Proceedings of the

29 May 2018, Tuesday
Arrival of participants to Logan

Hampton Inn by Hilton
1665 North Main Street
Logan, Utah 84341 USA
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18:30 – 20:30	Welcome Reception: The Italian Place: 48 Federal Ave, Logan, UT 84321

30 May 2018, Wednesday

09:00-09:30	Welcome to the conference by Dr. Paul Johnson, Department Head, Plants, Soils and Climate, Utah State University.
09:25-10:00	Introduction and logistics, Utah Bunt nursery details – David Hole
10:00-10:30	Break.

Scientific session:
Pathogens: Diversity, Pathogenicity, Methodology
Session chair: Hermann Bürstmayr

10:30-11:00	**iTRAQ-based Proteomic Analysis of Wheat Bunt Fungi *Tilletia controversa*, *T. caries* and *T. foetida*** Li Gao
11:00-11:30	**Classification of wheat bunt diseases (*Tilletia* spp.) and the importance of reliable reference material for the development of new detection methods** Monika K. Grundler

11:30-12:00	**Tracing *Tilletia caries* in wheat during the endophytic phase** Fabio Mascher

Lunch – 12:00 – 13:30
Session chair: Juliet Marshall

13:30-14:00	**Three new species of flag smut of grasses from the United States** Kyryll G. Savchenko
14:00-14:30	**Determination of the genome composition of *Sporisorium reilianum* f. sp. *reilianum* (Kühn) Langdon and Fullerton, the sorghum head smut pathogen** Chunlai Zhang
14:30-15:00	**Historical records of *Urocystis* in North America from the U.S. National Fungus Collections** Lisa A. Castlebury
15:00-15:30	Break

Sightseeing in Logan or hiking in Logan canyon to wind caves
16:00-

Dinner on your own

31 May 2018, Thursday

Scientific session:
Disease Control
Session chair: Anders Borgen

09:00-09:30	**Genome-Wide Association Mapping for Dwarf Bunt Resistance in the National Small Grains Collection** Tyler Gordon
09:30-10:00	**Virulence pattern of Czech bunt samples and sources of resistance** Veronika Dumalasová
10:00-10:30	**Evaluation of Nordic heritage varieties and NILs for resistance to common bunt (*Tilletia caries* syn. *T.tritici*)** Anders Borgen

10:30-11:00 Break
Session chair: Veronika Dumalasova

11:00-11:30	**Strategic use of virulence pattern to develop genetic markers for resistance to common bunt (*Tilletia caries*) in wheat** Anders Borgen
11:30-12:00	**New tools available to control the common bunt of wheat: development of an early detection test on plantlet by qPCR.** G. Orgeur.
12:00-12:30	**Mapping QTLs conferring additive resistance to Karnal bunt in bread wheat in two recombinant inbred lines populations** P.K. Singh

12:30 – 14:00 – Lunch
Session chair: Fabio Mascher

14:00-14:30	**Prospects and challenges for breeding bunt resistant wheat using molecular marker assisted selection** Rui Wang
14:30-15:00	**Comparative mapping of bunt resistance genes in winter wheat** Hermann Bürstmayr
15:00-15:30	**Organic methods of controlling common bunt at the farm level.** Lars Wiik

15:30-16:00 Break
Session chair: David Hole

16:00-17:00	Business Meeting - Discussion

Banquet – 19:00 – 21:00
Smithfield Golf Course Reception

01 June 2018, Friday

09:00-09:30	Travel to Logan Bunt nursery
10:30-12:00	Travel to Pocatello, ID
12:00-13:00	Lunch on own at Portneuf Valley Brewing
13:30-16:00	USDA small grains collection Aberdeen, ID **(Passport or Gov't ID required)**
16:00	Travel back to Logan or SLC airport hotels.

iTRAQ-based Proteomic Analysis of Wheat Bunt Fungi *Tilletia controversa*, *T. caries* and *T. foetida*

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This is the first study of proteomics of wheat bunt fungi *Tilletia controversa* (TCK), *T.caries* (TCT) and *T. foetida* (TFL) using the iTRAQ technique. Based on the relative quantities of specific proteins between each two pathogens, we found 50 up-regulated and 80 down-regulated protein genes in TCK compared to TFL, 62 up-regulated and 82 down-regulated protein genes in TCT compared to TFL, 47 up-regulated and 30 down-regulated protein genes in TCK compared to TCT, and there were 1 protein of up-regulated and 4 proteins of down-regulated in the three pairs. These protein data could be of great value for exploring the key proteins which play an important role in the interactions of these pathogens with their host. Some of them could be valuable for differentiating the three pathogens with monoclonal antibodies produced by the specific proteins and may enable in-site detection of the pathogens and performing routine monitoring as a diagnostic assay in wheat shipments.

Figure 1. The number of difference proteins between the three pathogens. Based on the relative quantities of specific proteins between each two pathogens, we found 50 up-regulated and 80 down-regulated protein genes in TCK compared to TFL, 62 up-regulated and 82 down-regulated protein genes in TCT compared to TFL, 47 up-regulated and 30 down-regulated protein genes in TCK compared to TCT.

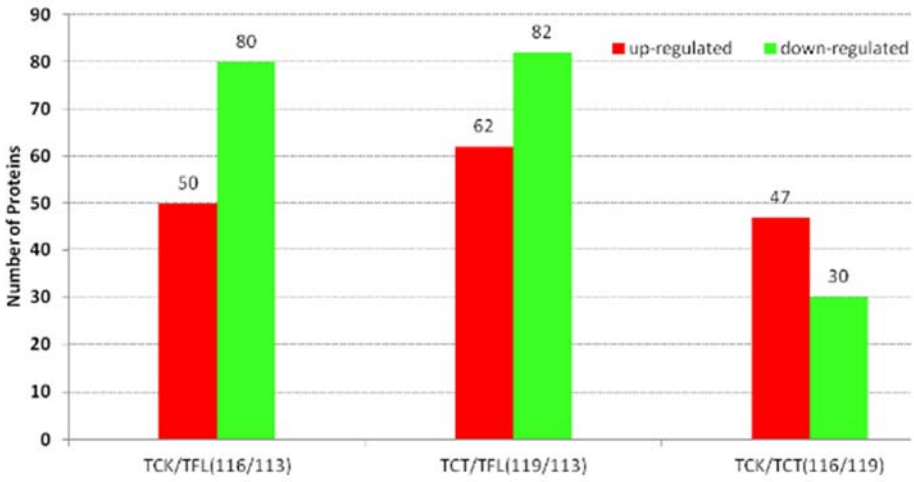
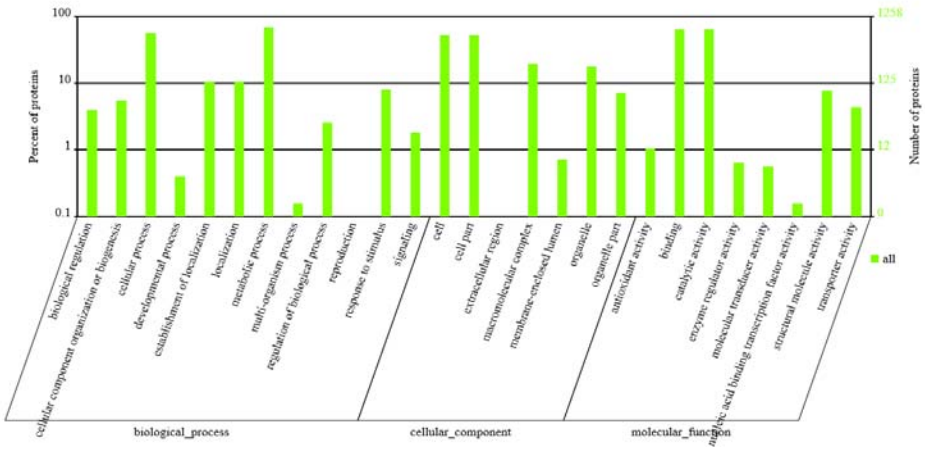


Figure 2. GO terms of identified proteins. The final selected differentially expressed proteins were analyzed using the GO database to determine their cellular component association, molecular function, and participation in biological process.



