Co-evolution of virulence and resistance in heterogeneous wheat populations

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Abstract

The pathogenesis of common bunt caused by *Tilletia caries* or *T.leavis* is that spores are introduced to a crop via farm equipment (Kristensen and Borgen 2001) or soil contamination (Borgen 2000A). Hereby, the new crop will be infected, and secondary infection will happen from year to year during threshing, as more plants will be infected and the infection rate is proportional with the amount of spores in the seed lot (Heald 1921). The speed of multiplication from year to year will depend on many factors, including equipment used for threshing (Kristensen and Borgen 2001) and seed cleaning (Borgen 2005), climatic conditions in particular in the period after sowing and the resistance of the variety and the virulence of the spores (Borgen 2000B).

Varieties with a single dominant resistance gene will according to the gene-for-gene relationship discovered by Harold Henry Flor (1942) either not be infected, if the spores are avirulent to the resistance gene, or it will be fully susceptible if the spores are virulent. This system is widely accepted as the dominant mechanism for pure line varieties with the known resistance genes summerised by Blair Goates (2012). However, little is known about what happens if a crop is not a pure line variety with a single resistance gene, or if the spores are not a single race with a specific virulence.

In the new EU Regulation for organic farming (EU 2018), a new term is introduced called Organic Heterogeneous Material (OHM), which is not a single variety but a mixture of plants with diverse genetic background. As OHM is introduced for organic farming, and common bunt is a main issue for organic wheat production, organic breeders of OHM often attempt to introduce resistance to common bunt in the populations. Among the OHM accepted in EU so far are Brandex and Liocharls from Dottenfelder Hof and Mariagertoba and Popkorn from Agrologica. These populations all have resistance to common bunt with Bt7 dominant in Brandex, Liocharls and Mariagertoba and with a broader mixture of resistances in Popkorn.

The aim of this trial is to study to dynamic of the epidemiology of common bunt over years in populations with multiple resistances.

8 varieties were used in the trial with differences in resistance genes:

1. NIL1 (Bt1)
2. NIL5 (Bt5)
3. Promesse (Bt5)
4. NIL6 (Bt9)
5. NIL9 (Bt9)
6. NIL10 (Bt10)
7. Magnifik (Bt9(+?))
8. Pi554121 (Bt3)

race		Variety/mixture	Frequency of avirulent genes	Actual Infection **2022** % in the plot	Actual Infection **2021** % in the plot	Expected infection in the mixture based on the infections in the components when tested alone
Vr10	2	NIL 1+10	50	57,4	15,0	23,5
					1,2	0,0
Vr10	2	NIL 5+10	50,0	33,3	22,1	23,5
Vr10	3	NIL 1+5+10	66,7	27,6	10,0	15,7
Vr10	4	NIL 1+5+10+6	75,0	12,1	6,1	11,8
Vr10	4	NIL 1+5+9+10	75,0	15,3	7,4	11,8
Vr10	6	NIL 1+5+10+6+9+Promesse	83,3	13,6	5,4	7,8
					6,5	6,7
Vr10	8	NIL 1+5+10+6+9+Promesse+Magnifik+Pi554121	87,5	14,6	5,1	5,9
Vr2	2	NIL 1+10	50,0	17,8	10,9	23,5
Vr2	2	NIL 1+5	50,0	17,4	8,0	23,5
Vr2	3	NIL 1+5+10	66,7	18,1	12,3	15,7
Vr2	4	NIL 1+5+10+6	75,0	15,5	6,1	11,8
Vr2	4	NIL 1+5+9+10	75,0	11,4		
Vr2	6	NIL 1+5+10+6+9+Promesse	83,3	19,6	0,0	7,8
Vr2	7	NIL 1+5+10+6+9+Promesse+Magnifik	85,7	18,9	2,5	7,8
Vr2	8	NIL 1+5+10+6+9+Promesse+Magnifik+Pi554121	87,5	5,5	0,0	6,7
					1,7	6,7
					3,9	5,9
Vr5	2	NIL 1+5	50,0	47,6	12,3	23,5
Vr5	3	NIL 1+5+10	66,7	21,6	16,4	15,7
Vr5	4	NIL 1+5+10+6	75,0	15,0	7,0	11,8
Vr5	4	NIL 1+5+10+9	75,0	23,9	5,2	11,8
Vr5	6	NIL 1+5+10+6+9+Promesse	66,7	12,5	10,5	15,7
Vr5	7	NIL 1+5+10+6+9+Promesse+Magnifik	71,4	9,3	2,4	13,4
Vr5	8	NIL 1+5+10+6+9+Promesse+Magnifik+Pi554121	75,0	10,5	6,6	11,8
Vr5+10	3	NIL 1+5+10	33,3	48,2	20,9	31,3
Vr2+10	3	NIL 1+5+10	33,3	32,0	21,1	23,5
Vr2+5	3	NIL 1+5+10	33,3	27,2	15,8	23,5
Vr2+5+10	3	NIL 1+5+10	0,0	29,7	13,0	23,5

