International Common Bunt Consortium – Newsletter No. 2 December 2024

Editors: Anders, Monika, Claire and Dennis

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Introduction

This is the second newsletter from the International Common Bunt Consortium (ICBC).

Many things happened during the last year leading to exciting progress and even more valuable interconnection between researchers working on common bunt.

We received reports during the season that the disease spread even more widely across Germany, Europe and the world. Breeding for complete resistance in adapted genetic background with high yield potential is becoming more and more important. Since we need complete resistance to this fiend, stacking of several genes together is the only durable and reliable strategy.

The last round of phenotypic data has been evaluated and results are described below in detail by Dennis gene by gene. Anders clarified status of virulences. The joint collaborative project “Brandresist” (Forschungsprojekte-Julius Kühn-Institut) started at Julius-Kühn Institute with Albrecht Serfling and a fresh enthusiastic PhD student Claire Ferreira. New trials have been sown and are growing in the greenhouse at JKI, but also in the fields at several partners.

Resistance genes have so far been denominated Bt-genes, and many researchers and breeders all over the world have used a differential set proposed by Blaire Goates (2012) for characterising resistance. A differential line in a proposed differential set is ideally a line with a single resistance gene. As described in further detail in a chapter Differential Set Update below, this is by far the case for the current differential set. So far, most researchers have worked with the 16 Bt- resistance genes mentioned by Goates (2012), but recent segregation studies combined with genetic mapping has demonstrated that some of these Bt-genes (eg. Bt11, Bt12) are actually not single genes, but a pyramid of other Bt-genes or combinations with so far unknown resistance genes. Some genes are called by the same Bt-number but actually being different genes (eg. Bt2 or Bt8). Some new genes have been described with no relation to any known Bt-genes. Some Bt-genes should therefore be deleted and other new genes be included. By the end of the day, the number of resistance genes has increased rapidly in the past year from the original 16 genes to at present 59 genes. This newsletter aims at giving an update and attempt to clear up the mess regarding the Bt- resistance genes.

We aim to describe all genes with a physical position on the wheat genome or with a link to one or more known markers, and to identifying lines or varieties with a single resistance gene to be used as differential line.

XXII International Workshop on Bunt and Smut Diseases 2023

The most recent update on the bunt research can be found in the following book of Abstracts: https://boku.ac.at/fileadmin/data/H03000/H97000/H97100/pdf/Book_of_Abstracts_Bunt_and_Smut_Workshop_2023.pdf

This is the summary of the workshop held at the BOKU Campus in Tulln, Austria. It took place from the 13th until the 15th of June 2023.

XXIII International Workshop on Bunt and Smut Diseases 2025

The registration for the coming workshop to be held in Sweden 2025 is now open (https://www.efpp2025.com/registration/).

As well as abstract submission (https://www.efpp2025.com/abstract-submission/).

We are already looking forward to seeing you all face to face.

List of consortium members / Welcome to New Members

1.	Anders Borgen	Agrologica
2.	Anja Hanemann	Breun
3.	Ahmed Jahoor	Nordic Seed
4.	Almuth Müllner	Saatzucht Donau
5.	Andreas Stahl	Julius Kühn Institut
6.	Andy Forbes	Brockwell Bake/wheat-gateway.org.uk
7.	Anna Berlin	SLU
8.	Carl Vollenweider	Dottenfelder Hof
9.	Dennis Kjær Christensen	Privat
10.	Felix Bemm	KWS
11.	Fluturë Novak	Uni Rostock
12.	Manuel Geyer	LFL Bayern
13.	Herbert Bistrich	Saatzucht Donau
14.	Herbert Voelkle	GZPK
15.	Hermann Bürstmayr	BOKU
16.	Jihad Orabi	Nordic Seed
17.	Karl-Josef Müller	Cultivari
18.	Kostya Kanyuka	NIAB
19.	Lorenz Hartl	LFL Bayern
20.	Magdalena Lunzer	BOKU
21.	Mahbubjon Rahmatov	SLU
22.	Wolfgang Maier	Julius Kühn Institut
23.	Marwan Alamrani	SLU

24.	Matilda Ciuca	Nardi
25.	Jenny Matthiesen	KWS
26.	N.N. Saulescu	Nardi
27.	Nadine Schreiber	Secobra
28.	Neele Wendler	KWS
29.	Nick Fradgley	CSIRO
30.	Pernille Merete Saarup	Nordic Seed
31.	Phil Howell	NIAB
32.	Monika Spiller	KWS
33.	Therese Bengtsson	SLU
34.	Tina Henriksson	Lantmannen
35.	Verena Weyermann	GZPK
36.	Veronika Dumalasova	VURV
37.	Jost Doernte	DSV
38.	James Cockram	NIAB
39.	Johanna Holmblad	Lantmannen
40.	Hendrik Hanekamp	Landwirtschaftskammer Niedersachen
41.	Jianli Chen	University of Idaho
42.	Guriqbal Singh Dhillon	University of Idaho
43.	Matias Dominguez	University of Idaho
44.	Pabitra Joshi	University of Idaho
45.	Albrecht Serfling	Julius Kühn Institut
46.	Claire Ferreira	Julius Kühn Institut
47.	Ronja Wonneberger	SLU
48.	Kevin Gauthier	Agroscope

Status for European Virulence Races

Infection in a resistant variety is not a final proof of the presence of a race being virulent to a resistance gene. Infection in a line may be caused by impurity in the line, or by the resistance gene being partly depending on environmental factors. To document presence of true virulence, spores need to be collected from resistant lines and used for re-inoculation to demonstrate increased infection rate.

Diseases that spread with soil, wind or by water are normally linked to a specific geographical region, but common bunt is mainly spread via seed and may therefore spread over long distances following seed trade rather than from neighbouring fields. Therefore, the virulence of common bunt is more relevant to relate to a seed system or continent rather than a single country or climatic zone. Seed is traded freely within the EU, and it is therefore relevant to consider Europe as one zone of bunt dispersal, and North America another, since seed exchange of infected seed between the continents are limited.

Dwarf bunt on the contrary is more likely to stay stable in virulence within a smaller region.

Virulence is found against most resistance genes in Europe. Bt11 and Bt12 have so far being considered safe genes in Europe, but as described below, these genes are not single genes, but combinations of several genes. The differential lines used to describe Bt11 contains a mixture of several genes, and it is still unknown if virulence is present in Europe against any of these single genes. The same is the situation with Bt12.

Several genes are called Bt8 but comes from different sources and are most likely quite different. The differential line for Bt8 suggested by Blaire Goates is PI 554120 (with PI 173438 as source). Virulence against "Bt8" in PI 554120 is present in Europe. In both USA and Europe, PI 178383 has been used in breeding as a source of "Bt8", but this "Bt8" is most likely different from the Bt8 in the differential line. It is unknown if virulence is present in common bunt in Europe against "Bt8" coming from PI 178383 or from the original source 'Yayla 305', but virulence against "Bt8" coming from PI 178383 is most likely present in dwarf bunt on the island Gotland (Sweden). More research is ongoing about the understanding of Bt8 complex and possible virulence to the genes involved.