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Chen, J.L., Guttieri, M,J,, Zhang, J,L,, David, H., Souza, E., Goates, B. 2016. A novel QTL associated with dwarf bunt resistence in Idaho 444 winter wheat. Theor Appl Genet 129(12): 2313-2322

Classification of wheat bunt diseases (*Tilletia* spp.) and the importance of reliable reference material for the development of new detection methods

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For more than 30 years the filtration method according to the International Seed Testing Association (ISTA) Working Sheet No 53 [1] combined with a microscopic examination has been used worldwide to determine and quantify the causal agents of common bunt (*Tilletia caries* (DC) Tul. syn. *T. tritici* (Bjerk.) G. Winter and *T. laevis* Kühn syn. *T. foetida* (Wallr.) Liro), and dwarf bunt (*T. controversa* Kühn), respectively. This method requires well-trained and experienced professionals to distinguish the different *Tilletia* species. In order to comply with seed multiplication, trading and quarantine regulations, a reliable and validated identification method is needed. The aim of this research project is to develop an efficient molecular testing method by means of quantitative Real-time PCR (qPCR) to support seed testing laboratories. In the course of developing the new method, the importance of reliable reference material became obvious. Consequently, several determination methods were combined to ensure the correct classification of the wheat bunt specimens used for the method development.

In this study over 100 specimens of *Tilletia* spp., mainly *T. caries*, *T. controversa* and *T. laevis*, were obtained from Germany, its neighboring countries and the USA. In general, the material consisted of infected spikelets and isolated bunt balls. All of these underwent a first morphological determination, followed by a detailed analysis based on morphology, germination behavior, PCR-RFLP and a characterization on sub-proteome level. Due to availability of only very small amounts of some specimens, these analyses could be performed for only 85 of more than 100 accessions. The morphological determinations were performed independently by seven experts using permanent slides of bunt teliospores. The germination behavior of the teliospores was tested at 5° C and 15° C, respectively. PCR-RFLP [2] was used as a genome-based classification technique. In addition, Matrix Assisted Laser Desorption Ionization — Time of Flight Mass Spectrometry (MALDI-TOF MS) was used for the proteome-based characterization of 67 out of the 85 analyzed specimens. Thus, the combined results were used to evaluate, characterize and identify *Tilletia* species in a polyphasic approach.

Two-thirds (57 of 85) of the examined specimens were consistently identified considering three independent criteria (morphology, germination behavior, PCR-RFLP). All seven experts unequivocally identified these 57 accessions as *T. caries*, *T. laevis* or *T. controversa,* respectively. In addition, teliospores of all 57 specimens germinated under temperature regime typical for each species supporting the morphological species identification. PCR-RFLP analysis resulted in two fragments for *T. controversa* and three fragments for *T. caries* and *T. laevis,* respectively.

Characteristic sub-proteome spectra for the 67 analyzed specimens were produced by MALDI-TOF MS clustering in two main groups. One group contained all *T. controversa* specimens and another consisted of two subgroups namely *T. caries* specimens originating from Europe and *T. caries* as well as *T. laevis* specimens from the USA, respectively. Three accessions grouped independently with no clear affiliation. 22 out of 85 specimens (26 %) differed in at least one of four tested criteria. Some of these specimens did not germinate and remained therefore unclassified regarding this criterion. Two instead of three PCR-RFLP fragments were observed in six specimens classified as *T. caries* by morphological characters and germination behavior. Three specimens were classified as *T. caries* by morphology and germination but as *T. controversa* by PCR-RFLP and MALDI-TOF MS.

Germination behavior as a method independent from professionals was the least contradictory of the tested criteria because it showed the highest consistency with the experts' identifications of specimens, even if morphology is the only method which is able to distinguish *T. laevis* from *T. caries*. The few accessions that did not germinate can be explained by reduced viability due to ageing or suboptimum storage conditions. Therefore, germination tests are a useful tool for classification of *Tilletia* spp. especially in combination with morphologic criteria. However, these tests are laborious, and the results are only available after several weeks. A fast method, although bearing a certain degree of uncertainty, is the PCR-RFLP. The results obtained suggest that small changes in the genome can result in determinations contradicting the other methods. MALDI-TOF MS now provides an innovative method for the characterization of *Tilletia* spp. teliospores. The results of MALDI-TOF MS allowed a clear separation of specimens into a dwarf bunt and common bunt group. However, *T. laevis* could not be separated from *T. caries*. Nevertheless, the common bunt specimens could be differentiated in specimens originating from Europe versus the USA. But these results also suggest a high variability within one species, challenging the development of a universally valid PCR-based detection method even more. To conclude, 72 specimens could be clearly characterized using the distinct methods. These specimens can be used as reliable reference material for the development of PCR-based detection methods. Verified species-specific molecular markers developed from this set of tester strains can then be used to classify the remaining specimens and to apply the new method to seed testing samples.

Acknowledgement: This project is funded within the framework of the Federal Organic Farming Scheme and other forms of sustainable agriculture (BÖLN) by the German Federal Ministry of Food and Agriculture (BMEL) based on a decision of the German Federal Parliament.

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Tracing *Tilletia caries* in wheat during the endophytic phase

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Common bunt of wheat is an important disease caused by infections with *Tilletia caries*. Besides yield losses, the pathogen spoils the harvest produce with a most disagreeable odour rendering it unsuitable for consumption or for use as seed. The causal agent is a basidiomycete that infects the seedling at the very first moments after germination. After successful infection, the fungus grows endophytically, remaining undetected until the first maturity stages. At that moment, packs of fungal teliospores form in place of kernels, in the spikes.

The disease can be avoided by the use of healthy seeds or the use of chemical, physical or certain biological seed dressings. Modern agriculture requires also more sustainable control methods that avoid additional costs. In this context, the use of plant resistance is gaining increasing interest. A series of resistance genes and resistance QTLs have been described. Several studies show that these genes do not necessarily confer total resistance and combinations of genes might provide higher resistance degrees. Yet, only little is known when plant resistance genes intervene in the disease cycle. The present project aims at tracing the fungus in the plant after infection in susceptible and resistant plants. Results and conclusions will be presented.

Three new species of flag smut of grasses from the United States

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Flag smut diseases are widespread on wild and cultivated grasses throughout the temperate and subtropical regions of the world. The pathogens (*Urocystis* spp., Urocystidales, Basidiomycota) cause a systemic infection on host plants, forming sori in the vegetative parts, most commonly in leaves, and present as narrow stripes between the leaf veins (Mordue & Walker, 1981). Most are believed to have a narrow host range on wild and cultivated grasses (Savchenko et al. 2017). Savchenko et al. (2017) clarified the taxonomy and phylogeny of flag smut pathogens of triticoid grasses using molecular and morphological analyses. Results indicated that there were several distinct lineages of flag smut pathogens and supported the distinction of *Urocystis tritici* as a separate species from *Urocystis agropyri*, which has important consequences for trade as the flag smut of wheat pathogen is currently subject to strict quarantine regulations in many countries.

The goal of the present study is to clarify the taxonomy and phylogeny of the flag smut pathogens on several additional grass hosts by analyzing morphological and molecular data. Specimens of *Urocystis* on several species of *Elymus, Schizachne, Bromus, and Poa* collected in the United States in 2015-2016 were examined and analyzed. DNA extracted from teliospores was used to amplify the ITS, TEF, RPB2, TIF2, and ATP2 loci used in phylogenetic analysis. The results of Bayesian analysis based on newly generated sequences, sequences from the previous study (Savchenko et al. 2017), and existing sequences from GenBank indicate that demonstrated that at least three undescribed species on *Elymus, Poa,* and *Schizachne* can be recognized and supported with morphological and host-specificity data. The new species on *Schizachne* is a first ever record of smut fungi on hosts from this genus. The new species on *Elymus* from New Mexico falls outside of the major clade of flag smut pathogens of grasses, representing a previously unknown lineage. The phylogenetic position of flag smut of wheat has not changed with increased sampling and the addition of sequences from Indian isolates from GenBank. Several other host-specific lineages were discovered in the phylogenetic analysis that will require additional sampling and further investigations.

References

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Determination of the genome composition of *Sporisorium reilianum* f. sp. *reilianum* (Kühn) Langdon and Fullerton, the sorghum head smut pathogen

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The smut fungi *Sporisorium* spp and *Ustilago maydis* are biotrophic plant pathogens that exhibit a very narrow host range includes sorghum, maize and sugar cane. *Sporisorium reilianum* (Kühn) Langdon and Fullerton occurs in two host-adapted formae speciales: *S. reilianum* f. sp. *reilianum* (SRS), which causes head smut on sorghum, and *S. reilianum* f. sp. *zeae* (SRZ), which induces disease on maize. Studies on the host specificity of *S. reilianum* have shown that SRZ colonizes sorghum leaves coupling with the production of the red phytoalexin luteolinidin which slows the fungus vegetative growth (Zuther K et al., 2012, Mol Plant Microbe Interact. 25:1230-7), whereas SRS proliferates non-efficiently on maize along with induction of less defensive response genes than by SRZ (Poloni A & Schirawski J, 2016, Mol Plant Pathol. 17:741-754). The genomic difference of SRS and SRZ has not been exploited.

