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Article

Race-Specific Reaction of Wheat Germplasm to Current European Virulence of Common Bunt (*Tilletia* spp.) and Fine-Mapping of Causal Resistance Genes Using SNP Markers

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Abstract

Common bunt of wheat (*Tilletia* spp.) remains a significant threat to wheat production in low-input and organic farming systems, where chemical seed treatments are restricted or avoided. Host resistance represents a key component of sustainable disease control, but its effective deployment requires detailed knowledge of race-specific virulence and the genetic basis of resistance. In this study, we analysed the reaction of a large and diverse wheat germplasm collection to current European populations of common bunt and mapped the underlying resistance genes using SNP-based approaches. A total of 2,731 wheat accessions were phenotyped from 2012 to 2025 using up to 42 purified bunt races with well-defined virulence profiles. Based on phenotypic responses to race-specific resistance patterns, accessions were grouped, and compared with established differential lines. A total of 1504 selected accessions were genotyped using Illumina 26k SNP arrays, and resistance loci were identified by genome-wide association studies followed by fine mapping using recombination analysis. All classical Bt resistance genes from *Bt1* to *Bt10* and *Bt13* were mapped to defined physical intervals, and the genomic positions of 16 additional race-specific resistance genes were identified in the panel of germplasm. Our results confirm that several historically defined Bt genes including *Bt11* and *Bt12* represent multi-gene resistance complexes rather than single loci. Also, genes established as separate genes may possibly be identical, including *Bt4* being identical to *Bt6*, *Bt10* being identical *BtZ* and *Bt9* possibly being identical to one of the genes in the *Bt11* complex. These findings highlight the need for revised nomenclature of genes and differential set of varieties. The identified resistance haplotypes provide an improved tool for marker-assisted selection, and support the development of wheat cultivars with durable resistance to common bunt.

Keywords: common bunt; Bt resistance genes; race-specific resistance; SNP genotyping; marker-assisted selection

1. Introduction

1.1. Common Bunt

Common bunt in wheat is an epiphytotic seed-borne disease caused by two closely related species (*Tilletia caries* (DC.) Tul. & C. Tul. syn. *T. tritici* (Bjerk.) Wint. and *T. laevis* J. G. Kühn syn. = *T. foetida* (Wallr.) Liro.). Recent research indicates that the two species are actually morphological forms of the same species [1–3] and there are no indications pointing to differences in reaction to race-specific resistance genes in wheat between the two species or to the third species *Tilletia controversa* (Kühn) syn. *T. contraversa* (Kühn) causing the predominantly soil borne disease dwarf bunt [4].

Wheat production needs control of common bunt. Wheat is and has always been one of the most grown and most significant crops for feed and for human consumption, today covering 20% of the human intake of calories and proteins [5]. Throughout the history of agriculture, common bunt has been considered one of the most significant diseases in wheat to control [6–8].

The impact of common bunt in wheat production significantly reduced after the invention of mercury products as a seed treatment in 1913 [9] and the implementation of Uspulun (phenylmercury acetate) by Bayer in 1914. Mercury products effectively controlled the disease for decades in Europe, whereas in less industrialised agrosystems and regions as in Iran and Azerbaijan, common bunt still caused average yield loss of 30% in the 1990's [10].

In the 1970's and 80's, the use of mercury for seed treatment was gradually banned and phased out in the EU and replaced by different synthetic fungicides. Probably as a combined result of increased wheat production and of a reduced effect of the pesticides replacing mercury-based products, the occurrence of bunt increased in Europe [11]. In organic farming and other farmers using home saved seed without seed treatment, common bunt still causes problems [12].

Significant epiphytotic outbreaks of common bunt are almost exclusively caused by use of infected non-treated seed. Infection in a seed system multiplies by a factor 100 from year to year in susceptible varieties [12] and hence even a small infection in a field will initiate epiphytotic development and after just a few years have multiplied to a significant level. As few as 100 fungal spores per gram of grain can in some cases be sensed by consumers, depending on age and moisture content of the spores [13], and this level in the grain can be caused by as little as a few infected plants per hectare [12,14]. *The accepted threshold for common bunt infestation in seed lots is thus very low, practically at the detection limit of seed analysis* [15], as even a small infection can reduce grain quality and will inevitably multiply and cause risk for neighbouring fields and the seed system in the region.

Pesticides for seed treatment in modern agriculture provide effective control against common bunt, and significant outbreaks of common bunt almost exclusively occur in untreated seed, whereas dwarf bunt occurs in regions like Sweden, The Alp region in Europe and Pacific Northwest in America where specific climatic conditions favour soil borne infections of this disease [16–18]. As a result, resistance to common bunt has been a low priority topic among geneticists and wheat breeders, in particular in Europe, and the disease has been categorised as a “hidden disease” neglected in both farmer's awareness and in research [18].

EU and many national authorities are concerned about the widespread use of pesticides in modern agriculture. Fungicides used for control of common bunt include a group of PFAS based pesticides including Fludioxil and Sedaxa, and another group of Carboxamid, Phenylamid and Stroburin. Authorities are implementing restrictions on these fungicides because of their persistence and eco-toxicity in the soil. Therefore, the most widely used group of fungicides in EU today is triazols, in Denmark covering 95% of the annual consumption [19]. Triazols are in focus as a risk factor for human health in particular in regards to development of fungicide resistance in human medicine used in treatment of Aspergillose. The European Farm to Fork and Biodiversity Strategies therefore aim to reduce the use and risk of chemical pesticides by 50% before 2030 [20,21]. Little has so far been achieved to meet this goal when it comes to control of seed-borne diseases.

If common bunt is to be controlled in modern farming with environmental and healthier alternatives to pesticides, there are a range of solutions, such as heat treatment, biological products and seed analysis combined with discarding contaminated seed lots [22]. In the current article, we focus exclusively on the progress made to use marker assisted selection in plant breeding to develop resistant varieties and with a focus on race-specific resistance.

1.2. Resistance Mechanisms

Host resistance is a well known strategy to control plant diseases, and resistance to common bunt was among the first breeding goals for breeders and plant pathologists in particular in Australia and North America [6,7].

As early as 1764, Tschanner observed that different types of spelt (*Triticum spelta*) differed in their susceptibility to common bunt [6], and also Kühn observed in 1880 varietal differences in susceptibility to bunt in bread wheat (*T.aestivum*) [23]. Already in the beginning of the last century there was basic knowledge on susceptibility and resistance, and successful breeding programmes were established in the beginning of the century by Farrer [24], Pye [25] and others. Based on studies on phenotypic reactions to different races of bunt, the resistant varieties were grouped into resistance factors, a fundamental work for the Bt-resistance genes known today.

Early research has shown that inoculating wheat varieties with spores from the same variety often results in higher infection than infecting with spores from other varieties [26–28], demonstrating the presence of race-specific resistance in wheat, and virulence specialisation in the pathogen. Hence, it has been known for about 100 years that race-specific resistance in both pathogen and wheat has a major impact on common bunt infections. Taking virulence into account is pivotal in the use of host resistance as a control strategy for common bunt, as resistant varieties may lose resistance when new races multiply within a region [29].

1.3. Genetic Origin of Common Bunt Resistance Genes

Today, the designation of race-specific resistance genes against common bunt is based on the Bt-classification system covering the genes *Bt1-Bt7*, [30], and later supplemented with the additional genes, including *Bt8* [31], *Bt9* [32], *Bt10* [33], *Bt11* [34], *BtZ* and *Bt12-15* [35] and *BtP* [36].

The Bt-genes were defined based on segregation and phenotypic studies, but novel development of different genetic tools, and knowledge about the genetic position of the genes and genetic markers have made it possible to study the Bt-genes and other bunt resistance genes in further detail.

The *Bt1* gene was discovered in the variety 'Martin', and therefore previously designated the Martin Factor [37]. The resistance of 'Martin' later turned out to be caused by a combination of two genes [38] and Briggs and Holton identified and designated them *M₁* and *M₂* [39]. *M₁* was later renamed to *Bt1*, and *M₂* to *Bt7* proposing Sel 2092 (PI 554101) and Albit as a differential line for *Bt1* [30]. *Bt1* has been mapped to Chromosome (Chr) 2B in 1960 [40] and a more precise position only recently presented [41].

At the same Chr 2B, resistance associated with bunt resistance has been mapped in two different diverse panels of wheat varieties, but it was not discussed if it was a mapping of a known gene [42,43]. In another study of a diverse set of varieties, the marker Ku_c71357_859 at position 581.70 Mbp on Chr 2B was associated with bunt resistance, and suggested to be *Bt1*, as the differential line for this gene, PI 554101, had a positive allele for this marker [44].

McCartney *et al.* associated resistance in the Canadian spring wheat variety 'Kenyon' with Chr 2B and argues that this resistance was a quantitative trait most likely inherited from 'Neepawa', and hence different from the race-specific Bt-genes such as *Bt1* [45]. In another Canadian spring wheat variety 'CDC Go', a QTL for resistance was also mapped to Chr 2B at 244 Mbp [46].

The gene *Bt2* was originally found in the variety 'Hussar' and designated the 'Hussar Factor' or *M3* [47], but this variety turned out to include also *Bt1*, and Metzger designated it to *Bt2* and proposed 'Selection 1403' and 'Selection 1075' as a differential lines for *Bt2* [30]. Also 'Canus' is mentioned as an original source of *Bt2* [34]. Goates changed the recommendation to the line 'Selection 1102' (PI 554097) as a differential line for *Bt2*, but the origin of this lines is not described [36].

We've previously made a preliminary mapping of *Bt2* from PI 554097 to Chr 1D [48]. At the same chromosome, a QTL, Qcbspa-1D, were mapped in 'Vesper', but it was characterised a quantitative trait for bunt resistance, and hence different from race-specific Bt-genes such as *Bt2* [49].

The gene *Bt3* was first found in varieties 'Florence' and 'Genoa' developed by Farrer from 'White Naples', 'Improved Fife', 'Hornblende' and an Indian wheat [24,50] and the resistance was studied in further detail by Churchward and Gaines demonstrating recessive inheritance [51,52]. The variety 'Ridit' (Wash. No. 2324, C. I. No. 6703) was developed at Pullman by Gaines in 1915 from a cross between 'Turkey' x 'Florence' and was registered in 1926 as a bunt resistant variety [53]. 'Ridit' was used as a differential line to separate bunt races already by Bressmann [54], and Metzger

designated the resistance *Bt3* with reference to resistance in 'Florence' and 'Ridit' noting that chromosomal position was unknown [30].

A resistance factor was by researchers at BOKU, Austria associated with bunt resistance in a diverse set of varieties to the position 473.96 Mbp at Chr 1A [44]. Later, researchers at the same institute mapped a QTL Qbt.ifa-1AL from the varieties 'Blizzard' and 'Bonneville' at Chr 1A and hypothesised that this may be the *Bt3* gene [55]. This hypothesis was supported by Lunzer *et al.* [56].

The gene *Bt4* was originally called the Turkey Factor by Briggs [57] and preliminary associated with Chr 1B using Monosomic Analysis [58]. The resistance was originally found in the varieties like 'Bison', 'Kaw', 'Nebred', 'Omaha', 'Oro' (along with *Bt7*), 'Turkey 2578', 'Turkey 3044' (along with *Bt7*), and in 'Turkey 1558', and resistance is linked with resistance *Bt6* [59] and also to at *Bt5* at Chr 1B [30,60]. 'Turkey 3055' was originally proposed as a differential line for *Bt4* [30], but it was later changed to CI 1558 (PI 11610) [35,36].

The *Bt5* gene was originally found in the varieties 'Hohenheimer Begrante' (En: awned) and 'Hohenheimer Unbegrante' (En: unawned), but it was shown that they were resistant only in Germany but susceptible in Pullmann, USA [61], and the resistance was inherited independently from resistance in 'Hussar' (*Bt1+Bt2*) [54,62].

It has long been known that 'Hohenheimer' (CI. 11458) has not only one, but at least two bunt resistance genes [63]. To solve this problem of dual genes in 'Hohenheimer' (CI 11458), Hoffmann and Metzger proposed using 'Selection R60-3432' ('Elgin'*'Hohenheimer') as a differential line for *Bt5* [4]. USDA-NSGC has no record of this accession. Goates returned to 'Hohenheimer' (CI 11458) as a differential line [35,36], and despite the documented dual resistance genes, this accession has been used in most studies since. 'Hohenheimer' (CI 11458) has a reaction to virulence demonstrating *Bt5* behaviour, but with a lower infection level than most other *Bt5* lines, confirming that an additional and most likely race-non-specific gene seems to contribute to the resistance [64,65].

The *Bt6* gene was originally found in the variety 'Rio' and was later identified in 'Turkey 10095', 'Turkey 10097' and in 'Columbia' (along with *Bt1*). The gene is also present in Hyslop' (along with *Bt7*), and resistance was very closely linked to resistance in Turkey (*Bt4*) with a recombination value of 2-4% [66]. Later, it was demonstrated by linkage studies and monosomic analyses that the 'Turkey' factors T(*Bt4*) and the Rio Factor R(*Bt6*) are linked with the gene that regulates red glume colour on Chr 1B [30,58,60]. Goates tested 'Rio' and the other Bt differential lines with 56 global races of common bunt, and 5 races could separate *Bt5* from *Bt4* and *Bt6*, but none of the races could separate *Bt4* from *Bt6* [36].

Other resistances related to Chr 1B

Leijerstam identified European winter wheat 'Trintella' as a highly resistant variety [67]. The resistance of 'Trintella' was confirmed by Denneken and Pedersen [68], and the dominating resistance factor in 'Trintella' was mapped to Chr 1BS [69].

Wang *et al.* identified a dominating resistance factor in variety 'Blizzard' associated with three markers Xgwm374, Xbarc128 and Xgwm264 at a 3.9cM interval at Chr 1BS [70], and the mapping was later improved to a position at 8–22 Mbp [55]. The authors argues that the 'Blizzard' resistance at 1B could be the same resistance found Canadian spring wheat variety 'AC Domain' on Chr 1BS [71] and in 'Carberry' [72] in the same region. The mapping of resistance in 'Carberry' has been refined to a position at 21.4 Mbp and it was concluded that it overlaps with resistance from 'Blizzard' at Chr 1B [46]. Bunt resistance has also been mapped to Chr 1B between the markers BS00086854_51 and wsnp_Ex_c5679_9976893 in Canadian spring wheat variety 'CDC Go' [73]. Association between marker BS00086854_51 and bunt resistance was confirmed in a diverse set of winter wheat varieties [44], but resistance associated with Chr 1B was not confirmed in a 'CDC Go' bi-parental population crossed with 'Attila' [46].

A resistance factor was mapped in a diverse panel of varieties to an interval 137.13–163.10 Mbp on Chr 1B, but it was not discussed if it was a mapping of a known gene [43].

