Genetic Resources for Disease Resistance Breeding in Wheat

Characterization and Utilization

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Abstract

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Bread wheat is affected by many diseases that cause yield and quality losses. The incorporation of resistance genes is a user- and environmentally friendly method to manage disease. It is highly desirable that the resistance is durable and that the genes have a positive or no influence on agronomic performance. The characterization of genetic resources for resistance is a prerequisite for their utilization in plant breeding.

The aim of the present thesis was to characterize resistance against the fungal diseases leaf rust and powdery mildew in (i) bread wheat landraces and cultivars, and (ii) a wheat-rye hybrid carrying a T2BS.2RL chromosome translocation. The methods included pathological tests, field trials and molecular DNA markers.

Nine leaf rust seedling resistance genes were identified in 47 of 84 North European wheat cultivars. Seven powdery mildew seedling resistance genes were identified in 21 of 155 Nordic landraces and cultivars. More than 50 Nordic seedling susceptible wheat accessions displayed high levels of adult plant powdery mildew resistance that could be effective in disease control.

The genetic diversity among 198 Nordic wheat landraces and cultivars from before 1900 to 2003 was studied using retrotransposon-based S-SAP markers. Diversity was clearly separated between spring and winter wheat. The genetic diversity decreased during the early period of plant breeding, increased after the late 1960s and resulted in a net gain. Useful characters could be present in landraces that are not found in modern wheat material.

The 2RL rye segment in the wheat-rye hybrid T2BS.2RL, SLU was found to confer seedling resistance against powdery mildew, leaf rust, stem rust; and adult plant resistance against stripe rust. The 2RL was associated with two to three days later flowering time and an increased number of spikelets per spike. It had no significant effect on several agriculturally important characters including yield, fertility and lodging. Microsatellite mapping and linkage analysis identified a major QTL for powdery mildew resistance located on 2RL. The T2BS.2RL appears promising for wheat improvement.

The wheat-rye hybrid and the wheat accessions with high levels of resistance constitute genetic resources that could provide durable and effective disease control in wheat.

Keywords: Triticum aestivum, genetic resources, powdery mildew, leaf rust, genetic diversity, wheat, rye, microsatellites, retrotransposon, translocation.

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It is your mind that creates this world.

Gautama Buddha

A thousand mile journey begins with one step.

LaoZi

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II. Hysing, S.C., Merker, A., Liljeroth, E., Koebner, R.M.D., Zeller, F.J. & Hsam, S.L.K. Powdery mildew resistance in Nordic bread wheat cultivars and landraces during a century of breeding. (Submitted)

III. Hysing, S.C., Säll, T., Nybom, H., Liljeroth, E., Merker, A., Orford, S. & Koebner, R.M.D. Temporal diversity changes among 198 Nordic bread wheat landraces and cultivars detected by retrotransposon-based S-SAP analysis. (Manuscript)

IV. Hysing, S.C., Hsam, S.L.K., Singh, R.P., Huerta-Espino, J., Boyd, L.A., Koebner, R.M.D., Cambron, S., Johnson, J.W., Bland, D.E., Liljeroth, E. & Merker, A. 2007. Agronomic Performance and Multiple Disease Resistance in T2BS.2RL Wheat-Rye Translocation Lines. *Crop Science*. In Press.

V. Hysing, S.C., Hsam, S.L.K., Merker, A. & Röder, M.S. Mapping of powdery mildew and leaf rust resistance genes on rye chromosome 2RL in wheat-rye translocation lines. (Submitted)

Supplementary information

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B. UPGMA dendrogram based on a Jaccard coefficient similarity matrix from S-SAP generated banding polymorphism in Nordic bread wheat (Paper III)

C. Abbreviations

1. Introduction

Bread wheat (*Triticum aestivum* L.) is one of the most important sources of food grain and animal feed in the world. It is the leading crop regarding the use of land area, followed by rice, maize and potatoes (FAOSTAT, 2006). The unique composition of the wheat kernel includes the elastic protein gluten that makes it possible for the dough to rise in leavened bread. Consumption worldwide has increased rapidly since the early 1960s, primarily in developing countries as a result of increased urbanization and a shift in preferences favouring wheat (Curtis, 2002). The world population growth rate from 1993 to 2000 is estimated at 1.5 percent per year, while the growth rate of wheat production from 1985 to 1995 was 0.9 percent (CIMMYT, 1996). As wheat is a leading global food source, there is a need to increase wheat production.

Wheat can be grown in a range of environments in temperatures between 3 to 32°C (optimum 25°C) and an annual precipitation of 250 to 1750 mm. Increased wheat production can be achieved by: (i) expanding the wheat area; (ii) improving the yield; and (iii) reducing pre- and post-harvest losses (Curtis, 2002). The area under cultivation has expanded from 200 million ha in the 1950s to 218 million ha in 2005, while the world wheat yield increased from about 1 t/ha in the 1950s to 2.9 t/ha in 2005 (CIMMYT, 1996; FAOSTAT, 2006). Plant breeding and improvement of cultural practices were largely responsible for this achievement. Yield stability has increased substantially across environments due to the breeding of management-responsive, high-yielding, disease resistant semidwarf wheat cultivars, improved agronomic practices and the use of nitrogen fertilizers and irrigation (Reynolds & Borlaug, 2006).

In the future, due to other agricultural and non-agricultural uses, an increased area of fertile agricultural lands is likely not available for wheat production. Improved cultural practices and breeding for cultivars adapted to abiotic stresses may allow the use of marginal environments such as acid and saline soils (Curtis, 2002). However, as wheat is continuously attacked by diseases and pests that cause crop and quality losses, approaches to increase and maintain genetic gains depend on the breeding for resistance. The evolution of complete or partial resistance in pathogens against major fungicides has further emphasized the need of host resistance (Wolfe, 1984). Disease resistance thus represents the most efficient, economically and environmentally sustainable method of control (Bennett, 1984).

Breeding for disease resistance depends on the availability of sources of resistance found in landraces, modern cultivars, breeding lines and close or distant relatives of wheat (Singh & Rajaram, 2002). Genetic erosion (Harlan, 1972) limits further improvement, and makes wheat more vulnerable to biological and environmental stresses. The collection, preservation and distribution of wheat germplasm held in national and international collections are vital to wheat breeding. In this perspective, it is hoped that this PhD study on the genetic diversity and the characterization of novel and traditional genetic sources for disease resistance contributes to present and future wheat breeding.

2. Background

2.1 Wheat genetic resources

Bread wheat

Bread or common wheat, *Triticum aestivum* L. (2n=6x=42, AABBDD), belongs to the tribe Triticeae in the family Poaceae. Morphological, cytogenetic and molecular studies have shown that the genomic evolution of the segmental allohexaploid occurred through the hybridization of *Triticum turgidum* L. (2n=4x=28, AABB) with *Aegilops tauschii* Coss. var. *strangulata* (Eig) Tzvelev (syn. *Triticum tauschii, Aegilops squarrosa*) (2n=2x=14, DD) (McFadden & Sears 1946; Kihara, 1944; Bowman *et al.*, 1983; Lafiandra *et al.*, 1992). The origin of the A^u genome is recognized as *Triticum urartu* Tum. ex. Gand. (Dvorak *et al.* 1993) while several S genome species in genus *Aegilops* sect. *sitopsis* have been suggested as candidates for the origin of the B genome (Giorgi *et al.*, 2003). The taxonomic treatment of van Slageren (1994) of the genera *Triticum* and *Aegilops* is used throughout this thesis.

Bread wheat arose around 7000 B.C. in the region extending from Transcaucasia to the southwest coastal areas of the Caspian Sea (Zohary & Hopf, 1993). The D genome in hexaploid wheat most likely contributed to a wider range of climatic adaption that facilitated the spread from the primary centre of diversity and area of origin (Feldman *et al.*, 1995). Today, wheat is grown from within the Artic circle to higher elevations near the equator.

The domestication of the early wheats progressed by (i) subconscious selection by the earliest growers to produce non-brittle spikes, simultaneous ripening of grains, rapid and synchronous germination, and free-threshing ears; (ii) deliberate selection among variable material in the field of the primitive farmer for increased yield, larger grain size and adaption to a range of climates and farming practices; (iii) planned breeding for uniformity, high yield, a semi-dwarf growth habit, increase in the number of florets, larger ear size, and resistance to biotic and abiotic stresses (Feldman *et al.*, 1995).

Figure 1. Bread wheat (from Köhler's Medicinal Plants 1887).

Genetic resources

Plant genetic resources have been categorized by Frankel (1977) and the Food and Agriculture Organization of the United Nations Commission on Plant Genetic Resources (FAO, 1983, 1998). The following categories are recognized:

- modern cultivars in current use;
- obsolete cultivars (often cultivars of the past and
- found in the pedigrees of modern cultivars);
- landraces;
- wild relatives of crop species;
- genetic and cytogenetic stocks;
- breeding lines.

An estimated 640 000 accessions of *Triticum* spp., *Aegilops* spp. and ×*Triticosecale* Wittmack. (triticale) are held in *ex situ* or *in situ* collections around the world (Skovmand *et al.*, 2002).

The traditional classification of plants according to the gene pool concept (Harlan & de Wet, 1971) reflects the ease of utlilization of genetic resources for plant breeding. The primary gene pool consists of the biological species, including cultivated, wild and weedy forms of a crop species. Gene transfer from the primary genepool is considered to be simple and F_1-hybrids are generally fertile with good chromosome pairing. The secondary gene pool includes biological species (coenospecies) that will hybridize with the primary gene pool species with some difficulty, resulting in F_1-hybrids with at least some fertility. The tertiary gene pool is composed of species with genomes non-homoeologous to bread wheat. Gene transfer is very difficult and special methods e.g. the use of bridging materials or ionizing radiation treatments are required. Hybrids tend to be anomalous, lethal or completely sterile and therefore require the application of methods e.g. embryo rescue to increase survival. The gene pools of bread wheat are shown in (Fig. 2).

The utilization of genetic resources from the primary gene pool occurs mainly through the use of modern wheat cultivars or breeding lines. Wheat landraces are morphologically recognizable, genetically diverse and dynamic populations (Harlan, 1975) that represent an important source of genetic variation in wheat. There have been several definitions of the term ‘landrace’. Zeven (1998) proposed the following: “an autochthonous landrace is a variety with a high capacity to tolerate biotic and abiotic stress, resulting in a high yield stability and an intermediate yield level under a low input agricultural system”. In the present thesis, ‘landrace’ is used in the broad sense as locally adapted material that has been genetically improved by traditional agriculturalists but has not been influenced by modern breeding practices.

Examples of useful characters transferred from landraces to modern cultivars include the *Rht* dwarfing genes from the Japanese landrace Shiro Daruma in the wheat line ‘Norin 10’ that improved lodging and yield (Kihara, 1983; Borlaug,

1988) and powdery mildew resistance gene *Pm24* from the Chinese landrace 'Chiyacao' (Huang *et al.*, 1997). Several genes for resistance to biotic and abiotic stresses have been identified in landraces (Skovmand & Rajaram, 1990; Hede *et al.*, 1999; Skovmand *et al.*, 2001). The majority of the thousands of landraces stored in seed banks are inadequately described for an efficient exploitation for breeding. Furthermore, the cost of time and resources associated with the search for useful characteristics, and the intensive pre-breeding required to transfer the desired genes into advanced breeding lines limits the use of landraces in modern plant breeding (Skovmand & Rajaram, 1990; Gollin *et al.*, 1998).

Wide-hybridization between wheat and non-*Triticum* (alien) species from the secondary and tertiary gene pools have been successfully accomplished between wheat and more than 40 species including rye *Secale cereale* (2n=2x=14, RR) (Jiang *et al.*, 1994; Mujeeb-Kazi & Villareal, 2002; McIntosh *et al.*, 2003; Mujeeb-Kazi & Rajaram, 2002). However, the alien chromosome segments often cannot compensate for the loss of wheat chromatin, or undesirable alien genes are linked to the desirable genes. Therefore, very few wheat-alien hybrids have led to the production of commercial cultivars (Jiang *et al.*, 1994; Friebe *et al.*, 1996).

The close homoeology between wheat and rye facilitates gene transfers from rye into wheat (Naranjo *et al.*, 1987; Devos *et al.*, 1993; Lukaszewski *et al.*, 2004). Several rye genes conferring resistance against biotic and abiotic stresses have been introgressed into wheat germplasm (Heun & Friebe, 1990; McIntosh *et al.*, 2003). This has contributed to wheat improvement mainly in the form of wheat-rye substitution and translocation lines. The most successful and widely used wheat-alien hybrids are the T1BL.1RS and T1AL.1RS translocations involving the long arm of wheat chromosome 1B or 1A and the short arm of rye chromosome 1R (Rabinovich, 1998). The 1RS from 'Petkus' rye carries a complex locus including resistance allele *Pm8* against powdery mildew (*Blumeria graminis* (DC) E.O. Speer f. sp. *tritici* Em. Marchal) (Hsam & Zeller, 1997), *Pm26* against leaf rust (*Puccinia triticina* Eriks.), *Yr9* against stripe rust (*Puccinia striiformis* Westend. f. sp. *tritici*), and *Sr31* against stem rust (*Puccinia graminis* Pers. f. sp. *tritici*) (Singh *et al.*, 1990) but the lack of wheat arm 1BS leads to sticky dough properties and poor bread making quality (Dhaliwal *et al.*, 1987). The translocations were spread mainly through derivates of German breeding lines from the 1930s and incorporated into modern wheat cultivars worldwide (Rabinovich, 1998).

Genetic diversity and erosion

The term 'genetic erosion' (Harlan, 1972) was first used to describe a potentially disastrous narrowing of the germplasm base used to improve crop plants. It has since become almost synonymous with the displacement of landraces by modern cultivars, detected as a dramatic shift in population structure or allele frequencies as a result of natural or human-led processes (Smale *et al.*, 2002). Expeditions to collect genetic resources were initiated in the 1970s to prevent the narrowing of genetic diversity through modern plant breeding (Frankel, 1970).

The narrowing of genetic diversity could negatively affect both future and present crop production and food supply. A diminishing genetic diversity would

impact the plant breeders' capacity to respond to unforeseen events. The large-scale sowing of genetically uniform crops with a limited genetic base for resistance to pests and diseases may cause the crop to become more vulnerable to epiphytotics, because genetic change at only one or a few loci in the pathogen would be required to overcome the resistance. The potato famine in Europe during the 19th century that led to mass emigrations to the United States, is an example of the devastating effects of low genetic diversity in disease resistance and a pathogen that acquired virulence (Browning, 1988).

Studies on temporal and geographical genetic variation in barley (Koebner *et al.*, 2003; Kolodinska-Brantestam *et al.*, 2004a; Kolodinska-Brantestam *et al.*, 2004b; Reeves *et al*., 2004); oats (Nersting *et al.*, 2006) and wheat (Donini *et al.*, 2000; Christiansen *et al.*, 2002; Huang *et al.*, 2002; Roussel *et al.*, 2004; Roussel *et al.*, 2005) have demonstrated the impact of plant breeding on cereals. The conclusions of these investigations are that the narrowing of genetic diversity seems to have occurred for some crops, during some time periods and in some regional or national selections of material under investigation. It is clear from the positive impact of genes such as the use of the *Rht* dwarfing genes and many disease resistance genes from close or distantly related species (Skovmand *et al.*, 2002), that genetic resources have played a significant role in wheat improvement and will continue to be needed to provide the variability for future wheat breeding. The characterization of the genetic variation present in wheat genetic resources is therefore important for their efficient utilization in wheat breeding.