The genome of *S. reilianum* f. sp. z*eae* strain SRZ2 has been sequenced by 454 Life Sciences (Schirawski J et al. 2010, Science 330 : 1546-1548). The genomic sequnce of *S. reilianum* f. sp. *reilianum* is not available which hindered the study on pathogenesis of SRS. Isolates of culture for SRS strain F4 were obtained from infected sorghum florets and DNA was extracted for DNA sequencing by Illumina HiSeq4000 technology which achieved 151 times coverage of the genome. The assembled genome of F4 is 20,259,439bp with GC content as 58.5% in comparison to 18,476,874bp of SRZ2 with GC content as 59.5%. By annotation of the genomic sequence of SRS F4, considerable genetic variation was discovered with 5971 genes have meaningful alterations: 5856 genes with non-synonymous SNP, 2655 genes with InDel, and 120 genes with structural variations. Sequence on the sex loci has been covered with one gene only 75% similar (E=7e-105） to the homeodomain transcription factor bE4. There are a number of putative effector genes related to pathogenesis carrying mutations include gene212 encode probable SAC1-recessive suppressor of secretory defect, gene1143 encodes secretory component protein SHR3, gene1295 and gene4827 encode cysteine-rich secretory protein family PRY2 and related to fruiting body protein SC7 respectively, gene1676 encode secretory pathway protein Sec39, gene1756, gene3677 and gene4373 encode secretory lipase, gene2168 encodes eukaryotic aspartyl protease, xylanase inhibitor N-terminal, related to secreted aspartic protease 2; gene2540 and gene4051 encode ABC transporter, related to positive effector protein GCN20, gene2850 and gene5852 encodes vacuolar protein sorting-associated protein 1 (vps1) with Dynamin GTPase effector domain, gene3394 and gene4156 encode glucose-repressible alcohol dehydrogenase transcriptional effector CCR4; gene4076 encodes late secretory pathway protein avl9, gene4432 encodes probable COP1-coatomer complex alpha chain of secretory pathway vesicles, gene4950 encodes probable PMR1-secretory pathway Ca^{2+}-transporting P-type ATPase, gene5589 encodes exocyst complex protein

Sec15, related to secretory pathway protein, gene6335 encodes related to SSP120-secretory protein vip1. Proteomic analysis of F4-infected sorghum florets has shown that several fungal proteins are transcibed. These provide a solid foundation to study pathogenicity and host specificity in *S. reilianum* f. sp. *reilianum.*

Acknowledgement: This study has been supported by Funds of Shanxi Organising Bureau Program (2014-18) and Shanxi Province Human Resources and Community Department and Natural Science Fundation of China (31470285).

Historical records of *Urocystis* in North America from the U.S. National Fungus Collections

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Urocystis (*Urocystidiales*, Basidiomycota) is a large and varied genus of smut fungi known to occur on hosts in more than 30 plant families [1]. More than 250 species have been described (MycoBank.org, retr. March 12, 2018) with more than 40 species reported to occur in North America [2]. Sori, the spore bearing structures, can be found in all plant parts and consist of spore balls with one or more brownish-colored spores covered by lighter-colored sterile cells [3]. Species identification is based on host plant taxonomy and the number, size, and shape of the spores contained in the spore balls. As a part of an effort to understand the systematics of the flag smut of wheat pathogen, *Urocystis tritici,* and facilitate the development of diagnostic assays or other means of identification for this important quarantine pathogen, a morphological and nomenclatural survey of the historical specimens from North America available in the U.S. National Fungus Collections (USNFC, Herbarium BPI), in conjunction with peer-reviewed literature reports, was undertaken.

Specimens and records from North America were located using the USNFC Fungal Databases (nt.ars-grin.gov\fungaldatabases) and sorted by host plant species. Currently accepted names for the host plants were based on the USDA Plants Database (plants.usda.gov), Tropicos (www.tropicos.org) and The Plant List (www.theplantlist.org). Initial efforts have focused on the *Urocystis* species associated with North American native grass hosts, with efforts to be expanded to non-poaceous hosts. Morphological examination and imaging of spore balls mounted in Shear's solution was performed using a Zeiss AxioCam HRc digital camera and a Zeiss Axioplan 2 compound microscope. Zeiss Axiovision version 4.7 software package used for image capture and measurement (Carl Zeiss Inc., Thornwood, NY).

1417 specimens on 12 host families were located with 795 specimens occurring on poaceous hosts [2]. Of these, approximately 40 North American native grasses were found to have had a *Urocystis* reported on them and/or to have a specimen deposited. Many of these have been identified as *Urocystis agropyri,* a taxon that serves as the default identification for many *Urocystis* species on grasses. Specimens from North American native grass hosts have been morphologically characterized and imaged. Efforts are ongoing to expand sampling to all North American native plant hosts for both morphological and molecular characterization. Initial results indicate that morphological distinctions exist for collections identified as *U. agropyri* on *Festuca idahoensis* and *Bromus ciliatus* among others.

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Genome-Wide Association Mapping for Dwarf Bunt Resistance in the National Small Grains Collection

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Dwarf bunt (DB), caused by the basidiomycete *Tillitia controversa* Kühn, can cause yield and quality losses on fall-planted bread wheat (*Triticum aestivum* L.) in regions with prolonged snow cover [1]. DB symptoms include dwarfing, increased tiller number, and bunted spikes where florets have been replaced by a fungal sorus. Fungicide seed treatments can control DB, but are not possible in organic wheat production systems [2]. Thus, the importance of breeding for DB resistance has grown with the market for organic wheat. The National Small Grains Collection (NSGC) in Aberdeen, ID is a repository for a worldwide collection of the small grains and holds 48,869 bread wheat accessions. Dwarf bunt resistance is rare among NSGC accessions. For example, of the 8,167 landrace accessions in the NSGC tested for DB, only 104 were classified as resistant [3]. Recently developed genomic tools have allowed for the identification of genetic loci underpinning resistance to DB. The purpose of this study was to 1) verify the DB resistance in the NSGC accessions with replicated field tests and 2) identify genetic loci associated with the resistance using a genome-wide association study (GWAS) approach.

A total of 291 bread wheat accessions were selected from the NSGC based on data from the Germplasm Resources Information Network (GRIN). This included accessions of varying improvement status, including landraces. Of the 291 selected, 144 were resistant based on GRIN data, and these were paired with a susceptible accession of similar geographic origin. Several susceptible checks and lines carrying each of the *Bt* genes (*Bt0* through *Bt15*) were also included as controls for testing. Two replications were sown in the fall of 2016 in a nursery in Logan, UT. Percentage of infected spikes (0 to 100%) were assessed after plant maturity. Genomic DNA was collected from individual plants of each accession, and genotyped using the 90K Illumina SNP platform. After filtering, 25,529 SNPs were used to perform Principal Component Analysis (PCA) and QK association testing using JMP Genomics.

Historical NSGC bunt ratings from GRIN and the data collected in 2017 were positively correlated with an R^2 value of 0.61. Of the 291 accessions tested, 126 accessions were resistant and 122 were susceptible in both tests. Resistant accessions originated from Turkey (29%), Iran (10%), and Serbia/Monenegro (8%). PCA revealed three distinct clusters: 1) landraces from Turkey and western Iran, 2) primarily landraces from Iran and Hakkari Province in eastern Turkey and 3) primarily landraces from Serbia and Montenegro. After controlling for kinship and population structure, GWAS revealed seven quantitative trait loci (QTL) with p-values <0.001, which

explained between 3 and 8% of the observed phenotypic variation. These QTL were on chromosomes 1AL, 2AS, 2BS, 3BS, 6BS, 6DS and 7AS. Previously reported QTL on 1AL [4], 6DS [5] and 7AS [6] were within two, one, and five centimorgans, respectively, of the QTL found in the present study. These results can assist with our understanding of the genetics of bunt resistance, and in developing resistant cultivars.

References

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Virulence pattern of Czech bunt samples and sources of resistance

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The contribution presents data on common bunt and data on dwarf bunt virulence obtained at the Crop Research Institute in Prague-Ruzyně.

Common bunt inoculation was done by shaking seed with teliospores in Erlenmayer flasks for 1 - 2 min. Inoculation and sowing (1 m long rows, 4 replications) was carried out in early October. For dwarf bunt tests rows 1 m long with 6 replications were sown in late October. Teliospores were evenly spread on the soil surface after sowing. In absence of a snow cover the plots covered with straw or white nonwoven fabric.

Samples of common bunt and dwarf bunt were tested in the field for virulence on a standard differential set of cultivars and lines carrying various genes of resistance (*Bt0-Bt13*). In the studied set of samples the presence of the most frequent virulence and avirulence genes were established.

Some of the tested sources of resistance proved high resistance to dwarf bunt. As they were recorded resistant to common bunt as well, they offer a suitable genetic material for resistance breeding both to the common and dwarf bunt.

