines- Siokra (derived from Tamcot SP37, carrying $B_2B_3B_7$) and Reba P279 (carrying $B_2B_3B_{9L}$). Both additive and dominance effects were detected for BB resistance, but resistance was determined primarily by additive effects with heritability estimates of 0.81 for F_1 and 0.76 for F_2.

The above generation-mean and diallel analyses estimated moderate to high heritabilities for BB resistance. However, using Griffing's approach to analyse a complete diallel with 56 F_1 hybrids from eight Upland parents from Cameroon, Côte d'Ivoire, Togo and Chad, Bachelier et al. (1992) in Chad detected the existence of specific combining ability (SCA) with a low heritability (0.24) for BB resistance. F_1 susceptibility was significantly higher than that of the parents, suggesting the existence of residual heterozygosity in certain parents. In a glasshouse study in New Mexico, Mahill and Davis (1978) evaluated BB resistance in seedlings of 28 F_1 hybrids from a North Carolina Design II, between 7 male and 4 Upland female parents. The 7 males included six *G. barbadense* lines—susceptible Pima S-4, Pima 8, E1124, and E1097 and resistant B_2B_6 × Pima 32 and K0210 and one resistant Upland Albar 637. The 4 Upland female parents included resistant strain 1–8 and susceptible Acala 4-42, and their cytoplasmic male sterile counterparts. Variances due to female and male parents (from additive effects) and female × male interaction (from SCA) were significant, and SCA was due to partial dominance effects. Resistant male parents B_2B_6 × Pima 32, Albar 637, and K0210 showed no difference in combining abilities, indicating equal effectiveness as sources of BB resistance.

Cytoplasmic effects

Because the tetraploid Upland cotton share a cytoplasm, similar to its cytoplasm donor—cultivated diploid species—*G. herbaceum* and *G. arboreum* (Wendel 1989), it was not surprising that Bachelier et al. (1992) did not detect any reciprocal effects in their complete diallel crosses using eight Upland parents. However, exotic cytoplasms from other *Gossypium* species may affect cotton growth and responses to abiotic and biotic stresses. Mahill and Davis (1978) demonstrated that *G. harknessii* cytoplasm enhanced BB resistance by 12%, as compared to Upland cytoplasm. Mahill et al. (1979) further showed that cytoplasms from *G. berbaceum* and *G. barbadense* slightly increased BB resistance, as compared to the cytoplasm from Upland cotton.

Molecular mapping of BB resistance genes

Wright et al. (1998) first used restriction fragment length polymorphism (RFLP) markers to map B_2, B_3, b_6 and B_{12} genes. Four F_2 populations, from crosses between susceptible *G. barbadense* Pima S-7 and four resistant Upland lines—Empire B2, Empire B3, and Empire B2b6 and S295, were used in the quantitative trait locus (QTL) mapping analysis. Seven QTL were identified including, one (corresponding to B_2) flanked by RFLP markers pAR 335b and G1219 (explaining 98.0% phenotypic variation for resistance to races 2 and 4) within a 4.3 cM region on chromosome c20 (LGD08) in the Empire B2 × Pima S-7 F_2 population. In the Empire B3 × Pima S-7 F_2 population, a RFLP marker pGH510a, located near the end of chromosome c20, explained 88.2% of the phenotypic variation in resistance to races 2 and 4. An earlier cytogenetic analysis also mapped B_3 to the end of the same chromosome (c20). Interestingly, the B_3 locus explained 53.4% of the phenotypic variation in resistance to Xcm races 7 and 18. In the Empire B2b6 × Pima S-7 F_2 population, similar to the Empire B2 × Pima S-7 F_2 population, the region (B_2) between the G1219 and pAR335 explained 92.2% of the

phenotypic variation in resistance to races 2 and 4. Interestingly, in this cross, four additional QTLs, which explained 56.4% of the phenotypic variation in reaction to races 7 and 18, were identified. These QTL corresponded to the recessive b_6 allele, b_{6a} on LGD02 (formerly LGU01), b_{6b} on c5, b_{6c} on c20 (formerly LGD04), and b_{6d} on c14. The authors suggested that the region near marker pAR1-28 on chromosome c5 mapped to a region that is homoeologous to the B_2 locus on chromosome c20. The authors further suggested that this region may correspond to the BB resistance gene B_4, identified in the diploid A genome species *G. arboreum*, and assigned to chromosome c5 using cytological stocks (Endrizzi et al. 1985). In the S295 × Pima S-7 F_2 population, B_{12} was mapped to a region near the DNA marker pAR043 within 11.4 cM on chromosome 14. This QTL accounted for 94.2% of the phenotypic variation in resistance to BB races 2, 4, 7, and 18. Although no closely linked RFLP markers were identified, results from this study have paved the way for follow-up genetic mapping studies focused on B_{12}.