Bt9 has been properly mapped confirming that it is a single gene. There are indications of infection in lines having Bt9, but the efficiency of Bt9 is partly climate dependent and it still needs to be confirmed that it is true virulence under field conditions, and not just random infection in off type plants.

Most other genes can be infected by bunt spores from Europe, including Bt1-7, Bt10, Bt13-15 and BtZ. We have not found any virulence to BtP, but little is known about this gene, based on the results from other genes, it may be worth investigating if BtP is really just a single gene or if it could be another example of a combination of other genes.

Status for the mapping of the Bt genes

Bt1

Bt1 has been mapped to chromosome 2B as presented at the Tulln Workshop 2023 (Christensen and Borgen 2023D).

See page 45 from the book of abstracts

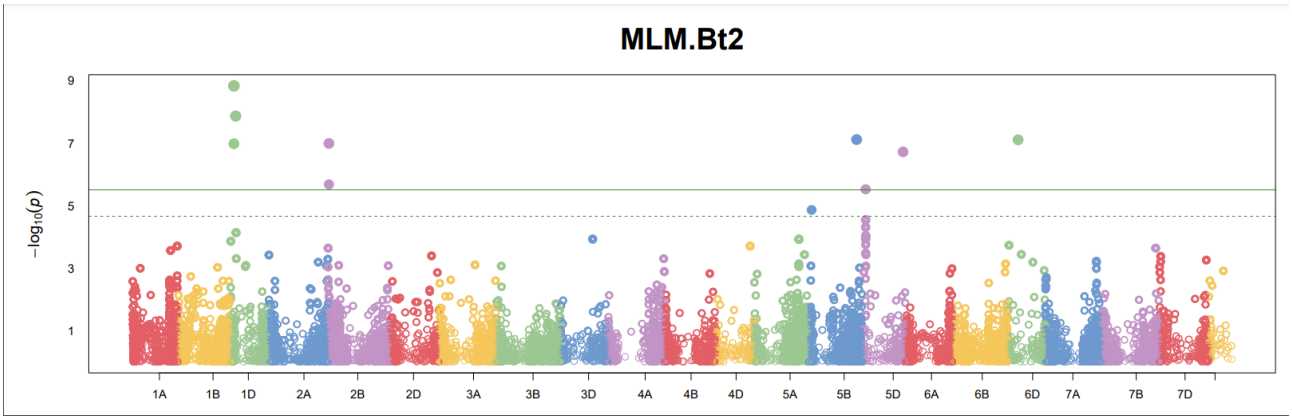
Recently, the mapping has been improved and a reduced interval of markers can be used for MAS.

Table 1 Markers that can be used to map Bt1

AX-158609666	A
Excalibur_c48404_59	C
wsnp_Ex_c15646_23969140	A
BS00065302_51	G
AX-94890379	G
BS00083998_51	G
Ra_c105904_187	C
Ra_c105904_1191	G
AX-158610188	A
AX-94808568	G
AX-158562114	C
Kukri_c49784_86	A

Bt2

Mapping Bt2 has been difficult and we finally have an idea why. So far, four different genes have been identified giving identical infection patterns in phenotyping with the used virulence races and two/three of them (Bt2 differential + Bussard (Bt_Bussard+ Bt_magnifik_5D)) even behave identically across all 44 virulences races in the PAN Europe experiment (Borgen *et al* 2023). This resulted in lack of power in the GWAS and the signals are still not impressive, but at least consistent between Blink, FarmCPU and MLM. Observe the lack of signal at 7A, where we have BtH from an earlier analysis.



The four genes with virulence pattern identical to Bt2 are:

1. The real Bt2 found in Hussar and present in the differential PI 554097 on Chromosome 1D
2. Then there is BtHereward/BtH/BtQ_7A. This gene is found alone in Skagen and Inthaler and some breeding lines.
3. One gene at 2B preliminary named Bt_Bussard_2B. This gene appears to be responsible for the Bt2-like resistnce in varieties such as 'Dream', 'Paroli' and 'Complet'.
4. One gene at 5D preliminary named Bt_Magnifik_5D. This gene is found alone in a few breeding lines descending from 'Skotte' or'Quebon', but it is not possible to say anything definite about the phenotyping pattern.

Fine mapping of the four genes is a work in progress and the quality is still low. Due to lack of parents/offspring triplets it is not possible to identify recombination events precisely and simple haplotype comparison is not as robust.

Virulence against Bt2 is present in ~50% of all bunt races in Europe (Borgen *et al* 2023A). This can be explained by the fact that the three new genes found to give the same virulence pattern as the original Bt2 is unintended present in many European varieties.

Some lines have markers for multiple "Bt2" loci and we do not know how many loci/genes are actually present. Some notable examples are:

- ‘Hereward’ with the markers for Bt2, Bt_Bussard_2B and BtH
- ‘Bussard’ with Bt_Bussard_2B and Bt_Magnifik_5D
- ‘Butaro’ with Bt_Bussard_2B
- Bt_Magnifik_5D(and Bt7)
- ‘Skotte’ with Bt_Bussard_2B and Bt_Magnifik_5D
- ‘Quebon’ and ‘Format’ with BtH, Bt_Bussard_2B and Bt_Magnifik_5D.

Plenty of material exist that can be used for mapping. ‘Hereward’ is a founder in the NIAB 8 founder MAGIC population and ‘Format’ + ‘Bussard’ are founders in the BMW MAGIC population. Hussar has been used in US wheat breeding and many lines descending from it are available from genebanks.

Bt3

Bt3 has been mapped to 1A, and is further described below in chapter “Dimenit and Bt11 Differential Genes”

Bt4 and Bt6

Bt4 and Bt6 are behaving identically with all virulence races so far in historic trials and in Anders, BOKU and Utah trials (100+). Both genes are at 1B and are either the same gene or tightly linked. Many historic and well characterized Bt4 containing lines were genotyped. We have a good mapping of Bt6 and studying haplotypes in an extended interval around the Bt6 interval in the Bt4 lines gave a reasonable mapping. The Bt4 and Bt6 intervals are overlapping and Bt4/Bt6 lines have identical haplotypes in the Bt6 interval.

For breeding and for the differential set there is no value in keeping both Bt4 and Bt6, and no harm.

Bt5

Dennis mapped the Bt5 to an interval on 1B (163,225,664 – 283,930,031 bp) (unpublished) and markers are available for MAS. Due to lack of marker polymorphism across the interval, we get many false positives. In our panel 30%. There is much better marker polymorphism just outside the interval and we can get the false positive rate down to 5% by adding some markers outside the interval, but at the cost of an increased false negative rate, from 5 to 10 %.

Our panel had very few lines where we had the parents as well, and it was therefore not possible to detect recombination events. We genotyped 90 lines described in the old papers this year and many of them have Bt2 in addition to Bt5. This helped improve the mapping very much.