The *Bt7* gene was discovered as one of two resistance factors, M and M₂, in the variety 'Martin' [39]. The M gene was later renamed to *Bt1*, and M₂ to *Bt7* [30]. The Martin factor M₂ (= *Bt7*) was

mapped using nullisomic and monosomic lines to Chr 2D [40]. 'Selection 50077' (PI 554100) is a selection from a cross of 'Martin' with 'Elgin', and is now used worldwide as the common bunt differential lines for *Bt7* [35,36].

Based on phenotyping reaction, *Bt7* is present in a range of commercial varieties in both spring wheat and winter wheat in Europe, including 'Tambor', 'Korrund', 'Xenos', 'Segor', 'Quarna', 'Fiorina', 'Thomaro', 'Sailor' [64,74,75].

The *Bt8* gene was originally found in the variety 'Yayla 305' (PI 178210). The gene was not mapped, but authors ruled out a position on Chrs 5A, 1B, or 2D [31]. 'Yayla 305' is a composite variety developed from local landraces in Turkey and released in 1939 [76]. Later, *Bt8* was also identified in PI 178383 along with other genes including *Bt9* and *Bt10* [77,78], and in PI 173438. 'M72-1250' (=PI 554120 (PI 173438*Elgin)) was proposed as a differential line for *Bt8* [35,36].

RAPD marker Psg3 is suggested to link with *Bt8*, but little evidence for this is provided, and information regarding a link to a chromosome or position is absent [79]. SSR marker Xgwm114 has been used in several studies to assess the presence of both *Bt8*, *Bt10* and *Bt11* [76,80].

SSR marker wmc112 was linked to bunt resistance on Chr 2D in wheat variety 'Lewjain'. Segregation demonstrated a single gene was responsible for resistance. Based on phenotyping with 40 races, *Bt7* and most other Bt-genes was ruled out in this variety, but *Bt8* was not, and it could be a novel gene for bunt resistance [81]. Except for *Bt7*, no other genes have been mapped to 2D.

Bokore *et al.* contradicts the mapping of *Bt8* to Chr 2D with an indication of *Bt8* to be at Chr 7A, but no detailed mapping has been presented [82].

The preliminary mapping of *Bt8* to Chr 7A is not supported in other studies, but many authors have identified resistance to common bunt associated with Chr 7A. Resistance factor Q.DB.ui-7AL derived from resistant line 'IDO835' was mapped to 7A [83], and in the highly resistant American sib line of 'IDO835' 'Blizzard', a QTL Qbt.ifa-7AL for bunt resistance has been mapped to a position of 722–737 Mbp on Chr 7A, and hence different from *Bt3* and *Bt6* also present in 'Blizzard' [55]. Ehn *et al.* was able to associate marker Ku_c5529_824 at position 335.99 Mbp to bunt resistance in a diverse panel of wheat varieties, and another marker RAC875_c23665_68 closer to Qbt.ifa-7AL [44]. However, the position of the second marker is contradicting in RefSeg 2.1 [84] and refers to different positions and different chromosomes including 3A.

In European winter wheat 'Trintella', a minor effect of bunt resistance has been mapped to Chr 7A [69], and also in Canadian spring wheat varieties, 'AC Domain' [71], 'Lillian' [49] and 'Carberry' [83] minor QTLs have been mapped to 7A. Virulence to *Bt8* is rare, and given that these mappings are reported as genes with minor effects, it is unlikely that they can express a confirmation of the mapping of *Bt8* to 7A.

Also at Chr 7A, resistance associated with bunt resistance has been mapped in two different diverse panels of wheat varieties, but it was not discussed if it was a mapping of a *Bt8* or other known genes [42,43].

The *Bt9* gene was originally found in early 1970'ties the landrace 'CI 7090' (along with *Bt7*) [32]. The gene has also been found in the landrace PI 178383, along with *Bt10* and other genes [77,78], and 'Selection M69-2094' derived from PI 178383*Elgin' was used as a differential line for *Bt9* from mid 1970'ties [4]. Today, a sib line 'R63-6968' (PI 554099) is used as a differential line in most studies [35,36].

Steffan *et al.* first mapped the *Bt9* gene to the distal end of 6DL using 7k DArT markers [85]. Wang *et al.* mapped a QTL on 6DL to the interval 469,8–470,3 Mbp in line 'IDO835' and hypothesise that this may co-locate with *Bt9* since it possibly is in the pedigree of 'IDO835' [83] and this mapping was confirmed and improved by Gordon *et al.* [86]. Qdb.ssdhui-6DL was mapped later at position 492.5 – 494.6 Mbp in variety 'UI Silver' and suggested also to be a mapping of *Bt9* [87].

The *Bt10* gene was originally identified in the varieties 'Greece 18' (PI 116301) and in 'Mocho' (PI 116306) [33]. In screening of virulence in American bunt races, the line 'M69-2094', a selection from the cross between 'Elgin' and PI 178383, was initially used as a differential line for *Bt10* [4], but

was later changed to the sib line 'R63-6982' (PI 554118), today widely used as a differential line for *Bt10* [35,36].

Bt10 has been mapped to 6DS and was the first Bt-gene to be mapped with markers useful for selection [88–90]. Cichy and Goates identified the RAPD marker 196 to be the best of three used markers in predicting the *Bt10* with a positive hit rate of 70%. However, the target alleles RAPD 196 were also present in some genotypes known not to have *Bt10* [79]. 'AC Cadillac' is a Canadian spring wheat carrying *Bt10* [91], and Singh *et al.* mapped a resistance gene to 6D in 'AC Cadillac' between flanking markers wPt-672044–wPt-5114 arguing that this gene is *Bt10* [72]. A refined mapping of *Bt10* resistance in spring wheat 'Peace' found the position to be at 7.4–7.6 Mbp [46]. This mapping was further refined in a diverse panel of varieties at 7.43 Mbp indicated to be a mapping of the *Bt10* gene [92]. Qdb.ssdhui-6DS was mapped at position 1.4 – 2.1 Mbp in the variety 'UI Silver' and suggest to be a mapping of *Bt10* [87].

The term *Bt11* was first used by Abdallah for a resistance gene identified in the landrace 'Dimenit' along with other resistance genes [34]. This landrace was collected in Tokat, Turkey, in 1948 by V. Taysi at the Turkey's Field Crops Research Institute and donated to USDA NSGC by J.R. Harlan, and recommended as a new source for breeding [93]. Abdalla describes *Bt11* as a gene in 'Dimenit' (PI 166910) additional to *Bt7* and *Bt9* also present in the landrace [34]. Goates proposed selection 'P68-1336-7' (PI 554098, ('Elgin'*PI 166910)) as a differential line for *Bt11* [35,36].

In the first attempt for a mapping of *Bt11* in 2011, resistance was associated with Chr 3B associated with marker loci Xbarc180, Xwmc623, Xwmc808 and Xgwm285, but the author notes that more precise studies of the association is necessary [94]. Cichy and Goates used SSR marker Xgwm114 to identify *Bt11*, and this marker has been used in several studies to assess the presence of *Bt11* in wheat varieties [76,80,95]. Resistance associated with Chr 3A has also been demonstrated in a diverse panel of varieties, but without reference to a specific resistance gene [43].

The association of *Bt11* with Chr 3B could not be confirmed in later studies, but the presence of multiple resistance genes on different other chromosomes in the donor of *Bt11* has been identified in several mapping populations [96]. A single QTL, *Qbt.ifa-6DL*, was found at Chr 6D at 482.8–495.2 Mbp, hence close to the position of the preliminary mapping of *Bt9* [85]. Since *Bt11* by the definition by Abdalla is a gene additional to *Bt9* [34], it can be speculated that *Qbt.ifa-6DL* is in fact *Bt9*. However, based on the position of peak markers and of contrasting alleles of markers within the mapped interval, Lunzer *et al.* argues that *Qbt.ifa-6DL* is different from *Bt9* and hence is the *Bt11* gene. On top of the mapped QTL at 6D, two other QTLs were mapped at Chr 4B, one of which, *Qbt.ifa-4BL*, was mapped to a region between 662.9 and 671.4 Mbp [96].

An additional factor for resistance in 'Dimenit' was mapped to Chr 7B at the position 10.1–12.8 Mbp [96]. In the Canadian spring wheat variety 'McKenzie', a QTL has also been associated with Chr 7B. This resistance is characterised as a minor factor of resistance [97]. A minor factor of resistance has also been associated with 7B variety in 'Trintella' [69].

A resistance factor was mapped in a diverse panel of varieties to a very large interval 18.10–703.15 Mbp on Chr 7B [43], but given the size of this interval, it cannot be concluded if it co-located with the mapping of resistance on Chr 7B in 'Trintella', 'McKenzie', or others.

On the short arm of Chr 2A, a QTL, (*Qbt.ifa-2A*) flanked by markers Tdurum_contig29983_490 and AX-94381641 2A was identified in a single mapping population between 'Dimenit' and the moderately resistant variety 'Mulan', but it was not presented if this QTL was inherited from 'Mulan' or from 'Dimenit' [96]. Also at Chr 2A, resistance was mapped in Canadian spring wheat 'Vesper' to a much higher position 745.40 – 746.74 Mbp [49], and therefore most likely different from *Qbt.ifa-2A*.

It seems contradictory that 'Dimenit' has *Bt9* in addition to *Bt11* [34], and at the same time that 'Dimenit' had only one major resistance gene on Chr 6D [96]. The contradiction however is not necessarily absolute, since 'Dimenit' is a landrace including genetic diversity, and the possibility exists that Abdallah may have analysed a selection of 'Dimenit' with *Bt9*, whereas Lunzer *et al.* may have analysed a selection without *Bt9*. Goates analysed virulence of 56 bunt races, and found only 2 races virulent to *Bt11*. One of these was also virulent to *Bt9*, whereas the other were only slightly virulent to *Bt11* but avirulent to *Bt9* [36].

The gene Bt12 was proposed by Goates using PI 119333 as a differential line [35,36]. PI 119333 is a landrace collected in Elazığ, Turkey, in 1937 and recommended as a new source for breeding [93].

Cichy and Goates associated *Bt12* with the marker Xbarc128 demonstrating a prediction of *Bt12* with a positive hit rate of 75%. However, the target alleles of Xbarc128 were also present in some genotypes known not to have the *Bt12* [79].

Müllner *et al.* has investigated PI 119333 in detail, concluding that the main factor of resistance QBT.ifa-7DS|Bt12 is caused by a gene within a 4.3 Mbp interval ranging from positions 6.47-10.84 Mbp at Chr 7D, and with additional resistance identified at Chr 4B [98].

In 'Blizzard', a resistance has been mapped to a position of 12.5–15.3 Mbp on Chr 7D [98] and this QTL has also been found named Q.DB.ui-7DS, in 'IDO444', a sibling of 'Blizzard' [99]. The markers associated with Q.DB.ui-7DS match the markers in differential line for *Bt12* and PI 173438 [86].

Singh *et al.* mapped a gene in Canadian spring wheat variety 'Carberry' at Chr 7D [72]. This resistance is considered race-non-specific and hence most likely different from the main factor of *Bt12* described as highly effective. Chen argues that the resistance at Chr 7DS in 'IDO444' is different from resistance at Chr 7D found in 'Carberry' [99].

The Bt13 gene was first described and studied by Goates found in the varieties 'Thule III' (PI 181463) [35,36]. The domination of the donor accession 'Thule III' is caused by mistake during transport data from NordGen to USDA NSGC or by a seed mixture accident, since the original variety 'Thule III' (NGB6714) is genetically and morphologically very different from (PI 181463) and showing another phenotypic reaction [100].

Bt13 has been mapped to 7D [101].

The Bt14 gene was proposed by Goates for a resistance gene found in the spring durum variety 'Doubbi' (CI 13711) [35] and also present in the hexaploid line 'Selection 186' (PI 172201) [102]. Goates later omitted the *Bt14* gene from the differential set because of an inconsistent reaction of the gene in different environments in particular regarding spring and winter sown trials [36]. Little is known about the physical position of the gene except that it must be on either the A or the B genome. Wang *et al.* identified QDB.ui-7AL in hexaploid wheat 'IDO835' in the position 732.8-736.7 Mbp associated with bunt resistant and pointed to the fact that it was present in differential lines of both 'Doubbi' and 'Carlton' [83].

The Bt15 gene was proposed by Goates [35] for a resistance gene found in the spring durum variety 'Carlton' (CI 12064). Similar to *Bt14*, Goates later omitted the *Bt15* from the differential set because of an inconsistent reaction of the gene in different environments [36].

The BtP gene was designated by Metzger, and Goates [36] included the landrace '7838' (PI 173437) in the differential set for bunt resistance. PI 173437 was collected in 1949 in the Hakkâri region in Turkey, a region that has been identified as one of the hot-spots of bunt resistance in their landraces [36]. No publications have investigated the genetic background of the resistance in further detail.

BtZ is a term first used by Goates referring to resistance from variety 'Zarya' [35]. This variety was developed in Soviet Union [103–105]. *BtZ* is supposed to be introgressed into *Triticum aestivum* from *Thinopyrum intermedium* via the line 'Hybrid 599' (W0480). The cultivar 'Zarya' has 'Hybrid 599' in its pedigree and is the main source of *BtZ* in European breeding material [106]. 'Zarya' has been widely used as a source of bunt resistance in particular by the German breeder Cultivari and included in variety 'Tilliko' [107].

Other identified resistance genes different from the Bt-genes

The designated Bt-genes *Bt1-Bt13* may as described above be associated with chromosomes 1A, 1D, 2A, 2B, 2D, 3B, 4B, 6D, 7A, 7B and 7D. However, bunt resistance has been mapped in different studies in different varieties also to other chromosomes.

A QTL for resistance was mapped to Chr 3A in the variety 'CDC Go' [73], and in a diverse panel of varieties, resistance has also been associated with Chr 3A [92]. Mourad *et al.* associated resistance in a diverse panel of varieties with Chr 3A, and also with Chr 3B and Chr 5A [43].

In Canadian spring wheat variety 'Lillian' resistance has been mapped to Chr 3D and Chr 5A [49], and in variety 'Carberry' resistance was associated with Chr 4D [72].

On Chr 5B, additional alleles for resistance has been mapped in 'Trintella' [69].

On Chr 5D, 6A and 6B, resistance has been associated with resistance in a diverse panel of varieties [43], and also on 6A, a study mapped resistance in the Canadian spring wheat 'Kenyon' Chr 6A arguing that resistance was inherited from 'Neepawa' [45].

On Chr 7A, resistance has been mapped in the American winter wheat varieties 'Blizzard' and 'Bonnevill' [55], 'Trintella' [69], 'AC Domain' [71], 'Lillian' [49] and 'Carberry' [83].

Research and breeding for resistance to common bunt have been performed for over 100 years. Varieties with resistance have been developed, but going through the published literature, the causal genes in both differential lines and in varieties bred for resistance are to a large extent unknown. Most research seem fragmented, focussing on single genes in bi-parental populations, or diverse panels of varieties with unknown resistance genes, making it difficult to compare findings in different studies. Hence, resistance breeding still rely on difficult phenotyping, and with unknown durability. No genes have been cloned, and no markers have been developed that are reliable for neither research or breeding to identify causal genes needed to estimate the resistance in the varieties.