Australia was very successful in incorporating BB resistance into commercial cotton cultivars (Kirkby et al. 2013). Pedigree records suggest that the resistance source was a set of related, so-called immune lines carrying the $B_2B_3B_7$ and B_{Sm} genes. The Australian resistant Upland cultivar, CS50, in an interspecific cross with susceptible Pima S-7, showed that resistance to race 18 segregated as a single dominant locus (Rungis et al. 2002). Using mapped RFLP markers in the interspecific cross, Rungis et al. (2002) suggested that the resistance locus for race 18 is not located on chromosome c20 near the B_2 or B_3 genes, as previously mapped by Wright et al. (1998), but co-segregated with a RFLP marker on chromosome c14. This marker is known to be linked to B_{12}, a gene originally from African cotton cultivars that provides broad-spectrum resistance to BB.

Xiao et al. (2010) at Monsanto reported closely linked portable PCR-based markers for B_{12}. In an $F_{4:5}$ population of 285 families from an intraspecific Upland cross between race 18 resistant Delta Opal and susceptible DP 388, four closely linked simple sequence repeat (SSR) markers (CIR246, BNL1403, BNL3545, and BNL 3644) flanking B_{12} in a 5.6 cM region were identified. These SSR markers, in turn, were further used to identify four single nucleotide polymorphism (SNP) markers (NG0207069, NG0207155, NG0210142, and NG0207159) spanning 3.4 cM that flanked the B_{12} region on c14. The primer sequences for the above SSR and SNP markers are listed in Table 4. Through a bulk segregant and segregation analysis in an F_2 population of 127 plants from Delta Opal × BRAS ITA 90 in Brazil, an 80 bp SSR marker, amplified by the BNL 2643 primers, was identified to be associated with the resistance in Delta Opal (Marangoni et al. 2013). However, Silva et al. (2014) reported that the 146 bp SSR marker from the CIR246 primers was not only PCR amplified in B_{12}-carrying S295 and Delta Opal, but also amplified in cotton carrying B_2B_3 (101-102B) and $B_{9L}B_{10L}$ (Guazuncho-2). Thus, both segregation and molecular analysis indicated that B_{12} in S295 was closely linked to the B_2B_3 locus which was homologous to or co-segregates with the $B_{9L}B_{10L}$ locus. Therefore, the CIR246 marker could be useful in identifying alleles for resistance up to races 1–18 but cannot be used to discriminate gene or gene complex involved in resistance within the same chromosomal region.

Using an interspecific F_2 population of S295 × Pima S-7 and the genome sequence of *G. raimondii*, Yang et al. (2015) delineated the B_{12} gene to a 354 kb region containing 73 putative plant disease resistance genes. Most recently, Zhang et al. (2019) has further narrowed the B_{12} gene to a region containing only a few putative genes using 550 multiparent advanced generation intercross (MAGIC) lines and more than 500,000 genotyping-by-sequencing based SNP markers (Thyssen et al. 2019; Zhang et al. 2020).

Elassbli et al. (2019) performed a genome-wide association study for more than 330 US Upland germplasm accessions based on a total of 26,345 SNPs from the CottonSNP63K array. A total of 55 SNPs on 9 chromosomes (c1, c5, c8, c10, c14, c15, c20, c22, and c24) were found to be associated with resistance to BB race 18, and each explained 13 to 52% of the phenotypic variation. Chromosomes c5, c14 and c24 had the highest number of SNPs associated with BB resistance. The QTL regions on c5, c14, and c20 are likely those reported by Wright et al. (1998).

Marker-assisted selection

Amudha et al. (2003) in India reported the use of random amplified polymorphic DNA (RAPD) markers to track the introgression of BB resistance gene

Table 4 Primer sequences for SSR and SNP markers linked to B_{12} with resistance to bacterial blight race 18

Marker	Forward primer	Reverse primer
BNL3545	AGTCAGTTTTTTGTTAGCAATATGC	AACCATTAATTCCCTATTTAACCG
BNL3644	GTGCTGTTTGGGCCTTACAT	TAAGCGCATTGACACACACA
CIR246	TTAGGGTTTAGTTGAATGG	ATGAACACACGCACG
BNL1403	TGAATTCATCACCGCAACAT	TGGAACCTCCTTCGGTACAC
NG0207069	CCCTCTCCCTCTACCCTTGATAAAG	CCAAGCATTCAACTTAGTGACCTATAGA
NG0210142	GGTAGGTTTTCTGTTGGCTTTTCAT	GCAGGATGGGAGAGGGCTA
NG0207155	CCAAAGTTGAGAGCATTTCGTTGAA	GCCCAAGTGGTAGCATAATTGTC
NG0207159	GGTCAGTGATAGGAGTTCAAAAGGT	CGGTTTCTCAAGCTATACTGATCATCA