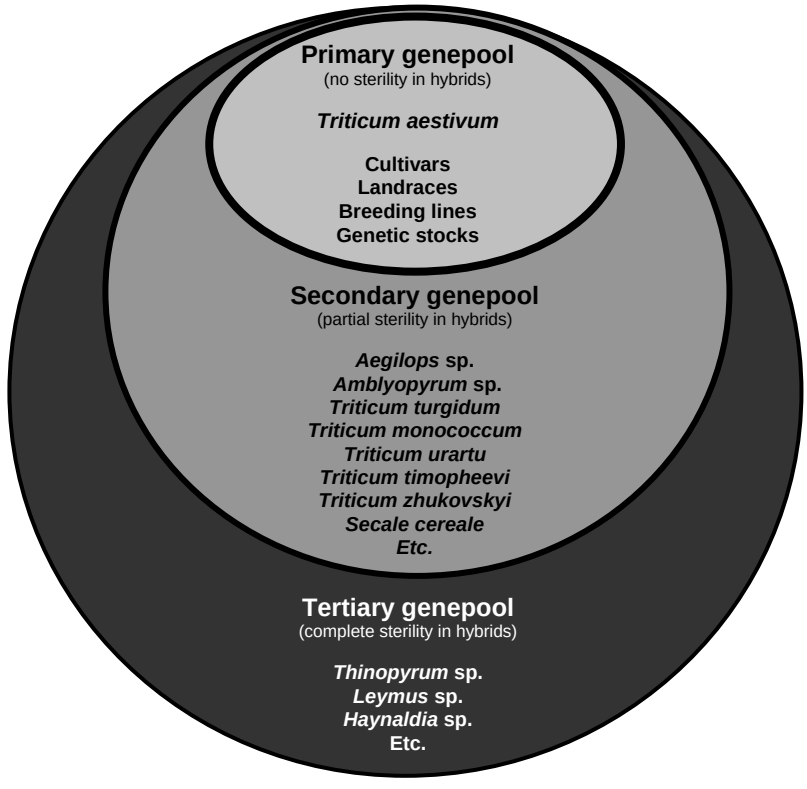

Figure 2. The gene pools of bread wheat (*Triticum aestivum*) according to the ease of gene transfer (after Harlan & de Wet, 1971).

2.2 Disease, resistance and breeding

General concepts

Plants are attacked by a number of pests and diseases. McIntosh (1998) listed 23 fungal diseases, five viral diseases and four bacterial diseases of economic importance for wheat production. The relative impact of a pathogen and disease varies with climatic conditions, crop management and the presence of resistance in prevalent cultivars. Reducing disease through the use of cultivar resistance and agricultural management contributes to increased yield and quality.

Plants are successfully attacked only by approximately 10 percent of all fungi and oomycetes in the world (Knogge, 1998). *Resistance* in general, has been defined as "the ability of an organism to withstand or oppose the operation of, or to lessen or overcome the effects of an injurious or pathogenic factor" (Federation of British Plant Pathologists, 1973). A basic compatibility (pathogenicity factors) between a plant and pathogen is required for a pathogen to recognize and overcome the *nonhost* or *basic* resistance of the host (Ellingboe, 1976). The capacity of a pathogen to cause disease is termed *virulence* and the plant response is *susceptible*. The incapability to cause disease is termed *avirulence* where the plant response is *resistant* (Heitefuss, 1997).

Biotypes of fungal pathogens with different host virulence spectra exist in the form of races (isolates or pathotypes) (Browder, 1971). Resistance against one or a few races is termed *race-specific resistance* (syn. differential, vertical). It is usually expressed qualitatively through a hypersensitive reaction (a rapid development of cell death at and immediately surrounding infection sites). Race-specific resistance is detectable at the seedling stage as *seedling resistance* and carried through all stages of the plant development. *Race nonspecific* (syn. non differential, horizontal) resistance is often detected only in the adult plant stage as *adult plant resistance* (van der Plank 1963; Heitefuss, 1997). Race-specific resistance is inherited monogenically and controlled by one 'major' gene with large observable effects. Race-specific or race nonspecific resistance is oligogenically inherited and controlled by a few genes that may be 'major' or 'minor'. Race-nonspecific resistance is polygenically inherited which implies a series of 'minor' genes that are expressed quantitatively (Heitefuss, 1997).

Partial resistance (Parlevliet, 1979) is a form of incomplete resistance that is characterized by a reduced epidemic rate and magnitude of spread despite a susceptible reaction. It is similar to *slow rusting resistance* (Caldwell, 1968) for rust pathogens or *slow mildewing* (Shaner, 1973) for powdery mildew pathogens where disease develops slowly and without hypersensitive reactions compared to a check cultivar (Roelfs *et al.*, 1992). A single slow rusting gene confers only a small to moderate reduction in disease progress, but the combination of several genes results in additive effects and markedly improved resistance. Partial resistance can be measured as a reduction in sporulation rate, pustule size, and numbers of pustules per square centimetre leaf area, an increase in incubation time (latent-period) and with intermediate to low levels of disease against all pathotypes of a pathogen (Ohm & Shaner, 1976).

Flor (1946, 1971) studied the inheritance of pathogenicity in the pathogen and the inheritance of resistance in its host. He concluded that for every resistance gene in the host there is a corresponding virulence gene in the pathogen. On the basis of Flor's ideas, Person (1959) defined the gene-for-gene concept where a host-pathogen relationship exists when the presence of a gene in one population is contingent on the presence of a gene in another population, and where the interaction between the two genes leads to a single phenotypic expression by which the presence or absence of the relevant gene in either organism may be recognized.

The genes for avirulence in the pathogen and resistance in the host are assumed to be dominant and interact directly or indirectly, resulting in incompatibility and resistance (low infection type). If a recessive virulence allele of the pathogen is not recognized by the host, the interaction does not occur, and the host and pathogen are compatible (high infection type). The host is then susceptible to the pathogen genotype. Most race-specific genes follow the gene-for-gene concept. However, there are a number of exceptions to the hypothesis including dominant, complementary genes; more than one avirulence gene that matches resistance encoded at a single locus; suppressor genes that prevent the production of a gene product or render it inactive; the interaction of genes with individually moderate effects that together give high levels of resistance; and effects depending on the genetic background (Knott, 1989; Ellingboe, 2001; Brown, 2002a)

Breeding for disease resistance

Breeding for disease resistance is the most economically and environmentally safe method to reduce crop losses (Bennett, 1984; Singh & Rajaram, 2002). Inherited disease resistance is also easy to use for the grower and reduces the need for other control methods including chemicals (Johnson, 1992). The long term success of breeding for disease resistance depends on (i) the nature of the pathogen and virulence spectra in the pathogen population; (ii) the availability, diversity and type of genetic resistance in the host; and (iii) the methodology for screening and selection for resistance.

Each disease requires unique selection approaches based on the level of knowledge of the genetic variability, life cycle, and heritability of resistance in the particular pathogen (McIntosh, 1998). Monitoring of the pathogen is therefore essential in any breeding programme. However, pathogens can be divided into broad categories depending on the nature of their relationships with their host(s). Biotrophic pathogens require living host cells and are often obligate, while necrotrophic pathogens require deceased host cells and are often facultative. Facultative pathogens e.g. the wheat fungal parasites *Mycosphaerella graminicola* causing Septoria tritici blotch, *Phaeosphaeria nodorum* causing Septoria nodorum blotch, *Pyrenophora tritici-repentis* (Died.) Drechs. causing tan spot, and *Fusarium graminearum* causing Fusarium head blight, can infect and multiply on many hosts. Obligate pathogens such as the rust fungi *Puccinia* spp., powdery mildews *Blumeria* spp. , bunts *Tilletia* spp. and smuts *Ustilago* spp. are highly specialized to a specific host or variety of a host. The populations of obligate pathogens often display a variable virulence gene spectra and the evolution of new

virulence through migration, mutation and recombination is more rapid than for the facultative pathogens. Breeding for resistance against obligate pathogens therefore often requires a more dynamic approach (Singh & Rajaram, 2002).

The availability and diversity of genetic resistance in the host relate to the germplasm sources in the three gene pools of wheat. Knowledge of the identity, efficiency and distribution of resistance facilitates the deployment in disease management. Identifying sources of resistance and resistant lines in a breeding program is dependent on a reliable screening methodology and environments favourable for disease development. The inclusion of check cultivars (tester or differential lines) with known resistance genes or levels of resistance under specified environmental conditions, is important in the assessment of degree and type of resistance. The methodology varies with the disease and type of resistance, and may include pathological seedling or adult plant greenhouse tests, field tests, and molecular DNA markers (Singh & Rajaram, 2002). Molecular markers can facilitate the combination, selection and tracking of resistance genes in breeding programs through marker assisted selection (MAS) (Bartos et al., 2002).

The nature and prevalence of the pathogen influences the longevity of resistance. Host resistance based on the use of race-specific genes has largely been ephemeral for obligate pathogens. For example, the race-specific resistance to rusts and powdery mildew is short lived, often lasting for an average of five years when deployed (Singh & Rajaram, 2002). Durable resistance (Johnson, 1979, 1981) has been defined as resistance that has remained effective in a cultivar during its widespread cultivation for a long sequence of generations or period of time in an environment favourable to the disease or pest. Van der Plank (1963) proposed that horizontal resistance would be more durable than vertical resistance as it was less likely that a pathogen would accumulate many mutations to overcome polygenically inherited resistance. Ellingboe (1981), however, argued that horizontal resistance is resistance that has not yet been shown to be vertical. Partial resistance has proven successful to extend the durability of resistance against rust and mildew pathogens (Shaner & Finney, 1977; Singh et al., 2001).

Since the discovery of the genetic basis of resistance by Biffen (1905), the utilization of the race-specific type of resistance has dominated in wheat breeding. The durability of resistance is unpredictable and relates the nature and virulence spectra of the pathogen, and to the diversity and type of host resistance. The evolution of new virulent races may lead to a boom-and-bust cycle where cultivars with effective race-specific genes have to be released continuously and are rapidly rendered susceptible (Kilpatrick, 1975; Knott, 1989). The probability of resistance breakdown is especially high with race-specific, monogenic or oligogenic resistance when the pathogen is highly variable, recombines sexually and similar resistance genes are spread over a large areas (McDonald & Linde, 2002). This has led to a search for methods to minimize the risk of rapid pathogen recombination e.g. combining genes and resistance types in a cultivar (Johnson, 1981, 1992), use of cultivar mixtures or multilines (Browning & Frey, 1981; Wolfe *et al.*, 1981); and regional and temporal deployment of cultivars (Priestley, 1981). Ultimately, knowledge of the pathogen and the availability of genetic resources are essential prerequisites for successful resistance breeding.

2.3 Leaf rust

The three cereal rusts on wheat, leaf (brown) rust (*Puccinia triticina* Eriks.), stripe (yellow) rust (*Puccinia striiformis* Westend. f. sp. *tritici*), and stem (black) rust (*Puccinia graminis* Pers. f. sp. *tritici*), are biotrophic pathogens belonging to the order Uredinales in Basidiomycota. Their relative economic impact on wheat cultivation in different areas depends mainly on the climate, prevailing winds carrying inoculum, and on the genetic resistance present in predominant cultivars (Saari & Prescott, 1985).

Leaf rust is the most common and widely distributed of the rust diseases. The disease symptoms are visible as isolated round, brown uredinia on leaf blades and occasionally leaf sheaths (Knott, 1989). Losses in grain yield are usually small (<10%) but can be severe (>30%) and are attributed to reduced floret set and grain shrivelling (Singh *et al.*, 2002). Quality losses include reduced protein levels and softness equivalent scores (Everts *et al.*, 2001).

Control strategies to manage the rust diseases have focused on the use of resistant cultivars (Johnson, 1981). Cultural practices such as the eradication of volunteer wheat to reduce the inoculum available for the next season has been practiced on a limited scale. Chemical control has been used in areas where the cost of fungicide applications could be motivated by high yield and wheat price expectations. Singh and co-workers. (2002) emphasized that a combination of cultural control practices, disease resistance, monitoring of the pathogen and fungicide applications under unusual circumstances would be the most effective means of controlling the cereal rusts.

Life cycle and symptoms

The life cycle of leaf rust includes both sexual and asexual stages with host alternation. The asexual cycle begins with the germination of urediniospores on the primary host i.e. bread wheat (*T. aestivum*), its immediate relatives or triticale. In contact with free moisture, leaf rust urediniospores initiate germination in temperatures from 2 to 30 ºC (optimum at 20 ºC) (Roelfs *et al.*, 1992). The germ tube penetrates through stomata and a haustorium develops inside the living host cell. Depending on the amount of host cells involved, the infection results in visible uredinia. New urediniospores are produced asexually in large numbers and can be carried by wind across considerable distances (Singh *et al.*, 2002). Temperatures of -4 to -6 ºC are required to reduce the viability of *Puccinia triticina* urediniospores to less than 5 % after 24 hours exposure (Eversmeyer & Kramer, 1995).

The first step in the sexual cycle of leaf rust is the teliospore stage. Teliospores develop under the epidermis on the primary host when conditions are unfavourable to the pathogen. The teliospore germinates in the presence of free moisture to produce a basidium. Haploid basidiospores are then formed in the basidium and released under humid conditions and spread a few metres to infect a nearby alternate host (Roelfs *et al.*, 1992). The primary alternate host for leaf rust is *Thalictrum speciosissimum* in the Ranunculaceae family (Anikster *et al.*, 1997). Its role in providing direct inoculum of infection to wheat is small but may it be

important for the genetic exchange between races. The basidiospores germinate in the presence of free moisture into pycnia. The mating of the sexual gametes, pycniospores and receptive hyphae, leads to the development of genetically heterogeneous aecia containing aeciospores that are spread by wind to the primary host. The aeciospores grow into uredinia that produce urediniospores and the sexual life cycle of leaf rust is completed at the end of the growing season by the formation of teliospores (Knott, 1989).

New pathogen races evolve through sexual recombination on the alternate host, mutation and somatic hybridization of germ tubes and hyphae (Samborski, 1985). Mutation is considered to be the most important source of variation in the rusts as a single uredinium can develop 3000 spores per day over a 20-day period in favourable conditions (Samborski, 1985; Roelfs *et al.*, 1992).

Host resistance

More than 50 genes and alleles of genes for seedling or adult plant expressed leaf rust (*Lr*) resistance have been identified and described at 51 loci (McIntosh *et al.* 1995; McIntosh *et al.*, 2003). The designated genes for resistance originate primarily from landraces and cultivars of *T. aestivum*, but some have been derived also from other *Triticum* species, and the genera *Aegilops*, *Secale* and *Thinopyrum* (McIntosh *et al.*, 2003). Thus, the designated gene *Lr9* was transferred from *Ae. umbellulata* Zhuk; *Lr14a* and *Lr14b* from *T. turgidum*; *Lr18* from *T. timopheevi* (Zhuk.) Zhuk; *Lr19*, *Lr24*, *Lr29,* and *Lr38* from *Thinopyrum intermedium* Host Barkworth & D. R. Dewey (syn. *Agropyron elongatum*); *Lr23* from *T. turgidum* ssp. *durum* (Desf.) Husnot; *Lr25*, *Lr26* and *Lr45* from *S. cereale*; *Lr28*, *Lr35*, *Lr36*, *Lr47* and *Lr51* from *Ae. speltoides* Tausch; *Lr21*, *Lr22a*, *Lr32*, *Lr39*, *Lr40, Lr42* and *Lr43* from *Ae. tauschii*; *Lr37* from *Ae. ventricosa* Tausch.; *Lr44* from *T. aestivum* ssp. *spelta* (L.) Thell; and *Lr50* from *T. aestivum* ssp. *armeniacum* (Jakubz.) MacKey. Molecular markers have been linked to several genes (McIntosh *et al.*, 1995; McIntosh *et al.*, 2003). Virulence most likely exists against most designated *Lr* genes, although the virulence against specific combinations may vary on a regional scale (Singh *et al.*, 2002).