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Evaluation of Nordic heritage varieties and NILs for resistance to common bunt (*Tilletia caries* syn. *T.tritici*)

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NordGen is the genebank covering the Nordic countries, storing and distributing germplasm of Nordic origin to potential users. Allkorn is a Swedish seed saver organisation making their own maintainance of cereals mainly of Nordic origin. Accessions from Allkorn may therefore be different from the accessions from NordGen with the same name.

Prof. James MacKey started a program many years ago, developing a differential set of NILs (Near Isogenic Lines) with *Bt*-resistance genes against common bunt based on the variety Starke II, backcrossing resistant lines with Starke II. However, he didn't manage to finish his work, and the material was stored at NordGen.

The aim of this stydy was to evaluate accesions from NordGen and Allkorn for the susceptibility of common bunt.

Materials and methods

In 2014-15, 165 lines from NordGen was tested at Agrologica by contaminating ~50 seed with abundance of spores of either a race with low frequency of virulence, or a mixture of different races [1]. The idea of testing with these two different spore samples was that if a variety has any resistance, they will have low level of infection when contaminated with spores without virulence, but a higher infection with spores of mixed virulence, and no infection at all if the variety has a resistance against all virulence races. Spikelets of spelt and emmer was dehulled before spore application to ensure contact between spore and kernel.

The expected NILs were provided by NordGen along with the original source of resistance. The NILs were contaminated with spores with and without virulence

Table 1: Study 2015 of the MacKey NILs from NordGen, developed from Starke II and lines with known Bt resistance genes against common bunt (Tilletia caries)

Accession	Spores used	Percent infected heads	95% conf interval
NGB11503 (NIL Bt1)	Vr: 1	8,3	(0,0 - 17,4)
NGB11503 (NIL Bt1)	Vr: 8	0,0	(0,0 - 0,0)
NGB11503 (NIL Bt1)	Vr: wes	0,0	(0,0 - 0,0)
NGB21283 (source Bt1)	Vr: 1	16,7	(0,0 - 37,8)
NGB21283 (source Bt1)	Vr: 8	0,0	(0,0 - 0,0)
NGB21283 (source Bt1)	Vr: wes	0,0	(0,0 - 0,0)
NGB11506 (NIL Bt10)	Vr: 1	5,9	(0,0 - 17,1)
NGB11506 (NIL Bt10)	Vr: 8	12,0	(0,0 - 24,7)
NGB11506 (NIL Bt10)	Vr: wes	54,2	(34,2 - 74,1)
PI-554-118 (Bt10)	Vr:1	0,0	(0,0 - 0,0)
PI-554-118 (Bt10)	Vr: 8	0,0	(0,0 - 0,0)
PI-554-118 (Bt10)	Vr: wes	53,6	(35,1 - 72,0)
Weston (Bt10)	Vr:1	0,0	(0,0 - 0,0)
Weston (Bt10)	Vr: 8	13,6	(0,0 - 28,0)
Weston (Bt10)	Vr: wes	52,6	(30,2 - 75,1)
NGB16160 (NIL Bt???)	Vr: 1	21,6	(8,4 - 34,9)
NGB16160 (NIL Bt???)	Vr: 8	12,9	(1,1 - 24,7)
NGB16160 (NIL Bt???)	Vr: wes	23,8	(5,6 - 42,0)
PI-554-114 (Bt7)	Vr:1	30,0	(9,9 - 50,1)
PI-554-114 (Bt7)	Vr: 8	3,7	(0,0 - 10,8)
PI-554-114 (Bt7)	Vr: wes	22,2	(6,5 - 37,9)
PI-554-100 (Bt7)	Vr:1	4,5	(0,0 - 13,2)
PI-554-100 (Bt7)	Vr: 8	9,1	(0,6 - 17,6)
PI-554-100 (Bt7)	Vr: wes	45,7	(29,2 - 62,2)
NGB16105 NIL Bt12)	Vr: mix	35,7	(21,2 - 50,2)
PI-119-333 (Bt12)	Vr: Mix	0,0	(0,0 - 0,0)
NGB11504 (NIL Bt6)	Vr: mix	3,4	(0,0 - 10,1)
Rio (Bt6)	Vr: Mix	0,0	(0,0 - 0,0)
NGB11505 (NIL Bt9)	Vr: mix	0,0	(0,0 - 0,0)
NGB21193 (Source Bt9)	Vr: mix	0,0	(0,0 - 0,0)
NGB16106 (NIL Bt5)	Vr: mix	0,0	(0,0 - 0,0)
NGB16106 (NIL Bt5)	Vr: 5	0,0	(0,0 - 0,0)
PI-554-104 (Bt5)	Vr: Mix	0,0	(0,0 - 0,0)
Promesse (Bt5)	Vr: 5	0,0	(0,0 - 0,0)

against the *Bt*-resistance-gene the NIL was expected to have. For one NIL there were no information of which resistance gene it should have. This line was therefore tested with different races hoping to gain information to give hints of the resistence gene in question.

In 2015-16, lines that in the first trial indicated some degree of resistance was tested again, this time with 8 different virulence races, and for comparison also a number of wheat varieties with known *Bt*-resistance genes.

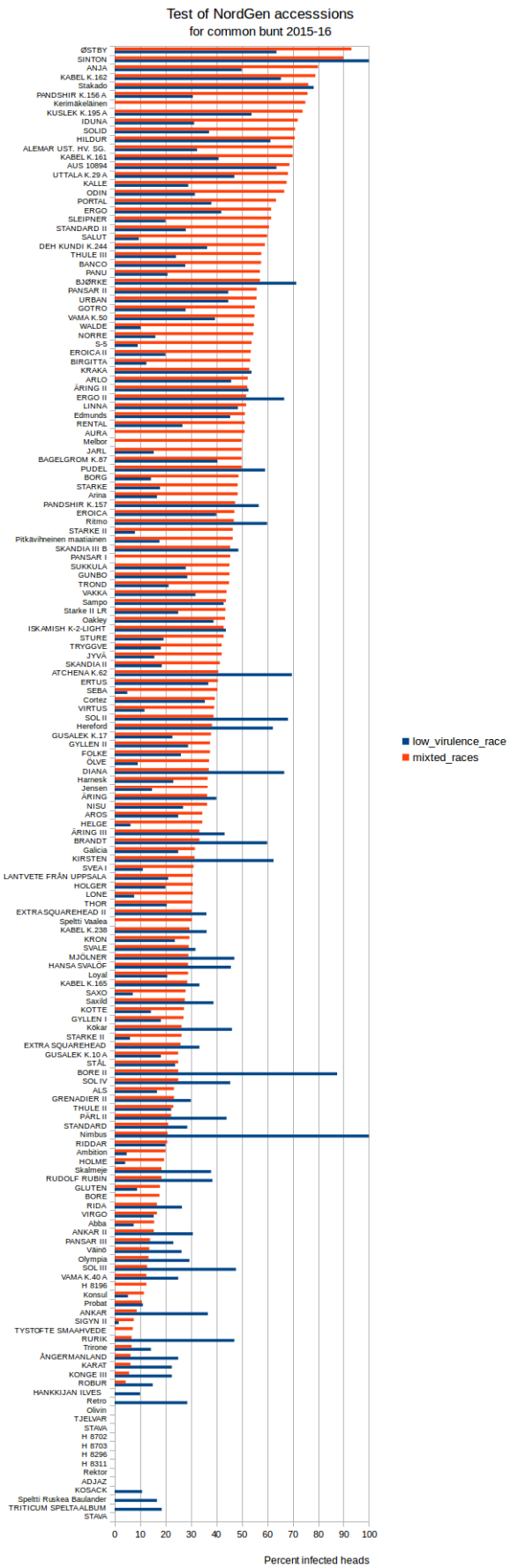
From MacKey´s NILs, 6-10 heads were collected and sown in head rows contaminated with spores of a race without virulance against the *Bt*-gene in question, in order to evaluate if the lines were homogenous.

In 2016-17, lines from Allkorn was tested in the same way with spores of low virulence. One line, Jacoby, was on beforehand expected to be resistant, and was therefore also tested with spores virulent to *Bt2*.

Results and discussion

The result of the 165 wheat accessions from NordGen showed infection leves from 0 to 100% (Figure 1). The infection level was in general higher using the mixed races compared with the race with low virulence, but there were exceptions most likely caused by the low number of tested plants. 16 accessions were selected for further studies the following year.