In our daily breeding and seed production of wheat for organic farming, we have experienced the challenges of preventing common bunt, but also seen the potential of resistant varieties and in particular varieties with multiple resistance genes.

The aim of the current work is to collate the phenotyping, genotyping and published information for the dual purpose of improving the practical use of standard markers for marker assisted resistance breeding, and also to prepare the mapping for cloning of causal gene sequences needed to develop reliable diagnostic markers.

2. Results

A diverse set of germplasm was collected from genebanks and plant breeders with both known and unknown reaction to bunt, including the differential lines with known Bt-genes *Bt1-Bt15* plus *BtP* and *BtZ* [36]. Selected lines were used as crossing partners developing segregating RIL-populations covering all the described Bt-genes, except *Bt14*, *Bt15*, and *BtP*.

Infections in resistant varieties with known resistance genes were tested for true virulence against the gene in question by re-inoculation spores from the varieties onto the differential lines and other varieties with similar resistance genes. We found infected plants in all the differential lines, and re-inoculation on the host varieties demonstrated true virulence against all the Bt-genes except in differential lines of *Bt9* (PI 554099), *Bt11* (PI 554098), *Bt12* (PI 119333), and *BtP* (PI 173437).

From initial bulk spores collected from farmers and research stations, we managed to purify races by multiplying them on different resistant varieties [65,74]. The purified races were used to phenotype the wheat panel, including the differential set with known resistance genes.

During the period 2012-2025, a total of 2,731 wheat accessions were phenotyped with one or more (up to 42) of the purified races of common bunt, resulting in a total of 23,736 field micro plots with 50 plant sown in each. Infection level ranged between 0-100% in all races, but infection differed depending on the interaction between race virulence and host resistance.

Based on infection to the different races, the varieties and breeding lines were grouped in categories with similar patterns of reaction to the different races. Categories including a differential line were postulated to carry the Bt-resistance gene. Categories with a reaction not including a differential line were postulated to possess a combination of genes or a new race-specific gene.

Each group of varieties with a Bt-resistance gene or with an undescribed resistance gene were analysed for significant association with TG26k SNP markers, and the resistance gene was preliminary mapped by GWAS, using the total panel of varieties for validation, and afterwards fine-mapped based on recombination analysis based on parental information.

Based on these analyses, we were able to map all the Bt genes *Bt1-Bt10* and *Bt13*, to positions on the physical DNA map (RefSeg 2.1) [84]. From phenotypic categories not including a differential line

with a known resistance gene, and hence postulated to potentially possess a new unknown race-specific resistance gene, we managed to map additional genes not covered by the standard collection of Bt-genes. We compared these mappings with previous publications to conclude if a new gene has been discovered or can be linked with already known resistance genes or previously described loci (Tables 1–13). We conclude that 16 new genes have been mapped, and that additional genes have been mapped in other studies not found in our panel of germplasm.

Mapping the causal genes in resistant varieties to a physical position is an important step towards development of Marker Assisted breeding. To further improve the findings for Marker Assisted Breeding (MAS), we selected a set of markers significantly associated with resistance in the GWAS analyses that can be used for MAS. A single or a few markers are rarely enough to identify a gene in a variety because of monomorphic markers in resistant and susceptible varieties. Therefore, haplotypes specific to resistant varieties are listed in Appendix 1. Hence, the intervals listed in Table 1-13 represent the intervals within which the gene is to be found, whereas the haplotypes in Annex 1 are markers giving the best hit rate for separating resistant from susceptible varieties in our diverse validation panel of both European, American varieties and landraces.

3. Discussion

From the initial collection of spores, we purified races by multiplying them on resistant varieties, and we managed to develop races with virulence to all the genes *Bt1-Bt8* and *Bt10*, *BtZ* and *Bt13*. This means that either virulence to almost all the genes have been in the initial spore collection from within Denmark, or have developed through mutation during the first 10 years of race purification. Whatever the reason was, it demonstrates that a single race-specific resistance gene is unlikely to be durable as a stand alone strategy to control common bunt if widely used in agriculture within a region. As a consequence, three strategies can be used: 1) many different genes can be used in different varieties in a region to minimise the risk of primary infection into a seed lot, or 2) several genes can be stacked into each variety, or 3) variety mixtures or populations of lines with different resistance genes can be mixed to reduce secondary multiplication. Whichever strategy is chosen, alone or in combination, it is pivotal to know the genes available for breeding, and to be able to identify and discriminate breeding lines with different resistance genes.

The resistance genes designated *Bt1-10* and the development of differential lines representing the genes has been a solid basis for research in resistance for common bunt. Today, novel genetic tools have been developed which were not available for the pioneers developing the Bt-designation system and selecting the differential lines. Using these tools to investigate the details of the mapping of the individual genes, new understanding of the genes has been revealed. Some resistances previously thought to be caused by single genes such as *Bt11* and *Bt12* have turned out to be combinations of multiple genes, whereas other genes thought to be different genes have turned out to most like being identical genes. Different genes with similar phenotypical reactions cause confusion in the statistical analysis of association between phenotyping and genotyping. We therefore consider it important to specify the original donor of the genes in question and track the heritage to prevent mistakes.

We have in this study focused on race-specific resistance genes, each with either major effect in avirulent races, or no effects in virulent races, and have therefore chosen the GABIT protocol as this tool is specifically designed to analyse the interaction between gene and environment (in our case different races) rather than analysing the effect on infection level [108]. We have used the method of postulation of resistance genes based on discrepancy in phenotypic reaction to different races. This method is less effective to identify minor race-non-specific genes, as a phenotyping result of medium infection in all races will in our system be assessed as susceptible even though the infection level may be lower than the most susceptible varieties. The fact that we have mapped only race-specific genes does not at all mean that only race-specific genes exist in our panel of varieties, but only that our method was not optimal for assessing minor genes. On the other hand, infection level is expected to be proportional to the concentration of virulent spores on the seed, and we believe that some genes

in other studies categorised as quantitative traits, may in fact in some cases be race-specific, if a significant proportion of spores in a bulk mixture of spores used for assessment are virulent while others are not.

On top of defining the intervals by markers with statistical association with resistance, we have used recombination mapping reducing the defined interval to areas demonstrated to be inherited from the resistant parent. This approach has the advantage of finding more precise intervals of the position of the actual gene, but bears the risk of concluding the final interval by just a few or a single line, and therefore with a significant impact of potential experimental errors.

Mappings on Chr 1A, including Bt3 summarised in Table 1

Bt3 has previously been found on Chr 3A [109]. We mapped the gene to a 4.8 Mbp interval. The mapping confirms and improves previous mappings of *Bt3*. Qbt.ifa-1A was mapped in ‘Blizzard’ and considered to be *Bt3*, but no phenotyping with racial differences were provided [55,56]. We tested the same material demonstrating *Bt3* phenotypic behaviour and genotypic similarity with the differential lines and the original donor, and our mapping confirms *Bt3* in ‘Blizzard’ and in the sister-line ‘Bonneville’.

In a previous study of the American wheat line ‘IDO444’, the resistance factor Q.DB.ui-1A was associated with marker Xcfa2129 at 74cM in Chr 1A [99]. Ehn. *et al.* mapped a resistance factor CB-1A to a position 473.96 Mbp and argues this to be identical to Q.DB.ui-1A [44]. We are convinced that these QTLs too are mappings of *Bt3*. ‘IDO444’ is a zip line of ‘Blizzard’ and ‘Bonneville’ and therefore most likely all have inherited *Bt3* from their common ancestor ‘Ridit’.

In a diverse panel of varieties from Nebraska, resistance was associated with position 497.93–499.86 Mbp [43], and also in a diverse panel of Canadian spring wheat, resistance was associated with Chr 1A at position 556.87 Mbp [92]. Based on the position, these two mappings could be mappings of *Bt3*.

We mapped a gene Bt_Mariann_1A different from *Bt3* at a position 1.23 – 10.42 Mbp on Chr 1A in breeding lines including spelt in the pedigree. We consider this being a new race-specific gene for bunt resistance. In Canadian spring wheat varieties, two resistance loci have been associated with Chr1A at the positions 13,37 – 14,03 Mbp and 4.38 Mbp [92]. Given the positions close to Bt_Mariann_1A, a common gene could have be involved in these marker-trait associations.

Table 1. Mapping of resistance on Chr 1A, including Bt3.

Gene/QTL name	Donor variety	Physical position	Comment	Reference
<i>Bt3</i>	‘Ridit’/‘Blizzard’	Chr1A: 495.06 – 499.90 Mbp		New mapping [109]
Q.DB.ui-1A	‘IDO444’	Chr1A: 503.31 Mbp	Possibly <i>Bt3</i>	[44,99]
Qbt.ifa-1A	‘Dimenit’	Chr1A: 355.2 - 515.2 Mbp	Possibly <i>Bt3</i>	[96]
NN	diversity panel	Chr1A: 473.97 Mbp	Possibly <i>Bt3</i>	[44]
Qbt.ifa-1AL	‘Blizzard’	Chr1A: 498.5–516.6 Mbp	Possibly <i>Bt3</i>	[55,56]
NN	diversity panel	Chr1A: 497.93–499.86 Mbp	Possibly <i>Bt3</i>	[43]
QCbt.dms-1A.3	diversity panel	Chr1A: 556.87 Mbp	Possibly <i>Bt3</i>	[92]
QCbt.dms-1A.2	diversity panel	Chr1A: 13.37 – 14,03 Mbp		[92]
QCbt.dms-1A.1	diversity panel	Chr1A: 4.38 Mbp		[92]
Bt_Mariann_1A	spelt	Chr1A: 1.23 – 10.42 Mbp		New mapping

Mappings on Chr 1B, Including Bt4, Bt5 and Bt6 Summarised in Table 2

We have fine-mapped both *Bt4* to a position 7.4-28.0 Mbp, and *Bt6* to a position 16.4 -28.0 Mbp at Chr 1B (Table 2). This confirms and improves previous mappings [58,59,110,111]. The genes *Bt4* and *Bt6* demonstrated the same phenotypic reaction when exposed to a diverse set of fungal races in both our own phenotypic trials and by others [34,36,64,74]. It cannot be finally concluded if the two genes are actually the same gene, but most indications point in that direction.

In variety 'Blizzard', a dominating resistance factor for resistance was identified between marker Xgwm374, Xbarc128 and Xgwm264 on Chr 1B [70], and the mapping was improved to a position 8–22 Mbp with a peak at gwm374 and gwm264, but these markers did not match in differential lines for *Bt4*, *Bt5* or *Bt6* gene [55]. Using our improved mapping of *Bt4* and *Bt6*, we can confirm that Qbt.ifa-1BS mapped in 'Blizzard' [55] and also mapped in 'Dimenit' [96] and in a diverse panel of varieties [44] are most likely mappings of the *Bt4* or *Bt6* gene.

We improved our previous mapping *Bt5* [112] to a decreased 120.7 Mbp interval at Chr 1B at 163.23–283.93 Mbp confirming it to be present in our accession of 'Hohenheimer' (Ci-11458) and in an accession of 'Hohenheimer' used at BOKU Austria provided directly to BOKU by Blair Goates (Hermann Burstmayer, pers. Comm.). The haplotype associated with the gene (Appendix A) is also present in a NIL line with *Bt5* developed by James McKay [113] and in a range of varieties including a number of European commercial varieties such as 'Genius', 'Promesse', 'Globus', 'Tommi', 'Bill' 'WPB Calgary', 'Apostel', 'Spontan', 'Bosporus', 'Tillsano', 'Initial', and 'Ikarus' with phenotypic reaction similar to this accession of 'Hohenheimer' [64,65]. The presence of *Bt5* in European breeding is in most cases not an effect of targeted breeding for resistance, but is most likely present randomly from original genetical background, and also the original German landrace 'Hohenheimer' may come from the same genetical background. However, when accession CI 11458 of 'Hohenheimer' is ordered from USDA NSGC today, a different line is provided with awns and susceptible to all the 10 races of bunt we use in our standard screening programme. We conclude that some error has occurred, and that CI 11458 today is not identical to the original accession of 'Hohenheimer' CI 11458 previously described in literature as a differential line for *Bt5* [35,63]. Despite the dual resistance genes in 'Hohenheimer' and the inconsistency in accession CI 11458, we are convinced our mapping is the *Bt5* gene referred to in the original publications [54,61–63].

The variety 'Trintella' has several factors for resistance, including a dominating factor on Chr 1B near to the centromere and closest to marker Xgwm273 [69]. The phenotypic reaction demonstrates that it is infected by several races avirulent to *Bt5*. Hence, the resistance in 'Trintella' is different from *Bt5*., despite having a resistance gene close to *Bt5*.

Mourad *et al.* associated resistance with Chr 1B at a position 137.13–163.10 Mbp in a diverse panel of varieties [43]. Several genes have been mapped to 1B, and it cannot be concluded if the close mapping to *Bt5* indicates involvement of the same genes or not.

Q Cbt.crc-1B.2 has been mapped in 'AC Domain' to 1BL and Q Cbt.crc-1B.1 to 1BS [71] and Qcbt.spa-1B was mapped in 'Carberry' to 1B [72]. The mapping of resistance in 'Carberry' to 1B was improved to a position at 21.4 Mbp [46]. Both Q Cbt.crc-1B.1 and Qcbt.spa-1B are in the vicinity *Bt6*, and could therefore be speculated to be *Bt6* [55]. However, 'AC Domain' are characterised to be only moderately resistant in Canada, and 'Carberry' descends from 'AC Domain' with no other parental lines with major resistance [71,72]. In a diverse panel of Canadian spring wheat varieties, resistance was associated with Chr 1B at position 21.0–21.4 Mbp, hence close to resistance mapped in 'Carberry' and 'AC Domain'. Despite the close chromosomal vicinity with the mapping of *Bt6*, the quantitative behaviour of resistances Q Cbt.crc-1B.1 or Qcbt.spa-1B in Canadian spring wheat varieties, demonstrate that these mappings of resistance are different from mappings of the *Bt6* gene, but only that the genes occur in the same chromosomal region.

Zou *et al.* mapped resistance in the Canadian spring wheat variety 'CDC Go' flanked by markers BS00086854_51 and wsnp_Ex_c5679_9976893 at Chr 1B [73] positioned at 517.23–551.90 Mbp. In a new study of 'CDC Go', resistance was not significantly associated with this position [46]. We have also not found race-specific resistance associated with this position at Chr 1B in our panel.

Galaev *et al.* introgressed dominant resistance into the intercalary region of Chr 1BL in bread wheat from the telomere region in *Ae. Cylindrica* [114]. No phenotypic reaction with different virulence races are presented, and based on the introgression from *Ae. Cylindrica*, it cannot be concluded if this is an already described gene in wheat or a new one.

Table 2. Mapping of resistance on Chr 1B, including *Bt4*, *Bt5*, *Bt6* a.o.