Dennis had to resort to haplotype comparisons and this is not as solid as detecting recombination events in parents-offspring triplets. Fortunately, we have new populations in the pipeline where the parents were selected to give good marker contrast in the candidate interval.

Also see the Bunt resistance breeding at Saatzucht Donau GmbH & CoKG (Probstdorf, Austria) in the chapter below.

Bt7

Bt7 has been mapped to 2D as presented at the Tulln Workshop 2023 (Christensen and Borgen 2023A).

Bt8

Bt8 was discovered in 'Yayla 305' (PI 178210) by Waud and Metzger (1970) and used as differential until the Goates (1996, 2012) update, where 'M72-1250' (PI 554120) replaced it. 'M72-1250' (PI 554120) inherits Bt8 from '7845' (PI 173438), which was shown to have it by a test cross to 'Yayla 305' (PI 178210). Neither of the two sources of Bt8 have been genetically mapped in either of these varieties and it is not firmly established that 'M72-1250' (PI 554120) is monogenic and its resistance is identical to Bt8 in 'Yayla 305' (PI 178210). Also the 'Yayla 305' (PI 178210) Bt8 gene is possibly not monogenic. The Turkish landrace '6256' (PI 178383) was shown to carry Bt8 and has served as the major source of Bt8 in especially US wheat breeding, but also in Sweden.

As a first step towards making a Starke II based Bt8 NIL, Anders has crossed Starke II and PI 554120. From this population we have genotyped and phenotyped 13 RILs. Two of these RILs are nearly NILs (98.6% identical), one with Bt8 and one without, and Dennis used them to get a few candidate intervals. All but one on 4B could be dismissed by including a few more RILs and the parents.

The phenotyping results give a clear indication that PI 554120 has two genes. We have no good mapping of the second gene.

From testing the 4B marker block in the entire panel, we conclude that it looks promising to be the dominating gene Bt8 from PI554120.There are approximately 200 Bt8 lines in the field in 2024. Approximately half descending from PI 554120 and half from Magnifik (and therefore from PI 178383 via Stava), which is another potential assigned carrier of Bt8.

We have no solid, or even semi solid, information on the original Bt8 described in 'Yayla 305' yet. The genebank accession of Yayla 305 seems heterogeneous which complicates the search for Bt8 in this original source.

Bt9

Bt9 has been mapped to 2B (Steffan et al 2017) and a refined mapping was presented at the Tulln Workshop 2023. (Christensen and Borgen 2023B)

Later research demonstrated that two of the markers published were wrong. They are not at 6D and have been removed (Thanks Almuth!). The interval has not changed but the new markers for MAS are:

Table 2 New Markers for mapping Bt9

Kukri_rep_c107605_164	T
wsnp_CAP8_rep_c4586_2232878	C
wsnp_CAP7_c1735_859875	G
wsnp_CAP7_c1735_859744	T

Bt10 and BtZ

Bt10 and BtZ are two genes with a lot in common and may even be identical. Phenotypically they are identical, and they are both present at Chromosome 6D. However, the mapping is not 100% identical.

Bt10 has been mapped to 6D (Laroche *et al* 2000, Menzies *et al* 2006) and a refined mapping was presented at the Tulln Workshop 2023. (Christensen and Borgen 2023C)

BtZ has been also been mapped to 6D as presented at the Tulln Workshop 2023. (Chrisenten and Borgen 2023E)

After the Tulln workshop, Dennis went back to the BtZ mapping and redid it getting a slightly different result.

Anders has made a small RIL population from the cross Starke II x Inna. Lines are called NIL-Z because they are the first step towards making a Starke II based BtZ NIL.

Table 3 Markers to potentially detect BtZ

RAC875_rep_c118305_446	T
BS00065960_51	C
Kukri_c73802_205	A

Orange markers are flanking the QTL interval potentially including BtZ. Green marker may be used for MAS, but monomorphic in this population

There are two RefSeq High Confidence genes in that interval

Table 4 RefSeq High Confidence genes

Chromosome	Phys Pos Min	Phys Pos Max	Gene	Function
Chr6D	4363458	4366232	TraesCS6D03G0022800	Receptor-like protein kinase
Chr6D	4533591	4537917	TraesCS6D03G0023800	F-box family protein

Bt10 and BtZ behaves identically with all virulence races used in the PAN Europe trial (Borgen *et al* 2023A) and in all previous trials by Anders and they map to near identical intervals at 6D. The original BtZ interval presented last year in Tulln overlapped with the Bt10 interval, but the new one presented here does not. Dennis is convinced that the new Bt10 interval is inaccurate and that BtZ = Bt10 = TraesCS6D03G0022800. Phenotyping 75 Saatsucht Donau lines from a Bt10 x BtZ cross we found 1-2 lines with a single or a few infected heads. It remains to be investigated whether this is true segregation or an error.

BtZ is supposed to be present in Zarya as a *Thinopyrum intermedium* introgression, inherited from AG.IN via PPG-599 and Lutescens.126-65.

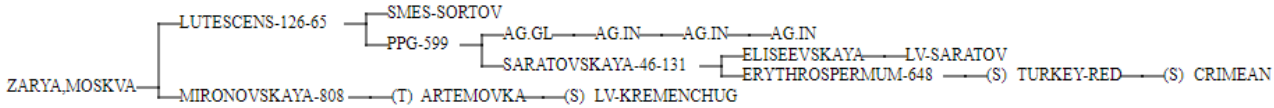


Figure 1 Phylogenetic tree

It seems a bit unlikely that such an introgression should contain Bt10 and end up at 6D. To investigate that, Mironovskaya 808 and PPG-599 will be phenotyped. Lutescens-126-65 and Smes-Sortov are available from the Vavilov Institute (https://www.wheat-gateway.org.uk/search.php?send=1&per=50&search=sortov&bunt_a=1&genes=1&simple=1). Thank you to Andrew Forbes for the information!

Bt11 and Dimenit Genes

Lunzer *et al* 2023 mapped a number of loci/genes in the Bt11 differential line 'M82-2123' (PI 554119) and in 'Dimenit' (PI 166910).

Four biparental mapping populations were used: M82 2123 x Mulan, Rainer x Dimenit, Dimenit x Rainer and Dimenit x Lukullus. Three loci at 4BS, 4BL and 6DL were mapped in M82-2123 and a fourth locus at 2A was segregating in that population. In the Rainer x Dimenit and Lukullus x Dimenit populations two loci at 4BS and 6DL were mapped. Dimenit was the donor. It turned out that Dimenit was not homogenous and in the Dimenit x Rainer population, and three (or four) additional loci at 1A, 1B and 7B were found. It is uncertain whether the 4BL locus was identified in this population as the 4B QTL spanned the entire chromosome.

All phenotyping and genotyping data were made public – excellent! Thank you BOKU!

Dennis (unpubllished) did a detailed analysis of the data and found that our Bt3 markers detected the gene at 1A, and the Bt6 markers detected the gene at 1B. The Bt7 markers were also validated. Bt7 is not very effective against the virulence used in the BOKU study and was therefore not detected, but still this gene was segregating in the population.