Rust resistance based on race-specific genes has been largely ephemeral (Singh *et al.*, 2002). However, partial leaf rust resistance has been maintained in a few cultivars e.g. 'Americano 25' and 'Frontana' (Roelfs, 1988) and the partial resistance genes *Lr34* and *Lr46* are considered to be durable (Singh *et al.*, 1998; Singh *et al.*, 2001). For example, the co-segregating genes *Lr34* and *Yr18* (leaf rust and stripe rust resistance) have remained effective for more than 50 years (William *et al.*, 2003). Cultivars with *Lr34* and two to three additional genes have shown a stable environmental response and final disease ratings lower than five percent under heavy disease pressure (Singh *et al.*, 2001). Yield losses of around 7-10% for such cultivars are comparable to 6-10% yield loss in cultivars carrying race-specific types of resistance under high disease pressure (Sayre *et al.*, 1998). The management of disease through the breeding and deployment of cultivar resistance depends on the type of resistance and monitoring of the virulence spectrum in the pathogen.

2.4 Powdery mildew

Powdery mildew on wheat is caused by the fungus *Blumeria graminis* (DC) E.O. Speer f. sp. *tritici* Em. Marchal (syn. *Erysiphe graminis* f. sp. *tritici*) of the order Erysiphales in Ascomycota. It is an obligate, biotrophic parasite that is potentially destructive under humid rainfed and cool temperature conditions and in dryland areas under irrigation. The disease does not appear to have been important on cereals until late in the 19th century (Hermansen, 1968). High seeding rate, nitrogen fertilization and a semidwarf growth habit has increased the severity (Tompkins *et al.*, 1992). Grain yield losses are related to reductions in grain size and number per unit area and range from 5 to 34% and above 40% (Lipps & Madden, 1988; Kema *et al.*, 1995). Powdery mildew infection may cause decreased flour protein levels but milling and baking quality appear to be unaffected (Johnson *et al.*, 1979).

The disease management to reduce crop losses includes cultural practices, the application of fungicides and host resistance in combination with monitoring of the virulence range in the pathogen. Cultural practices such as the removal of voluntary wheat plants, restricted use of nitrogen fertilizers to reduce lodging, and the choice of planting date are used to restrict the development and spread of disease. Cultivar mixtures that carry several different resistance genes have been shown to reduce disease and improve yield but have not been widely accepted since they lack in the general requirement of uniformity (Pink, 2002).

Although foliar fungicide such as difenoconazole and strobilurins are effective (Leath & Bowen, 1989; Lucas, 2003), the development of fungicide insensitivity is a concern in areas of intensive use. A combination of partial (quantitative) and race-specific resistance (qualitative) has been proposed to provide broad-spectrum and potentially durable resistance (Hsam & Zeller, 2002).

Life cycle and symptoms

The life cycle of powdery mildew comprises asexual and sexual stages on the same host. The parasitic stage is haploid, in contrast to the dikaryotic stage in the rust fungi, and allows for immediate expression of mutations for virulence (Schafer, 1987). Powdery mildew is favoured by temperatures of 10 to 22 °C (optimum 20 °C, 100% humidity) but does not require free moisture for germination (Schafer, 1987). Germination of asexual conidia leads to the development of appressoria from which a specialized hyphae, the penetration peg, attempts to breach the host cuticle cell wall. If successful, a specialized feeding structure, the haustorium, develops within the host epidermal cell followed by the formation of a colony on the plant surface. Wind-dispersed conidia develop singly or in chains on short conidiophores, giving colonies a powdery appearance (Green *et al.*, 2002). Disease symptoms begin as pustules i.e. small white circular patches of fungal mycelium most visible on the underside of the leaf. New pustules with conidia are produced every seven to ten days at optimal conditions and provide repeated cycles of spores, easily dislodged and spread by wind and rain. Powdery mildew is most prevalent on lower leaves but can also cause blighting of the upper leaves, heads and awns of susceptible cultivars (Cunfer, 2002). After crop

maturity, sexually derived ascospores develop in dark round cleisthothecia and serve as survival structures. Ascospores may infect host plants but their role in initiating disease is much less important than that of the conidia in most environments (Green *et al.*, 2002). Cleistothecia may over-winter on autumn sown wheat or as early stage infections in surviving wheat plants (Schafer, 1987).

Host resistance

Race-specific resistance has been the primary means to manage powdery mildew. To date, 53 genes and alleles for powdery mildew resistance (*Pm* genes) have been described at 34 gene loci (Hsam *et al.*, 2003; McIntosh *et al.*, 2003; Huang & Röder, 2004; Miranda *et al.*, 2006). The designated resistance genes mostly originate from *Triticum aestivum* but also from other genera (McIntosh *et al.*, 2003). The genes *Pm1b* and *Pm25* were transferred to bread wheat from *T. monococcum*; *Pm4a* and *Pm5a* from *T. turgidum* ssp. *dicoccum* (Schrank *ex* Schübler) Thell.; *Pm16*, *Pm26* and *Pm30* from *T turgidum* ssp. *dicoccoides* (Körn. *ex* Asch. & Graebner) Thell.; *Pm4b* from *T. turgidum* ssp. *carthlicum* (Nevski *in* Kom.) Á.Löve & D.Löve; *Pm6* and *Pm27* from *T. timopheevi*; *Pm5c* from *T. aestivum* ssp. *sphaerococcum* (Percival) MacKey; *Pm7*, *Pm8*, *Pm17* and *Pm20* from *Secale cereale*; *Pm2* and *Pm19* from *Aegilops tauschii*; and *Pm21* from *Dasypyrum villosum* (L.) Borbas (syn. *Haynaldia villosa*); *Pm29* from *Aegilops ovata* L. and *Pm12* and *Pm32* from *Aegilops speltoides* (Hsam & Zeller, 2002; Hsam *et al.*, 2003). Molecular markers have been linked to *Pm* genes and alleles (McIntosh *et al.*, 2003, 2006) as well as quantitative trait loci (QTL) (Chantret *et al.*, 2000; Keller *et al.*, 1999).

Most *Pm* genes deployed in cultivars have been overcome by evolving virulence in the mildew pathogen (Hsam & Zeller, 2002). For example, the development and spread of virulence against the commonly used gene *Pm8* transferred from rye in a T1BL.1RS translocation led to a breakdown of resistance (Liu *et al.*, 2000; Hsam & Zeller, 2002). Studies on the presence of race-specific *Pm* genes in cultivars and virulence spectra in the pathogen have shown that common mildew races are often replaced by new races with virulence against cultivars that were resistant at the time of introduction (Svec & Miklovicova, 1998). However, higher frequencies of virulence have also been found in the powdery mildew population due to *Pm* genes not known to be widely deployed (Niewoehner & Leath, 1998).

The single deployment of major genes may provide effective protection only for a short period of time. Resistance to powdery mildew can also be accomplished by using partial (slow mildewing) resistance, where multiple genes with additive effects slows the rate of disease progress (Roberts & Caldwell, 1970). Effective partial resistance that has remained durable was identified in e.g. cv. 'Knox' (Shaner, 1973). Recently, line 'Saar' has been shown to have high levels of partial resistance (Lillemo *et al.*, 2006). Griffey and Das (1994) found two to three minor genes provided long-lasting adult plant resistance in wheat cultivars. The pyramiding of quantitative genes with qualitative genes may improve the durability of the resistance (Wang *et al.*, 2005). The continued identification of sources of powdery mildew resistance and regional virulence surveys of the pathogen are needed to allow an effective utilization of resistance in breeding.

2.5 Wheat in the Nordic countries

The Nordic countries Sweden, Norway, Denmark, Finland and Iceland, are situated in Northern and Northwestern Europe (Fig. 3). The climate is greatly influenced by the proximity to the Gulf stream. Bread wheat is grown in all the Nordic countries except for Iceland. The main use is as animal feed. Less than one quarter of the total wheat harvest is used for human consumption (Donner & Mesdag, 2000).

The history of wheat breeding in the Nordic countries began in the period from the late 19th century to the turn of the century. The area under production and yield of bread wheat has increased from 232 000 ha and 2.0 t/ha in 1924-1926 to 1'329350 ha and 5.4 t/ha in 2005 (FAOSTAT, 2006) (Fig. 4 and 5). The prevalence of wheat diseases in different Nordic regions is partially determined by the climate. Septoria and Fusarium are common, while powdery mildew can be severe in areas with a maritime climate. Leaf rust is currently of minor importance due to cold winters (Berg, 2006).

The Nordic Gene Bank, founded in 1979, currently has more than 800 Nordic bread wheat accessions (NGB, 2007). The documented history of wheat breeding in the Nordic countries provides valuable insights into the use of genetic resources and the basis for future resistance breeding.

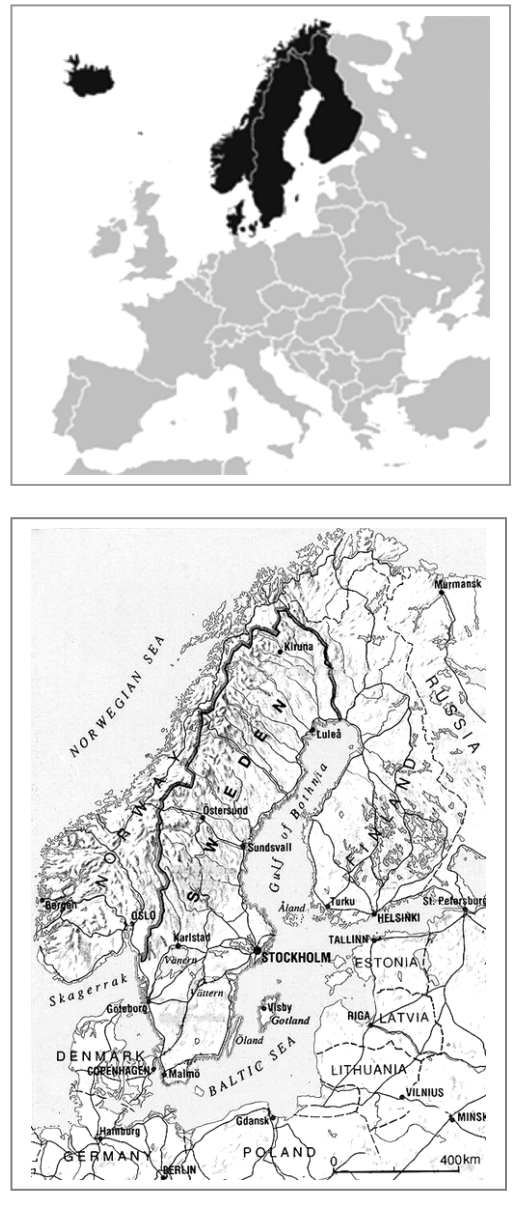

Figure 3. Map showing the Nordic countries Iceland, Norway, Denmark, Sweden and Finland in Europe (top) and a magnification of Denmark, Norway, Sweden and Finland (bottom).

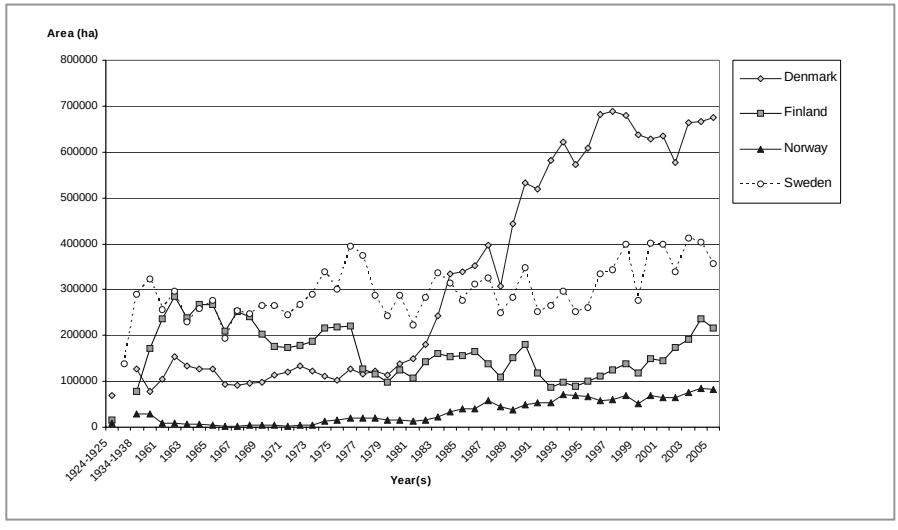

Figure 4. Total area under wheat production (ha) of spring and winter wheat in the Nordic countries 1924-2005 (Donner & Mesdag, 2000; FAOSTAT, 2006). Figures for 1925-1925 and 1934-1938 represent means across years.

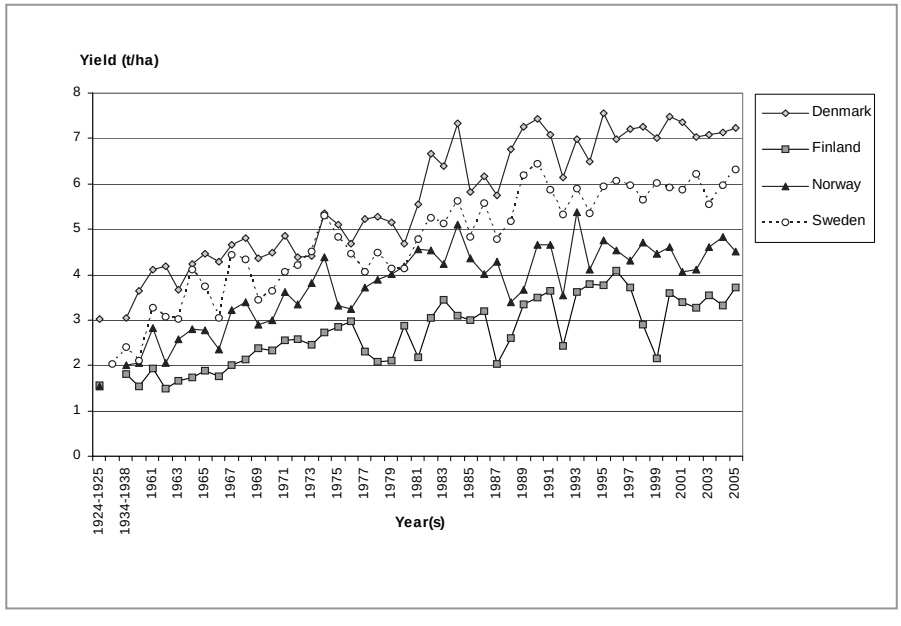

Figure 5. Average yield (t/ha) of spring and winter bread wheat in the Nordic countries 1924-2005 (Donner & Mesdag, 2000; FAOSTAT, 2006). Figures for 1925-1925 and 1934-1938 represent means across years.

Sweden

In Sweden, the annual rainfall and temperatures varies considerably with latitude and altitude. The climate is prevailing maritime in the south and around the coastline, while continental in the central and northern regions. The vegetation period is 210 to 120 days from south to north (Åkerberg, 1965). Plant breeding was initiated during the late 19th century. Foreign cultivars were adopted in the early 19th century e.g. Squarehead wheats with higher yield and lodging resistance than indigenous landraces but with low winter hardiness and baking quality. Landraces with good winter hardiness continued to be popular in northern Sweden until around 1915 (Hagberg, 1965; Olsson, 1997; Svensson, 1997).