Gene/QTL name	Donor variety	Physical position	Comment	Reference
<i>Bt6</i>	‘Rio’	Chr 1B: 16.38 – 28.02 Mbp		Confirmed mapping [111]
<i>Bt4</i>	PI 11610	Chr 1B: 7.48 – 28.02 Mbp		Confirmed mapping [110]
QBt.ifa-1B	‘Dimenit’	Chr 1B: 7.60 - 18.00 Mbp	possibly <i>Bt6</i>	New mapping
NN	‘Blizzard’	Chr 1B: 8–22 Mbp	possibly <i>Bt6</i>	[55]
QBt.ifa-1BS	‘Dimenit’	Chr 1B: 2.2 and 46.9 Mbp	possibly <i>Bt6</i>	[96]
NN	diversity panel	Chr 1B: 11.18 Mbp	possibly <i>Bt6</i>	[44]
NN	‘Blizzard’	Xgwm374 Xbarc128 and Xgwm264	possibly <i>Bt6</i>	[70]
<i>Bt5</i>	Tommi/Starke NIL- <i>Bt5</i>	Chr 1B: 163.23 -283.93 Mbp		Confirmed mapping [112]
NN	diversity panel	Chr 1B: 137.13–163.10 Mbp	possibly <i>Bt5</i>	[43]
QCbt.dms- 1B.2	‘CDC Go’	Chr 1B: 551.90 – 517.23 Mbp	Minor effect gene	[73]
QCbt.dms-1B	‘Carberry’	Chr 1B: 21.4 Mb	Minor effect gene	[46]
QCbt.dms-1B	diversity panel	Chr 1B: 21.0-21.4 Mbp	Minor effect gene	[92]
QCbt.spa-1B	‘Carberry’	Flanking: wPt-667763–wPt-731722 (= 517.23 Mbp)	Minor effect gene	[72]
Q Cbt.crc-1B.2	‘AC Domain’	Xgwm403	Minor effect gene	[71]
Q Cbt.crc-1B.1	‘AC Domain’	Xgwm374.1 (173.53 Mbp) and Xwmc818b	Minor effect gene	[71]
NN	‘Trintella’	45 cM Xgwm273 near the centromere (Xgwm273 = WMS273: 218.88 Mbp)	Minor effect gene	[69]

Mappings on Chr 1D and 2A, Including *Bt2* Summarised in Table 4

Resistance gene *Bt2* was mapped to a 2.9 Mbp interval at 41.70 – 44.67 Mbp on Chr 1D (Table 4), and confirming and refining our previously mapping [48]. The mapping was confirmed to fit with the differential line PI 554097 and other lines descending from the original donor of *Bt2* ‘Hussar’.

Accession PI 554097 is the globally used differential line for *Bt2* [35,36], but the origin of this line is not described. Kinship analysis and morphological appearance indicate that it is a selection from a cross between ‘Elgin’ and PI 554102, and that PI 554102 is a selection from ‘Hussar’*‘Hard Federation’. Despite the unknown origin, we consider PI 554097 as a true carrier of the *Bt2* gene inherited directly from the original donor of *Bt2*.

The QTL Qcbt.spa-1D identified in spring wheat ‘Vesper’ was mapped to Chr 1D [49]. ‘Vesper’ was not included in our mapping panel, but position demonstrate that Qcbt.spa-1D is different from *Bt2*. However, the author mapped the gene to Chr 1D using RefSeg 1.0 [115], but the updated RefSeg 2.1 places the linked marker BS00066855_51 on Chr 1B [84] in the vicinity of the mapping of QCbt.dms- 1B.2 mapped in ‘CDC Go’ [73].

Table 3. Mapping of resistance on Chr 1D.

Gene/QTL name	Donor variety	Physical position	Comment	Reference
<i>Bt2</i>	‘Hussar’ /PI 554097	Chr 1D: 41.70 – 44.67 Mbp		Confirmed mapping [48]
QCbt.spa-1D	‘Vesper’	Chr 1D: 595.40 Mbp	Position uncertain	[49]

Mappings on Chr 2A (Table 4)

Qcbt.ifa-2A was mapped in a mapping population of ‘Mulan’ x ‘Dimenit’ between the markers Tdurum_contig29983_490 and AX-94381641 [96]. We fine-mapped this position to the apical end of Chr 2A beneath 35 Mbp. From recombination analysis we conclude that the resistance is inherited

from ‘Mulan’ and not from ‘Dimenit’, and hence not a part of the *Bt11* complex defined by the established differential line.

Qcbspa-2A was mapped in the Canadian spring wheat ‘Vesper’ to an interval 745.41-746.75 Mbp at Chr 2A 49. The mapping is considered distinct from resistance in ‘Mulan’ because of chromosomal distance of the mappings.

Table 4. Mapping of resistance on Chr 2A.

Gene/QTL name	Donor variety	Physical position	Comment	Reference
QBt.ifa-2A	‘Mulan’	Chr 2A: 0.3 - 35.09 Mbp		New mapping
QBt.ifa-2A	‘Mulan’*‘Dimenit’	Chr 2A: 259.75 - Mbp	Tdurum_contig29983_490 and AX-94381641	[96]
QCbt.spa-2A	‘Vesper’	Chr 2A: 745.40 – 746.74 Mbp		[49]

Mappings on Chr 2B, Including *Bt1* Summarised in Table 5

The *Bt1* gene was mapped to position 799.98 – 804.81 Mbp at Chr 2B (Table 5) and thereby confirming previously mapping [41].

On the same chromosome, Qcbsph-2B has previously been mapped in a diverse panel of varieties including varieties with *Bt1*, by two makers at position 566 and 1591 Mbp [42], and hence on each side of our new mapping of *Bt1*. In another study of a diverse panel of varieties, resistance was mapped to the interval 787.82–785.91Mbp, slightly beneath our mapping of *Bt1* [43]. Ehn *et al.* associated marker Ku_c71357_859 at the position 581.70 Mbp with bunt resistance and suggested that this could be *Bt1* [44]. The genetic position of these mappings in the vicinity of our mapping supports that these resistance-marker associations could indeed be caused by the presence of *Bt1*.

Q.DB.ui-2B was mapped in ‘IDO444’ [99]. Position associated with Xwmc317 at 14cM and parental information of ‘IDO444’ indicate that this is different from *Bt1*.

In Canadian spring wheat variety ‘CDC Go’, a QTL was also mapped to Chr 2B at 244 Mbp associated with bunt resistance [46]. The position and phenotypic behaviour indicate that this is different from *Bt1*.

The varieties ‘Hereward’, ‘Bussard’, ‘Skotte’ ‘Complet’, ‘Paroli’ and several other well known varieties used in European breeding over the past century have been shown to have a phenotypic reaction similar to *Bt2* [64,74]. Genotypic analysis of these varieties from the European genepool reveal that they have not inherited the *Bt2* gene from ‘Hussar’ mapped to Chr 1D, but have another race-specific resistance genes mapped to Chr 2B. We mapped this resistance, Bt_Bussard_2B, in ‘Bussard’, ‘Hereward’ and other European varieties to 8,70 – 13,12 Mbp at Chr 2B. The difference in position and phenotypic behaviour demonstrate it to be different from *Bt1* and other resistance genes identified on Chr 2B.

Table 5. Mapping of resistance on Chr 2B, including *Bt1*.

Gene/QTL name	Donor variety	Physical position	Comment	Reference
<i>Bt1</i>	‘Martin’/PI554 100	Chr 2B: 799.98 – 804.81 Mbp		New mapping [41]
NN	diversity panel	Chr 2B: 787.82–785.91Mbp	Possibly <i>Bt1</i>	[43]
NN	diversity panel	Chr 2B: 581.70 Mbp	Possibly <i>Bt1</i>	[44]
QCbt.cph-2B	diversity panel	Chr 2B: 655 – 1591Mbp	(position according to reference)	[42]
Bt_Bussard_2B	‘Bussard’	Chr 2B: 8.70 – 13.12 Mbp	Phenotypical identical to <i>Bt2</i>	[48]
QCbt.dms-2B	‘CDC Go’	Chr 2B: 244.0 Mb		[46]
Q.DB.ui-2B	‘IDO444’	Chr 2B: Peak: Xwmc317 14cM		[99]

Mappings on Chr 2D, Including Bt7 Summarised in Table 6

We have mapped *Bt7* to position 616.02 - 621.07 Mbp at Chr 2D, confirming and refining previous mappings [40,75]. Phenotypic reaction and presence of associated markers demonstrate that *Bt7* is present in a range of commercial varieties of both spring wheat and winter wheat in Europe, including ‘Tambor’, ‘Korrund’, ‘Xenos’, ‘Segor’, ‘Quarna’, ‘Fiorina’, ‘Thomaro’, ‘Sailor’ [64,74]. No other *Bt* genes have been mapped to Chr 2D.

Bunt resistance in ‘Lewjain’ was shown to be caused by a single major gene linked to SSR marker *wmc112* on Chr 2D, but based on phenotyping with different races, the authors argued this resistance to be different from *Bt7*, but possibly be *Bt8* [81]. We have not analysed ‘Lewjain’, but our mapping of *Bt8* to Chr 4A (see below) can dis-confirm the authors speculation of the resistance in ‘Lewjain’ pointing at *Bt8*. As no other genes have been mapped on Chr 2D and excluding *Bt7*, ‘Lewjain’ may have a hitherto unknown strong gene yet to be studied in further detail.

Table 6. Mapping of resistance on Chr 2D

Gene/QTL name	Donor variety	Physical position	Comment	Reference
Bt7	‘Martin’/PI554100	Chr 2D: 616.02 - 621.07 Mbp		Improved mapping [75]
NN	Lewjain	<i>wmc112</i>	Position uncertain	[81]

Mappings on Chr 3A, 3B and 3D Summarised in Table 7

None of the *Bt* genes were mapped to Chr 3A, but in the variety ‘Stephens’, we mapped two genes, one on Chr 3A a the position 683.12-688.69 Mbp and a second on Chr 7B (see below). This confirms previous findings pointing at ‘Stephens’ carrying one or more minor resistance genes effective against a few races [63]. In a diverse panel of Canadian spring wheat varieties, resistance was mapped to Chr 3A a position 671.29 Mbp [92]. Given that resistance in ‘Stephens’ and in Canadian spring wheat are described as minor additive genes, and the close vicinity of the mappings, it is possible that our mapping of *Bt_Stephens_3A* is the same gene associated with resistance in Canadian spring wheats. In the same study, additional resistance was associated with to Chr 3A at a position 10.28 Mbp [92]. In another study of a diverse panel of varieties, resistance was associated with positions 51.29–53.74 Mbp and also to a wide interval 69.957–742.47 Mbp [43]. The first position at Chr 3AS is definitely dfferent from *Bt_Spephens_3A*, but give the wide span of the latter, it cannot be excluded to co-locate with our mapping of resistance in ‘Stephens’.

Zou *et al.* mapped resistance in Canadian spring wheat variety ‘CDC Go’ associated with markers *RAC875_c17453_896* and *RAC875_c57584_240* at Chr 3A [73]. This correspond to position 1.48 – 251.79 Mbp [84]. It is difficult to interpret if this wide span between the markers is an interval or two different resistance factors, but re-mapping of ‘CDC Go’ could not confirm resistance significantly associated with Chr 3A [46].

In a diverse panel of varieties, resistance was mapped to Chr 3B [43] and in the variety ‘Lillian’, resistance was mapped to Chr 3D [49]. We found no resistance in our panel associated with Chr 3B or 3D, and we can therefore not correlate this finding to other genes.

Table 7. Mapping of resistance on Chr 3A, Chr 3B and Chr 3D.

Gene/QTL name	Donor variety	Physical position	Comment	Reference
Bt_Stephens_3A	‘Stephens’	Chr 3A: 683.12 – 688.69 Mbp		New mapping
QCbt.dms-3A.2	diversity panel	Chr 3A: 671.29 Mbp		[92]
NN	diversity panel	Chr 3A: 69.957 – 742.47 Mbp		[43]
NN	diversity panel	Chr 3A: 51.29 – 53.74 Mbp		[43]
QCbt.dms-3A.1	diversity panel	Chr 3A: 10.28 Mbp		[92]
QCbt.dms- 3A	‘CDC Go’	Chr 3A: 1.48 – 251.80 Mbp	Not confirmed in	[73]

		a later study	
		[46]	
NN	diversity panel	Chr 3B: 0.85 - 6.95 Mbp	[43]
QCbt.spa-3D	'Lillian'	Chr 3D: 3.12 - 3.98 Mbp	[49]

Mappings on Chr 4A, Including Bt8

We mapped *Bt8* close to the end of Chr 4AS below 16.86 Mbp. *Bt8* was originally found in Turkish mixed line 'Yayla-305' [31], but the differential line used for *Bt8* today is PI 554120 descending from landrace PI 173438 [35], and also present in landrace PI 178383 extensively used in American wheat breeding. We can confirm that the haplotype recommended for MAS (Appendix A) is present in both 'Yayla-305', in PI 554120, in PI 1 173438 and in PI 178383 confirming that the same *Bt8* gene is present in all three sources of resistance. No other studies have mapped bunt resistance genes on Chr 7A.

Our mapping of *Bt8* to the position at Chr 4AS contradicts the findings by Bokore *et al.* proposing *Bt8* to be at 7A [82], and also dis-confirm the hypothesis that the mapping of a single dominating resistance gene linked to Wmc112 on 2D in the variety 'Lewjain' co-locate with *Bt8* [81]. This however, does not rule out the presence of *Bt8* in 'Lewjain', as the marker may have been positioned on a wrong chromosome.

Mappings on Chr 4B, Including Genes Associated with Bt11 and Bt12 Summarised in Table 8

As described in the introduction, the donors and differential lines of *Bt11* and *Bt12* each include several bunt resistance genes, and it is therefore debatable which of them should be selected as *Bt11* and *Bt12*.

The landrace 'Dimenit' is the donor of *Bt11* in the differential line PI 554098, and Qbt.ifa-4BL was mapped in 'Dimenit' [96]. We re-mapped this gene to a position at 657.89 – 662.87 Mbp. This gene is not present in differential line PI 554098.

Qcvt.ifa-4BS was identified as resistance factor in 'Dimenit' on Chr 4B, but a position was not presented [96]. We mapped this gene to 15.74 – 17.79 Mbp confirming that it is distinct from Qbt.ifa-4BL. Qcvt.ifa-4BS is present in differential line PI 554098 and hence contributing to the excellent resistance of this line.

Also the differential line for *Bt12* PI 119333 include several bunt resistance genes, making it debatable which should represent *Bt12*. Qcvt.ifa-4B mapped in PI 119333 to a very large interval covering most of the chromosome 20.6–706.5 Mbp [98]. We investigated this in further detail and mapped two different genes in each opposite ends of Chr 4B, Bt_PI119333_4BL at 650,38 – 670,63 Mbp and another Bt_PI119333_4BS at 1,31 – 15,86 Mbp. We found virulence in our spore collection to both these resistance genes.