The varieties were selected with the hope that they all had different resistance genes, but later trials have shown that this was not quit true. Promesse, NIL6 and Magnifik was included as they were expected to have Bt4, Bt6 and Bt8 respectively, but it turned out that they did not. The NIL's (described in Borgen *et al* 2018) were used as these being closely related are expected to compete in the population mainly based on their differences in response to common bunt and not on agronomic performance in the field.

All varieties were tested for response to bunt of 3 different races of bunt.

Mixtures were made by mixing the varieties in equal amounts with increasing degree of diversity regarding resistance genes, or rather increasing expected degree of resistance as some varieties did not truly have the resistance genes expected at the time of experimental design.

Each mixture were then infected with one of the three races of bunt spores, and with a combination of races. After assessment and at maturity of the wheat, healthy and infected plants were harvested separately, and spores of the infected plants were then applied to the seed of the healthy plants for regrowing in the following season. This design was chosen to avoid the problem of contaminating threshing equipment and thereby mixing up the spores in the different seed lots. The design however has the limitation that it does not represent true agricultural practice, which may affect the epidemiology of the disease.

Based on the infection of the pure line varieties by each race, an expected infection rate can be calculated by the proportion of the different varieties in the mixtures. In some cases, the actual infection differed from the expected infection (Table 1). These differences can be affected by epidemiological effects of the mixtures.

In the first year (2021), the actual infection in the mixtures was generally slightly lower what was expected from the infection in the pure lines and the proportion of the varieties in the mixtures. In the second year (2022) it was higher.

When a plant is infected with common bunt, the grain yield of the infected plant will be reduced by 50-100% as infected heads will produce no seed. Some plants of a susceptible variety will not be infected. In conclusion, it is expected that selection pressure in disfavor of susceptible varieties will reduced these by some 50% in comparison with the resistant varieties within the mixtures. From year to year, it would therefore be expected that the mixtures gets more and more dominated by resistant varieties and the frequency of susceptible varieties will decrease. After two years, this effect seems not to be recorded in the actual infection rate. There is a possibility that spores from other races have contaminated the mixtures by mistake, or that the fungal races develop and adapt within the mixtures faster than the dynamic between wheat varieties. The most dominant effect however may be that each year spores are re-applied to the mixtures by artificial spore application. In real life, the amount of spores will depend on the frequency of infected plants in the field. It is possible that the actual infection rate under farm conditions will be so low that the amount of spores will be limiting the infection rate, whereas in this experiment, that same amount of spores are applied to all mixtures, disregarding the differences in infection rate in the previous season. The chosen design has the presumption that a few percent of infected plants will produce so many spores that it will matter little if there are more than a few percent infected plants, but maybe this is not really the case.

Even though the infection rate is still high after selection pressure, this means that secondary infection is still able to maintain infection. It is likely the composition of the mixtures have indeed changed and increased resistance, and that this will give increased protection against new primæary infection, if the secondary infection is eliminated by seed treatment.

The experiment will continue for another year to follow the development.

Keywords

winter wheat, common bunt, organic agriculture, population dynamics, Organic Heterogeneous Material.

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