The breeding work during the early 1900s focused on combining the high yielding Squarehead and Shiriff wheats with good winter hardiness from Swedish landraces. Pure line selection resulted in e.g. 'Grenadier I' (1898) and 'Extra Squarehead' (1900)' from English material; and 'Sammet' (1910), Sol (1911) and 'Bore' (1902) Swedish landraces. The pedigree method was used to produce the cultivars 'Jarl' (1925), 'Ankar' (1928), 'Fylgia I' (1933) and 'Ergo' (1934) from crosses between pure line selected wheat cultivars (MacKey, 1963; Svensson, 1997). Combination breeding involving crosses of indigenous and foreign material was introduced around 1910. This became more prevalent during the 1920s to 1930s when breeding for improved baking quality was initiated using Swedish landraces and foreign cultivars e.g. the Hungarian line Bankuti 178 and North American Marquis type wheats (Olsson, 1997).

The breeding objectives and methods changed to incorporate powdery mildew and rust resistance during the 1950s and exotic germplasm during the late 1960s. This was in part due to the devastating stem rust epiphytotic in 1951 that led to an increased focus on resistance breeding in the Nordic countries through virulence studies and the establishment of resistance research laboratories. Stem rust, stripe rust and powdery mildew have historically been the most important fungal pathogens on wheat in Sweden. At the turn of the 19th century, stripe rust caused severe damage about every fourth year in southern Sweden (Lundin, 1997).

The wheat area under cultivation in Sweden is mainly sown with winter wheat, while the area under spring wheat is highly variable. A few cultivars have dominated wheat production since the 1950s. For example, 'Starke' (1959) covered about 95% of the Swedish wheat area during many years. 'Holme' and 'Starke' breeding lines with resistance against powdery mildew were crossed with the Ukrainian cultivar 'Mironovskaya 808' to produce 'Kosack', the most successful cultivar during the 1980s and 1990s (Svensson, 1997). Yields (Fig. 5) have increased from 2 t/ha during the 1920s (Donner & Mesdag, 2000) to 6.4 t/ha in 1995 (FAOSTAT, 2006).

Norway

The climate in Norway shows great variations due to the close proximity to the Gulf Stream and high mountain ranges. Annual rainfall varies from 400 to 2000 mm and the vegetation period from 200 to 120 days (Åkerberg, 1965). The wheat breeding work started early in the 20th century to improve winter hardiness and earliness. Hybridization replaced pure line selections around 1920. Selection for resistance to powdery mildew was started in 1920 by crosses with a resistant line (J-03) found in a local variety (Wexelsen, 1965). The early breeding programs utilized Norwegian selections as well as Swedish, Finnish and Russian material. A series of crosses between the Norwegian cultivars 'Fram I' and 'Fram II', and the foreign cultivars 'Pika', 'Marquis', 'Sopu' and 'Diamant II' were made in 1936 and led to 'Norrøna' and 'Nora' (released in the 1950s).

During 1945 to 1965, wheat production decreased because the old cultivars were unsuitable for combine harvesting. A breeding programme emphasizing selection for earliness and high yield in combination with resistance to sprouting, shattering, lodging and various diseases was initiated in 1959 and resulted in the release of 'Rollo', 'Runar' and 'Reno' in the 1960s to 1970s. Semidwarf wheats were introduced in 1968 and incorporated into e.g. 'Bastian' (1989). The importance of domestic wheat production increased during the 1980s and 1990s leading to additional breeding objectives such as yield, further disease and lodging resistance, and bread-making quality (Donner & Mesdag, 2000). A number of Swedish cultivars have also been important for wheat production in Norway e.g. winter wheat 'Folke' during the 1990s. However, spring wheat dominates the market. Average grain yield (Fig. 5) has increased from 2 t/ha in the 1930s to a maximum of 4.8 t/ha in 2004 (FAOSTAT, 2006).

Denmark

The climate in Denmark is characterized by mild winters, cool summers and evenly distributed rainfall, with a vegetation period between 200 to 220 days (Åkerberg, 1965). Plant breeding started late in the 19th century by the formation of various associations that later led to the many private plant breeding enterprises of today. Cereal cultivars were imported to Denmark before the turn of the 19th century and grown alongside the indigenous landraces. The first state experiment station was founded in 1885 at Tystofte. Among the early cultivars produced, 'Tystofte Småhvede', a winter hardy line selected in English Squarehead wheat in 1901, became the principal cultivar for many years. Similarly, the line that became 'Trifolium 14' was selected in Dutch Wilhelmina wheat. These were later replaced by 'Ideal' (1929) and 'Als' (1937) (Frandsen, 1965). During the 1920s and 1930s, a number of high-yielding cultivars were bred that together with the increased use of fertilizers, contributed to a raise in the agricultural output in Denmark (Frandsen, 1965). Average grain yields (Fig. 5) have increased from 3 t/ha during the 1920s (Donner & Mesdag, 2000) to 7.6 t/ha in 1995 (FAOSTAT, 2006). Wheat cultivation in Denmark has been characterized by a high proportion of foreign cultivars compared to indigenous cultivars. Bread wheat is one of the main agricultural crops and winter wheat dominates (98%) over spring wheat (2%) (Donner & Mesdag, 2000).

Finland

The climate in Finland is mainly continental but maritime along the coast, and with a vegetation period from 170 to 120 days (Åkerberg, 1965). The principal breeding objectives for wheat have been yielding ability, earliness, baking quality, straw stiffness and resistance to sprouting (Kivi, 1965). During the first years of the 1900s, many cultivars e.g. 'Diamant' were imported from Sweden, a tradition that continues today.

Plant breeding began at the turn of the 19th century and was initially practiced through pure line selections from landraces with good baking quality and early maturity and the incorporation of frost resistance from indigenous and Russian material (Kivi, 1968). This led to e.g. spring wheat cultivar 'Ruskea' (1919), selected from a Swedish landrace (Halland). The trend continued during the 1910s, when crosses with foreign cultivars such as Canadian 'Marquis' and Australian 'Aurore' were used to improve yield, quality and disease resistance (Donner & Mesdag, 2000). During the 1970s and 1980s, spring wheat breeding was largely based on domestic cultivars from earlier periods. In winter wheat during the 1960s, the need for stiffer straw characteristics and resistance to sprouting led to an increased use of foreign material for breeding.

A few winter wheat cultivars have been popular during extended periods of time in contrast to the higher turnover of spring wheat cultivars. For example 'Vakka' was grown for more than 30 years (1953 to 1987) and Aura for 20 years (1979 to 1996) (Donner & Mesdag, 2000). Average grain yields (Fig. 5) have increased from 1.5 t/ha during the 1920s (Donner & Mesdag, 2000) to 4.1 t/ha in 1990 (FAOSTAT, 2006). However, there is a great annual fluctuation in the area under cultivation (Fig. 4) due to the climate (Donner & Mesdag, 2000). The change in weather conditions has had an impact on both agriculture and breeding where indigenous material was discarded during the warmer period of the 1930s (Kivi, 1965). Most wheat grown in Finland today is of the spring type as the growing season is short and a high degree of winter hardiness is required to survive the cold and long winters.

3. Genetic resources for disease resistance breeding in wheat – Characterization and Utilization

3.1 Design of study

Rationale

To maximize the economic and environmental benefits of using disease resistance in wheat, it is highly desirable that the resistance is durable and the genes have no adverse effects on agronomic performance. Resistance to pests and diseases transferred from the three gene-pools of wheat has substantially contributed to increased yield and quality (Bennett, 1984; Singh & Rajaram, 2002). However, the use of race-specific resistance has often been ephemeral and many resistance genes from alien sources are associated with negative effects on agronomic characters. For example, the wheat-rye T4BS.4BL-2R has not been used in agriculture as it decreases yield and has low levels of resistance (Hsam & Zeller, 2002).

Partial resistance has been shown to be more durable than most race-specific genes because genetic change at several virulence loci is required in the pathogen to overcome its quantitative nature (Singh *et al.,* 2001). Combinations of race-specific genes and partial resistance would also extend effectiveness (Liu *et al*., 2000; Wang *et al*., 2005). Effective partial resistance can be identified in the adult plant stage in lines without race-specific resistance or where resistance has been broken (Singh & Rajaram, 2002). Studies on the presence of seedling resistance in wheat germplasm are therefore a prerequisite for further characterization of partial resistance and the identification of useful donors.

Wheat-alien translocations have provided effective resistance to several diseases (Friebe *et al*., 1996). An example of durable resistance transferred from an alien species into is the wheat - *Thinopyrum intermedium* translocation carrying stem rust resistance gene *Sr26* that occurs in many Australian cultivars although it is associated with approximately 9% yield penalty (The et al., 1988). Compensating wheat-rye translocations with effective resistance in a wheat background with high levels of partial resistance therefore seem a promising avenue to extend the durability of resistance and maintain or improve agronomic performance.

Powdery mildew and leaf rust are fungal diseases of economic importance in many areas in the world. Both pathogens are parasites where virulence can change rapidly due to sexual recombination, mutation or spread of inoculum from neighbouring areas (McDonald & Linde, 2002). There is therefore a continuous need for the monitoring of virulence frequencies in the pathogen population and incorporation of disease resistance. The variation in powdery mildew and leaf rust host resistance genes has been studied in landraces and modern wheat cultivars used in Western and Eastern Europe (Bartos & Valkoun, 1988; Broers & de Haan, 1994; Heun & Fischbeck, 1987; Hovmøller, 1989; Herrera-Foessel, 2001; Park *et al*., 2001; Peusha *et al*., 2001; Warzecha, 1992; Winzeler *et al*., 2000). The

conclusion of these works is that a broadening of the genetic basis of powdery mildew and leaf rust resistance is desirable.

The history of wheat breeding in the Nordic countries currently spans more than a century. Resistance breeding against powdery mildew and leaf rust has different histories. Powdery mildew has been of historical significance in several Nordic areas (Vik, 1937; Leijerstam, 1962). Early breeding in Norway emphasized field selections to increase powdery mildew resistance (Vik, 1937) while screening and incorporation of race-specific resistance was practiced in Sweden since the late 1950s (Leijerstam, 1962, 1965, 1972a). Leaf rust is of importance only during years with mild winters (Wiik, 1991). The climate change towards a warmer and more humid weather will likely increase the economic impact of these diseases on wheat production (Burdon *et al.*, 2006). The methodology for gene identification through gene-postulation is well established for both pathogens and there is a wide range of available pathotypes and tester lines with known genes of resistance (Roelfs *et al.*, 1992; Hsam & Zeller, 2002). Powdery mildew and leaf rust were therefore identified as suitable candidates for the studies on diversity changes and the identification of resistance sources in Nordic wheat germplasm.

Partial resistance and wheat-rye translocations without negative effects on agronomic performance appear promising sources for effective and potentially durable resistance to powdery mildew and leaf rust. An integrated approach including investigations on disease resistance, agronomic characteristics, genetic diversity and the identification of molecular markers for resistance is proposed to target useful resistance for wheat breeding.

Aim and objectives

The overall aim of the PhD project was to characterize genetic resources for disease resistance and identify effective sources for resistance against powdery mildew and leaf rust that can be utilized in wheat breeding. The study focused on material from two gene pools: (i) wheat landraces and cultivars, and (ii) a wheat-rye T2BS.2RL translocation.

Specific objectives were to:

- determine the presence and distribution of leaf rust seedling resistance and resistance genes in North European wheat cultivars (Paper I).
- determine the presence and distribution of powdery mildew seedling and adult plant resistance and resistance genes in Nordic wheat (Paper II).
- determine the genetic relationships among accessions, and the spatial and temporal distribution of genetic diversity in Nordic wheat (Paper III).
- determine the resistance and agronomic characters in a T2BS.2RL wheat-rye translocation (Paper IV).
- develop a molecular map of a rye 2RL segment and identify molecular markers for leaf rust and powdery mildew resistance in a T2BS.2RL wheat-rye translocation (Paper V).

Plant materials

The material from the primary gene pool consisted of landraces and cultivars bred and/or grown in the Nordic countries Sweden, Norway, Denmark and Finland. The wheat accessions were chosen to represent different time periods and geographic distribution. The plant material varied between studies according to objectives and practical constraints e.g. limited availability of seed or quarantine restrictions prohibiting further use of the material in other countries. In all, 84 North European cultivars released between 1992 to 2002 were used in the study on seedling resistance in leaf rust (Paper I); 155 Nordic landraces and accessions from before 1900 to 2001 were assessed for seedling and adult plant powdery mildew resistance (Paper II); and genetic retrotransposon diversity was studied in 198 Nordic landraces and cultivars from before 1900 to 2003 (Paper III). The seed material was donated by The Nordic Gene Bank, The John Innes Centre, and the plant breeding companies Svalöf-Weibull AB in Sweden, Graminor A/S in Norway, Abed Fonden and Pajbjerg Fonden in Denmark and Boreal PB in Finland. Appendix C contains a summary of the accessions used in Papers I to III.

The material derived from the secondary gene pool comprised wheat-rye translocation lines with rye chromosome arm 2RL. Wheat-rye T2BS.2RL translocation and non-translocation lines were produced at the Swedish University of Agricultural Sciences (SLU) and originated in a mildew resistant wheat-rye substitution line with 1R and 2R crossed with the winter wheat cultivars 'Holme' and 'Kraka'. The material has been described by Merker & Rogalska (1984), Merker & Forsström (2000), Forsström & Merker (2001), and Forsström, Merker & Schwarzacher (2002). F_2-derived F_4-F_6 T2BS.2RL and non-translocation sister lines were evaluated for disease resistance and agronomic characteristics (Paper IV). In order to map powdery mildew and disease resistance in the 2RL segment, an F_2 crossing population was produced from a cross between a T2BS.2RL translocation line and a T2AS.2RL translocation line (P66) by Dr. Sai L. K. Hsam at Lehrstuhl für Pflanzenzüchtung, TUM, Germany (Paper V).

Methodological aspects

The main methods used to characterize the material included (a) pathological tests and gene-postulation, (b) cytological investigations, (c) field trials and (d) molecular DNA markers. The methods will briefly be introduced below. Please see the individual papers (Appendix I-V) for further details.

(a) *Pathological tests and gene-postulation.* The methodology for pathological seedling and/or seedling leaf segment tests and gene-postulation for powdery mildew (Lutz *et al.*, 1995) and leaf rust (Singh & Rajaram, 1991; Lutz *et al.*, 1995) are largely similar although the specific conditions for inoculation depend on the pathogen. If a set of pathotypes with differing virulence spectra and host tester lines with known resistance genes are available, the presence of designated resistance genes in a material can be postulated using a successive elimination procedure (Vallavielle-Pope *et al.*, 1990). The avirulence/virulence formulas of the pathotypes are first identified by observing the infection types on the tester lines. The putative resistance genes are subsequently determined in the host accessions according to the gene-for-gene hypothesis (Flor, 1956; Person, 1959).

The comparison of compatible and incompatible interactions between the host and the pathogen is used to eliminate the presence of resistance genes. A high infection type (susceptible response reaction) signifies that the accession could have any of the genes for which the pathotype is virulent or an unidentified resistance gene(s) for which the isolate is also virulent. A low infection type (resistant response reaction) signifies that the host has a resistance gene against which the pathotype is avirulent. The infection type is then compared with the infection types of the tester lines with known resistance genes. The hypothesis that a certain resistance gene is present in a host is supported if a corresponding incompatible interaction is observed with an isolate that is avirulent for this gene, the infection type produced after infection by an isolate that is virulent to the gene is similar to that of a tester line carrying the gene, and the results are supported by pedigree information.