Both 'Dimenit' and PI 119333 have resistance genes in each of the opposite ends of Chr 4B mapped to overlapping intervals. The haplotype developed for MAS differs, but this could be due to the different donors of origin affecting the population structure of the markers. The genes have not been isolated into lines with only a single gene, and we have therefore not been able to test with different virulences if they demonstrate identical phenotypic reactions. We therefore cannot at present finally conclude if there are two, three or four genes involved in the *Bt11/Bt12* complex at Chr 4B.

Singh *et al.* also mapped Qcvt.spa-4B conferring common bunt resistance to a region on the short arm of Chr 4B in 'Carberry' [72]. This mapping was flanked by wPt-744434 – wPt-617 which does not overlap with our mapping, and hence most likely different from the genes mapped in 'Dimenit' and in PI 119333.

Table 8. Mapping of resistance on Chr 4B.

Gene/QTL name	Donor variety	Physical position	Comment	Reference
Qbt.ifa-4BL	'Dimenit'	Chr 4B: 657.89 – 662.87 Mbp	Not present in diff. line PI 554098	Improved mapping
Qbt.ifa-4BL	'Dimenit'	Chr 4B: 662.9 and 671.4 Mbp		[96]
Bt_PI119333_4BL	PI 119333	Chr 4B: 650.38 – 670.63Mbp		Confirmed mapping [116]
Bt_Dimenit_4BS	PI 554119/'Dimenit'	Chr 4B: 15.74 – 17.79 Mbp		New mapping
NN	'Dimenit'	not specified	different from Qbt.ifa-4BL	[96]
Bt_PI119333_4BS	PI 119333	Chr 4B: 1.31 – 15.86 Mbp		Confirmed mapping [116]
QBt.ifa-4B	PI 119333	Chr 4B: 20.6–706.5 Mbp		[98]
QCbt.spa-4B	'Carberry'	Position uncertain (167.48 Mbp)	flanking: wPt-744434 – wPt-617	[72]

Mappings on Chr 5A, 5B, 5D, 6A and 6D Summarised in Table 8

None of the Bt genes was mapped to Chr 5A, 5B or 5D, but new genes were mapped to these chromosomes.

In the original donor of *Bt8* 'Yayla 305', we identified two additional genes different from the *Bt8* gene, and not found in the differential line for *Bt8* PI 554120.

Bt_Yayla305_5A was mapped at 572.16 – 597.23 Mbp on Chr 5A demonstrating race-specific behaviour. In a diverse panel of varieties, resistance was mapped to Chr 5A: 568.05 - 613.55 Mbp [43], and in the spring wheat variety 'Lillian', resistance was mapped to Chr 5A at 659.01 - 658.82 Mbp in addition to resistance at Chr 7A (see below) [49]. Given the close mappings in these studies, it is possible that these are mappings of the same gene.

We mapped an additional gene Bt_Yayla305_5B in 'Yayla 305' on Chr 5B at a large interval 36.90 - 324.49 Mbp. Resistance in variety 'Trintella' was associated with marker Xgwm408 at Chr 5B, but genotyping demonstrate that 'Trintella' does not have the Bt_Yayla305_5B.

We did not find any genes on Chromosome 5D, 6A or on 6B and could therefore not confirm or dis-confirm mappings published to these three chromosomes [43,45,92].

Table 9. Mapping of resistance on Chr 5A, 5B and 5D.

Gene/QTL name	Donor variety	Physical position	Comment	Reference
Bt_Yayla305_5A	'Yayla 305'	Chr 5A: 572.16 – 597.23 Mbp		New mapping
	diversity panel	Chr 5A: 568.05 - 613.55 Mbp		[43]
Qcbt.spa-5A	'Lillian'	Chr 5A: 659.01 - 658.82 Mbp		[49]
Bt_Yayla305_5B	'Yayla 305'	Chr 5B: 36.90 - 324.49 Mbp		New mapping
NN	'Trintella'	Chr 5B: 0–19 cM, nearest marker Xgwm408		[69]
NN		Chr 5D: 544.28 – 545.10 Mbp		[43]
QCbt.dms-5D.1	diversity panel	Chr 5D: 244.10 Mbp		[92]
QCbt.dms-5D.2	diversity panel	Chr 5D: 565.87 Mbp		[92]
NN	diversity panel	Chr 6A: 431.92–611.86 Mbp		[43]
NN	'Kenyon'	Chr 6A: Not specified		[45]
NN	diversity panel	Chr 6B: 461.41–708.26 Mbp		[43]

Mappings on Chr 6D, Including Bt9, Bt 10 and Bt11 Summarised in Table 10

We mapped *Bt9* to the distal end of the Chr 6D above 490.7Mbp, confirming and refining previous mapping [85,117]. We can confirm that resistance mapped to Chr 6D in ‘IDO835’ [83,86] and in ‘UI Silver’ [87] are indeed mappings of the *Bt9* gene.

PI 178383 has been widely used as a donor of *Bt9* in both USA and Europe, and *Bt9* is confirmed to be present in varieties such as ‘Stava’, ‘Hallfreda’ and ‘Magnifik’. Using our proposed haplotype for MAS (Appendix A), we can confirm that the gene is present also in the original donor of *Bt9* ‘CI 7090’ confirming that the *Bt9* genes found in PI 178383 is identical in to the *Bt9* originating from ‘CI 7090’.

Qdb.ssdhui-6DL was mapped in ‘UI Silver’ to a position at 492.5 – 494.6 Mbp [87] and we improved the mapping to a position from 492.57 Mbp to the end of the chromosome. Based on parental information and position, we believe this mapping is a mapping of *Bt9*.

Virulence against *Bt9* is present in USA [99]. Infections in the differential line of *Bt9* are rarely reported in Europe [118,119], but low infections in the differential line for *Bt9* are seen in some trials [56,65,120]. When infected spikes of breeding lines or varieties with *Bt9* were observed in our nursery, spores were used to re-inoculate differential lines with *Bt9* to investigate if infection was a sign of emerging virulence. We also required infected plants with *Bt9* from other European countries, but we never managed to confirm virulence or to maintain the disease on the differential line with *Bt9* from spores from any of these infected plants. We consider it questionable if virulence to *Bt9* is present in Europe.

In the original donor of *Bt11*, the landrace ‘Dimenit’, a major factor for resistance, Qbt.ifa-6DL, has been mapped Chr 6D at 482.8–495.2 Mbp on top of the resistances mapped on Chr 4B [96]. We mapped this resistance in the differential line of *Bt11*, PI 554098, and in ‘Dimenit’ to an interval identical to the mapping of *Bt9* using haplotype recombination mapping. The question still remains if *Bt9* and Qbt.ifa-6DL from ‘Dimenit’ is the same gene. Qbt.ifa-6DL has not been isolated into a line without additional bunt resistance genes on Chr 4B, and no virulence against *Bt9* has been found in our spore collection enabling us to compare phenotypic reaction of *Bt9* and Qbt.ifa-6DL. Only one race (D-19) has been demonstrated to be virulent to *Bt11*, and this race is also virulent to *Bt9* [36]. We cannot on the current basis finally conclude if the two genes are identical or not.

We mapped *Bt10* to Chr 6D at position to 2.05 - 3.34 Mbp, confirming and refining previous mapping [72,88–90,121]. The recommended haplotype for MAS (Appendix A) confirms *Bt10* to be present in PI 178383 and also present in the original donors of *Bt10* ‘Greece 18’ (PI 116301) and ‘Mocho’ (PI 116306) [33]. Given the parental information available, and the position of the mappings, we can confirm that mapping of resistance factor Qdb.ssdhui-6DS in ‘UI Silver’ [87], the mapping of Qcbt.spa-6D in ‘AC Cadillac’ [72], ‘IDO835’ [86], and Qcbt.dms-6D in both ‘Peace’ [46], and in a diverse panel of Canadian spring wheat varieties [92] co-locate with our mapping of *Bt10*.

The term *BtZ* was introduced by Goates referring to resistance from the variety ‘Zarya’ [35]. We’ve mapped *BtZ* to an interval identical to the mapping of *Bt10*. Indeed, *BtZ* resembles *Bt10* in many respects including identical phenotypical reactions to all tested fungal races. If *BtZ* is caused by an introgression from *Thinopyrum intermedium*, we would expect failing markers on the standard TG26k chip in the vicinity of the *BtZ* gene indicating the alien origin compared with the standard genetic setup from bread wheat, but this has not been observed. We therefore conclude that bunt resistance in ‘Zarya’ is caused by a source in the pedigree other than introgression ‘Hybrid 599’. We cannot finally conclude if *BtZ* and *Bt10* are identical genes or two different genes.

Table 10. Mapping of resistance on Chr 6D.

Gene/QTL name	Donor variety	Physical position	Comment	Reference
<i>Bt10</i>	PI 178383	Chr 6D: 2.05 – 3.34 Mbp		Improved mapping [121]

<i>BtZ</i>	‘Zarya’/‘Tilliko’	Chr 6D: 2.05 – 3.34 Mbp		Improved mapping [122]
QCbt.dms-6D	‘Peace’	Chr 6D: 7.6 Mb	= <i>Bt10</i>	[46]
QCbt.dms-6D	diversity panel	Chr 6D: 7.43 Mbp	= <i>Bt10</i>	[92]
QCbt.spa-6D	‘AC Cadillac’	Flanking: wPt-672044–wPt-5114 (=6.17 Mbp)	= <i>Bt10</i>	[72]
Qdb.ssdhui-6DS	‘UI Silver’	Chr 6D: 1.4 – 2.1 Mbp	0	[87]
DB-6D1	‘IDO835’	Chr 6D: 1.77 Mbp	0	[86]
DB-6D2	‘IDO835’	Chr 6D: 6.97 to 7.29 Mbp	0	[86]
Qdb.ssdhui-6DL	‘UI Silver’	Chr 6D: 492.5 – 494.6 Mbp	0	[87]
<i>Bt9</i>	PI 554099	Chr 6D: 492.64 – 495.16 Mbp		Improved mapping [85,117]
Qbt.ifa-6DL	‘Dimenit’	Chr 6D: 482.8–495.2Mbp		[96]
Qbt.ifa-6DL	‘Dimenit’	Chr 6D: 492.57 - 492.64 Mbp		Improved mapping

Mappings on Chr 7A, Summarised in Table 11

Quebon is a French winter wheat with a broad resistance to common bunt [120], and including *Bt5*. We mapped *Bt_Quebon_7A* to Chr 7A position 671.34 – 676.63 Mbp. *Bt_Quebon_7A* has a race-specific behaviour similar to *Bt2* and *Bt_Bussard_2B*.

Qcbt.spa-7A was mapped in the Canadian spring wheat in ‘Lillian’ to a position at 693.40 - 598.87 Mbp Chr 7A [49], close to *Q Cbt.crc-7A* mapped in ‘AC Domain’ [71]. Both ‘Lillian’ and ‘AC Domain’ as well as the closely related ‘Neepawa’ and ‘Thatcher’ are known to have a quantitative type of resistance. Virulence to *Bt2* was found in 80% of American bunt races, a frequency much higher than virulence to other *Bt*-genes [123]. Testing varieties with a mixture of spores including both virulent and avirulent spores to a gene can give phenotypic results of race-specific genes with reduced infection in all races similar to quantitative genes. The presence of virulence to *Bt2* in Canada may explain the quantitative behaviour of resistance in Canadian spring wheat varieties. Therefore, it cannot be finally excluded that *Bt_Quebon_7A* can be involved in resistance in Canadian spring wheat.

Qbt.ifa-7AL was mapped in ‘Blizzard’ at position 722–737 Mbp [55], and *QDB.ui-7AL* was mapped in ‘IDO835’ to a position at 732.05 to 736.56 Mbp [83]. We have refined the mapping in ‘Blizzard’ to 717.92 – 735.89 Mbp confirming that this is distinct from *Bt_Quebon_7A*.

The fact that three genes, including *Bt2* at Chr 1D, *Bt_Bussard_2B* at Chr 2B, and *Bt_Quebon_7A* at Chr 7A gives the same phenotypic reaction similar to *Bt2* and are widely distributed in European winter wheat breeding, there may have been a selection pressure on the pathogen towards virulence against these genes in Europe. It is confirmed that half of the races collected in Europe have virulence against *Bt2*, a frequency far higher than virulence to other known *Bt*-genes [65].

In three different diverse panels of varieties, resistances have been mapped at Chr 7A to positions at 444.4 Mbp [42], 298.99 Mbp [43] and at 335.99 Mbp and 633.77 Mbp [44]. Also in ‘Trintella’ resistance has been associated with Chr 7A [69]. Resistance associated with single markers with different marker systems in particular in diversity panels are difficult to evaluate and compare, but the systematic findings of resistance associated with Chr 7A may indicate some similarities that could refer to a single or few common genes widely distributed in the genepool of modern varieties.

Table 11. Mapping of resistance on Chr 7A.

Gene/QTL name	Donor variety	Physical position	Comment	Reference
<i>Bt_Quebon_7A</i>	‘Quebon’/‘Hereward’	Chr 7A: 671.34 – 676.63 Mbp.		New mapping
<i>QBt.ifa-7AL</i>	‘Blizzard’	Chr 7A: 717.92 - 735.89 Mbp		Improved mapping
<i>QBt.ifa-7AL</i>	‘Blizzard’	Chr 7A: 722–737 Mbp		[55]
<i>QDB.ui-7AL</i>	‘IDO835’	Chr 7A: 732.05 – 736.57 Mbp		[83]
<i>QCbt.spa-7A</i>	‘Lillian’	Chr 7A: 598.87-693.40 Mbp		[49]

Q Cbt.crc-7A	‘AC Domain’	Chr 7A: 686.3–688.6 Mbp		[71]
QCbt.cph-7A,	diversity panel	Chr 7A: 444.4 Mbp		[42]
NN	diversity panel	Chr 7A: 335.99 Mbp		[44]
NN	diversity panel	Chr 7A: 633.77 Mbp	Position and Chr uncertain	[44]
NN	‘Trintella’	Uncertain position	32.7–48.5 cM nearest marker	[69]
NN	diversity panel	Chr 7A: 298.99 – 298.99 Mbp	Xpsp3050	[43]

Mappings on Chr 7B, Summarised in Table 12

Qbt.ifa-7B was identified as an additional resistance factor in ‘Dimenit’ (PI 166910) on Chr 7B at 10.07 -12.80 Mbp [96]. We have confirmed this mapping and conclude that it is not present in the differential line for *Bt11* (PI 554098) and hence not involved in the *Bt11* gene complex.

We also mapped another gene on Chr 7B from variety ‘Stephens’ at 630.88-638.50 Mbp. Given the genetic chromosomal distance, we consider this resistance distinct from Qbt.ifa-7B. Resistance in ‘Trintella’ has also been associated with 7B [69], but genotyping demonstrates that ‘Trintella’ does not have resistance from ‘Stephens’.