During the initial population validation, Dennis discovered that the Dimenit selection with the extra genes was identical to the one we have later genotyped. Dennis also discovered lines having the wrong parents and lack of inbreeding/selection, especially in the Dimenit x Lukullus population.

The detailed analysis identified recombination events that could be used to refine the intervals from the QTL mapping, and in most cases gave much smaller intervals. However this was not possible for the 4BL and 7B loci as these were masked by the other loci in most lines and not very effective against the used inocula.

The 6D locus gives immunity to both inocula used by Lunzer *et al* (2023), but interestingly, the 4BS locus was highly resistant to the BOKU "house keeping" inoculum, but the "aggressive" inoculum used in 2022 was able to give infections in the 12-24 % range.

Unfortunately we have not managed to get lines from the mapping populations with each gene isolated, into the field trial of 2024, except for a few exceptions; U11.15 with the 6D locus and U11.50 with the 7B

locus. From the 2022 trial, we know that breeding lines from Anders with the 6D locus alone is fully resistant to all eight virulence races in Anders´ core set.

Getting lines with 4BS and 4BL alone in the 2024 trial was a high priority, but we did not succeed in finding such lines.

Two lines have recombination in the Bt3 interval (U11.114 and U11.84) but they also have the 6D gene that is masking the presence or absence. Perhaps we can find a virulence race virulent on 6D but not on Bt3 to lift the mask. We could also cross the lines to susceptible varieties to test if Bt3 segregates. **Any volunteers? ;)**

A big question is whether the 6D QTL in Dimenit may be identical to the Bt9 gene. We have this evidence so far:

1) Abdallah *et al*. (1984) found Bt7, Bt9 and Bt11 in Dimenit
2) Bt9 and Dimenit 6D both are at 6DL
3) The haplotypes of Bt9/Bt11 lines are very different around the mapped intervals
4) There is no overlapping of Dennis's intervals for Bt9 and Dimenit 6D so far

Additional evidence can be collected by phenotyping 6D lines with the common bunt races L-21/T35 (Goates 2012) which are virulent to Bt9, but not to Bt11. **Any volunteers?**

Based on our analysis, we preliminarily suggest replacing Bt11 by two new assigned Bt gene numbers and also exchange the line in the differential set by the separate QTL carriers. Nevertheless, a bit of validation work has to be done still.

Bt12 and other PI 119333 Genes

Bt12 was mapped by Muellner *et al* (2020) to 7DS in the interval 6.5 – 10.8 Mbp in RefSeq 1.0 positions. Chromosome 4B was found to contribute some resistance too. The 7DS interval was found not to be 100% linked to Bt12, meaning that it is a signal only and actually an exclusion interval.

BOKU kindly gave Dennis access to genotypic and phenotypic data, which he analysed.

Anders phenotyped 37 lines from the mapping populations with 7-9 virulence races and we found that two genes at 7D and two at 4B could explain the infection patterns.

To get a mapping at 4B, Dennis analysed markers across the chromosome and found 18 recombination events, giving 19 intervals. Interval 1 at 1,306,080 – 15,855,852 bp was found to be harbour some resistance gene(s). Intervals 2-14 were not related to the presence of resistance and interval 15-19 at 648,869,446 – 670,633,612 bp (end of chromosome) again provided resistance.

In this table, we see the infection patterns for the four genes and for their combinations. It was very difficult to figure this out based on the patterns from the 37 lines only and there may be errors in it. There may also be inaccuracies and errors in the phenotyping and the table should not be taken as the final word.

Table 5 Infection pattern from Bt4, Bt12 and their combinations

	Vr0	Vr2	Vr-3	Vr-5	Vr10	Vr-13	Vr-Z	Vr-DOT	Vr-Iran
Bt4B_1			Bt4B_1		Bt4B_1				
Bt4B_2		Bt4B_2		Bt4B_2	Bt4B_2				
Bt12A	Bt12A		Bt12A	Bt12A					
Bt12B	Bt12B			Bt12B	Bt12B				
Bt4B_1+Bt4B_2					Bt4B_1+Bt4B_2				
Bt12A+Bt12B	Bt12A+Bt12B								
Bt4B_1+Bt12A Bt4B_2+Bt12A			Bt4B_1+Bt12A						
Bt4B_1+Bt12B	Bt4B_1+Bt12B				Bt4B_1+Bt12B				
Bt4B_2+Bt12B				Bt4B_2+Bt12B	Bt4B_2+Bt12B				
Bt4B_1+Bt4B_2+Bt12A									
Bt4B_1+Bt4B_2+Bt12B					Bt4B_1+Bt4B_2+Bt12B				

We see that the perceived strength of "Bt12" comes from the good combining ability of the four genes in PI 119333.

Bt13

Bt13 has been mapped to 7D as presented at the Tulln Workshop (Christensen and Borgen 2023F). There is so far no indications that Bt13 is identical to other Bt-genes found at 7D.

Bt14

Differential set line "Doubbie" - a durum- carrying resistance Bt14 has been crossed into hexaploidy wheat by Farrer in Australia (Watson, 1958) and germplasm from this cross, called accession '186' (PI 172201), is available. This accession is heterogenous, six head selections have been made and they have been genotyped and phenotyped this year. The lines turned out not to be homogenous and phenotyping results are not conclusive. It appears that three genes are segregating. Further line creation, phenotyping and genotyping is in progress. With a bit of luck, we will be able to make enough pure lines to map the genes.

Bt15

Bt15 is found in durum wheat Carleton. But only little is know about this gene.

BtP

BtP is present in the line PI 173437. Little is known about this gene, but no virulence has so far been found in Europe. In our humble opinion it is very doubtful that BtP is one gene.

New Genes

Many "new" genes are in the pipeline. Too many that we know very little about, but a few deserve to be mentioned.

Bt_Mariann_XX

A RIL population from the cross (Magnifik x Tataros) x (Spelt x Sebesta Blue) is called Mariann. Phenotyping of 33 lines with the core virulence set in Anders nursery identified a unique infection pattern in three RILs. The underlining gene is called Bt_Mariann_XX and we have no mapping for it. Two lines showed a Bt2'ish pattern, probably from Bt_Magnifik_5D.

Bt_Mariann_XX can explain the full resistance in the remaining lines together with Bt_Magnifik_5D, but "Bt8" from PI 178383 via Magnifik can also be present. 100 more lines are in the pipeline.

Bt_Blizzard_7A (QBt.ifa-7AL)

Muellner et al (2020B) mapped four loci in 'Blizzard' and 'Bonneville'.

Anders phenotyped 24 lines from the mapping population selected to carry single genes and we found infection patterns consistent with Bt3, Bt6, Bt7, and in addition this pattern for the 7A locus.

No lines with the 7D locus alone was found, but a number of lines show complete resistance that cannot be explained by the other genes.

Mapping Overview

Mapping status for all the 59 genes we are currently working on is collected in a spreadsheet "MappingOverview.xlsx". Is being maintained by Dennis. Please do not hesitate to ask for it.