(b) *Cytological investigations.* The C-banding technique reveals the location of two classes of chromatin, heterochromatin (dark-staining regions) and euchromatin (light-staining regions) along the chromosome axis. Genomic affinity of individual chromosomes is determined by meiotic pairing analysis or by sequential banding and *in situ* hybridization (Gill *et al.*, 2002). The standard karyotype of wheat (Gill *et al.*, 1991) and rye (Sybenga, 1983) allow the unambiguous identification of both the short and long chromosome arms using C-banding. Chromosome C-banding (Merker, 1973) and Genomic *in situ* hybridization (GISH) (Schwarzacher & Heslop-Harrison, 2000) was applied to the T2BS.2RL and non-translocation lines to verify the homozygous presence or absence of the rye segment.

(c) *Field trials.* The agronomic characters of several T2BS.2RL translocation and non-translocation lines were measured in three years of randomized block-design field trials (2001, 2002 and 2003) using hill plots of approximately 15 plants per line. The field was located at Lönnstorp, near Alnarp, in southern Sweden. The following parameters were recorded for each hill: lodging, heading, straw length, yield, thousand kernel weight, grain volume weight, spike size, fertility, grain alpha amylase activity, grain starch content and grain protein content.

(d) *Molecular DNA markers.* Bread wheat has a genome size of 16 billion base pairs (bp) of DNA organized into 21 pairs of chromosomes, seven pairs belonging to each of the genomes A, B and D (Gill & Friebe, 2002). DNA markers are abundant, unaffected by the environment and have a much higher level of allelic variation than morphological and isozyme markers. The following applications of molecular markers and methods are used in the thesis:

(i) The presence of the T1BL.1RS chromosome translocation in the Nordic wheat accessions was determined by a using a PCR-based assay with a pair of primers directed at the spacer region of rye *Nor-R1* (Paper II).

Polymerase chain reaction (PCR) -based identification of rye chromatin in wheat is an efficient and rapid diagnostic tool compared to cytological methods. The nucleolar organizer region with the *Nor-R1* locus is located in the rye chromosome arm 1RS. Primer pairs directed at the locus have been shown to be correlated with

the rye segment in wheat backgrounds (Koebner, 1995). The marker was used as a diagnostic tool for the 1RS segment in wheat accessions (Paper II). The presence of the T1BL.1RS translocation was also verified by C-banding.

(ii) Genetic diversity was assessed through the use of a retrotransposon based sequence-specific amplified polymorphic DNA (S-SAP) method that generated banding polymorphisms in Nordic wheat accessions (Paper III).

The interspersed repetitive elements called retrotransposons, are among the most prevalent class of eukaryotic transposable elements. They are characterised by their ability to transpose via an RNA intermediate, which they convert to DNA by reverse transcription prior to insertion (Waugh *et al.*, 1997). Retrotransposon integration sites are stably inherited, and therefore integration sites shared between accessions are likely to have been present in their common ancestors. Polymorphism may be the results of transpositional activity of retroelements, a restriction site polymorphism, or both (Soleimani *et al.*, 2005).

The LTR (long terminal repeat) retrotransposon superfamily is the best characterized and probably most abundant in crop species (Sabot *et al.*, 2004). The LTRs are relatively conserved and if two or more retrotransposons reside in close vicinity to each other, outward-facing LTR-specific primers can be used to amplify the intervening DNA sequence (Weising *et al.*, 2005). Molecular methods have been designed based on PCR with outward facing primers matching LTRs in combination with primers corresponding to another retroelement (inter-retrotransposon amplified polymorphism IRAP method; Kalendar *et al.*, 2004), a microsatellite (retrotransposon-microsatellite amplified polymorphism method REMAP; Kalendar *et al.*, 2004), or a restriction site amplified fragment length polymorphism (AFLP) adaptor (sequence-specific amplified polymorphism S-SAP method; Waugh *et al.*, 1997). In S-SAP, the initial steps of DNA digestion and ligation are similar to standard AFLP (amplified fragment length polymorphism). Only one restriction site specific AFLP primer is then used in the final amplification step, and a second primer is complementary to a defined DNA sequence. Selective bases at the 3'-end of the AFLP primer can be used to reduce the complexity of the banding patterns. The S-SAP markers are dominant and the presence of a band could be the result of a heterozygous or homozygous allele state (Waugh *et al.*, 1997).

The S-SAP method with the barley *Sukkula*-9900-LARD retrotransposon primer was used in the study on genetic diversity in Nordic wheat (Paper III). The LARD (large retrotransposon derivative) *Sukkula* elements in barley belong to the LTR family, are non-autonomous and probably dependent on other elements for their transposition. LARDs appear to be recent and active elements in the Triticeae genomes and probably originate from *gypsy* LTR retrotransposons after numerous cycles of deletions, recombinations and mutations (Kalendar *et al.*, 2004). The relative activity and order of copy number of *Sukkula* elements in barley have been estimated to be similar to *BARE-1* (Shirasu *et al.*, 2000, Leigh *et al.*, 2003). Figure 6 shows an example of an S-SAP autoradiogram from the *Sukkula-9900*-LARD primer applied to wheat.

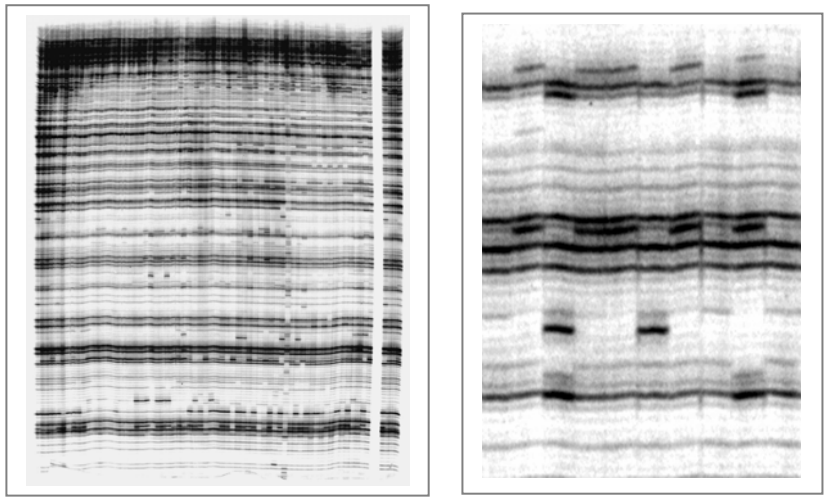

Figure 6. S-SAP autoradiogram (left) and close-up (right) using the *Sukkula*-9900-LARD retrotransposon primer and one primer extension of three selective bases.

(iii) Genetic linkage mapping using wheat and rye microsatellites (simple-sequence repeats, SSRs) were applied to construct a map of the 2RL region determining powdery mildew and leaf rust resistance in the T2BS.2RL translocation line and investigate the association between disease resistance and marker genotype (Paper V).

Microsatellites or Simple Sequence Repeats (SSRs) are tandemly repeated DNA sequences with a basic motif of one to six base pairs. They are abundant, polymorphic and randomly distributed in eukaryotic genomes (Tautz & Renz, 1984). The repeat numbers are detected via PCR using primers designed to match the flanking sequences of a specific microsatellite locus. Microsatellite markers are codominant i.e. both alleles are detected and homo- or heterozygous individuals can be distinguished. Hundreds of genomic derived expressed sequence tag (EST) microsatellite loci have been identified in wheat (Röder *et al.*, 1998; Stephenson *et al.*, 1998) and rye (Hackauf & Wehling, 2002; Bolibok *et al.*, 2006). SSRs have been applied to near isogenic lines ; double haploid populations, recombinant inbred lines, F_2 populations, and for bulked segregant analysis (BSA) in numerous genetic diversity, mapping and linkage studies to find associations to useful traits. Recent examples include, rye microsatellites linked to aluminium tolerance genes in rye (Matos *et al.*, 2005); mapping of QTLs for adult plant powdery mildew resistance in wheat (Liang *et al.*, 2006), wheat microsatellites linked to the durable stem rust resistance gene *Sr2* (Hayden *et al.*, 2006a), powdery mildew resistance gene *Pm34* from *Aegilops tauschii* (Miranda *et al.*, 2006); and leaf rust resistance genes *Lr19* (Gupta *et al.*, 2006) and *Lr34* (Bossolini *et al.*, 2006). Expressed sequence tag derived microsatellites have also been shown to be transferable between cereal species (Zhang *et al.*, 2005), allowing their utilization in cross-species mapping and linkage studies.

3.2 Disease resistance and genetic diversity in Nordic wheat (I-III)

The major findings of leaf rust and powdery mildew disease resistance and genetic diversity, in regard to the history of wheat breeding in the Nordic countries and possible implications for future resistance breeding are discussed below.

In order to assess the current resistance against leaf rust in wheat, a selection of 84 Nordic and European cultivars from 1992 to 2002 were inoculated with twelve Mexican pathotypes (Paper I). The pathotypes were chosen to reflect a wide range of virulence, as there is currently no information available on the virulence in the Nordic leaf rust population. The results showed that 89% of the cultivars carried seedling resistance against leaf rust. Gene-postulation identified the race-specific genes *Lr1*, *Lr2a*, *Lr3*, *Lr10*, *Lr13*, *Lr14a*, *Lr17*, *Lr23* and *Lr26* in 56% while 33% had only unidentified resistance. There appears to be a wide range of identified and unidentified leaf rust seedling resistance genes present in cultivars grown in Northern Europe that could have influence on the pathogen population.

The proportion of leaf rust resistant cultivars for individual countries largely corresponded to differences in climatic conditions and prevalence of disease (Paper I). For example, Denmark with the mildest weather also showed the highest proportion of resistant cultivars. Denmark is also closer to the continent and is most likely affected by the spread of inoculum from neighbouring areas to a larger extent than Finland. This has been shown for stripe rust in Denmark (Hovmøller, 2001). Financial and temporal constraints made it impossible to include a larger number of Nordic cultivars also from other time periods, and therefore no conclusions can be made regarding the influence of breeding on the presence of seedling leaf rust resistance.

In the study on powdery mildew resistance, leaf segments from 155 Nordic wheat accessions were inoculated using eleven European powdery mildew isolates, in addition to scoring of adult plant resistance to a mixture of isolates (Paper II). The results showed that mildew seedling resistance was present in 42% of the material and adult plant resistance in 92% of the 109 accessions in the adult plant tests. The results showed that there is widespread adult plant resistance in contrast to seedling resistance. Gene-postulation identified the race-sepcific genes *Pm1a*, *Pm2*, *Pm4b*, *Pm5*, *Pm6* and *Pm9* in 21 lines. Unidentified genes, with or without identified genes, were present in 45 accessions. A limiting factor in the gene-postulations was the choice of isolates, as several accessions with different pedigrees showed similar results for resistance that could be due to the same or different genes or alleles of genes. Resistance tests using a wider selection of isolates might allow this question to be resolved.

Seedling resistance to powdery mildew was more frequent in spring than winter wheat, while the distribution of resistance by country or during time had varied (Table 2). These results seem to reflect the history of powdery mildew breeding and the prevalence of the disease in individual Nordic countries. In Sweden, powdery mildew was a minor disease at the turn of the 19th century but gained in importance with the increased use of nitrogen fertilizers, and became the dominating cereal pathogen during some years (Lundin, 1997). Therefore, virulence surveys and systematic breeding for race-specific powdery mildew

resistance were initiated in the 1950s (Leijerstam, 1962). In contrast, powdery mildew was recognized as a significant disease of wheat early in the breeding history of Norway, and selection for mildew resistance was practiced since the 1920s (Vik, 1937). Due to the cold climate in Finland, powdery mildew is of little importance. The situation in Denmark is similar to that in southern Sweden, although the history of breeding is more difficult to trace due to the many private plant breeding enterprises.

A shift in the presence of resistance, as measured by the eleven isolates, seems to have occurred across six different time periods detected as a decrease in seedling resistance during 1910 to 1929 and a subsequent increase to resistance against all isolates (Table 2). Powdery mildew is currently a disease of importance in the southern and coastal regions of the Nordic countries. The emphasis on selecting for race-specific seedling resistance since the 1950s has had a clear impact. The efficiency of the adult plant and seedling resistance during different time periods is difficult to estimate as little is known about the virulence spectrum of the pathogen. The results for seedling disease resistance against powdery mildew and leaf rust in Nordic cultivars during from 1992 to 2002 show that seedling resistance is common, most likely due to wheat breeding efforts (Papers I and II).

Comparisons of historical notes about resistance or susceptibility in cultivars (Leijerstam, 1962, 1965, 1972a, 1972b; Wolfe & Schwarzbach, 1975), information on the proportion infected leaf area from recent disease surveys (Bengtsson *et al.*, 1991; Carlsson *et al*., 1992; Larsson & Magnét, 1996; Larsson *et al.*, 1997; Skinnes, 2002; Larsson *et al.*, 2003; Åssveen *et al.*, 2003) and results from gene-postulations (Papers I and II) showed that virulence to all the postulated genes and many unidentified genes most likely exists in the current leaf rust and powdery mildew pathogen populations in the Nordic countries. There also appears to have been an evolution in the virulence profile of the powdery mildew pathogen in Sweden between the 1960s and the 1980s most likely due the emphasis on breeding for seedling resistance using race-specific genes (Paper II). The presence of extensive seedling resistance is clearly no guarantee for resistance to the prevalent races in the field. These results indicate that the race-specific resistance genes have been overcome and there is a need to monitor the pathogen and incorporate new sources of resistance.

DNA fingerprinting using the retrotransposon based S-SAP was selected for a more unbiased estimate of the genetic diversity in Nordic bread wheat in contrast to postulated leaf rust and powdery mildew resistance genes. In many accessions, genes for seedling resistance could not be postulated because several isolates were avirulent and therefore the conclusions regarding the distribution of genotypic resistance characters based on resistance are incomplete. The use of molecular markers linked to powdery mildew or leaf rust resistance genes could have resolved the question in some cases, but many of the accessions most likely carried undesignated race-specific resistance genes. Therefore, the study using the S-SAP method and retrotransposon primer *Sukkula*-9900-LARD, became an important source of information on the relationships and distribution of genetic diversity in Nordic bread wheat.

The S-SAP method and *Sukkula*-9900-LARD retrotransposon primer with six primer extensions were highly efficient in unambiguously genotyping 198 Nordic bread wheat accessions from before 1910 until 2003. Comparisons of average gene diversity, cluster and principal coordinates analysis showed that there was a clear separation between winter and spring germplasm, but less for different countries. This is consistent with a frequent exchange of germplasm between the Nordic countries as also seen in many of the pedigrees (Paper V). The results on disease resistance against powdery mildew and partially also the results for leaf rust resistance, showed a difference in resistance to number of isolates between countries, growth habit and time periods (Papers I and II). There was a somewhat clearer separation between countries as measured by disease resistance compared to retrotransposon diversity most likely due to different focus on leaf rust and powdery mildew resistance breeding in the individual Nordic countries. In agreement with previous studies on genetic diversity in wheat germplasm (Donini *et al*., 2000; Christiansen *et al.*, 2002; Koebner *et al*., 2003; Roussel *et al.*, 2005), the general loss of genetic diversity was found to be negligible though allele loss is presumed to have occurred at some loci. Thus, there appears to have been a genetic shift also in Nordic wheat where genetic diversity was reduced through mass and pure line selection in landraces and increased markedly after the 1960s through the incorporation of exotic germplasm.

The data on S-SAP gene diversity (Nei, 1973) was analyzed and compared to the presence of seedling resistance against the eleven powdery mildew isolates for 152 accessions that were included in both Papers II and III (Table 1 and Fig. 7). This showed a strikingly similar pattern for presence of resistance and gene diversity regarding growth habit and time periods. The results strengthen the hypothesis that breeding activities have influenced gene diversity and disease resistance in Nordic wheat. The result for geographical areas is more difficult to interpret as the number of accessions was highly variable between the countries (Table 1). The high proportion of seedling resistance in the Norwegian accessions could be due to the importance of this disease for wheat cultivation in this region. The high proportion of genetic diversity in landraces is an indication that useful characters may be present in this category.