Qcbt.spa-7B.1 was mapped in spring wheat variety ‘McKenzie’ and associated with bunt resistance with peak markers Xgwm573 and Xwmc17 on Chr 7B [97]. In a diverse panel of varieties, resistance was associated with Chr 7B at position 703.15 Mbp [43]. Given the use of different marker systems and uncertain positions, it cannot be concluded if these mappings refer to the same resistance genes as listed above.

Table 12. Mapping of resistance on Chr 7B.

Gene/QTL name	Donor variety	Physical position	Comment	Reference
QCbt.ifa-7B	‘Dimenit’	Chr 7B: 10.07 -12.80Mbp		Improved mapping
QBt.ifa-7B	‘Dimenit’	Chr 7B: 7.1 -26.4 Mbp		[96]
NN	diversity panel	Chr 7B: 18.1 Mbp		[43]
Bt_Stephens_7B	‘Stephens’	Chr 7B: 612.10 - 643.48 Mbp		New mapping
NN	diversity panel	Chr 7B: 703.15 Mbp		[43]
QCbt.spa-7B.1	‘McKenzie’	Position uncertain	Peak: Xgwm573 and Xwmc17	[97]
NN	‘Trintella’	Chr 7B: 417 - 544 Mbp		[69]

Mappings on Chr 7D, Including *Bt12* and *Bt13* Summarised in Table 13

PI 119333 is the differential line representing *Bt12* [35], and Qbt.ifa-7DS was mapped in PI 119333 to the interval 6.47 – 10.84 Mbp [98]. We’ve mapped several genes in PI 119333, including *Bt7* and two genes at Chr 4B (see above). Our mapping of Qbt.ifa-7DS indicates a slightly elevated position at 14.62-20.93 Mbp. Within lines with this chromosomal interval inherited from PI 119333, we find three different virulence patterns, only one of which confers total immunity. We therefore conclude that the mapped interval at Chr 7D includes two different and independent resistance genes, and that total immunity to races in our collection only occurs when both genes are expressed.

‘Blizzard’ is another variety carrying multiple resistance genes including resistance mapped to Chr 7D at 12.5–15.3 Mbp [98]. In wheat line ‘IDO444’, a resistance factor Q.DB.ui-7DS has been mapped to position 5.88 Mbp and this is likely a mapping of the same gene, as ‘IDO444’ is a sibling of ‘Blizzard’ [99]. Chen *et al.* argues that Q.DB.ui-7DS fits with the differential line for *Bt12* [99].

We mapped the resistance in ‘Blizzard’ to a the position on Chr 7D at 16.71 - 21.84 Mbp overlapping with resistance in PI 119333 but with different haplotypes for lines with different origins of resistance. Therefore, we’re still unable to definitely determine if the Chr 7D resistance mapped in ‘Blizzard’ and ‘IDO444’ is identical to resistance mapped to Chr 7D in PI 119333.

The *Bt13* gene was mapped to an interval beneath resistance from ‘Blizzard’ and PI 119333 at 5,01 – 9,64 Mbp on Chr 7D, confirming previous mapping [101]. The phenotypical reaction and the chromosomal distance demonstrate that *Bt13* is different from the genes at Chr 7D found in ‘Blizzard’ and PI 119333.

A resistance factor Qcbt.spa-7D gave minor effect on bunt incidence in Canadian spring wheat variety ‘Carberry’ and was mapped to Chr 7D flanked by X664136 and Xwmc273 on [72]. Resistance associated with Chr 7D in ‘Carberry’ was not confirmed in a later study [46]. The *Bt13* gene and the genes on Chr 7D in PI 119333 demonstrate a clear race-specific behaviour and the chromosomal distance demonstrate that Qcbt.spa-7D mapped in ‘Carberry’ is different from resistance factor from our mapping.

In a diverse panel of varieties, a single marker on Chr 7D at 595.91 Mbp was associated with resistance [43], but given the position there is no indication of co-location with *Bt13* or resistance from ‘Blizzard’ or PI 119333 at 7D.

Table 13. Mapping of resistance on Chr 7D.

Gene/QTL name	Donor variety	Physical position	Comment	Reference
Bt_PI119333_7D-1	PI 119333	Chr 7D: 14.16 - 20.93 Mbp		New mapping
Bt_PI119333_7D-2	PI 119333	Chr 7D: 14.16 - 20.93 Mbp		New mapping
QBt.ifa-7DS	PI 119333	Chr 7D: 6.47 - 10.84 Mbp		[98]
QCbt.ifa-7D	‘Blizzard’	Chr 7D: 16.71 - 21.84 Mbp	=QBt.ifa-7DS	Improved mapping
Q.DB.ui-7DS	‘IDO444’	Chr 7D: 5.88 Mbp	=QBt.ifa-7DS	[99]
QBt.ifa-7DS	‘Blizzard’	Chr 7D: 12.5–15.3 Mbp	=Q.DB.ui-7DS	[55]
<i>Bt13</i>		Chr 7D: 5.01 - 5.66 Mbp		Improved mapping [101]
NN	diversity panel	Chr 7DL: 595.91 Mbp		[43]
NN	‘Carberry’	Position uncertain	Flanked: X664136 and Xwmc273	[72]

BtP

The landrace ‘7838’ (PI 173437) is included as a differential line to represent *BtP* [36]. We have not studied segregating RILs of crosses with PI 173437 and neither have we found literature references studying of *BtP* in any detail. The haplotypes recommended for MAS of the other genes described in this article (Appendix A) indicates that *Bt2* and *Bt7* may be present in PI 173437. Goates studied 56 bunt races, and found 12 races virulent to PI 173437. All 12 had also virulence to both *Bt2* and *Bt7*, but many other races virulent to both *Bt2* and *Bt7* were avirulent to PI 173437 [36]. In our own phenotyping, none of the races were virulent against PI 173437, but several had virulence to the combination of *Bt2* and *Bt7*. This indicates but does not definitely prove that PI 173437 has a combination of *Bt2*, *Bt7* and in addition one or more known or unknown genes. The term *BtP* as an expression of a specific gene must therefore be used with precaution and not be confused with the expression of the donor line PI 173437 as this may express the effect of a combination of several different resistance genes.

Race-specific resistance normally follows the gene-for-gene precepts [124–126], and it has been confirmed that this also is valid for the Bt-genes in the bunt-wheat interaction [123,127,128]. When studying the virulence of the pathogen against race-specific Bt-genes in wheat, it is important to take this into account. This means that infection levels in the differential set and other varieties governed by race-specific resistance genes will have a reaction either similar to a resistant line infected by an avirulent race (often close to zero) or a reaction similar to a susceptible line (often >40%) depending on environmental factors and minor race-non-specific resistance). Deviations from this binary response therefore indicate the influence of factors other than the direct interaction between Bt resistance genes and corresponding virulence genes.

A factor often causing problems when testing race-specific resistance, is heterogeneity in either the spores or in the wheat line, causing ambiguous results where it is difficult to conclude if the spores

are virulent or avirulent to the resistance of the wheat varieties tested, and also difficult to conclude if a given wheat variety has a resistance gene or not. If a line has a few infected plants, it cannot be finally determined if virulences are present or not, as the infection may have been caused by other factors.

The virulence of spores to a resistant line can in practice only be tested by taking spores from the few infected plants of a potentially resistant variety, and re-inoculating the varieties with these spores as proposed by Flor [126]. If the infection level is higher from this test than inoculation with the original spore sample, then the small infection was caused by presence of virulence in the spores, and that the original spores were a mixture of virulent and avirulent spores. If the infection level remains at the same level, then the low infection is a sign that virulence is absent, and that the wheat variety may either be genetically impure (being a mixture of resistant and susceptible lines), or the infection level is governed by race-non-specific resistance or environmental factors.

Common bunt is in practice predominantly seed borne, but this only covers secondary infections during seed multiplication from year to year. The initial introduction of infectious spores into the crop system comes from other sources, including infection from soil [129] or from spores dispersed by wind from neighbouring fields during harvesting, or from combine harvesters and other farm equipment [12,14]. This crucial distinction between primary infection and secondary spread of common bunt is important to take into account when evaluating the effect of different types of resistance mechanisms, in particular regarding race-specific versus race-non-specific resistance.

When spores are first introduced into the crop, race-specific resistance will either prevent infection or not, depending on the virulence of the spores. Therefore, race-specific resistance genes will reduce the risk of primary infection to a degree relative to the proportion of spores virulent against the resistance gene. If the crop is infected by virulent spores, the race-specific resistance will have no effect on the subsequent multiplication in later generations. In contrast, race-non-specific resistance will reduce the level of primary infections and will also reduce the rate of secondary multiplication from year to year, but race-non-specific resistance genes will only reduce, but rarely prevent primary infections in the pathogenesis of the disease, unless combined with genes or other control measures.

As infections increase by a factor 100 from year to year in susceptible varieties, common bunt must be reduced by 99% by the combined control mechanisms each year to prevent this general multiplication rate. As the threshold for bunt is extremely low, a single gene of a race-non-specific resistance will rarely be able to prevent multiplication. Race-specific resistance can prevent multiplications only against avirulent spores, but will have no effect against virulent spores. Therefore, a single resistance gene can rarely alone control common bunt, but a combination of genes, or resistance combined with other measures such as monitoring and discarding seed lots with primary infections can in combination control the disease and replace or reduce the need for fungicide application.

4. Materials and Methods

Origin, Development and Maintenance of Spores

Spores used in phenotyping of germplasm in this study was originally collected in the ORGSEED project (FØJO 2001). Spores were collected from infected seed samples submitted to The Danish Plant Directorate seed health laboratory for seed health analysis. The seed samples included both certified seed and home saved seed in the period 2002-2005. With such a broad material, we conclude that this material includes all virulences present in Denmark during this period. The material was at start not systematically tested against the differential set, but resistant varieties like '3540' (Bt1), 'Hereward' (Bt_Bussard_2B 4.1-40.9%), 'Tommi' (Bt5), 'Bill' (Bt5 0.2-21.0%) and 'Globus' (Bt5), 'Trintella' (1.8-64%) and 'Stava' (Bt9+Bt10 0.0-1.6%) showed low infections most years, but increasing infections from year to year 1999-2003 seen in 'Hereward', 'Bill' and 'Trintella' indicate presence of virulence [130-132].

To test the material for virulence, spores from infected plants of resistant varieties in the BIOBREED experiment and later follow up research were collected and used to re-innoculate this and other varieties expected to have the same resistance gene. In cases where infection increased to the level of susceptible controls, it was concluded that virulence was present and the spores used in further field trials as a new race with virulence against the Bt-gene. In this way, new races of common bunt can be developed, in line with [77]. After a few years, races with virulence against *Bt1*, *Bt2*, *Bt3*, *Bt5*, *Bt7*, *Bt10* and *Bt13* were developed [64,74]. Later, virulence against *Bt4/6* and *Bt8* have in the same way been developed in the disease nursery leaving only *Bt9* as a single Bt-resistance gene without virulence.

Phenotyping of Germplasm

Germplasm in the current study is study included resistance lines from the BIOBREED project [42] and differential lines with known resistance genes demonstrated in other research, additional germplasm from genebanks, research institutions, and RILs from breeders developing bunt resistant wheat and our own biparental RIL populations developed to map resistance gene. Each year, differential lines and other lines with known Bt-genes and other resistant varieties were included. A total of 2,731 lines was phenotyped for bunt infections by adding an excess of dry spores into paper bags with 50 seeds, and shaken, and sown by hand directly from the bags into the soil to avoid mixing of spores with different races through sowing equipment. After heading, each tiller was assessed for infection by visual inspection with a focus on development of sori in the head, and the percentage of infected tillers recorded [133]. Tillers with partially infected heads were assessed as infected whereas tillers expressing only leaf symptoms without sori development were recorded as healthy.

Comparing the infection level in each variety with each race, the varieties were grouped into categories with similar reactions to the different races. Varieties included in the same categories were postulated to have the same resistance gene(s). Comparing the groups with differential lines with known resistances, matching lines have been postulated to have the published Bt-genes, and none matching accessions have been postulated to have an undescribed resistance gene or a combination of resistance genes [64,74].

Based on phenotypic reaction to the different races, 1504 of the phenotyped accessions were selected for SNP genotyping using TG26k chip Illumina Infinium by Trait Genetics GmbH (Gatersleben, Germany, <https://www.traitgenetics.de>). Additional 589 accessions were genotyped and included in the analysis based on literature information about phenotypic reaction. Supporting data on phenotypic reaction and SNP genotyping from 183 accessions on the TG15k chip was provided by NORDGEN [113] and the ECOBREED project at BOKU, Austria [55,96,98]. Phenotyping results and SNP genotyping on the 90k chip was kindly provided by Idaho University [83,87].

Initial Resistance - Marker Association

For most of the Bt-genes *Bt1-Bt13*, NILs with 'Red Bob', 'Starke-II' or 'Prins' as the recurrent parent, have been developed and are available in USDA Small Grains Collection or NordGen [113,134]. In particular NILs from 'Starke-II' are very near isogenic and gave relevant information as to the position of the basic Bt-genes on the physical map RefSeq 2.1 [84].

Statistical analysis was based on the gene postulation based on phenotypic reactions where each line was characterised as resistant or not towards each virulence race. Lines with a postulated resistance gene were tested against the rest of the dataset using several different mapping methods to get approximate locations for postulated genes: NIL Mapping, GWAS, Composite NIL mapping and mappings from literature. GWAS against genepostulates was performed with the R-package GABIT, mainly using the MLM method, but also GLM, FarmCPU and Blink [114], with a FDR corrected P value of 0.05 used for significance. Physical positions and chromosome assignments for marker data were obtained by BLASTing markers against the IWGSC RefSeq v2.1 [84]. Markers with positive association to both phenotypic reaction and to a specific stable physical position were used

to estimate a brutto interval within which the gene must be positioned. We consider this only as the initial step of the mapping.

Second Step of Gene Mapping and Development of Haplotypes for MAS

Based on the preliminary mapping of the genes, each line with phenotypic reaction of the gene was analysed in further detail using a triplet analysis of each line with their two parents (if known) comparing the markers in the physical vicinity of the interval with markers statistically significantly associated with resistance postulation were inherited from the resistant or from the susceptible parent. In this way, it was possible to identify the crossover events and track the origin of the haplotype interval identified in the initial mapping of the genes. Tracking recombinations of all lines, a refined mapping of the genes were assigned to the smallest interval between crossover events. Examples and further details of the description of the refined mapping procedure are presented in previous publications [121]. Missing parental information was imputed [135].

A problem when working with SNP markers is the presence of monomorphic markers between susceptible and resistant lines. A small interval gives the best position of the gene itself, but is not always optimal for MAS if dominated with monomorphic markers. For MAS, a selection of markers statistically best linked to the resistance were chosen, and a haplotype of markers was developed with closest linkage to the resistance giving a minimum of false positive and false negative results. Haplotypes for MAS may in some cases include markers outside the mapping interval of the genes, in particular in cases where markers in the mapped interval are small or dominated by monomorphic markers.