Differential Set Update

Today, most researchers use the same differential set and Bt-gene denomination, and refer to Blair Goates (2012) as the key reference. As mentioned above and debated in Borgen *et al* (2023B) in the Tulln Bunt Workshop 2023, novel discovery in genetic research calls for a revision of the differential set and Bt-gene denomination, as the existing set of differential lines also has a few other problems including that

- The differential line for Bt5 'Hohenheimer' probably has not only Bt5 but also another gene at 1B.
- The differential line for Bt11: 'M82-2123' (PI 554119) also has an additional gene.
- The differential line for Bt12 '' (PI 119333)) has two (or more) genes at 7D and two (or more) at 4B.

The lines are very un-adapted to actual (European) field conditions and highly susceptible to yellow rust and powdery mildew.

Figure 2: Bt Differential set grown in untreated, unfertilized field close to Einbeck Germany season 2023 only surving by being bend and fixed to sticks, adapted modern wheat in the background

Anders and Dennis are therefore working on developing a new set of differential lines with single gene resistances.

The origin of resistance and differential lines

Already Tschaner (1764) observed that spelt varieties (*Triticum spelta*) differed in susceptibility to common bunt, and Kühn (1880) could see varietal differences in susceptibility to bunt in bread wheat (*T.aestivum*).

Already in the beginning of last century there was basic knowledge on susceptibility and resistance, and successful breeding program were put up by Farrer (1901, 1904), Pye (1909) and others. Resistant varieties at the time included 'Hussar', 'White Odessa', 'Martin', 'Ridit', 'Banner Barkley', 'Turkey' and 'Florence' (Hurd-Karrer 1925), but heritage differences is resistance was not known at the time.

The genetic differences in resistance between resistant varieties was discovered by Fred N. Briggs in 1926 demonstrating that 'Martin' and 'Hussar' had different resistances, and the resistance in the variety 'Martin' was denominated the 'Martin Factor' or MM factor (Briggs 1926). However, already Gaines (1925) was convinced that the bunt resistance in all 'Martin', 'Hussar' and 'White Odessa' was due to not only a single factor, but multiple factors. This was demonstrated in 'Martin' by Crépin *et al.* (1937). The Martin Factor was therefore divided and denominated M1 and M2 (Briggs and Holton 1950). The M1 gene was renamed Bt1 and the M2 gene was renamed Bt7 by Metzger (1970).

The second factor for common bunt resistance was found in the variety Hussar (Briggs 1926, 1929). It was called the HH factor and later renamed Bt2 by Metzger (1970). It was known from the start, that 'Hussar' along with Bt2 also had the Martin Factor (Bt1), and Briggs (1933) identified 'Selection 1403' and 'Selection 1418' as having only the Hussar factor (Bt2) without the Martin Factor (Bt1).

A third resistance factor was found in 'Turkey 1558' and 'Turkey 3055' and was designated as the Turkey Factor or TT (Briggs 1933). This factor was renamed Bt4 by Metzger (1970).

Stanford (1941) identified a fourth genetic factor for bunt resistance in a series of 'Rio' crosses. He designated it as the Rio factor, RR and it was later renamed to Bt6 by Metzger (1970).

'Florence' and later 'Ridit', which is derived from a cross of 'Florence' and 'Turkey', was found to be resistant as early as 1908 (Sutton 1908). Churchward (1931) working with the cross 'Florence' X 'Hard Federation' obtained evidence which seemed to show the presence of a factor difference for resistance to bunt. 'Florence' resistance has been further investigated by several authors (Gaines 1933, Vogel 1938, Vogel 1944) through the times and is now known as Bt3 (Metzger 1970).

'Hohenheimer' was first used as a differential by Gaines (1933). Hohenheimer resistance was named Bt5 by Hoffman and Metzger (1976).

The second factor in 'Martin' was named MM2 by Briggs and Holten (1950) and later renamed Bt7 (Metzger 1970). Schaller *et al* (1960) investigated the inheritance of the gene and identified 'Selection 50077' (CI13561=PI 554100) from a cross between 'Martin' and 'Elgin' as having only the MM2 factor. Also the varieties 'Onas', 'Baart' and 'Federation' (CI4734) was identified as having the same resistance.

Bt8 was discovered in 'Yayla 305' (PI 178210) in 1970 by Waud and Metzger (1970). Later, Bt8 was also found in '7845' (PI 173438) and in PI 178383. When Blair Goates updated the differential lines in the review 1996, he proposed 'M72-1250' (PI 554120) as differential line deriving from '7845' (PI 173438). However, as mentioned above in this newsletter, it is uncertain if these Bt8 is the same gene(s).

Bt9 was identified in the Georgian landrace 'CI 7090' (PI 57143), which also has Bt7 (Metzger *et al.* 1979). Bt 9 is also found in other sources, including PI 178383. The Bt9 differential line proposed by Goates (1996) is R63-6968 / PI 554099 which is a selection from the cross Elgin / PI 178383. By genetic markers it is confirmed that the Bt9 is the same regardless if it derives from 'CI 7090' or PI 178383.

Metzger and Silbaugh (1971) identified a new gene for bunt resistance, designated Bt10 in the varieties 'Mocho' (PI 116306) and 'Greece 18' (PI 116301). Bt 10 is also found in other sources, including PI 178383, and the differential line R63-6982 / PI 554118 is a selection from the cross Elgin / PI 178383 (Goates 2012).

Differential lines for Bt1-Bt10 are shown in (Hofmann and Metzger 1976), but Goates later changed some of them for unknown reasons (Goates 1996, 2012).

The latest update to the differential set was done by Goates (1996, 2012) and here differential lines for Bt11, Bt12, Bt13 and BtP were added.

The Turkish landrace 'Dimenit' (PI 166910) was found to have three genes, Bt7, Bt9 and a new gene, Bt11 (Abdallah 1984). 'Dimenit' was crossed to 'Elgin' in that study, and one line, 'M82-2123' (PI 554119), was selected as differential line for Bt11 (Goates 1996, 2012).

Bt12 was found in the Turkish landrace '1696' (PI 119333) and proposed as differential (Goates 1996, 2012). '1696' (PI 119333) was crossed to 'Elgin' and a line from this cross, 'P78-24' (PI 554106), has also been used as a differential for Bt12 by some researchers.

Bt13 was found in PI 181463, incorrectly named 'Thule III' in the USDA genebank and first mentioned by Goates (1996, 2012).

The spring durum 'Doubbi' (PI 224657) has Bt14. A series of wheat crosses were studied by Pugsley (1953). He found one major recessive gene in a bunt resistant tetraploid wheat variety, 'Doubbi' (Kanbertay 1982).

The spring durum variety 'Carleton' (PI 352397) contains Bt15. It is first mentioned by Goates (2012A).

To the authors knowledge nothing has been published about the genetic basis of resistance in '7838' / PI 173437. It is suggested to have one gene called BtP (Goates 1996, 2012).

In the past century, different differential set have been used to describe virulence races and to compare breeding material to identify factors of resistance.