The investigations on powdery mildew and leaf rust resistance in Nordic and North European bread wheat, identified 59 seedling susceptible but adult plant resistant accessions that could carry partial resistance (Paper II). Comparisons of postulated genes and annual ratings of diseased leaf area found five cultivars with leaf rust resistance that could still be effective in the Nordic countries (Paper I). The resistance could be due to qualitative or quantitative genes. For example, the Swedish cultivar ‘Kosack’ most likely has effective quantitative partial resistance as it was shown to carry only one postulated qualitative *Pm* gene that has been overcome in Sweden and displayed low figures for infected leaf area in the field (Paper II). These accessions are of interest for future breeding and the putative partial resistance should be assessed and quantified under field conditions in areas with high disease pressure.

Table 1. *Summary of number (N) of accessions, gene diversity (*H*) measured by S-SAP polymorhpisms of retrotransposon* Sukkula-*9900-LARD, percentage (R) of accessions with powdery mildew (Pm) seedling resistance , and presence (+) of powdery mildew resistance against individual isolates by (growth) habit, country and time period, in a total of 152 Nordic bread wheat accessions*

Category	**N**	***H***	**R**	**Pm isolate**										
			%	**2**	**5**	**6**	**9**	**10**	**12**	**13**	**14**	**15**	**16**	**17**
Habit														
Spring	68	0.214	69	+	+	+	+	+	+	+	+	+	+	+
Winter	84	0.181	20	+	+	+	+	+	+	+	+	+	+	+
Country														
Sweden	107	0.213	41	+	+	+	+	+	+	+	+	+	+	+
Norway	17	0.187	94	+	+	+	+	+	+	+	+	+	+	+
Denmark	15	0.160	20	+	+	+	+	+	+	+	+	+	+	+
Finland	13	0.156	8		+		+	+	+			+		
Time														
Before 1910	32	0.209	48	+	+	+	+	+	+	+	+	+	+	+
1910-1929	20	0.174	0											
1930-1949	30	0.189	23	+	+	+	+	+	+	+	+	+		+
1950-1969	28	0.196	32	+	+	+	+	+	+	+		+	+	
1970-1989	25	0.239	76	+	+	+	+	+	+	+	+	+	+	+
1990-2001	17	0.224	10	+	+	+	+	+	+	+	+	+	+	+
In Total	*152*	*0.227*	*41*	+	+	+	+	+	+	+	+	+	+	+

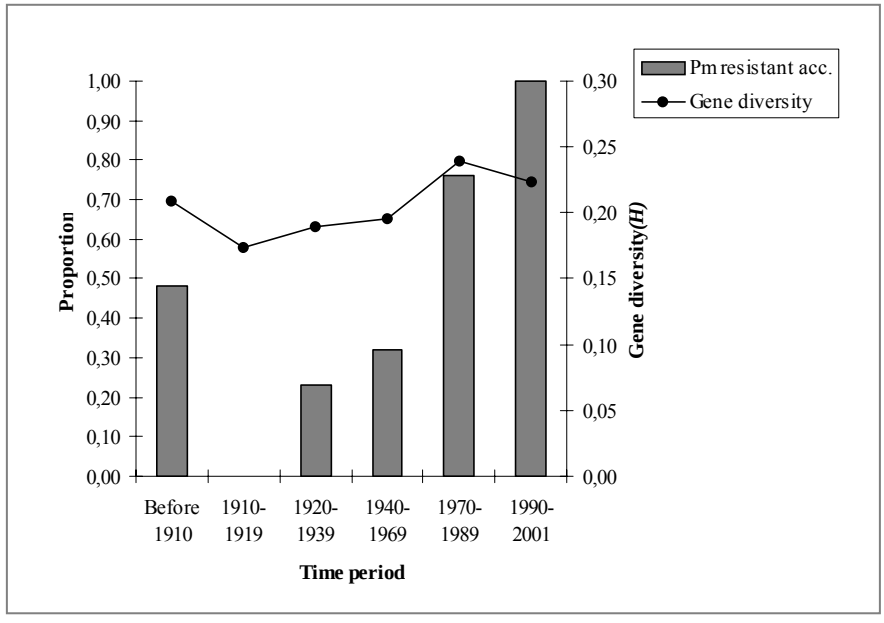

Figure 7. The proportion powdery mildew resistant accessions and gene diversity during different time periods in Nordic wheat.

3.3 Disease resistance, agronomic performance and mapping of T2BS.2RL (IV-V)

The major findings of the investigations on the T2BS.2RL, SLU wheat-rye translocation and possible implications for future resistance breeding are discussed below.

Genetic and physical mapping has shown wheat chromosome arm 2BL to be homoeologous to the long arm of rye chromosome 2R (2RL) (Naranjo *et al.*, 1987; Devos *et al.*, 1993; Lukaszewski *et al.*, 2004). Wheat 2BL lacks the storage protein genes that affect baking quality (Knackstedt *et al.*, 1994). The presence of resistance genes and good agronomic performance in combination with quality aspects that could be improved by selection, makes the use of rye chromosome arm 2RL highly suitable for wheat improvement (Lahsaiezadeh *et al.*, 1983; Friebe *et al.*, 1990; Ehdaie *et al.*, 2003).

In the present study, the T2BS.2RL, SLU translocation was investigated for agronomic performance, disease and pest resistance (Paper IV). Resistance against 17 powdery mildew isolates, 14 leaf rust- and one stem rust pathotype could unambiguously be ascribed to 2RL. The rye segment showed no resistance to Hessian fly or seedling stripe rust infection. However, stripe rust infection of adult plants under field conditions showed the translocation lines to be resistant and the non translocation lines susceptible. The T2BS.2RL lines were also observed to have a deep red coleoptile color that could be a simple marker for screening in some wheat backgrounds, in contrast to the green coleoptiles of the non translocation lines. Figure 8 displays some of the results of the investigations.

The two-year field trials on agronomic performance of translocation and non translocation lines showed that the 2RL was associated with two to three days later flowering time and an increased number of spikelets per spike. The translocation had no significant effect on yield, straw length, lodging, volume weight, thousand-kernel weight, fertility, alpha amylase activity, starch or protein content (Paper IV). This is in agreement with investigations of agronomic characteristics of T2BS.2RL translocations derived from 'Blanco' and 'Chaupon' rye (Lahsaiezadeh *et al.*, 1983; Ehdaie *et al.*, 1991, 1998). The source of rye chromatin and the wheat genetic background can influence the variation and expression of resistance genes as well as agronomic characteristics such as protein content and grain yield (Kim *et al.*, 2004). The effect on number of seeds per spike, grain yield and harvest index of the Hamlet T2BS.2RL has been shown to be dependent on the wheat background (Fritz & Sears, 1991; Ehdaie *et al.*, 2003). The results on disease resistance and agronomic performance for the T2BS.2RL, SLU translocation are promising for wheat breeding, however, it would be important to study these effects and those of bread-making quality also in various wheat backgrounds to determine the stability of these characters.

Molecular mapping of populations derived from parents differing in traits of interest is a prerequisite for linkage analysis of valuable genes. A screening test of the SLU T2BS.2RL, Hamlet T2BS.2RL ('Karl' wheat - 'Chaupon' rye; Fritz & Sears, 1991), and P66 T2AS.2RL ('Chinese Spring' wheat - 'Imperial' rye; Sears, 1968) translocations using more than 20 powdery mildew and eight leaf rust isolates showed that the SLU line was completely resistant to all isolates, line Hamlet was resistant to all powdery mildew but no leaf rust isolates and P66 was susceptible to all isolates (Hysing & Hsam, 2004 unpublished results). This indicates that chromosome 2RL from sources of various origin may carry different resistance genes or alleles of genes. A molecular map was created of the 2RL segment from microsatellite polymorphisms in a F_2 T2BS.2RL, SLU × T2AS.2RL, P66 population (Paper V). Linkage analysis located a major QTL for powdery mildew resistance associated with one of the rye microsatellite markers (Paper V).

Due to the close homoeology between rye 2RL and wheat 2BL, only three rye microsatellite markers among more than 100 wheat or rye SSRs were found to be chromosome specific and polymorphic between the 2RL segments (Paper V). This illustrates the difficulty in developing molecular maps for wheat-alien translocations involving closely homoeologous chromosome segments. A denser map of 2RL may have allowed the leaf rust resistance to the two isolates to be mapped with higher accuracy and could facilitate confirmation of the markers in generation F_3 bulked segregant analysis.

A strategy in finding polymorphic markers for molecular mapping of alien chromosome segments lies in the simultaneous employment of several marker methods. Steed and co-workers (2006) used the S-SAP method and *Sukkula*-9900 LARD-retrotransposon to compensate for a paucity in polymorphic microsatellites in a mapping and linkage study on Fusarium head blight resistance transferred from *T. aestivum* ssp. *macha* (Dekapr. & Menab.) MacKey. However, the resistance was associated with a microsatellite marker and the mapped S-SAP

markers were located 50 cM apart from the microsatellite markers, much closer to the assumed centromere. This is in agreement with a high-density cytogenetic map of the *Aegilops tauschii* genome incorporating retrotransposons and defense-related genes, where retrotransposons were found to cluster in the pericentromeric (proximal) chromosomal regions while resistance and defense-response genes were clustered in the telomeric (distal) regions (Boyko *et al.*, 2002). Although some barley retrotransposon S-SAP markers have been found to be broadly distributed among all wheat chromosomes (Queen *et al.*, 2004), other studies have shown a tendency for clustering in barley (Rodriguez *et al.*, 2006).Therefore S-SAP markers would presumably be useful for mapping studies but not for linkage analysis of resistance traits associated with the T2BS.2RL translocation.

Sequence-tagged microsatellite profiling (STMP) has been used to develop sequence-tagged microsatellite (STM) primers that target compound repeat motifs of two or more tandem repeats in wheat. The technique has been demonstrated as a rapid and economical alternative for the development of anonymous SSRs to increase marker density in the genetic map of wheat (Hayden *et al.*, 2006b). Bossolini and co-workers (2006) used rice expressed sequence tags (ESTs) with homoeology to wheat and derived new polymorphic microsatellites that were closely linked to leaf rust resistance gene *Lr34*. Similar methods might facilitate the development of SSRs also in rye and increase marker density in the wheat-rye translocation 2RL map.

The development of molecular maps and markers for 2RL and agronomically significant traits could greatly facilitate the tracking of the genes of interest through marker assisted selection. The use of SSRs were here shown to be a feasible albeit difficult method to produce a map of the 2RL segment and find associations with QTLs for disease resistance. The durability of the highly effective resistance conferred by the T2BS.2RL, SLU translocation cannot be predicted but will likely be determined by the method and extent of its deployment in wheat cultivation. Introgression of the translocation into wheat backgrounds with high levels of partial resistance could extend the longevity of the putative race-specific genes and make the cultivar less prone to sudden yield reductions due to the breakdown of major resistance genes.

A further strategy to promote resistance durability and maximize the utility of the translocation, lies in the possibility of homologous recombination between 2RL segments from different rye sources in wheat. Thus, for example, the Hessian fly resistance from the T2BS.2RL, Hamlet line could be combined with powdery mildew and leaf rust resistance in the T2BS.2RL, SLU line. Molecular markers linked to genes or alleles of interest in the various 2RL sources could facilitate the selection in breeding. The results of the present study on the T2BS.2RL, SLU translocation revealed multiple disease resistance and minor negative effects on agronomic performance that could be improved through selecting for earliness or possibly by using a different wheat background. The translocation has been distributed to several countries, and is currently being introgressed into wheat breeding lines. If the yield performance continues to be stable across wheat backgrounds, and the negative effects on baking quality are found to be minor, the T2BS.2RL, SLU could have a positive impact on wheat production.

The present study identified several sources of potentially effective disease resistance present in landraces, cultivars and the wheat-rye T2BS.2RL, SLU translocation. As resistance has been observed to be subject to erosion, there is a need to find and incorporate additional sources. Studies on near-isogenic lines in barley with major resistance genes against powdery mildew have shown that none of the resistance genes were associated with difference in yield, suggesting that the fitness cost of pyramiding qualitative disease resistance genes is low (Brown, 2002b). The combination of quantitative and qualitative resistance genes could therefore counteract the ephemeral nature of qualitative race-specific resistance while maintaining agronomic performance.

It has been argued that the use of comparatively primitive breeding material, such as landraces or distant relatives of wheat, is a slow and costly process as many generations of backcrosses are needed to produce an agronomically suitable cultivar. Molecular markers are valuable for the estimation of genetic relatedness and diversity, genome and trait mapping in crop germplasm. The retrotransposon based S-SAP method used in the present study showed high levels of polymorphism and could be extended using different types of retrotransposons for molecular fingerprinting, mapping and linkage analysis. The technique is similar to AFLP, rapid, cost-effective and reproducible. As retrotransposon ESTs have been found to match across several grass genera (Vicient *et al.*, 2001), retrotransposon primers identified in one species could be useful also in other species.

The cloning and sequencing of race-specific and partial resistance genes is important for understanding their structure and function. Resistance genes have been isolated from model and crop plants and are categorized depending on the presence of typical structural motifs, such as nucleotide binding domains (NBs) and leucine rich repeats (LRRs) (Dangl & Jones, 2001). The majority of resistance genes have been found to contain regions of amino acid conservation in these domains that can be used across genera for mapping, linkage analysis and gene isolation (Collins *et al.*, 2001; van der Linden *et al.*, 2004). Comparisons of sequences from the ten *Pm3* alleles for powdery mildew resistance in wheat, have shown that resistance specificites are based on one or a few amino acid changes by gene conversion or mutation (Yahiaoui *et al.*, 2006). The use of TILLING (targeting induced local lesions in genomes), where large populations are screened to obtain an allelic series with numerous point mutations, could be a strategy in finding resistance genes. Adapted wheat genotypes can be used as starting material for the mutated population and as few as four backcrosses may suffice to derive lines similar to the parent (Slade *et al.*, 2005).

The effectiveness of resistance genes against a disease or pest depends on the prevalence of corresponding genes for virulence in the pathogen population. The spectra of avirulence and virulence in pathogen populations change as a consequence of mutation, recombination, selection and migration (McDonald & Linde, 2002). During the last decades, two factors have gained in influence on pathogen and host evolution: the increased mobility of humans that contributes to a broadening of the migration patterns; and the global climate change that may

alter the potential for survival, germination, infection and reproduction for pathogens and resistance in hosts (Coakley *et al.*, 1999; Burdon *et al.*, 2006).

In gross terms, climate change is predicted to lead to an increase in temperature, atmospheric carbon dioxide concentration, humidity and annual precipitation. The effects in Northern Europe and the Nordic countries include an increased mean annual temperature, humidity, precipitation, and possibly soil nitrogen availability (Burdon *et al.*, 2006). Low temperatures has been one of the limiting factors for plant pathogens in the northern hemisphere and a raise in temperature is predicted to lead to increased over-wintering of inoculum and a higher number of epidemic cycles per season. The postulated effects on the plant hosts include increased leaf area and expression of resistance genes induced by high temperatures. The combined influence of climate change on pathogens and their hosts may affect the evolution in virulence, resistance and host-pathogen dynamics especially in sexually recombining biotrophic fungi such as the rusts and powdery mildew (Burdon *et al.*, 2006; Garrett *et al.*, 2006).