5. Conclusions

Differential lines have been developed to discriminate races with different virulences, and for this purpose, differential lines may include single genes or combinations of multiple genes. However today, differential lines are often also used to represent and define specific genes. For this latter purpose, differential lines should ideally only have a single resistance gene. Regarding the differential lines representing the Bt-genes, we conclude that the use of the terms *Bt11*, *Bt12* and maybe *BtP* are misleading, as the differential lines have multiple resistance genes different from other described Bt-genes. Some of the Bt-genes may be identical. *Bt10* may be identical to *BtZ*, *Bt4* may be identical to *Bt6* and one of the genes in the differential line for *Bt11* at Chr 6D may be identical to *Bt9*. Both the donors of *Bt11* and *Bt12* resistances carry resistance genes on Chr 4BS that may be identical, and the same on Chr 4BL.

We have mapped a couple of new genes not included in the classic Bt-genes, and literature supports the conclusion that not only the classical Bt-genes are relevant in control of common bunt. Mappings of genes after the sequencing of the wheat genome have improved the credibility of mappings, and mappings supported by different independent studies including ads to credibility. In particular, we consider 9 genes relevant candidates to evaluated in further detail with regard to expand and revise the differential set:

- Bt_Mariann_1A
- QBt.ifa-2A (from 'Mulan')
- Bt_Bussard_2B
- Bt_Stephens_3A
- Bt_Yayla305_5A
- Bt_Yayla305_5B
- Bt_Quebon_7A
- Qcbt.ifa-7B (from 'Dimerit')
- Bt_Stephens_7B

More genes than these have been mapped, and in particular in Canadian spring wheat, genes have been mapped on Chr 1BS, 1BL, 1DL, 2AL, 3DS and 5DL. These genes, or doners of these genes

have not been thoroughly tested in our trials, and this is also the case for and the *Bt14* and *Bt15*. It would be relevant to investigate these genes in further detail, including to test the potential effect of the genes also in winter wheat, including to test the specificity to a broad range of races to check their quantitative, environmental dependency or race-specific behaviour.

Mapping bunt resistance genes using SNP markers is developing work in progress, but there is a limit as to how close it's possible to get to the genes if using only this method. The next step is therefore to clone the genes and to develop gene-specific markers for accurate MAS.

Common bunt was along with other seed-borne diseases called the forgotten diseases neglected by breeding and research [18]. We believe that the research described in this paper in different institutions in different countries and continents around the globe has returned common bunt resistance breeding on the scene as a significant tool to solve at the same time both agricultural, environmental and human health problems related to the strategy for control of both common bunt and dwarf bunt.

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Abbreviations

The following abbreviations are used in this manuscript:

Base pair	bp
Recombinant Inbred Line	RIL
Near Isogenic Line	NIL
Mega base pair	Mbp

ChromosomeChr

Recombinent Inbred LinesRIL

Nomen nescioNN

Appendix A

Table A1. This is a table caption.

Gene name	Chr	Minimum position	Maximum position	SNP Marker
Bt1	2B	799.991.847	803.471.859	1: Excalibur_c48404_59; Value: C
				2: wsnp_Ex_c15646_23969140; Value: A
				3: BS00065302_51; Value: G
				4: AX-94890379; Value: G
				5: BS00083998_51; Value: G
				6: Ra_c105904_187; Value: C
				7: Ra_c105904_1191; Value: G
				8: AX-158610188; Value: A
				9: AX-94808568; Value: G
				10: AX-158562114; Value: C
Bt2	1D	41.696.687	44.673.116	1: BS00011451_51; Value: G
				2: AX-94692978; Value: C
				3: tplb0044p22_2330; Value: C
				4: tplb0044p22_2257; Value: G
				5: AX-158571869; Value: C
				6: D_GBUVHFX01ANH44_230; Value: T
				7: wsnp_CAP11_rep_c4017_1896951; Value: T
				8: AX-94458136; Value: G
Bt3	1A	495.266.486	499.647.119	1: IACX5821; Value: G
				2: AX-94679138; Value: C
				3: AX-109834411; Value: T
				4: AX-158605760; Value: T
				5: AX-94632321; Value: A
				6: Ra_c62804_1641; Value: C
				7: Excalibur_c48379_413; Value: C
				8: Excalibur_c48379_116; Value: G
				9: Kukri_c27693_551; Value: G
				10: Excalibur_c23992_436; Value: C
				11: AX-94504536; Value: C
				12: IAAV5535; Value: C
				13: AX-158531952; Value: T
				14: RAC875_c102223_220; Value: C
				15: IACX3047; Value: G
				16: AX-158537471; Value: T
				17: AX-109401732; Value: A
				18: AX-94462466; Value: A
				19: AX-158564927; Value: T
				20: AX-111506455; Value: G
				21: Tdurum_contig32437_257; Value: T
				22: AX-109405334; Value: T
				23: IACX3595; Value: G
				24: wsnp_Ex_c55986_58282517; Value: T
				25: Ra_c105707_788; Value: T
				26: BS00089894_51; Value: C
				27: AX-158556605; Value: T
				28: AX-94462123; Value: T
				29: AX-158569077; Value: C
				30: wsnp_Ex_rep_c109742_92411838; Value: C
				31: Kukri_c73734_175; Value: C

				32: AX-158536689; Value: G
				33: wsnp_Ku_c21316_31053745; Value: T
				34: AX-86174938; Value: G
				1: Excalibur_c30569_384; Value: C
				2: BS00005004_51; Value: T
				3: AX-94668775; Value: G
				4: wsnp_BE405749B_Ta_2_1; Value: C
				5: Kukri_c36151_170; Value: G
				6: Kukri_c44392_212; Value: G
				7: BS00083562_51; Value: G
				8: AX-94997422; Value: G
				9: AX-94692514; Value: C
				10: BS00004903_51; Value: C
				11: AX-94926658; Value: A
				12: Ku_c1312_1194; Value: C
				13: AX-158607271; Value: G
				14: BS00064829_51; Value: C
				15: AX-94759371; Value: C
				16: Tdurum_contig58525_304; Value: MustFail
				17: AX-158540316; Value: C
				18: AX-158545235; Value: MustFail
Bi4	1B	7.480.290	28.018.966	19: IAAV2147; Value: G
				20: TA005766-0499; Value: G
				21: AX-158570866; Value: MustFail
				22: BS00108057_51; Value: MustFail
				23: BS00089524_51; Value: C
				24: AX-94602901; Value: C
				25: BS00074962_51; Value: MustFail
				26: BS00011695_51; Value: A
				27: AX-108893457; Value: A
				28: AX-158540300; Value: MustFail
				29: AX-94570104; Value: G
				30: AX-94800893; Value: MustFail
				31: TGWA25K-TG0215; Value: C
				32: Tdurum_contig50667_306; Value: C
				33: AX-111603368; Value: MustFail
				34: BS00110121_51; Value: G
				35: AX-94733902; Value: G
				36: Excalibur_c3270_1566; Value: A
				37: Excalibur_c3270_1566; Value: A
				1: BS00066165_51; Value: G
				2: BS00022625_51; Value: G
				3: BS00106581_51; Value: G
				4: AX-158556915; Value: A
				5: AX-110422876; Value: C
				6: AX-95248610; Value: A
				7: RAC875_rep_c72356_51; Value: C
				8: AX-158555575; Value: T
				9: Kukri_c19709_383; Value: T
Bi5	1B	164.434.858	271.683.038	10: AX-158561091; Value: C
				11: wsnp_BE637864B_Ta_1_1; Value: G
				12: AX-158540267; Value: C
				13: Kukri_c147_1620; Value: G
				14: BS00022317_51; Value: A
				15: AX-158561110; Value: G
				16: AX-158570854; Value: C
				17: AX-110035800; Value: A
				18: Kukri_rep_c105316_262; Value: T
				19: AX-158520903; Value: G

				20: AX-109447012; Value: C 21: BS00091191_51; Value: G 22: AX-108921445; Value: G 23: AX-158540090; Value: A 24: AX-111514779; Value: C
<i>Bt6</i>	1B	16.466.442	25.179.826	1: BS00011695_51; Value: A 2: AX-108893457; Value: A 3: AX-158540300; Value: MustFail 4: AX-94570104; Value: G 5: AX-94800893; Value: MustFail 6: TGWA25K-TG0215; Value: C 7: Tdurum_contig50667_306; Value: C 8: AX-111603368; Value: MustFail 9: BS00110121_51; Value: G 10: AX-94733902; Value: G 11: Excalibur_c3270_1566; Value: A
<i>Bt7</i>	2D	621.068.206	622.542.240	1: RAC875_c30919_311; Value: G 2: RAC875_rep_c114621_200; Value: C 3: wsnp_Ex_c42970_49408712; Value: A
<i>Bt8</i>	4A	1.887.532	16.857.414	1: AX-94471577; Value: C 2: AX-158542180; Value: A 3: BS00021716_51; Value: C 4: BS00106545_51; Value: A 5: AX-108808218; Value: G 6: AX-158542161; Value: C 7: BS00043286_51; Value: G 8: wsnp_Ex_c14478_22481430; Value: C 9: AX-94381780; Value: T 10: BS00108852_51; Value: T 11: AX-111488229; Value: G 12: AX-95104288; Value: C 13: BS00065863_51; Value: T 14: AX-94715337; Value: A 15: AX-94409394; Value: C 16: wsnp_BE405275A-Ta_1_1; Value: G 17: wsnp_RFL_Contig2771_2524880; Value: G 18: AX-94514459; Value: G 19: AX-89419039; Value: C 20: AX-158524876; Value: C 21: wsnp_Ex_c28429_37553452; Value: A 22: AX-158542163; Value: C
<i>Bt9</i>	6D	494.217.075	495.158.655	1: AX-109985406; Value: T 2: RAC875_rep_c104893_620; Value: C 3: RAC875_c2910_1562; Value: G 4: AX-109853614; Value: A 5: BS00070856_51; Value: T 6: RFL_Contig2615_982; Value: T 7: RFL_Contig2615_700; Value: A 8: AX-94841369; Value: A 9: AX-94747666; Value: T 10: AX-109917993; Value: C
<i>Bt10</i>	6D	2.388.914	3.140.478	2: Kukri_c55362_75; Value: A 3: AX-108746724; Value: C 4: Excalibur_c7731_2743; Value: A 5: AX-158531240; Value: C 6: wsnp_Ex_c18664_27540364; Value: G
<i>Bt13</i>	7D	5.542.839	7.432.654	1: Ra_c30952_531; Value: T 2: AX-158544378; Value: T 3: AX-94708419; Value: G

				4: TA001746-1415; Value: G
				5: Kukri_c80931_147; Value: A
BtZ	6D	2.388.914	3.140.478	1: wsnp_Ex_c14439_22426200; Value: T
				2: Kukri_c55362_75; Value: C
				3: AX-108746724; Value: C
				4: Excalibur_c7731_2743; Value: G
				5: AX-158531240; Value: T
Bt_Blizzard_7A	7A	623.904.095	728.815.254	1: IAAV5550; Value: C
				2: BS00084193_51; Value: C
				3: Kukri_c28968_130; Value: G
				4: BS00105531_51; Value: C
				5: BS00027226_51; Value: A
				6: BS00002510_51; Value: T
				7: BS00067682_51; Value: C
				8: BS00068033_51; Value: C
				9: Kukri_c51453_406; Value: T
Bt_Blizzard_7D	7D	16.714.316	21.837.392	1: RAC875_rep_c104791_336; Value: G
				2: Kukri_c20949_503; Value: T
				3: AX-95229555; Value: T
				4: Kukri_c12113_837; Value: A
				5: AX-94411546; Value: T
				6: AX-158601965; Value: C
				7: BobWhite_c20306_88; Value: A
				8: BobWhite_c11327_185; Value: T
				9: BobWhite_c11327_248; Value: G
				10: TA005893-0466; Value: G
				11: BobWhite_c30138_69; Value: T
				12: GENE-4277_295; Value: C
				13: Excalibur_c1310_414; Value: G
				14: Excalibur_c16355_712; Value: T
				15: AX-158559855; Value: T
Bt_Dimenit_6D_1	6D	494.217.075	495.158.655	1: AX-109985406; Value: C
				2: RAC875_rep_c104893_620; Value: C
				3: RAC875_c2910_1562; Value: G
				4: AX-109853614; Value: G
				5: BS00070856_51; Value: G
				6: RFL_Contig2615_982; Value: C
				7: RFL_Contig2615_700; Value: A
				8: AX-94841369; Value: T
				9: AX-94747666; Value: T
				10: AX-109917993; Value: T
Bt_Dimenit_6D_2	6D	494.217.075	495.158.655	1: AX-109985406; Value: C
				2: RAC875_rep_c104893_620; Value: T
				3: RAC875_c2910_1562; Value: A
				4: AX-109853614; Value: G
				5: BS00070856_51; Value: G
				6: RFL_Contig2615_982; Value: C
				7: RFL_Contig2615_700; Value: G
				8: AX-94841369; Value: T
				9: AX-94747666; Value: T
				10: AX-109917993; Value: T
Bt_PI119333_7D	7D	14.163.614	20.292.538	1: Excalibur_c833_1405; Value: G
				2: AX-94735600; Value: G
				3: AX-111070966; Value: A
				4: Ex_c25027_535; Value: T
				5: wsnp_Ra_c8297_14095831; Value: T
				6: BS00110642_51; Value: T
				7: AX-94930280; Value: A
				8: AX-94398131; Value: A

				9: BobWhite_c8454_782; Value: G
				10: RAC875_rep_c104791_336; Value: G
				11: Kukri_c20949_503; Value: T
				12: AX-95229555; Value: T
				13: Kukri_c12113_837; Value: A
				14: AX-94411546; Value: T
				15: AX-158601965; Value: C
				16: BobWhite_c20306_88; Value: A
				17: BobWhite_c11327_185; Value: T
				18: BobWhite_c11327_248; Value: G
				1: AX-158564576; Value: C
				2: Tdurum_contig81460_347; Value: T
				3: AX-94572741; Value: G
				4: AX-109946437; Value: G
				5: Tdurum_contig11733_825; Value: T
				6: AX-158538742; Value: A
				7: BS00039935_51; Value: G
				8: BS00039936_51; Value: A
				9: BS00063809_51; Value: G
				10: AX-158542337; Value: T
				11: AX-95190182; Value: G
				12: AX-158598944; Value: A
				13: BS00060041_51; Value: T
				14: AX-95143067; Value: T
				15: AX-158538739; Value: A
				16: AX-158542312; Value: A
				17: AX-109388531; Value: A
				18: AX-158538740; Value: A
				19: AX-111481149; Value: A
				20: AX-158564641; Value: A
				21: AX-158583365; Value: A
				22: AX-158542333; Value: C
				23: AX-89538793; Value: G
				24: Tdurum_contig10322_1908; Value: A
				25: wsnp_Ra_c9755_16200944; Value: T
				26: Tdurum_contig93710_409; Value: A
				27: BS00037094_51; Value: A
				28: AX-158564494; Value: G
				29: AX-158583339; Value: G
				30: AX-158542402; Value: C
				31: Tdurum_contig47622_234; Value: A
				32: AX-110907280; Value: T
				33: Tdurum_contig67399_676; Value: A
				34: AX-110382283; Value: C
				35: AX-158542410; Value: G
				36: Tdurum_contig76559_124; Value: G
				1: Tdurum_contig9893_492; Value: C
				2: RAC875_c51375_394; Value: C
				3: AX-94448564; Value: G
				4: AX-95258779; Value: G
				5: AX-109865770; Value: T
				6: AX-110958367; Value: T
				7: BobWhite_c4256_213; Value: A
				8: AX-158598904; Value: C
				9: BS00104279_51; Value: T
				10: wsnp_Ex_c4148_7494801; Value: T
				11: AX-111537772; Value: G
				12: BS00034148_51; Value: T
				13: AX-111607064; Value: T