from *G. anomalum* into Upland MCU5 and identified two RAPD markers. However, the SSR marker CIR 246 identified by Xiao et al. (2010) was proven to be most useful. Three SSR alleles (146,156, and 166 bp) were amplified by the CIR 246 primers. Xiao et al. (2010) showed that, two lines (03Q060 and X 3163) with the homozygous 146 bp alleles were all resistant; 5 heterozygous allele cotton genotypes including PMX 1144 and 660 with 146 bp and 156 bp or 146 bp and 166 bp were segregating in resistance; and 6 homozygous 156 bp lines including Acala Maxxa or 166 bp genotypes or 3 heterozygous 156 bp/166 bp genotypes including X9269 were all susceptible. The presence of the 'resistance' 146 bp allele was consistent with the SNP haplotype (A–C–T–T) from four SNP markers in all the nine resistant lines, while the susceptible allele 155 bp or 165 bp was consistent with the SNP haplotype (G–G–C–A) in all the nine susceptible lines tested. Silva et al. (2014) further showed that the CIR246 SSR primers amplified the 146 bp fragment in race 18 resistant S295 (carrying B_{12}), Delta Opal (carrying B_{12}), 101-102B (carrying $B_2B_3B_{Sm}$) and Guazuncho-2 (carrying $B_{9L}B_{10L}$), while the 156 bp fragment was amplified in race 18 susceptible Memane B1 (carrying B_2B_{Sm}) and ST 2B-S9 (carrying B_{Sm}), and the 166 bp fragment was amplified in susceptible Acala 44 carrying no known *B* genes. Due to outcrossing over generations, advanced breeding lines as well as commercial cultivars, may no longer be homozygous for resistance to race 18. Faustine et al. (2015) used the SSR marker CIR 246 (1.8 cM from B_{12}) and SNP marker NG0207155 (0.6 cM from B_{12}) to screen individual plants in three Tanzania and four Brazilian Upland cultivars and found that the resistance gene B_{12}—linked marker allele frequency ranged from 69–75% for UK91 and 25–86% for UK08, and to 0% for Cedro. Results suggested that the cultivars tested were not homozygous in the B_{12} locus. Wheeler et al. (2016) reported that water soaked symptoms of susceptibility were found in 8–15% of the seedlings in resistant transgenic cultivars PHY 375WRF, FM 1830GLT and FM 2484B2F when inoculated in the greenhouse.

Summary and concluding remarks

There are currently 20 races of Xcm recognized in bacterial blight of cotton, and these have been effectively controlled in many cotton-producing countries by planting resistant cultivars. However, the disease has resurged in recent years in the US due to the popularity of susceptible, transgenic cultivars. Since the 1940s, more than 20 major resistance *B* genes (B_1 to B_8, B_{9K}, B_{9L}, B_{10K}, B_{10L}, B_{11}, B_{12}, B_{In}, B_n, B_s, and more than 4 unnamed genes), and at least two polygene complexes (B_{Sm} and B_{Dm}), have been identified. Actions of the resistance *B* genes may be dominant, partially dominant, or even recessive, with additive or epistatic effects, depending on *B* genes. Many major *B* genes can be detected using quantitative genetic approaches. One *B* gene may be resistant to one or multiple Xcm races, and pyramiding of several *B* genes can enhance resistance to one or multiple Xcm races. However, allelic relationships and interactions among some of the *B* genes are currently unknown. For example, the exact chromosomal locations of most of the *B* genes including B_2B_3 have not been determined. Although the notion that there are two complexes (B_{DM} and B_{SM}) has been

widely accepted by the cotton community, no further work has been attempted to clarify their existence, and their relationship with one another, and with other *B* genes. In addition, other major resistance B genes have been identified in germplasm lines in the US, India and Pakistan, but no allelic tests were performed.

Many *B* genes in tetraploid cotton were identified in resistant lines selected from susceptible cultivars. Residual genetic variation in obsolete lines and as well as modern transgenic commercial cultivars exist, due to heterozygosity or natural outcrossing. Some lines may not be homozygous in their resistance or susceptibility to BB. Therefore, pedigree selection within existing susceptible cultivars is not out of date and should still be an effective way to identify BB resistant genotypes.

Genetic resources possessing different *B* genes should be collected and well maintained. Resistance to BB exists in both wild and cultivated diploid *Gossypium* species and should be transferred to cultivated tetraploid cotton. For examples, many accessions of *G. arboreum* and *G. herbaceum* are known to be immune to BB; however, no introgressed Upland or *G. barbadense* lines with immunity have ever been developed and reported. Because susceptible *G. arboreum* and *G. herbaceum* lines exist, genetic and molecular studies can be performed to identify *B* gene(s) that confer immunity in crosses within the diploid species. Markers can then be used to trace their transfer to Upland cotton through interspecific hybridizations.

Although it is difficult to perform allelic tests among all the *B* genes, gene mapping using molecular markers should provide a quick avenue to locate them onto chromosomes. Among the *B* genes, B_{12} appears to confer resistance to most of the Xcm races, including races 18 and 20, and therefore, B_{12} is the focus in current breeding and genomic studies for BB resistance. Markers closely linked to B_{12} have been developed and can be used for marker-assisted selection in BB resistance. Candidate genes for B_{12} have been further identified through high resolution molecular mapping using SNP markers. Currently, however, no *B* genes have been cloned, isolated and sequenced. It is expected that B_{12} will soon be cloned and sequenced, facilitating a better understanding of the molecular genetic basis of bacterial blight resistance in cotton.

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