Bressmann (1931) was the first to propose a differential set of wheat varieties to characterise the different resistance factors known at the time:

- Hybrid 128 (CI. 4512) susceptible check,
- Bt1 Albit (CI. 8275)
- Bt1 White Odessa (CI. 4655)
- Bt1 Banner Berkeley (CI. 7362)
- Bt1 Regal (CI. 7364)
- Bt1+Bt2 Hussar (CI. 4843)
- Bt3 Ridit (CI. 6703)
- Turkey x Bearded Minnesota 48
- Bt4+Bt7 Oro (CI. 8220)
- Bt1+Bt7 Martin (CI. 4463)

Gaines and Smith (1933) successfully reduced the differential set and added Bt5 to the list:

- Hybrid 128 (CI. 4512) susceptible check,
- Bt1 Albit (CI. 8275)
- Bt1 White Odessa (CI. 4655)
- Bt1+Bt2 Hussar (CI. 4843)
- Bt3 Ridit (CI. 6703)
- Bt4 Turkey (C.I. 6I75)
- Bt5+? Hohenheimer
- Bt1+Bt7 Martin (CI. 4463)

Flor (1933) was able to separate 13 races of bunt using a differential set of 9 varieties of wheat. The resistance of Turkey (C.I. 6I75) seemed to differ from the other varieties and from other differential sets used at the time:

- Hybrid 128 (CI. 4512) susceptible check,
- Bt1 Albit (CI. 8275)
- Bt5+? Hohenheimer CI11458
- Bt4+Bt7 Oro (CI. 8220)
- Bt3 Ridit
- Turkey (C.I. 6I75)
- Turkey sel. (CI7366)
- Bt1+Bt7 Hussar (CI. 4843)
- Bt1 White Odessa (CI4655)

Metzger and Hoffmann (1976) made changes in Bt2 and Bt5 and added Bt8, Bt9 and Bt10:

- Hybrid 128 (CI. 4512) susceptible check
- Bt1 Albit CI8275
- Bt2 Selection PS60-1-1075(Elgin*Selection 1403)
- Bt3 Ridit
- Bt4 Turkey CI1558
- Bt5 Selection R60-3432 (Elgin*Hohenheimer)
- Bt6 Rio CI10061
- Bt7 Selection 50077 CI13561=PI 554100
- Bt8 Yayla 305(PI 178210)
- Bt9 Selection M69-2073(Elgin*CI7090) (Not PI 554099!)
- Bt10 M69-2094 (PI 554118) (Elgin*PI 178383)

Goates (1996) proposed additional differentials to the previous list, but also changed a few existing differential lines, including Bt0, Bt1, Bt2, Bt5 and Bt8. Goates (1996) also mentioned BtZ as a new gene, but did not appoint a differential line for this gene.

- Bt0 Heines VII (PI 209794)
- Bt1 Sel 2092 PI 554101
- Bt2 Sel 1102 PI 554097
- Bt3 Ridit CI 6703
- Bt4 CI1558 CI 1558
- Bt5 Hohenheimer CI 11458
- Bt6 Rio CI 10061
- Bt7 Sel 50077 PI 554100
- Bt8 PI 173438 x Elgin PI 554120
- Bt9 Elgin X PI 178383 PI 554099
- Bt10 PI 178383 x Elgin PI 554118
- Bt11 Elgin x PI 166910 PI 554119
- Bt12 PI 119333
- Bt13 Thule III PI 181463
- Bt14 Doubbi CI 13711
- Bt15 Carleton CI 12064

Goates (2012) basically used the same differential set as Goates (1996), but added an additional line 7838 (PI 173437) to represent a new gene BtP.

The evolution of differential set includes improvements when lines with dual resistance genes are exchanged with lines having only a single isolated resistance gene, whereas the opposite to the opinion of

the authors must be considered as a setback in differential quality. In addition, exchanging differential lines with origin in a previous differential set with a lines of a different origin of the resistance gene always include a risk of the two genes not being identical.

Current Differential Set

Table 6 Overview of the current differential set

Gene	Name	Gene Bank ID	Pedigree
Bt0	Heines VII	PI 209794	
Bt1	Sel 2092	PI 554101	
Bt2	Sel 1102	PI 554097	
Bt3	Ridit	CI 6703	
Bt4	CI 1558 / Turkey	PI 11610	
Bt5	Hohenheimer	CI 11458	
Bt6	Rio	CI 10061	
Bt7	Sel 50077	PI 554100	
Bt8	M72-1250	PI 554120	PI 173438/Elgin
Bt9	R63-6968	PI 554099	Elgin/PI 178383
Bt10	R63-6982	PI 554118	Elgin/PI 178383
Bt11	M82-2123	PI 554119	Elgin/PI 166910
Bt12	1696	PI 119333	
Bt13	"Thule III"	PI 181463	
Bt14	Doubbi	CI 13711	
Bt15	Carleton	CI 12064	
BtP	7838	PI 173437	

Proposed Differential Set

Table 7 Overview of the proposed differential set

Gene	Name	Gene Bank ID	Pedigree
Bt0	Heines VII	PI 209794	
Bt1	Sel 2092	PI 554101	
Bt2	Selection 2075	PI 554103	Selection 1403/Elgin
Bt3	Ridit	CI 6703	
Bt4	CI 1558 / Turkey	PI 11610	

Bt5	Starke II NIL		selection in: NGB 16106
Bt6	Rio	CI 10061	
Bt7	Sel 50077	PI 554100	
Bt8	M72-1250	PI 554120	PI 173438/Elgin
Bt9	R63-6968	PI 554099	Elgin/PI 178383
Bt10	R63-6982	PI 554118	Elgin/PI 178383
Bt11	None		
Bt12	None		
Bt13	"Thule III"	PI 181463	
Bt14	Doubbi	CI 13711	
Bt15	Carleton	CI 12064	
BtP	7838	PI 173437	
Bt16	P106.13		PI 199333/Rainer
Bt17	S13.10		PI 199333/Midas
Bt18	P106.54		PI 199333/Rainer
Bt19	P106.41		PI 199333/Rainer

Ongoing Research by Consortium Partners

Bunt resistance breeding at Saatzucht Donau GmbH & CoKG (Probstdorf, Austria)

Saatzucht Donau uses genomic selection (GS) and marker assisted selection (MAS) to decrease the time interval between wheat generations and speed up the breeding process. Due to its organic wheat breeding program, Saatzucht Donau has been active in bunt resistance breeding for more than a decade now. 'Tillexus' and 'Tillstop' are examples for the successful use of MAS to introgress Bt10 from Weston into adapted breeding material. 'Tillsano' and 'Axaro' were identified as bunt resistant "by chance" in artificially inoculated (common) bunt trials. Subsequently, both were suggested to carry Bt5, based on race tests with Agrologica.

Our main interests regarding bunt resistance breeding include the implementation of MAS to efficiently develop high performing wheat cultivars with an improved and durable bunt resistance. Bunt resistant material of particular interest to SZD, including breeding lines developed at BOKU in recent years, has been directly integrated into the wheat breeding program of Saatzucht Donau and KASP markers, whenever available, have been used successfully for the pre-selection of bunt resistant material.