				14: AX-158550172; Value: A
				15: AX-94492644; Value: C
				16: AX-158582661; Value: C
				17: AX-94433424; Value: G
				18: AX-158550140; Value: C
				20: Tdurum_contig29983_490; Value: C
				21: AX-109964399; Value: C
				22: Ex_c19516_3687; Value: C
				23: RFL_Contig174_406; Value: A
				24: AX-158573559; Value: G
				25: BobWhite_c13373_250; Value: G
				26: TGWA25K-TG0117; Value: T
				27: TG0117; Value: T
				28: AX-94446514; Value: A
				29: wsnp_Ex_c11950_19164191; Value: T
				30: AX-94956032; Value: G
				31: wsnp_Ku_c33374_42877546; Value: C
				32: Kukri_c33374_1048; Value: T
				33: tplb0032i02_1388; Value: C
				34: Excalibur_c12980_2621; Value: A
				35: Excalibur_c12980_2392; Value: A
				36: RAC875_c42847_141; Value: T
				37: IAAV8501; Value: T
				38: RAC875_c2300_1021; Value: A
				39: RAC875_c63883_76; Value: A
				40: BS00022760_51; Value: C
Bt_Mulan_2A	2A	259.719	34.967.976	41: AX-94944993; Value: G
				42: IACX6178; Value: C
				43: CAP12_c259_307; Value: T
				44: AX-94679104; Value: C
				45: AX-94906650; Value: C
				46: RAC875_c829_1143; Value: C
				47: RAC875_c829_355; Value: T
				48: IACX11417; Value: A
				49: Kukri_c29358_277; Value: T
				50: BS00039973_51; Value: T
				51: BS00021706_51; Value: A
				52: AX-95126447; Value: G
				53: TA003766-0683; Value: G
				54: BS00093990_51; Value: C
				55: AX-158540813; Value: T
				56: RAC875_rep_c111906_144; Value: A
				57: AX-94381659; Value: T
				58: AX-94717890; Value: A
				59: CAP8_rep_c8022_270; Value: C
				60: BobWhite_c2022_245; Value: G
				61: CAP11_c2293_200; Value: G
				62: wsnp_Ex_c61879_61748626; Value: A
				1: Tdurum_contig76559_124; Value: G
				2: Excalibur_c7581_791; Value: C
Bt_Dimenit_4BS	4B	15.848.436	17.149.339	3: Excalibur_rep_c79414_306; Value: A
				4: AX-110579601; Value: G
				5: Tdurum_contig76213_958; Value: C
				6: Tdurum_contig82942_681; Value: G
				1: TA003210-1094; Value: A
Bt_Dimenit_4BL	4B	658.074.570	662.257.892	2: wsnp_BE403378B-Ta_2_1; Value: C
				3: BS00027054_51; Value: G
				1: AX-94463677; Value: A
Bt_Quebon_7A	7A	656.861.151	679.827.267	2: AX-158566892; Value: T

				3: AX-158566889; Value: T
				4: AX-94721829; Value: C
				5: Excalibur_c84687_162; Value: G
				6: RAC875_c20121_561; Value: A
				7: AX-94531661; Value: T
				8: AX-158590659; Value: A
				9: AX-158589980; Value: A
				10: BobWhite_c1215_240; Value: G
				11: AX-158567102; Value: C
				12: BS00088825_51; Value: G
				13: BobWhite_c15352_394; Value: G
				14: BS00071478_51; Value: T
				15: AX-158553168; Value: C
				16: RAC875_c37085_317; Value: A
				17: Kukri_c24408_743; Value: T
				18: JD_c149_1700; Value: T
				19: BobWhite_c12302_389; Value: T
				20: Ra_c14761_1348; Value: T
				21: wsnp_Ku_c42539_50247333; Value: C
				22: BobWhite_c1201_384; Value: T
				23: wsnp_Ku_c42539_50247426; Value: A
				24: AX-158567056; Value: A
				25: BS00023128_51; Value: A
				26: AX-158543577; Value: G
				27: wsnp_Ex_c9428_15641609; Value: C
				28: wsnp_Ex_c9428_15641639; Value: A
				29: AX-158556233; Value: T
				30: BS00021657_51; Value: T
				31: AX-158559587; Value: A
				32: AX-158625860; Value: G
				33: Excalibur_c95707_285; Value: T
				34: AX-158556230; Value: G
				35: BS00026622_51; Value: G
				36: AX-158537273; Value: C
				37: wsnp_JD_c20555_18262317; Value: A
				38: RAC875_c19111_628; Value: C
				39: AX-108837168; Value: A
				40: AX-94439426; Value: G
				41: IAAV6957; Value: G
				42: tplb0045p11_893; Value: T
Bt_Bussard_2B	2B	8.697.088	13.124.351	1: RAC875_rep_c71112_400; Value: A
				2: Excalibur_c34937_710; Value: T
				3: AX-94831339; Value: C
				4: AX-94880001; Value: A
				5: AX-109959677; Value: A
				6: BS00023068_51; Value: C
				7: AX-158562561; Value: T
				8: BS00044332_51; Value: C
				9: BS00084668_51; Value: A
				10: wsnp_Ex_c1996_3754394; Value: T
				11: RAC875_rep_c115433_378; Value: C
				12: AX-94505732; Value: T
				13: TGWA25K-TG0159; Value: A
Bt_Mariann_1A	1A	1.208.845	10.424.272	1: RAC875_c95364_259; Value: A
				2: Tdurum_contig44888_837; Value: C
				3: BS00033749_51; Value: G
				4: BS00026456_51; Value: T
				5: AX-89562713; Value: G
				6: TGWA25K-TG0108; Value: T

				7: TG0108; Value: T
				8: Ku_c28007_1398; Value: A
				9: AX-111569969; Value: A
				10: AX-158555547; Value: A
				11: AX-158560734; Value: C
				12: AX-158569633; Value: G
				13: BS00073243_51; Value: T
				14: BS00023201_51; Value: G
				15: BS00022355_51; Value: C
				16: AX-110068701; Value: C
				17: wsnp_Ex_c57982_59470152; Value: A
				18: AX-110004070; Value: C
				1: RAC875_c10194_673; Value: C
				2: AX-95132491; Value: A
				3: Excalibur_c29600_173; Value: C
				4: AX-158533015; Value: G
				5: wsnp_Ex_c1894_3575749; Value: G
				6: RAC875_c15003_377; Value: C
				7: BS00063696_51; Value: A
				8: Kukri_c8465_54; Value: G
				9: AX-89724344; Value: A
				10: AX-109295307; Value: T
				11: BS00039498_51; Value: G
				12: wsnp_Ex_c12341_19693570; Value: G
				13: wsnp_Ex_c12341_19693090; Value: A
				14: AX-108911182; Value: T
				15: AX-158538215; Value: A
				16: wsnp_Ex_c27317_36522052; Value: C
				17: wsnp_CAP11_rep_c4226_1995152; Value: G
				18: IAAV1410; Value: T
Bt_Stephens_3A	3A	683.220.645	688.690.639	19: IAAV5370; Value: A
				20: wsnp_Ex_rep_c66357_64540428; Value: T
				21: Ex_c66357_866; Value: C
				22: wsnp_Ra_c132_291198; Value: G
				23: wsnp_Ex_rep_c66357_64540369; Value: T
				24: Kukri_c12079_204; Value: A
				25: Tdurum_contig59585_656; Value: A
				26: wsnp_JD_c29019_23208279; Value: A
				27: AX-158538218; Value: T
				28: AX-158538236; Value: T
				29: BS00081610_51; Value: T
				30: AX-95003297; Value: C
				31: BS00023337_51; Value: G
				32: BS00060029_51; Value: A
				33: BS00088756_51; Value: C
				34: BS00088755_51; Value: T
				35: AX-86168015; Value: G
				36: BobWhite_c5337_225; Value: T
				37: AX-158523254; Value: G
				1: AX-158554033; Value: G
				2: AX-94676341; Value: C
				3: RAC875_c4834_694; Value: T
				4: AX-158554039; Value: G
Bt_Stephens_7B	7B	612.396.641	643.412.748	5: AX-94505411; Value: T
				6: Ku_c5351_1820; Value: G
				7: BobWhite_rep_c66630_331; Value: C
				8: AX-158592651; Value: C
				9: BobWhite_c12256_96; Value: T
				10: AX-158567766; Value: C

				11: AX-158592634; Value: A
				12: AX-108870188; Value: T
				13: AX-89599935; Value: C
				14: BS00022045_51; Value: C
				15: wsnp_Ex_c10550_17231294; Value: C
				16: AX-110432367; Value: A
				17: RAC875_c27548_417; Value: G
				18: BS00089942_51; Value: G
				19: RAC875_c27548_234; Value: T
				20: AX-158592661; Value: G
				21: wsnp_Ku_c17161_26193994; Value: A
				22: wsnp_Ku_c17161_26193672; Value: T
				23: GENE-4624_79; Value: C
				24: AX-158567774; Value: A
				25: Kukri_c51101_351; Value: T
				26: RAC875_c24101_284; Value: C
				27: AX-158544029; Value: C
				28: AX-95021316; Value: C
				29: AX-110369629; Value: G
				30: RAC875_c21489_908; Value: C
				31: IACX486; Value: C
				32: AX-158591808; Value: C
				1: AX-94406443; Value: A
				2: Tdurum_contig71499_211; Value: G
				3: wsnp_Ex_c7266_12475249; Value: C
				4: wsnp_Ex_c1138_2185522; Value: A
				5: AX-158584923; Value: G
				6: AX-158542656; Value: C
				7: Excalibur_c45297_316; Value: A
				8: AX-109433082; Value: T
				9: wsnp_Ex_rep_c69647_68598463; Value: G
				10: AX-158620334; Value: G
				11: AX-158558755; Value: A
				12: Tdurum_contig82190_124; Value: C
				13: AX-94432465; Value: C
				14: AX-109331427; Value: T
				15: AX-158551020; Value: C
				16: Tdurum_contig44343_1039; Value: G
				17: AX-158542740; Value: C
				18: AX-158551088; Value: G
Bt_Yayla305_5A	5A	572.654.222	597.231.033	19: AX-108744896; Value: C
				20: AX-110426237; Value: C
				21: Ku_c19858_2078; Value: C
				22: AX-94619088; Value: A
				23: BS00076246_51; Value: C
				24: Tdurum_contig86202_145; Value: C
				25: Tdurum_contig86202_175; Value: A
				26: BobWhite_c23736_153; Value: A
				27: Tdurum_contig52695_388; Value: T
				28: AX-109849058; Value: A
				29: AX-89764932; Value: G
				30: GENE-3601_145; Value: C
				31: AX-94837642; Value: T
				32: AX-158550911; Value: C
				33: AX-158565171; Value: T
				34: BS00044408_51; Value: C
				35: AX-89311025; Value: T
				36: wsnp_Ku_c20011_29589514; Value: T
				37: wsnp_Ku_c20011_29589289; Value: A

				38: wsnp_Ku_c20011_29589089; Value: G
				39: Excalibur_c37943_221; Value: T
				40: AX-158550736; Value: A
				41: AX-95629509; Value: C
				42: wsnp_Ra_c12183_19587379; Value: G
				43: Kukri_c29560_455; Value: G
				44: BS00065481_51; Value: T
				45: AX-158584285; Value: G
				46: AX-111040754; Value: G
				47: AX-158584445; Value: T
				48: AX-94442743; Value: G
				49: AX-111483425; Value: G
				50: AX-158538907; Value: G
				51: AX-158538950; Value: A
				52: AX-111072968; Value: A
				53: RAC875_c86041_91; Value: C
				54: TG0053; Value: C
				55: TGWA25K-TG0053; Value: C
				56: TG0020; Value: C
				57: TGWA25K-TG0041; Value: G
				58: TG0041; Value: G
				59: BS00075959_51; Value: A
				60: AX-158584403; Value: G
				61: AX-158542533; Value: A
				62: BS00088851_51; Value: C
				63: CAP11_c3209_76; Value: A
				64: AX-158585018; Value: C
				65: AX-94391667; Value: A
				66: AX-158584526; Value: T
				67: AX-109435061; Value: A
				1: AX-158534310; Value: A
				2: AX-158526437; Value: T
				3: AX-94612603; Value: G
				4: Tdurum_contig25068_259; Value: T
				5: Kukri_s113060_116; Value: C
				6: AX-158526315; Value: A
				7: AX-94691166; Value: G
				8: AX-94467784; Value: T
				9: wsnp_Ku_c7872_13484038; Value: G
				10: Ex_c2571_987; Value: G
				11: wsnp_Ex_c58012_59490259; Value: C
				12: GENE-0782_747; Value: A
				13: BS00074315_51; Value: G
				14: JD_c16284_736; Value: C
Bt_Yayla305_5B	5B	48.668.280	284.765.351	15: BobWhite_c4852_323; Value: G
				16: AX-110419826; Value: C
				17: TA006084-0922; Value: A
				18: wsnp_Ku_c32477_42087329; Value: G
				19: wsnp_Ex_rep_c104986_89538820; Value: C
				20: BS00067028_51; Value: G
				21: wsnp_Ra_c5210_9289264; Value: T
				22: wsnp_BQ166999B-Ta_2_1; Value: T
				23: Kukri_c13224_551; Value: A
				24: wsnp_Ex_c12431_19823475; Value: G
				25: wsnp_Ex_c24577_33826666; Value: G
				26: AX-158526448; Value: G
				27: RAC875_c96137_101; Value: C
				28: Kukri_c40388_844; Value: A
				29: IAAV8999; Value: A

	30: IAAV4590; Value: G
	31: wsnp_Ex_c18519_27369737; Value: C
	32: AX-158565742; Value: C
	33: AX-158526336; Value: C
	34: AX-158525931; Value: C
	35: Excalibur_c97201_294; Value: G
	36: wsnp_Ex_c658_1294003; Value: G
	37: AX-158539087; Value: C
	38: AX-158534082; Value: G
	39: AX-158533793; Value: C
	40: wsnp_CAP7_c2086_1018815; Value: G
	41: BobWhite_c36054_53; Value: A
	42: wsnp_Ex_c39535_46808105; Value: G
	43: Kukri_c31961_630; Value: T

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