The result of the first test of the NILs is presented in Table 1. NGB11506 reacts in somewhat the same way as Weston and PI554118 both having *Bt10*. However, the line was also tested at Lantmännen in Sweden and at BOKU in Austria concluding that the line did not have *Bt10* (data not shown). The line NGB16160 with unknown resistance did not show any conclusive results, but there is some similarity with *Bt7*. NGB16105 has high infection to s mix of virulences of which none had virulence to *Bt12*. Therefore it is unlikely that this line has *Bt12*. NGB11504, NGB11505 and NGB16106 had low infection to the mixed races of which none had virulence to *Bt6, Bt9* or *Bt5*. Therefore it is likely that these lines indeed have the resistance genes indicated. However, the low infection in



NGB11504 may indicate that this line was not quite pure. Based onthese results, further studies the following year with head rows gave more detailed information. The results shows that the line

Table 2. Test of resistant wheat accessions against 8 different virulence races. Similar reaction to different virulence races indicates that the lines may have the same resistance genes.

	Vr: 1	Vr-2	Vr-G	Vr: 10	Vr: Q	Vr-5	Vr-13	Vr: 4
Stava	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0
PI-554-117 (Bt6)	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0
Rio (Bt6)	0,0	0,0	2,9	0,0	1,7	4,2	0,0	0,0
NGB-11504 NIL Bt6	6,5	2,2	3,7	0,0	7,7	0,0	5,0	7,8
NGB-11505 (NIL Bt9)	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0
PI-554-112 (Bt9)	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0
Pi 554099 (Bt9)	0,0	0,0	1,5	0,0	0,0	0,0	0,0	0,0
PI-554-098 (Bt11)	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0
PI-554-119 (Bt11)	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0
PI-554-106 (Bt12)	2,2	0,0	0,0	0,0	2,1	0,0	0,0	0,0
PI-119-333 (Bt12)	2,9	0,0	0,0	0,0	0,0	0,0	0,0	17,5
Hohenheimer Ci-11458 (Bt5)	0,0	7,4	8,3	7,0	4,3	0,0	4,8	0,0
NGB-16106 NIL Bt5	0,0			0,0	1,7		0,0	
PI-554-104 (Bt5)	0,0	0,0	0,0	0,0	0,0	0,0	0,0	3,9
Nebred (Bt4)	3,0	0,0	12,9	0,0	0,0	0,0	0,0	1,1
Promesse (Bt5)	0,0	0,0	34,3	0,0	0,0	0,0	0,0	0,0
PI-554-115 (Bt4)	7,8	0,0	26,1	5,7	0,0	13,5	0,0	3,0
PI-554-110 (Bt8)	16,7	0,0	10,0	16,7	0,0	0,0	0,0	0,0
PI-554-111 (Bt8)	66,7	2,7	55,0		30,0	17,9	0,0	0,0
NGB-17140 Speltti Ruskea Baulande	52,3	2,1			44,2	18,8	5,7	23,6
Pi173437 (Btp)	2,8	0,0	0,0	21,1	0,0	37,1	0,0	0,0
Thule III (Bt13)	0,0	0,0	0,0	3,3	0,0	0,0	45,7	0,0
Weston	0,0	5,9	5,9	43,8	0,0	2,8	0,0	0,0
PI-554-118 (Bt10)	0,0	1,3	11,3	38,8	3,8	4,1	0,0	2,8
PI-554-109 (Bt10)	0,0	0,0	25,0	28,6	0,0	16,7	0,0	0,0
NGB-11506 NIL Bt10	43,8	44,4	42,1	42,9	50,0	42,1	50,0	20,4
Inna (BtZ)	0,0	0,0	53,0	69,4	1,7	0,0	0,0	0,0
Nemchinovskaya 25 (BtZ) PI 591981	14,7	0,0	19,6	25,0	4,7	18,1	0,0	33,3
PI-554-101 (Bt1)	37,8	54,5	3,3	0,0	0,0	13,5	0,0	0,0
NGB-11503 NIL Bt1 (Albit)	81,3	63,6	0,0	7,3	0,0	6,3	0,0	31,9
PI-554-103 (Bt2)	14,6	44,4	0,0	4,0	0,0	0,0	2,0	22,0
PI-554-097 (Bt2)	17,4	43,6	0,0	30,0	24,0	0,0	5,0	0,0
NGB-7484 Rubrik	50,0	63,2	30,4	11,1	72,1	12,1	8,7	0,0
NGB-26 HELGE	48,7	29,5	28,6	19,0	72,1	31,7	5,5	39,6
Pi554-121 (Bt3)	5,8	0,0	0,0	0,0	61,3	3,6	0,0	5,9
PI 554-116 (Bt3	5,9	10,0	0,0	0,0	50,0	0,0	0,0	10,0
Ridit (Bt3)	0,0	3,2	0,0	0,0	27,5	0,0	0,0	0,0
NGB-473 SIGYN II	42,4	8,9	45,6	17,6	43,8	19,4	4,9	36,2
Erythrosperum 5221 (Bt14)	20,0	14,6	16,1	12,5	0,0	18,1	5,6	14,3
Lutescens	13,6	41,2	41,4	38,8	44,7	12,5	9,4	13,6
PI-554-114 (Bt7)	31,6	68,4	13,6	12,5	17,5	31,4	28,4	3,8
PI-554-100 (Bt7)	16,2	72,7	48,7	63,6	0,0	72,4	27,0	0,0
NGB-16160 NIL Bt?	22,7	29,0	21,2	32,1	51,7	37,8	31,1	39,3
PI 172201 (Doubbi resistens)	20,0	18,2	53,8	21,4	57,1	0,0	75,0	14,3
Gluten	40,7	12,2	61,2	26,4	75,4	49,0	22,7	59,6
NGB-347 AURA	60,7	45,5	65,2	34,0	38,7	42,9	26,5	37,3
NGB-16909 Probat	66,7	51,4	53,4	36,7	72,9	16,1	34,1	29,7
NGB-16852 Konsul	60,0	54,3	60,7	37,1	68,3	36,4	18,2	65,9
NGB6728 Seba	69,2	20,0	54,5	55,7	73,5	70,2	37,7	68,4
NGB-22 STARKE II	58,1	54,2	58,7	46,5	75,9	55,3	47,9	66,7
NGB-4 ANKAR	78,1	42,2	90,0	57,1	74,6	17,3	80,4	65,3
NGB-17139 Speltti Vaalea	65,4	57,4	86,1	67,6	83,3	83,3	41,0	33,3
NGB-13445 ÅNGERMANLAND	77,3	81,3		45,9	87,4	57,5	50,7	75,0
NGB-10883 TRITICUM SPELTA ALB	100,0	54,2	83,0	64,3	63,2	60,5	56,9	80,0

NGB-11503 is homogeneous and is resistant most like having *Bt1*, NGB-16106 is homogeneous and is resistant most like having *Bt5* and NGB-11505 is homogeneous and is resistant most like having *Bt9*. None of the tested lines of NGB16105 had resistance, whereas NGB-11506, NGB-11504 and NGB-16160 seems to be heterogeneous, with some lines being resistant.

Accessions from NordGen that seems to have some degree of resistance including the NILs in the first trial was included in a more detailed study in 2015-6 and tested with 8 different virulence races, and the results are presented in Table 2. These results shows that the lines Stava, NGB-11504 (*Bt6*), NGB-16106 (*Bt5*) and NGB-11505 (NIL *Bt9*) are indeed very resistant against all the races. Stava has in other studies show presence of the marker for *Bt9* [2], but may also have resistance gene Bt8. NGB-11506 (*Bt10*) do not react similar to the the lines with *Bt10*, but as shown in Table 3, this is most likely caused by diversity in the sample. Selected lines with resistance are now purified and will be tested again.

NGB-11503 (*Bt1*) has similarities in reaction with differential line PI-554-101 having *Bt1* confirming that they both have *Bt1*.

NGB-16160 with an unknown resistance gene reacts somewhat similar to *Bt7*, but as the line is diverse (Table 3), the indication must be taken with precaution.