Studies on allelic diversity in landraces and modern wheat cultivars have shown that modern breeding has had a strong selection pressure in the D genome (Hao *et al.*, 2006), which implies that the D genome in landraces may be a rich source for widening the genetic diversity in modern wheat (Andolfatto, 2001). The D genome also most likely contributed to a wider range of climatic adaption during the evolution of hexaploid wheat (Feldman *et al.*, 1995). In order to study the effects of climate change, wheat landraces, cultivars and TILLING populations could be grown in target environments where the current climate conditons correspond to a predicted climate scenario. Investigations on the wheat plant, pathogen and resistance responses could provide valuable information on the effect of the prevalent conditions, that may be of particular interest in expanding the genetic basis for wheat breeding in regard to climate change.

In conclusion, there is an incentive for the monitoring of potentially significant pathogens of the future, and continued characterization and utilization of genetic resources from the gene pools of wheat. In this regard, the present PhD study has contributed towards the understanding of the genetic diversity and presence of powdery mildew and leaf rust resistance in North European wheat, the impact of plant breeding strategies, and the finding of putative sources of effective disease resistance present in landraces, cultivars and the T2BS.2RL, SLU wheat-rye translocation. The findings constitute part of the research needed for the effective implementation of genetic resources for successful plant breeding and future wheat production.

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I

Leaf rust (*Puccinia triticina*) resistance in wheat (*Triticum aestivum*) cultivars grown in Northern Europe 1992–2002

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Diversity of resistance to leaf rust caused by *Puccinia triticina* can be enhanced in wheat (*Triticum aestivum*) cultivars through a better knowledge of resistance genes that are present in important cultivars and germplasm. Multi-pathotype tests on 84 wheat cultivars grown in Denmark, Finland, Norway and Sweden during 1992–2002 and 39 differential testers enabled the postulation of nine known genes for seedling resistance to leaf rust. Genes *Lr1*, *Lr2a*, *Lr3*, *Lr10*, *Lr13*, *Lr14a*, *Lr17*, *Lr23* and *Lr26* were found singly or in combination in 47 of the cultivars (55.9%). The most frequently occurring genes in cultivars grown in Sweden were *Lr13* (20.4%), *Lr14a* (14.8%) and *Lr26* (14.8%). *Lr14a* was the most common gene in cultivars grown in Norway (18.7%), *Lr13* in Denmark (35.5%) and *Lr10* in Finland (20.0%). Although 28 cultivars (33.3%) exhibited a response pattern that could not be assigned to resistance genes or combinations present in the tester lines, several pathotypes carried virulence and hence these genes or combinations are of limited use. Nine cultivars (10.7%) lacked detectable seedling resistance. One cultivar was resistant to all pathotypes used in the study.

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Leaf or brown rust, caused by the fungus *Puccinia triticina* Eriks., is one of the most common diseases of bread wheat (*Triticum aestivum* L.) in the world. Yield losses in Mexico can vary from 6.6% in resistant cultivars to 62.7% in susceptible cultivars under high disease pressure (Sayre et al. 1998). Quality losses due to leaf rust include reduced protein levels and softness equivalent scores (Everts et al. 2001). In the North European countries Denmark, Finland, Norway and Sweden, the disease generally appears late in the crop season. Yield losses are rarely severe but can be substantial during years when susceptible cultivars are used, winters are mild and conditions for development of leaf rust continue to be favorable throughout the season (Wiik 1991). In Sweden, yield losses of up to 10% have been noted in field trials (Sandnes and Waern 1991; Wiik 1991).

Genetic resistance is one of the most effective, environmentally sound and economic means of control of diseases on wheat (Pink 2002). Knowledge of the identity and diversity of leaf rust resistance genes in cultivars and commonly used germplasm in breeding programs can improve the efficiency of developing new resistant cultivars. More than 50 resistance genes to leaf rust have been identified in wheat (McIntosh et al. 2003, 2004). However, new pathogen races rapidly overcome most race-specific resistance genes. For example in Australia the release of cultivars with single genes for resistance to *Puccinia triticina* during 1938–1964 was followed by increased frequencies of pathotypes with matching virulence (Park et al. 2001). There is a continuous need to identify and incorporate effective resistance genes into cultivars of wheat.

The distribution of leaf rust resistance genes has been investigated for cultivars grown in Western and Eastern Europe (Bartoš and Valkoun 1988; Warzecha 1992; Broers and de Haan 1994; Bartoš et al. 1996; Mesterházy et al. 2000; Winzeler et al. 2000; Park et al. 2001; Singh et al. 2001) but only for a few cultivars grown in Northern Europe (Broers and de Haan 1994; Winzeler et al. 2000; Herrera Foessel 2001).

The objective of the present study was to investigate the occurrence and distribution of genes for seedling resistance to leaf rust in 84 bread wheat cultivars commonly grown in Denmark, Finland, Norway and Sweden during 1992–2002. Information on the genetic basis of resistance could contribute to a better understanding of the durability of resistance and facilitate the accumulation of effective resistance genes into cultivars.

MATERIAL AND METHODS

Host material

The host material represents 84 of the most widely cultivated spring and winter wheat cultivars in Denmark (31 cultivars), Finland (25 cultivars), Norway (16 cultivars) and Sweden (54 cultivars) between 1992 and 2002 (Table 1). Cultivars with the highest quantity of certified seed were selected based on figures obtained from the following seed control agencies or plant breeding companies: Plantedirektoratet in Denmark, Statens utsädeskontroll in Sweden, Landbrukstilsynet in Norway and Boreal PB in Finland. Svalöf-Weibull AB in Sweden, Graminor A/S in Norway and Boreal PB in Finland kindly provided seed. Information on pedigrees and year of release was provided by plant breeding companies, The Nordic Gene Bank or obtained from the Wheat Pedigree and Identified Alleles of Genes On Line database (http://genbank.vurv.cz/wheat/pedigree/).

A set of differential tester lines that carry known leaf rust (*Lr*) resistance genes were also included (Table 2). The tester lines, mostly developed by P.L. Dyck, Agriculture Canada, Winnipeg, Man., Canada, are maintained at CIMMYT (International Wheat and Maize Improvement Center) in Mexico.

Pathogen material

Twelve Mexican pathotypes of *Puccinia triticina* were used to evaluate the testers and cultivars. The nomenclature of the pathotypes follows the system of Long and Kolmer (1989), with two additional supplementary sets (Singh 1991). The pathotypes used were the following: BBB/BN, BBG/BN, CBJ/QB, CBJ/QL, CCJ/SP, MBJ/SP, MCJ/QM, MCJ/SP, MFB/SP, NCJ/BN, TBD/TM and TCB/TD (Table 3).

Inoculation, disease assessment and gene postulation

A set of 6–8 seeds per cultivar and tester line was used in the test with each pathotype. Ten-day-old seedlings were inoculated by spraying with urediniospores suspended in a light-weight mineral oil (2–3 mg ml^{-1}). Inoculated plants were placed in a dew chamber overnight at 18–20°C and then transferred to greenhouse chambers at 18–22°C. (Singh and Rajaram 1991). For pathotype NCJ/BN, avirulent to *Lr13*, seedlings of cultivars and tester lines were inoculated after 14 days since expression of *Lr13* is known to be clearer when older seedlings are inoculated (Singh and Rajaram 1991). Infection types (IT) were recorded 9 to 12 days after inoculation according to the 0 to 4 scale described by Stakman et al. (1962) and Roelfs (1984) where 0 = no macroscopic symptoms, ; = hypersensitive necrotic or chlorotic flecks, 1 = small uredinia surrounded by necrosis, 2 = small to medium uredinia often surrounded by necrosis or chlorosis, X = random distribution of variable-sized uredinia on single leaf, 3 = medium-sized uredinia that may be associated with chlorosis, 4 = large uredinia without chlorosis, + = uredinia somewhat larger than normal for the infection type, − = uredinia somewhat smaller than normal, C = more chlorosis than normal, N = more than usual degree of necrosis. Discrete infection types on different plants of the same line were separated by a comma (e.g. 4, or 1,3). A range of infection types on a single leaf, not adequately described by X, was recorded using more than one infection type with the predominant infection type listed first (e.g. 3C3, 12^{-}). Infection types 3 and 4 (susceptible host/virulent pathotype) were considered high and 0–2 were considered low (resistant host/avirulent pathotype).

The presence of leaf rust resistance genes in the cultivars was postulated by comparing the high and low infection types displayed by a cultivar with the infection type of known *Lr* genes in the tester lines. Based on the gene-for-gene concept, lines susceptible to a pathotype cannot have an *Lr* gene for which the pathotype is avirulent. *Lr* genes were considered present if the low infection type produced on a cultivar by one or more pathotypes, matched the infection type of the corresponding tester line. If the low infection type produced on a cultivar was lower than the corresponding tester lines, then the cultivar was considered to have one or more unidentified *Lr* genes (Statler 1984; Modawi et al. 1985; McVey 1989).

RESULTS

The 84 selected cultivars, estimated to be the most commonly grown in Northern Europe 1992–2002 based on quantities certified seed, are presented in Table 1. Several cultivars were grown in more than one country. A few of the most popular cultivars were not available to the present study: 'Ebi' and 'Lars' in Sweden; 'Stakado Abed', 'Sleipner' and 'Hereward' in Denmark; 'Folke', 'Portal', 'Kalle', 'Rida', 'Rubin', 'Lars' and 'Skjaldar' in Norway; 'Ramiro' and 'Gunbo' in Finland. According to literature, 'Sleipner' carries *Lr26* (McIntosh et al. 1995) and 'Hereward' has *Lr13* (Goyeau and Park 1997).

The infection types produced on the tester lines with known *Lr* genes after inoculation with twelve Mexican leaf rust pathotypes are presented in Table 2. The low infection types of the tester lines were in accordance with low infection types reported by McIntosh et al. (1995). The presence of genes *Lr3ka*, *Lr9*, *Lr14b*, *Lr16*, *Lr19*, *Lr20*, *Lr22b*, *Lr25*, *Lr29*, *Lr30*, *Lr34*,

Table 1. *Cultivars grown in Denmark, Finland, Norway and Sweden 1992–2002 and postulated genes for seedling resistance after inoculation with twelve pathotypes of Puccinia triticina.*

Cultivar	Pedigree[1]	Year[2]	Locality[3]	Postulated *Lr* gene(s)[4]
Winter wheat				
Aura	Ertus/Vakka	1975	F*	*u*
Ballad	Sv85297/Sv85568	2000	S	*u*
Bill	(DH) Multicross	n/a	D/S*	*Lr3+Lr17+Lr23+u*
Bjørke	SvU75630/Rida	1998	N*	*u*
Brigadier	Squadron/Rendezvous	1992	D/S	*Lr26+u*
Citadel	Composite cross of 24 cvs with main cv Tadorna	1983	D	*Lr13*
Dirigent	Ritmo/Reaper	n/a	D	*u*
Flair	Ares/Marabu; Ares/3/Rabe/Jubilar//Armada	1996	D/S	*u*
Florida	Caribo/Disponent	1985	D/S	*Lr26*
Grommit	Apostle/Torfrida//Hereward	n/a	D/S	*Lr3+Lr10+Lr17+u*
Haven	Hedgehog/Norman//Moulin	1988	D*/S	*Lr26+u*
Holme	Starke/WW-1014-55	1972	S	*u*
Hurtig	Severin M8B8/Sperber//Urban/3/Konsul	2003	S	*u*
Hussar	Squadron/Rendezvous	1991	D*/S	*Lr26+u*
Ilves	Hja b356/Vakka	1984	F*	*none*
Kamerat	Rida/Moulin//Disponent/2*Rida	n/a	N	*Lr26*
Kartesch	Severin M8B8/Sperber//Urban/3/Konsul	2003	S	*u*
Konsul	Ertus/Norre//Holme-M/3/Cerc.Res	1990	D/S	*Lr13+u*
Kosack	Mironovskaya-808/Starke-M//Holme-M	1984	D/N*/S*	*u*
Kris	Hereward/Rendezvous//Torfrida	1999	D*/S*	*Lr10+Lr13+u*
Linna	Panu/Hja04519/Virtus	1976	F	*none*
Lynx	Rendezvous/CWW-4442-64	1992	D*	*Lr17+Lr26+u*
Magnifik	Composite cross of German and Swedish lines	2004	N/S	*Lr1*
Marabu	LP-6077-71/Monopol//Kronjuwel	1988	D/S	*Lr26+u*
Marshal	Kontiki/Brigadier	2001	S	*Lr26+u*
Meridien	Starke/Norre/3/2*Ertus/Norre//Holme/4/Wampum/5/Moisson	1993	D/S*	*Lr13+u*
Mjølner	TL340/Starke/W25458	1996	N*/S	*Lr10+u*
Nova	Angela/TJB-330-1491//Arminda	1993	D/S	*Lr14a+u*
Otso	Elo/Vakka	1995	F	*u*
Pagode	Composite cross of 36 cultivars	1986	S	*Lr13*
Pepital	ROC/VDH-040-71-B; ROC-109-751/VDH-040-71-B; ROC/VDH-1040-71-B	1989	D*/S	*Lr10+Lr13*
Pitko	Ta 05901/Vakka	1985	F*	*u*
Rental	Sv70355/M. Huntsman	1993	S	*u*
Residence	Obelisk//Cebeco-8451/Arminda	1998	S	*Lr13+u*
Revelj	Kanzler M15M28	2000	D/S	*Lr13+u*
Rialto	Haven (sib)/Fresco (sib)	1993	D	*Lr13+Lr26+u*
Ritmo	Hobbit//Line-1320/Wizard/3/Marksman/4/Virtue	1990	D*/S*	*Lr13+u*
Stava	Helge-M7D1/Helge-M7D2//WW-31254	1995	D/S*	*u*
SW Gnejs	KosackMB/3*Kraka/4/Kurier	2001	S	*Lr1*
SW Harnesk	WD-linje/Konsul	2001	S	*Lr13+u*
Tarso	Taras/Hadmerslebener 13313-80	1992	D*/F/S*	*Lr26+u*
Terra	Kraka/TJB730-3637	1992	D*/N/S	*Lr13*
Tjelvar	Sture D/4/StureM3bM5M7	1984	S	*Lr26*
Toronto	Disponent/Weihenstephaner 616–67//Kronjuwel	1990	D	*Lr26*
Trintella	CB-239/VDH-256-81//RPB-48-75-A/Moulin	1994	D	*Lr13*
Tryggve	Riley/Holme//18614/3/Helge	1990	F/S	*u*
Urban	Kranich/Diplomat	1981	S*	*none*
Urho	Nisu/Tsitsin	1999	F	*u*
Vakka	Varma/Kehra	1960	F	*u*
Virke	n/a	1999	S	*none*
Spring wheat				
Anniina	Satu/Polkka	2001	F*	*u*
Avle	22279M15/20299M12//Canon	1996	N*/S	*Lr14a*
Bajas	Bastian/Sport	n/a	N/S	*u*

Table 1 (*Continued*)

Cultivar	Pedigree[1]	Year[2]	Locality[3]	Postulated *Lr* gene(s)[4]
Bastian	Bajio-66/Runar/4/Yaktana/Norin10/Brevor/3/Moystad/5/ Rollo/Magnif/4/Sonora/Tezanos-Pintos-Precoz//Nainari/ 3/Moystad	1989	F*/N*	*none*
Bjarne	SvB87293/Bastian	2002	N	*Lr1*
Canon	Sicco/2*WW-12502//2*Sappo/3/Kadett	1990	S	*Lr14a*
Curry	Canon s/Nemares//Kadett Mp1	1994	S*	*Lr14a+u*
Dacke	P18/17269//19151	1990	S	*none*
Drabant	CI-12633/6*Ring	1973	S	*Lr14a*
Dragon	Sicco/WW-12502//Sappo/5*Kadett	1988	D*/S*	*Lr14a+u*
Hanno	Banjo/Hermes	n/a	D/N	*u*
Heta	Hja a1105/Hja a4431	1988	F*	*Lr10*
Hugin	Dragon (sib)/Nemares	1996	D/S	*Lr13+u*
Jondolar	Sicco/Tilly//VDH-1166-76-M	1990	D	*Lr14a*
Kadett	Kolibri/WW 439-66/Pompe-M	1981	F*/S	*u*
Kruunu	Mahti/Hja23471	2001	F*	*Lr10*
Laari	Villa Glofi/Tuoko	1990	F	*u*
Lavett	WW118466/Kadett//Dragon	1992	S	*u*
Leguan	ST-324-84/ST-174-83	1997	D*	*Lr14a+u*
Luja	Svenno//Hopea/Tammi	1981	F*	*u*
Mahti	Cebeco/Hja20519	1995	F*	*Lr10*
Manu	Ruso/Runar	1992	F*	*u*
Polkka	Sv70415/Snabbe//Norrena/Karn-2/3/Snabbe	1992	F*/N*/S	*u*
Runar	Els/Rollo; Els/7*Rollo	1972	N	*u*
Ruso	Reward/Pika	1967	F	*Lr10*
Satu	Snabbe/Drabant//T-106/Snabbe	1990	F/S	*none*
Sport	P18/17269//19151	1991	S	*u*
SW Vals	Can.M12 M14 M18 B9 B10/ Can.M14 M15 B9	2001	S	*Lr2a+u*
Tapio	Hja c3929/Kolibri	1980	F	*Lr10+u*
Thasos	Miron.808/ Bastion// Minaret; Max /Ze73.1331.2/Minaret	1994	S	*none*
Tjalve	Reno/KolibriM//15432	1981	F/N*/S	*none*
Triso	SaxoArgan/Kadett	1996	S	*u*
Vinjett	Tjalve M14/Tjalve M15//Canon	1998	D*/F/N*/S*	*Lr14a*
Zebra	Ralle/Dragon	2001	F/N/S	*Lr14a*

[1] Alternative pedigrees are separated by a semi-colon (;); n/a = not available.
[2] n/a = not available.
[3] D = Denmark, F = Finland, N = Norway, S = Sweden; * = total quantity certified seed 1992-2002 was among the ten highest for the country and cultivar.
[4] *u* = unidentified gene(s).