Currently, we are using GWAS and QTL mapping to map so far 'unmapped' Bt-genes, such as Bt5, and develop/improve KASP markers for specific Bt-genes. Using GWAS, we recently mapped Bt5 to chromosome 1B in a population of >200 lines carrying Axaro in their pedigree. An additional resistance locus was identified on chromosome 1D, which does not show up in Spontan and Tillsano, other "confirmed" carriers of Bt5. SNP markers linked to the Bt5 locus and polymorph in SZD breeding material will be turned into KASP markers and used for the large-scale analysis of breeding material. Although not diagnostic, these KASP markers will help us to enrich Bt5 carriers in the breeding material, which will be tested in yield and bunt resistance trials next year. A similar strategy will be used to develop/improve KASP

marker for all other Bt-genes present in our breeding material. The interval for Bt5 overlaps with the most current interval identified by Dennis Christensen (Agrologica). The observation that most SNP markers identified by Agrologica linked to Bt5, however, were not polymorph in our material and underlines the importance of further pinning down Bt5 to an improved interval. To that end, we will use two bi-parental Bt5 populations of >180 lines each for classical QTL mapping, which show high marker contrast in the Bt5 interval, in 2024. This will hopefully leave us with an improved interval and SNP markers for Bt5.

Bt5 itself is not providing full resistance against the full spectrum of European bunt races, however, it will be an important resistance factor in combination with other Bt-genes.

In a small side project, 75 F4 lines of a Tilliko (BtZ) x Tillstop (Bt10) cross are tested at Agrologica with an avirulent race (Vr0). Finding any infected plants would mean that BtZ and Bt10 are –contrary to the current hypothesis – two different genes that can therefore segregate into susceptible offspring. Since BtZ and Bt10 – if different genes – are expected to be closely linked, segregation will be a rare event and the number of tested lines is rather small. Citing Dennis Christensen (Agrologica), finding a single susceptible line would be sufficient to gain further insight by genotyping this line. If necessary, the same experiment could be repeated with larger numbers and more homozygous plants next year.

For Saatzucht Donau, the most important goal is to develop high performing cultivars which on top display durable bunt resistance.

Researches for wheat resistance to common bunt at NARDI Fundulea, Romania

National Agricultural Research & Development Institute Fundulea, Romania is the largest Agricultural Research institution in Romania, was founded in 1957, has continued the Research of the Agronomic Research Institute of Romania, founded in 1927 and has been involved in wheat breeding from 1958. The winter wheat cultivars obtained at NARDI Fundulea occupy now about 40% of total wheat area in Romania.

The arable land of NARDI is a part of a transition area between Vlasia and Southern Baragan Plain, along the Mostistea river. Its geographical coordinates are: 44°33' Northern latitude and 24°10' Eastern longitude. The relief is generally flat, having the average altitude of 68 m.

The effort of the team of researchers on wheat bunt resistance is generally focused on: (1) continuously checking the efficiency of known bunt resistance genes against local populations (isolates) of bunt; (2) improving the competitiveness of bunt resistant germplasm by repeated cycles of crossing with adapted cultivars and selection for both bunt resistance and agronomic type, plus to stack resistance genes and (3) searching for new bunt resistance genes (in special from synthetic amphiploids).

Currently, the main activities in this field follow: to identify molecular markers associated with resistance to bunt originated from F000628G34-1 line that carries a 1RS:1AL translocation; to stack resistance genes to ensure durable resistance to common bunt in wheat (like the effect of the rye translocation 1RS:1AL from Consecvent winter wheat cultivar (obtained by Otilia x F000628G34-1) cross with adapted breeding lines that carry different *Bt* resistance genes).

Table 8 Crossing for stack of resistance genes to ensure durable resistance to common bunt in wheat (F1 generation)

Common bunt resistance gene source	Crossing	
Bt5	FDL94889GM1-31	Consecvent (1RS:1AL)
Bt12	FDL94895GM1-21	Consecvent (1RS:1AL)
Bt13	FDL95601GM37	Consecvent (1RS:1AL)

Bt10	FDL15001GM1	Consecvent (1RS:1AL)
WGRC23	FDL96915G1-1	Consecvent (1RS:1AL)

The FDL94895GM1-21 (Bt12), FDL95601GM37 (Bt13), F00628G34-1 (1RS:1AL translocation) and F96915G1-1 (WGRC23) breeding lines have kept the resistance over time and across fungal races covering a broad range virulences occurring in Europe. The resistance source, in WGRC23, probably comes from *Triticum monococcum* accessions PI 266844 or/and PI 355520.

Another activity is based on searching new resistance source. In this respect, last year, 27 synthetic amphiploids (SHWs) were tested under artificial inoculation with mix of *Tilletia spp*. local races, in Romania at NARDI Fundulea, the phenotypic observation showed six SHWs free of infection (0%): E10A, E15A, E22A, E30A, E32A and E34A. Two of them (E10A and E15A) have the same accession, *Ae. squarrosa* -2454, as parent. For these six SHWs we continue the validation of resistance. Also, for E10A, E15A and E34A we have synthetic lines.

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Crop Research Institute in Prague – Ruzyne, Czech Republic:

This is a small introduction of Crop Research Institute in Prague – Ruzyne, Czech Republic:

The Institute conducts basic and applied research, ranging from traditional studies of genetics, plant breeding, plant nutrition, agroecology, plant health, and the safe storage of crops and agricultural produce, to the fast-developing fields of molecular biology, biotechnology, food and feed safety, and the use of biowastes and biomass for energy production. Moving to the forefront of CRI research are the issues of sustainable agriculture production and organic farming with the aim to maintain high soil fertility, support natural processes and biodiversity, reduce water pollution and overall minimise the negative impacts of agricultural production on the environment and human health.

The Institute coordinates two national programmes on the conservation of genetic resources, National Programme on Conservation and Utilization of Plant Genetic Resources and Agro-biodiversity and National Programme on Protection of Genetic Resources of Economically Significant Microorganisms and Tiny Animals.

I am a part of the Genetics and Breeding Methods research team, focused on genetics of disease resistance of cereals, mostly wheat, barley and oat. In field trials, varietal reactions to rusts, fusarioses, common bunt and dwarf bunt, eyespot, leaf spot, powdery mildew and viroses are tested.