Table 3. NILs based on Starke II tested for presence of Bt-genes

	Virulence Race	Percent Infected	
NGB-11503 NIL Bt1	Vr13	0,0	Most likely having Bt1
2	Vr13	0,0	
3	Vr13	0,0	
4	Vr13	0,0	
5	Vr13	0,0	
6	Vr13	0,0	
7	Vr13	0,0	
8	Vr13	0,0	
9	Vr13	0,0	
10	Vr13	0,0	
NGB-16106 NIL Bt5	Vr:Mix	0,0	Most likely having Bt5
2	Vr:Mix	5,7	
3	Vr:Mix	0,0	
4	Vr:Mix	0,0	
5	Vr:Mix	0,0	
6	Vr:Mix	0,0	
NGB-11505 (NIL Bt9)	Vr:Mix	0,0	Most likely having Bt9
2	Vr:Mix	0,0	
3	Vr:Mix	0,0	
4	Vr:Mix	0,0	
5	Vr:Mix	0,0	
6	Vr:Mix	0,0	
7	Vr:Mix	0,0	
NGB16105 NIL Bt12)	Vr:Mix	46,2	None of the tested lines had Bt12
2	Vr:Mix	65,2	
3	Vr:Mix	53,7	
4	Vr:Mix	84,1	
5	Vr:Mix	35,9	
NGB-11504 NIL Bt6	Vr:Mix	24,4	There seem to be some diversity among the lines, so maybe some of them do indeed have Bt6.
2	Vr:Mix	14,7	
3	Vr:Mix	0,0	
4	Vr:Mix	2,0	
5	Vr:Mix	7,4	
6	Vr:Mix	14,8	
NGB-11506 NIL Bt10	Vr:2	3,7	There seem to be some diversity among the lines, so even most of them are susceptible, maybe some of them do indeed have Bt10.
2	Vr:2	71,7	
3	Vr:2	76,0	
4	Vr:2	42,2	
5	Vr:2	59,5	
6	Vr:2	65,0	
7	Vr:2	1,6	
8	Vr:2	54,2	
9	Vr:2	57,4	
NGB-16160 NIL Bt?	Vr:4	0,0	There seem to be some diversity among the lines, so maybe some of them do indeed have a resistane gene.
2	Vr:4	82,1	
3	Vr:4	94,1	
4	Vr:4	88,4	
5	Vr:4	79,1	
6	Vr:4	0,0	
7	Vr:4	84,6	
8	Vr:4	56,4	

The variety Sigyn II does not react as any other variety, and this may indicate that this line do not have any of the known *Bt*-genes. Helge and Rubrik has some similarities with lines having *Bt2*. Speltti Ruskea Baulander (NGB-17140) has some resistance which is interesting because little has been done to identify resistance genes in spelt wheat. There are inconsistencies in the reactions between the lines with *Bt8*, and it is therefore not conclusive that Speltti Ruskea Baulander has indeed *Bt8*, and these results should be confirmed before any final conclusions are taken.

Heritage varieties maintained by the Allkorn also showed high susceptibility to common bunt as shown in Table 4. Only the German variety Jacoby and the Swiss spelt variety Oberkulmer Rotkorn can be considered resistant whereas all the other varieties of Nordic origin were susceptible. Jacoby has previously been shown to have resistance, and the test with spores virulent to *Bt2* indicate, that the line has resistance gene *Bt2*.

Conclusion

Most accessions from NordGen and Allkorn were susceptible to common bunt which is in line with other screening experiments of randomly selected varieties. However, a few lines not previously known to be resistant were shown to have different resistance genes. None of them however were resistant to all the virulence races tested. The MacKey´s NILs were evaluated and there seem to be a potential for using some of them as differential lines for resistance gene *Bt1*, *Bt5*, and *Bt9*. Purification based on resistant head rows may also develop NILs with resistance to *Bt6* and *Bt10* and possible also *Bt7* from NGB16160. A crossing program of the remaining *Bt*-genes was started in 2016. The NILs may be used also to support development of genetic markers of the resistance genes.

Table 4. Susceptibility of heritage varieteis maintained by Allkorn

	Infected heads %	95% Conf interval
Jacoby	0,0	(0,0 - 0,0)
Jacoby (infected with spores virulent to Bt2)	31,8	(12,4 - 51,3)
Oberkulmer Rotkorn (spelt)	3,3	(0,0 - 9,8)
Odin	21,2	(7,3 - 35,2)
Walde	23,5	(9,3 - 37,8)
Holger	25,0	(14,7 - 35,3)
Eroica	26,1	(13,4 - 38,8)
Eroica, white kernels	46,9	(29,6 - 64,2)
Red Prolific	29,6	(12,4 - 46,9)
Spelt Gotland	34,2	(19,1 - 49,3)
Lv Gotland	34,9	(20,6 - 49,1)
5113	37,5	(20,7 - 54,3)
Rauweizen	38,5	(12,0 - 64,9)
Erbe (white kernels)	38,5	(23,2 - 53,7)
Erbe (red kernels)	41,0	(25,6 - 56,5)
Holger brun	40,9	(26,4 - 55,4)
Börstvete Gotland	43,3	(25,6 - 61,1)
Robur	43,5	(23,2 - 63,7)
Svart Emmer (Hulless seed)	43,5	(23,2 - 63,7)
Svart Emmer	53,8	(38,2 - 69,5)
Starke	46,3	(31,1 - 61,6)
Svale	50,0	(33,7 - 66,3)
Aros	52,6	(36,8 - 68,5)
Banko	55,3	(41,1 - 69,5)
Sol	58,7	(44,5 - 72,9)
Sol (large kernels)	72,2	(51,5 - 92,9)
Ure	61,3	(44,1 - 78,4)
Lv Halland	78,4	(65,1 - 91,6)

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Strategic use of virulence pattern to develop genetic markers for resistance to common bunt (*Tilletia caries*) in wheat

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When assesssing races of common bunt for virulens pattern within a region, it is important to take into account that collected spores may represent a diverse population of different virulence races. When screening spores on a differential set of wheat lines with known resistance genes, a low infection rate on a resistant wheat variety does not necessarily demonstrate that virulence is absent in the spore collection, but could be a sign that virulence is present, but only present in a low frequency among the spores. If just a few spores within a spore sample are indeed virulent, they may infect some plants and from there multiply the virulence quite rapidly next years. Previous studies have shown that virulence against most resistance genes were present in Denmark after purifying races of common bunt (*Tilletia caries*) on resistant varieties. So far, only wheat differential varieties with *Bt4, Bt6, Bt9, Bt11* and *Bt12* cannot be infected with bunt races purified from Danish collections [1, and later own unpublished data]. Virulence against *Bt4, Bt6* and *Bt9* has been found in other European studies [2], and *Bt11* may not be only one gene but a combination of at least two genes [3]. Therefore, *Bt12* seems to be the only gene for which virulence have not been found in European population of common bunt. This leads to the conclusion that if resistance breeding shall safely control common bunt in wheat, we need not only one effective gene, but a combination of pyramided genes. Since it is very difficult to test if a resistant line has only one gene or more genes, the most effective tool to achieve this at present are genetic markers.

Using Genome Wide Association Studies (GWAS) to find QTLs and markers for the major resistance genes in wheat have so far led to only few commercial useful markers. Till now, only markers for *Bt9* [6] and *Bt10* are used in practice, but a marker for *Bt12* [4] and Blizzard [7] have also been found. One of the problems in developing markers for bunt resistance have been that spores used in GWAS trials have been divers or unknown in virulence, and that phenotypic results not distinguishes between different resistance genes. Therefore, the most successful studies have used segregating populations of single crosses where the resistance gene is known on before hand [5].

In the LIVESEED project, we have the ambition to develop genetic markers on several different resistance genes at the same time. We will do so by testing segregating populations of several different crosses between varieties with 7 different resistance genes, and infect them with 7-11 different virulence races of common bunt able to distinguish between the resistance genes. A total of 300 varieties will be pheno- and genotyped. Using this experimental design, we attempt during

2018 and ‘19 to develop markers for *Bt1, Bt2, Bt5, Bt7, Bt13, BtZ* and Quebon-resistance, and hopefully also a couple of minor QTLs.

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New tools available to control the common bunt of wheat: development of an early detection test on plantlet by qPCR.

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Common bunt, mainly caused by *Tilletia caries* and *T. foetida*, is a re-emerging disease since 2007 in France. This disease causes important yield reductions, has an impact on the seed health quality and demonstrates a significant capacity for dissemination.

GEVES, throughout a research project with ITAB and a European project named TESTA, developed a pathotest in control conditions with early detection by PCR on plantlets, correlated with expression of the symptoms on ears in the field. This early detection allows to evaluate transmission rate of the pathogen from seed to plant and to determine the threshold damage. A practical implementation was to define the derogation of the certification standard for bunt in seed lot by the ministry. A second implementation could be to evaluate the efficiency of the seed treatments.

Possible use of this pathotest in early evaluation of resistance of the varieties is ongoing in a new research project CASDAR: ABBLE, in collaboration with Arvalis, l'ITAB, FNAMS, CA26 and FREDON. Currently only 10% of varieties are considered as resistant and, little work has been done on the varietal resistance due to a lack of selection tools. In conventional farming, seed treatments resolve the problem, but this is not authorized in organic farming.

The program ABBLE was created to study the variability in populations of common bunt in France and to identify the main virulences present in France. In parallel an early detection method using qPCR was developed on plantlets. A resistance test was carried out in controlled conditions with detection at an early stage (2 leaves) and compared with symptom expression for similar varieties. Currently, the method allows the differentiation of susceptible varieties and resistant varieties.

Evaluation of the resistance of the varieties mainly used in organic farming will allow to acquire a better knowledge of the current resistance against the virulence mainly present in France. The development of an early, rapid and reliable resistant test will provide breeding with a new tool for screening resistant varieties.

These new tools, adaptable to other pathotest, are currently available to control the common bunt of wheat.

Mapping QTLs conferring additive resistance to Karnal bunt in bread wheat in two recombinant inbred lines populations

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Karnal bunt (KB) of wheat, caused by the fungus *Tilletia indica* Mitra [syn. *Neovossia indica* (Mitra) Mundkur], is one of the biggest challenges to the wheat grain industry. Yield loss is usually limited by this disease, but quality deterioration is a big concern, since merely 1-4% of kernel infection is sufficient to make wheat grain unpalatable and 5% of kernel infection causes distinct deterioration in flour quality. Additionally and more important, quarantine regulations restrict international movement of wheat germplasm, seed distribution, and wheat grain exports. Despite the fact that resistant donors have been identified in primary wheat gene pool, only a few have been subjected to genetic analysis. Earlier studies on genetics of resistance have indicated the presence of oligogenic rather than monogenic resistance, which complicates the introgression of multiple genes in elite cultivars. The scarcity of genetic studies on KB in wheat makes it imperative that researchers identify and map additional genes to widen the genetic base. The purpose of this research was to identify and map quantitative trait loci (QTL) conferring resistance to KB in hexaploid wheat from two recombinant inbred line (RILs) populations.

The first bi-parental population (POP1) derived from a cross between CIMMYT breeding lines 'BLOUK #1' and 'WHEAR/KUKUNA/3/C80.1/3*BATAVIA//2*WBLL1', was developed through single seed descent method and advanced to F_6 generation, with 165 progenies. The female parent 'BLOUK #1' is a KB susceptible line, whereas the male parent is a moderately resistant line, showing consistently better resistance than the female parent in several experiments. The second population (POP2) was developed using a similar approach with 'Huirivis#1' as female parent (susceptible), and 'Mutus' as male parent (moderately resistant), with 275 progenies. The populations were phenotyped in 2015-2016 and 2016-2017 growing seasons in an artificially inoculated KB nursery at Obregon, Mexico. The populations had two dates of planting: mid-November and early December. Field inoculations were done via injecting 1 ml of inoculum with a concentration of 10,000 sporidia/ ml into the boot at the booting stage, and five randomly selected spikes were inoculated in each plot. The inoculated spikes were separately harvested and manually threshed, then infected and total grain numbers were scored for each spike for the calculation of disease severity. Averaged KB severities over the five spikes were used in subsequent analysis. The populations were genotyped with the DArTseq genotyping-by-sequencing (GBS) platform. The SNP loci with missing genotypes in >20% RILs and showing segregation distortion from expected 1:1 ratio (based on chi-square test) were discarded before genetic mapping. Genetic linkage maps were constructed using JoinMap ver. 4.0 at a minimum LOD score of 3.0 using Kosambi and Holdane mapping function. Linkage groups were assigned chromosome names by

comparing published GBS consensus map. Least squares means of the percent KB infection from two seeding dates from two environments were used in the QTL analyses using MapQTL ver. 6.0. A simple interval mapping model was first performed to identify markers significantly associated with the traits. The final QTL analyses were performed using the closest markers identified by simple interval mapping as co-factors. The phenotypic data was analysed for analysis of variance using MIXED procedure in SAS ver. 9.4, where RIL entry was treated as a fixed factor, and environment and RIL entry by environment interaction as random factors.

The phenotype of parents in both populations was clearly distinct in both environments indicative of sufficient disease pressure. RIL entry, environment and their interaction were all significant for percent KB infection, with significant correlation among KB severities of RILs across environments. KB severity was quantitatively expressed as reported in previously published studies and transgressive segregation was observed in both populations. Multiple stable QTLs were mapped in both populations, which were detected in three or more environments. In POP1, three QTLs on 2B and two QTLs on 3D chromosomes were detected, explaining 6.7-11.1% and 6.7-9.6% phenotypic variation in different environments, respectively. In addition to these stable QTLs, a QTL on 7A (explaining 6.7% phenotypic variation) and two on 6A (explaining 7.3% and 6.7% phenotypic variation), were detected in 2016-2017 growing seasons' planting date 1 and 2, respectively. Except for the 6A QTLs, all others were contributed by the moderately resistant parent. In POP2 population, multiple QTLs with minor effect (explaining <8% phenotypic variation) were detected on 2D (1), 3B (2), 4A (2), 4B (3), 5A (1) 5B (3), 7A (2), and 7B (2). However, only two QTLs on 5B (4.1-7.5% phenotypic variation), one on 4A (4.6-5.2% phenotypic variation), and two on 3B (4.2-5.2% phenotypic variation) were detected in at least three of the four environments. Except for the QTLs on 2D and 3B, all others inherited resistance alleles from the male parent 'Mutus'. The majority of the QTL loci have an additive effect to improve resistance in resistant RILs in both populations. Hence, selection for genomic regions rich in KB resistance QTL can result in development of improved germplasm.

Prospects and challenges for breeding bunt resistant wheat using molecular marker assisted selection

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Dwarf bunt caused by *Tilletia contraversa* J.G. Kühn and common bunt caused by *T. caries* and *T. foetida* are two destructive diseases of wheat (*Triticum aestivum* L.). They can reduce grain yield and quality by replacing wheat grains by bunt balls, fungal sori filled with brown-black, unpleasant smelling spores. In the past several decades, with the advent of seed treatments, most breeding programs de-emphasized common bunt and dwarf bunt resistance selection. However, bunt host resistance has regained world-wide interest due to the increase in organic farming and concerns for more sustainable production methods in agriculture [1]. Lack of effective organic certified seed treatments has lead to increasing incidence and damage from common bunt. Development of organic certified treatments for dwarf bunt is even more difficult due to the timing of infection by soil borne innoculum and the necessity of anti-fungal activity that can persist throughout a lengthy infection period.

The three *Tilletia* species that cause these two bunt diseases are closely related [2], and both diseases in wheat are partly controlled by shared genes (*Bt*) in a gene-for-gene system [3, 4]. Fifteen resistance genes (*Bt1* to *Bt15*) were proposed based on phenotypic evaluation of differential lines [4], but none have been well characterized at the molecular level. Current dwarf bunt resistance breeding efforts are mainly based on phenotypic selection, which is extremely difficult in the field since disease development requires a period of snow cover and low temperature of 3-8 C for spore germination [3] and the disease can only be assessed when plants are mature. Molecular marker-assisted selection (MAS) is an alternative method and an efficient way of pyramiding multiple genes to achieve a high level of resistance that is potentially durable.

The present study used advance genotyping platforms (DArT, 9K or 90K SNP) and dwarf bunt resistance assessment in field nurseries in Logan and Green Canyon, Utah, USA in three winter wheat bi-parental populations. One novel QTL *Q.DB.ui-7DS* was identified on chromosome 7DS in a recombinant inbred line population (IRRIL, 159 lines), which was derived from a cross between a susceptible hard white winter cultivar 'Rio Blanco' and a resistant hard red winter line 'IDO444' [5]. The 7DS QTL explained up to 53% of phenotypic variation for dwarf bunt resistance. Two additional major QTL were identified on 6DL (*Q.DB.ui-6DL)* and 7DL *(Q.DB.ui-7DL)* in a doubled haploid (DH) population (IMDH, 130 lines) derived from a cross between the resistant hard white winter line IDO835 and the susceptible hard red winter cultivar 'Moreland'.

The two QTL explained up to 41% and 39% of phenotypic variation in dwarf bunt resistance, respectively (data to be published). Based on the most recently released Chinese Spring reference sequence (RefSeq v1.0) and the associated physical map, the QTL *Q.DB.ui-6DL* co-located with the *Bt9* gene locus, which was mapped by Steffan et al. (2017) for common bunt resistance [6]. The QTL *Q.DB.ui-7DL* is located at the flanking region of the long arm of chromosome 7D, where Singh et al., (2016) found a minor QTL for common bunt resistance [7]. In a third DH population (UI Silver x Shaan89150, SSDH, 130 lines), the QTL *Q.DB.ui-6DL* and QTL *Q.DB.ui-1AS* were detected and explained 10% and 9% of phenotypic variation, respectively (data to be published).

The peak SNPs of the three major QTL were converted to KASP markers (Kompetitive Allele Specific PCR) and genotyped in a set of bunt differential lines, known resistance sources, and resistant cultivars to identify bunt gene-sepecific KASP markers. The results demonstrated potential use of the KASP markers in breeding for dwarf bunt resistance and suggested future research priorities.