University of Idaho Wheat Breeding and Genetics Program/ University of Utah activities

Jianli Chen, Professor, Senior Wheat Breeder, jchen@uidaho.edu

Idaho wheat production contributes significantly to domestic and overseas wheat markets. Nationally, Idaho ranks in the top eight states for wheat production. Wheat is the second cash crop behind Potato in Idaho, comprised of approximately 60% winter wheat and 40% of spring wheat. About 85% of wheat is under irrigated production in southeastern Idaho, while majority of wheat production is under rainfed in Northern Idaho. Dwarf bunt is an endemic disease of dryland winter wheat production in the regions with high elevation and prolonged snow cover. Historically, dwarf bunt caused severe yield loss and quality reduction when epidemics occurred in some dryland farmland such as in southeast Idaho and North Utah. Breeders at University of Idaho (UI) and Utah State University (USU) have collaboratively worked on the disease for many decades and jointly released over 20 dwarf bunt resistant cultivars, some of which have been used in breeding programs worldwide. Dr. Jianli Chen, the senior wheat breeder at UI collaborating with the retired breeder Dr. David Hole has generated solid preliminary results on QTL mapping for dwarf bunt resistance and published three papers in the prestigious journal *Theoretical and Applied Genetics.* Currently, Dr. Chen led a USDA-NIFA grant identifying the candidate genes underlying the QTL previously identified, implementating of genomic selection for dwarf bunt resistance in collaboration with a newer breeder Dr. Margaret Krause at USU and another molecular physiologist Dr. Fangming Xiao at UI.

Candidate genes underlaying the QTL on the 7DS and 6DL are being identified using fine-mapping, exome capture and PACBio sequencing of common wheat, and PanGenome sequencing of *Ae. Taschii*. These research projects are in collaboration with Drs. Brande Wuff, Herman Buestermayr, Steven Xu, Anders Borgen, and Dennis Christensen. Molecular markers for 6DL and 7DS QTL are developed and will be shared with the consortium after papers are published this year. Dr. Chen's program is also closely collaborating with Drs. Herman and Borgen evaluating genetic response of bunt differential lines to their local races of common bunt. In addition, Dr. Chen's program established a protocol for screening common bunt in greenhouse and currently assess common bunt resistance in responses to three single races L-18, L-19, and T33. Dr. Chen's program currently has two postdoc, two PhD students, one senior technician, and three seasonal help. Two post doc Drs. Amandeep Kaur and Guriqbal Dhillon, and one Ph.D student Pabitra Joshi are working on dwarf and common bunt projects. Ms. Joshi will graduate in December of 2024. Dr. Krause left for a new faculty position at Oregon State University. Dr. Krause's MS student Mr. Will Krause will graduate in May of 2024. The USU is filling a new wheat breeder in the summer of 2024.

Agrologica

Anders Borgen screens breeding lines and varieties with the purpose to identify genetic markers using a set of virulence races. The focus in 2024-25 is Bt2 and Bt8. Until 2024, the research was partly funded by projects BOOST and DIVERSILIENCE. New funding to the GRAINGOOD project enables further research in 2025-2027.

Dennis Christensen continues to fine map genes identified in phenotyping from Anders or from other sources.

Julius Kühn Institut, BrandResist project

Claire Ferreira, Albrecht Serfling & Andreas Stahl

The goal of the BrandResist project is to find and use previously unused resistances from diverse genetic materials. To screen for susceptibility, several pathogen isolates of European origin will be tested to analyse the genotypes. To identify QTL´s genotyping and genome-wide association studies (GWAS) will be done. Using NAM populations, we can develop molecular markers for resistance QTL´s. Resistance-specific KASP markers can be derived from these, which significantly speed up the breeding of resistant varieties.

Finishing the first year of the project, we can report that the first field trial was successful and the first GWAS data are being analysed right now. The second field trial is growing slowly and will, hopefully, give us more insides and some more interesting QTL´s to look at.

Nordic Seed

Update from Amalie Poulsen

Nordic Seed is doing some crosses, which are now in the F3 Population.

270 plants from seven crosses and 216 plants from cross no. 2 are under vernalization until 13.12.2024.

Table 9 Overview of the crosses from Nordic Seed

Cross Nr.	Habit	Parents	Description
1	W*W	Tillexus*Hallfreda	Polymorphic in the Bt10 interval (and possible recombinations near Bt9)
2	W*W	PI 554097*Thule-III	Polymorphic in the Bt13 interval (and possible to study also Bt2)
3	W*W	PI 554101*S.Th.Cia-2	Polymorphic in the Bt13 interval (and to study Bt1)
4	S*S	S.Th.Cia-3*OD-19	Polymorphic in the Bt13 interval
5	S*S	S.Th.Cia-2*CCP-7	Polymorphic in the Bt13 interval (and possible recombinations near Bt7)
6	W*W	Tommi*Tillexus	Polymorphic in the Bt5 interval (and possible

			recombinations near Bt10)
7	S*W	Østby*Tommi	Polymorphic in the Bt5 interval
14	W*S	Erythrospermum 5221*Mirakel	Made to study the Erythrospermum-resistance

New Line development

Anders made crosses based on Dennis' analysis on polymorphism in selected haplotypes. Nordic Seed is making 8 x 200 RILs from these. Breun is making recombinant lines detected by KASP markers from three crosses and populations from two crosses.

Populations at Nordic Seed

Østby (Bt0) x Tommi (Bt5)

Erythrospermum 5221 x Mirakel (Bt0)

Tillexus (Bt10) x Hallfreda (Bt9)

PI 554097 (Bt2) x Thule III (Bt13)

PI 554101 (Bt1) x S.Th.Cia-2 (Bt13)

S.Th.Cia-4 (Bt13) x OD-19 (Bt0)

S.Th.Cia-3 (Bt13) x CCP-7 (Bt7)

Tommi (Bt5) x Tillexus (Bt10)

A total of 50 F1 plants are now at the heading stage.

Populations at Breun

Tilliko (BtZ) x Yayla 305 (Bt8/BtX/Bt0)

PI 554098 (Bt11) x Ridit (Bt3)

Bosporus (Bt5) x PG3540 (Bt1)

Trintella (Bt_Trintella_1B/Bt_trintella_5B/Bt_Trintella_7A/Bt_Trintella_7B) x Mirakel (Bt0)

Mirakel (Bt0) x Dimenit (Bt3, Bt6, Bt7, Bt_Dimenit_4BS, Bt_Dimenit_4BL, Bt_Dimenit_6D, Bt_Dimenit_7B)

Since Breun cannot continue all of their work, we are on the lookout for partners who want and can take over a part or all of it. If you want to know, more feel free to contact Claire (claire.ferreira@juliu-kuehn.de).

Pipeline Overview

As seen from the previous chapters we have much material with these genes in the pipeline: Bt1, Bt2, Bt3, Bt5, Bt6, Bt7, Bt9, Bt10, “Bt11”, Bt13, BtZ, BtH, Bt_Bussard_2B, Bt_Magnifik_5D, Bt_Mariann_XX and also Erythrospermum 5221, Trintella and Dimenit Resistance.

The amount of “Bt8” material from PI 178383, Yayla 305 and PI 173438 is a bit unknown.

Some “Bt12” material is in the field for phenotyping and Utah is working on it.

We have no or very little Bt14, Bt15 and BtP material in the pipeline.

Future References perspectives

Perhaps we should have a meeting and/or a survey with the purpose of aligning our expections for the consortium.

What should we do?

Should we have a steering committee?

Should we coordinate it differently?

How should we share information?

How much information should be shared?

Should we do prebreeding?

Should we do yield /observation trials with resistant lines?

Who should analyse (and publish findings) from the new populations.

Etc.

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