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Comparative mapping of bunt resistance genes in winter wheat

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Bunts of wheat were a serious threat to wheat before the advent of effective fungicides. With the recent increase in organic crop acreage, however, bunt diseases re-emerge as relevant wheat diseases. The major bunt pathogens in Europe are *Tilletia tritici* and *Tilletia laevis* causing common bunt and *Tilletia controversa* causing dwarf bunt. Infested wheat crops display varying levels of yield losses and quality losses due to the fishy smell of bunt spores caused by trimethylamine. Most current wheat cultivars are highly susceptible to bunt. Resistance to bunt diseases is therefore of increasing relevance, particularly for organic wheat production.

We evaluated three RIL mapping populations for bunt resistance developed from crosses between parents with high resistance to common bunt and dwarf bunt and the susceptible Austrian winter wheat cultivar Rainer. The resistant parents were Bonneville and Blizzard, two North American winter wheat cultivars with unknown resistance; and PI119333, a landrace which is described as carrier of the bunt resistance gene *Bt12*.

All mapping populations were evaluated in nurseries inoculated with either common bunt or dwarf bunt, using local spore mixes during 2-3 seasons. Bunt severity was determined as percentage of bunted (infected) spikes over the total spikes within a plot. Phenotypic data collected were highly informative with broad sense heritabilities of 0.94-0.98 and 0.83-0.85 for common bunt and dwarf bunt severity, respectively. High density genotyping of all parental and RI lines was performed using a 15K Illumina SNP array. Marker data were used for linkage map construction and QTL mapping.

Both large effect and small effect QTL were detected. Blizzard and Bonneville share a large effect QTL for common bunt and dwarf bunt resistance on chromosome 1A, and a large effect QTL for common bunt resistance on 1B. Several small effect QTL for dwarf bunt resistance mapped to 2D, 7A and 7B. The *Bt12* donor line PI119333 was tested for common bunt resistance only and possesses a large effect QTL on 7D and a small effect QTL on 4B.

In summary, several large effect alleles for resistance to dwarf bunt and/or common bunt were detected and mapped. These are promising targets for marker assisted selection and rapid introgression of bunt resistance into locally adapted wheat cultivars.

Organic methods of controlling common bunt at the farm level.

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In wheat crops, appropriate seed treatment fungicides have very good effect against seed-borne common bunt, caused by *Tilletia tritici* or *T. laevis*. However, in organic farming, other methods than chemical pesticides must be used, such as resistant varieties, removal or killing of the seed-borne spores. In an ongoing project (2017-2019) in Sweden the purpose is to compare some ecologically acceptable methods of treatment and sanitation against common bunt. The goal is to control common bunt by using organic methods at farm level. Methods and preliminary results will be presented at the XXth International Workshop on the Smuts and Bunts.

Sources of resistance to a common bunt - the results of an international wheat germplasm testing

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Common bunt (CB) is one of the most destructive diseases of wheat worldwide, causing considerable yield loss and reduction in seed quality. Therefore its management was intensively studied. The review of the studies indicates that at the present stage of the development of wheat breeding that the most feasible approach for managing the bunt diseases of wheat is to use resistant cultivars. Such cultivars directly benefit farmers by assuring production [Blair J., Goates; Harold E. Bockelman, 2012]. In organic agriculture systems growing resistant cultivars is the most economic and environmentally friendly option in bunt control, especially important because of likely decrease in the use of chemical seed treatments [Ciuca M; Saulescu N. 2008]. The goal of ongoing research was the search for new sources for the creation of donors of resistance to common bunt.

Material and methods

As a research material for screening were used the accessions from 2 International nursery: 1) CAC (2012-2014) and 2) Common Bunt Resistant Nursery (CBUNT- RN 2015-2016). The CAC Nursery formed by CIMMYT for the countries of Central Asia and the Caucasus contained realized varieties of winter wheat from 9 countries: Kazakhstan; Kyrgyzstan; Uzbekistan; Tajikistan; Armenia; Azerbaijan; Russia; Mexico, Turkey. Common Bunt Resistant Nursery (CBUNT- RN 2015-2016) was prepared and distributed by the National Wheat Improvement Program of Turkey (CIMMYT/ICARDA). This nursery will combine different type of germplasm resistant to common bunt (75 accessions from 7 countries - Iran, Kazakhstan, Mexico, Romania, Russia, Turkey and USA). The resistance to *Tilletia caries* has been identified through at least two cycles of held evaluation under artificial inoculation conducted by Transitional Zone agricultural Research Institute (Eskisehir, Turkey). Majority of the entries will possess resistance to yellow rust. The essential part of research was an assessment of the resistance to common bunt (*Tilletia caries*). Immunological evaluation of the tested material was carried out by artificial pre-seed contamination of seeds with spores of common bunt of the local population before sowing. Bunt incidence is observed at plant maturity as the percentage of infected heads.

Results

The results of the assessment of the resistance to common bunt of the CAC nursery material on an artificially created infectious background has shown 5 different reaction class (% of infection incidence): 1) R – resistant (0-10%) - Sultan (Turkey); 2) MR – moderately resistant - (11-30%) -

Sathheni (Armenia), Naz, Krasnovodopadskaya 25 (Kazakhstan), Sanzor 8, Ulugbek 600 (Uzbekistan), Adyr (Kyrgyzstan), Umanka (Russia); 3) MS - moderately susceptible (31-50%) - Steklovidnaya 24, Karlygash, Yuzhnaya 12, Zhetysu (Kazakhstan), Sharora (Tajikistan), Kiyal (Kyrgyzstan), Nairi 149 (Azerbaijan) and Scifyanka (Russia); 4) S - susceptible (50-70%) – Almaly (Kazakhstan); 5) HS – highly susceptible (over 70%) - Oktyabrina 70, Arap (Kazakhstan), Ani 591 (Azerbaijan).

By studying of the CBUNT-RN nursery accessions for two years (2016-2017) on an infectious background were allocated: 1) 46.7% of resistant (R); 2) 30.7% of moderately resistant (MR); 16.0% of moderately susceptible (MS); 5.3% of susceptible (S); 1.3% of highly susceptible (HS). The level of infection incidence of Nasibey (TCI) and Mufitbey (TCI) varieties used as resistant standards was from 2 to 10% (R). The variety Gerek 79 (TR-ESK) used as a susceptible standard was infected up to 39.8% (MS). The Table 1 shows the data of the CBUNT-RN nursery accessions infection level by *Tilletia caries* in the context of countries, 2017.

Table 1. Distribution of CBUNT–RN nursery accessions on reaction by *Tilletia caries* infection, 7 accessions from 35 resistant showed yields at the level of local standards (over 5 t/ha).

Country	Reaction class/number of accessions					
	R	MR	MS	S	HS	Total
Iran	3	4	2	-	-	9
Kazakhstan	3	-	-	-	-	3
Mexico - TCI	-	2	1	-	-	3
Romania	3	1	1	-	1	6
Russia	2	1	-	-	-	3
Turkey- TCI	22	14	6	3	-	45
USA - TCI	2	1	2	1	-	6
Total	35	23	12	4	1	75

Table 2. Productivity of CBUNT–RN nursery resistant accessions, 2017

Original number	Cross	Country	Yield (t/ha)	Infection level (%)		
				YR	LR	CB
22	Krasnodar/Frtl/6/Ngda146/4/Ymn/ Tob//Mcd/3/Lira	TCI	6.2	30S	15MS	8.6
66	Bezostaya/Tr.militinae//Tr.militinae-4	KAZ	5.7	0R	15MR	0
35	Ks902709-B-5-1/Burbot-4	TCI	5.6	0R	10MS	0
44	Rina-6/Bez/Nad//Kzm(Es85.24)/3/F900k	TCI	5.4	0R	10MS	0
38	Batera//Kea/Tow/3/Tam200/4/494j6.11 /Trap#1/	TCI	5.3	5MS	10MS	5.9
23	362k2.111//Tx71a1039.Vi*3/Ami/3/Es 14/130l1.12.	TCI	5.2	0R	10MS	10
43	Rana96/Gansu-3	TCI	5.1	20MS	15MR	0
1	Mufitbey (Check-R)	TCI	6.1	10MR	20MR	10.1
2	Nasibey (Check-R)	TCI	2.2	10MR	15MS	2
3	Gerek 79 (Check-S)	ESK	2.6	5MS	15MS	39.7
	Almaly (Local Check–S)	KAZ	5.2	15MS	25MS	68.3

Conclusions

Ongoing studies have shown that 46.7% of accessions of the CBUNT-RN nursery were characterized by high immunological indicators of resistance to common bunt (R - 0-10% of infection). The presence among the resistance and moderate resistance gene pool the productive varieties of Kazakhstani breeding - Naz, Krasnovodopadskaya 25 and new constant line - Bezostaya/Tr.militinae//Tr.militinae-4 - indicates the perspective of the direction of the local breeding for resistance to a common bunt, while maintaining high productivity and quality. The promising material will be used as sources of genetic resistance to common bunt in local breeding programs.