Lr36, *Lr37* and *LrB* could not be determined with the twelve pathotypes used in the study because all were avirulent or virulent to these genes.

The results of the gene-postulations for different cultivars are presented in Table 1. A summary of the identified and unidentified genes in the material is presented in Table 4. Comparisons of infection types displayed by the cultivars and tester lines allowed the postulation of nine known leaf rust genes in the material: *Lr1*, *Lr2a*, *Lr3*, *Lr10*, *Lr13*, *Lr14a*, *Lr17*, *Lr23* and *Lr26*. Of the 84 cultivars tested, 9 had no detectable seedling resistance; 47 lines had one or more known *Lr* genes including 26 cultivars that had one or more known *Lr* genes and one or more unidentified *Lr* genes. The infection types of the 28 cultivars exhibiting only a response pattern not corresponding to the tester lines and postulated to carry only unidentified *Lr* genes are presented in Table 5. Eleven different combinations of known and unknown *Lr* genes were detected in the material. Ten cultivars were resistant to more than eight pathotypes including one cultivar ('Lavett') that was resistant to all pathotypes used in the study (Table 5).

The results for the 54 cultivars commonly grown in Sweden 1992–2002 are presented in Tables 1 and 4. The cultivar 'Kosack' (*u*) was released in 1984 and has dominated the seed market to the present day. The winter wheat cultivars 'Ritmo' (*Lr13+u*), 'Stava' (*u*), 'Tarso (*Lr26+u*), 'Kris' (*Lr10+Lr13+u*), 'Meridien' (*Lr13+u*), 'Urban' (*none*), 'Bill' (*Lr3+Lr17+Lr23+u*), 'Ebi' and 'Lars' and spring wheat cultivars 'Dragon' (*Lr14a+u*), 'Curry' (*Lr14a+u*) and 'Vinjett' (*Lr14a*) were the most extensively grown during 1992–2002 estimated from quantities certified seed

Table 2. *Seedling infection types*[1] *displayed by tester varieties with known Lr genes when tested with 12 different pathotypes of Puccinia triticina.*

Lr gene (s)	Tester	Pathotype											
		BBB/BN	BBG/BN	CBJ/QB	CBJ/QL	CCJ/SP	MFB/SP	TBD/TM	TCB/TD	MCJ/QM	MCJ/SP	MBJ/SP	NCJ/BN
1	R.L.6003	0;	0;	0;	0;	0;	3+	3+	3+	3+	3+	3+	3+
2a	R.L.6016	0;	1	;	0;	0;	0;	3+	3+	0;	0;	0;	1
2b	R.L.6019	;	1+	1−	0;	;	;	3+	3+	0;	0;	;	1+
2c	R.L.6025	;1	3C3	1+3C	;1−	;	1−	3+	3+	;	0;	;	3
3	R.L.6002	12	;1−	3+	23−	3	3+	3+	3+	23C	23C	3+	;
3bg	R.L.6042	;1	;	23C	3+	;12	3C3	3+	3+	3C3	;1−	3+	;
3ka	R.L.6007	;1	;1−	23C	;1−	;12−	12−	12	;12	;12	3C	23C	12
9	R.L.6010	0;	0;	0;	;	0;	0;	0;	0;	0;	0;	;	0;
10	R.L.6004	3+	3+	;1	3+	3+	3+	3+	;1−	3+	3+	3+	3+
11	R.L.6053	13C	4	4	4	3+	3C3	1+3C	1+3	3+	3+	3+	3+
12	R.L.6011	X+, 3+	3C	3+	4	3+	3+	3+	X+3	3+	3+	3+	XX+
13	Manitou	X	1, 1+	3+	3+	3+	3+	3+	3+	3+	3+	3+	11+
14a	R.L.6013	X	X	3+	4	3+	3+	3+	3+	3+	3+	3+	3+
14b	R.L.6006	3+	3+	3+	4	3+	3+	3+	3+	3+	3+	3+	3+
15	R.L.6052	;1−	;1−	0;	;	3+	3+	3+	3+	;	3+	3+	;1−
16	R.L.6005	1	1	1	1+	1+3C	1	1	1	1	1+3C	1+3C	11+
17	R.L.6008	;	;	3+	X+	3+	;1−	3+	;1−	3+	3+	3+	2+3C
18	R.L.6009	23C	23C	3+	3+	23C	22+	3+	3+	23C	3C	12	2
19	R.L.6040	;	;	0;	0;	;	0;	;	;	0;	;	;	0;
20	W.203	3+	3+	3+	3+	3+	4	3+	3+	3+	3+	3+	3+
21	R.L.6043	12	;1	12	12	12	;12−	;1	;12	12	23C	12	3+
22a	R.L.6044	3+	3+	3+	3+	3+	3+	3+	3+	3+	3+	3+	12
22b	C.I.10003	3+	3+	3+	3+	3+	3+	3+	3+	3+	3+	3+	3+
23	R.L.6012	3+	3+	;1	;1	3+	3+	23C	3+	;1	3+	3+	3+
24	R.L.6064	;	;	;1−	;1−	;	23C	0;	;	0;	;	;	;1−
25	R.L.6084	0;	0;	0;	;	0;	0;	0;	0;	0;	0;	;	0;
26	R.L.6078	23C	0;	1+3C	;1−	3+	3+	;1−	3+	3+	3+	11+	3+
27+31	Baviacora 92	X−	;1	X	X−	X+	3	3+	X−	3+	3+	3+	X
27+31+10	Gatcher	X−	;1−	;	X−	X+	3+	3+	;	3+	3+	3+	;1
28	R.L.6079	X+	;1−	0;	0;	0;	3+	3+	X+	0;	0;	0;	3C3+
29	R.L.6080	;1−	;1−	;1	;1−	;1−	1	;1	;1	;1	;1−	;1−	1
30	R.L.6049	2−	12	23C	;12	;12	12	23C	23	23C	;12−	23−	12−
32	R.L.5497 −1	;1	;1−	3C3	3+	23	;12−	3	;1	3C3	12	12	12−
33	R.L.6057	3	3+	23	3+	23	3C	12	;23	3	12	23	23−
34	R.L.6058	3+	3+	3+	3+	3	3	3	3+	3+	;23=	3	3
35	R.L.5711	3+	3C3	3+	3+	3	3	3	3+	1+3C	3C	3+	3+
36	E.84018	;1	;1	;1−	12	X	;	;1	;1	1	12	13C	;1−
37	R.L.6081	3+	3C3+	3+	3+	3+	3+	X+3	3+	3+	3+	3+	2+3C
B	R.L.6051	3+	3+	3+	3+	3+	3+	3	3+	3+	3+	3+	3+

[1] 0 = No uredinia or other macroscopic signs of infections;; = no uredinia, but hypersensitive necrotic or chlorotic flecks of varying size present; 1 = small uredinia often surrounded by necrosis; 2 = small to medium uredinia often surrounded by chlorosis or necrosis; 3 = medium-sized uredinia that may be associated with chlorosis or rarely necrosis; 4 = large uredinia without surrounding chlorosis; X = random distribution of variable-sized uredia on single leaf; + = uredinia somewhat larger than normal for the infection type; − = uredinia somewhat smaller than normal; C = more chlorosis than normal; N = more necrosis than normal. Discrete infection types on plants of the same line are separated by a comma. A range of infection types on a single leaf is described using more than one infection type with the predominant infection type listed first.

Table 3. *Avirulence/virulence formulae for leaf rust genes based on seedling reactions for 12 Puccinia triticina pathotypes used in the study*[1].

Pathotype	Avirulence to *Lr* genes	Virulence to *Lr* genes
BBB/BN	*1,1c,2a,2b,2c,3,3bg,11,13,14a,15,17,18,21,24,26,27+31*	*10,22a,23,28*
BBG/BN	*1,1c,2a,2b,2c,3,3bg,11,13,14a,15,17,18,21,24,26,27+31,28*	*10,22a,23*
CBJ/QB	*1,1c,2a,2b,2c,10,15,18,21,23,24,26,27+31,28*	*3,3bg,11,13,14a,17,22a*
CBJ/QL	*1,2a,2b,2c,15,18,21,23,24,26,27+31,28*	*3,3bg,10,11,13,14a,17,22a*
CCJ/SP	*1,1c,2a,2b,2c,18,21,24,28*	*3,3bg,10,11,13,14a,15,17,18,22a,23,26,27+31*
MFB/SP	*2a,2b,2c,11,17,18,21*	*1,3,3bg,10,13,14a,15,22a,23,24,26,27+31,28*
TBD/TM	*1c,11,21,23,24,26*	*1,2a,2b,2c,3,3bg,10,13,14a,15,17,18,22a,27+31,28*
TCB/TD	*1c,10,11,17,21,24,27+31,29*	*1,2a,2b,2c,3,3bg,13,14a,15,18,22a,23,26,28*
MCJ/QM	*1c,2a,2b,2c,15,18,21,23,24,28*	*1,3,3bg,10,11,13,14a,17,22a,26,27+31*
MCJ/SP	*1c,2a,2b,2c,21,24,28*	*1,3,3bg,10,11,13,14a,15,17,18,22a,23,26,27+31*
MBJ/SP	*1c,2a,2b,2c,21,24,26,28*	*1,3,3bg,10,11,13,14a,15,17,18,22a,23,27+31*
NCJ/BN	*2a,2b,3,3bg,10,13,15,18,22a,24,27+31*	*1,2c,10,11,14a,17,21,23,26,28*

[1]All pathotypes were avirulent in seedling stage for genes *Lr3ka*, *Lr9*, *Lr16*, *Lr19*, *Lr25*, *Lr29*, *Lr30*, *Lr36*; and were virulent for genes *Lr14b*, *Lr20*, *Lr22b*, *Lr34*, *Lr37*, *LrB*.

(Nilsson and Andersson 1998; Andersson et al. 2002). Of the cultivars tested, 11.1% were susceptible; 61.1% were postulated to carry one or more known *Lr* genes with or without additional unidentified genes. 68.5% showed infection types not corresponding to the tester lines and were postulated to have one or more unidentified *Lr* genes with or without known *Lr* genes. The most commonly occurring genes were *Lr13* (20.4%), *Lr14a* (14.8%) and *Lr26* (14.8%) while *Lr2a*, *Lr3*, *Lr10*, *Lr17* and *Lr23* occurred in less than 10% of the cultivars.

The results for the 31 cultivars commonly grown in Denmark are presented in Tables 1 and 4. Wheat is mainly of winter type in Denmark and there is a rapid change in cultivar distribution. The two most commonly grown cultivars typically account for more than 50% of the area under cultivation e.g. 'Ritmo' was grown on more than 50% of the wheat area 1998 and 1999 (Hovmøller 2001). 'Pepital' (*Lr10+Lr13*), 'Haven' (*Lr26+u*), 'Hussar' (*Lr26+u*), 'Ritmo' (*Lr13+u*), 'Lynx' (*Lr17+Lr26+u*), 'Kris' (*Lr10+Lr13+u*), 'Terra' (*Lr13*), 'Sleipner' and 'Hereward' have been the most common winter wheat cultivars during 1992–2002. 'Dragon' (*Lr14a+u*), 'Leguan' (*Lr14a+u*) and 'Vinjett' (*Lr14a*) have been the most popular spring wheat cultivars (Plantedirektoratet 1992–2002). The infection response patterns of the selected cultivars and tester lines enabled postulation of *Lr* genes in 83.9% of the material while a total of 77.4% displayed a resistance response not matching the testers. *Lr13* occurred in 35.5%; *Lr26* in 29.0%; *Lr14a* in 16.1%; *Lr10* and *Lr17* in 9.7%; *Lr3* in 6.4% and *Lr23* in 3.2% of the material.

The results for the 16 cultivars commonly grown in Norway are presented in Tables 1 and 4. Cultivars 'Avle' (*Lr14a*), 'Zebra' (*Lr14a*), 'Bjarne' (*Lr1*), 'Tjalve' (*none*), 'Bastian' (*none*), 'Polkka' (*u*), 'Mjølner' (*Lr10+u*), 'Terra' (*Lr13*), 'Bjørke' (*u*), 'Magnifik' (*Lr1*) and 'Folke' have been the most commonly grown during 1992–2002 (Åssveen et al. 2003). Of the 16 cultivars grown in Norway, 50.0% contained one or more of genes *Lr14a* (18.7%), *Lr1* (12.5%); *Lr10*, *Lr13* or *Lr26* (6.2%) while 37.5% had an infection type pattern not matching the tester lines and 12.5% were susceptible.

The results for the 25 selected cultivars grown in Finland are presented in Tables 1 and 4. In Finland, the cultivars 'Tjalve' (*none*), 'Mahti' (*Lr10*), 'Vinjett' (*Lr14a*), 'Kruunu' (*Lr10*), 'Bastian' (*none*), 'Zebra' (*Lr14a*), 'Manu' (*u*) and 'Anniina' (*u*) have dominated the spring wheat market. The most common winter wheat cultivars during 1992–2002 were 'Tryggve' (*u*), 'Urho' (*u*), 'Tarso' (*Lr26+u*), 'Aura' (*u*), 'Ilves' (*none*), 'Gunbo' and 'Ramiro' (M. Jalli, pers. comm). Gene-postulation showed the presence of *Lr10* (20.0%); *Lr14a* (8.0%) and *Lr26* (4.0%) in 32.0% of the material while 48% displayed only a resistance reaction different from that of the tester lines and 20.0% were susceptible to all twelve leaf rust pathotypes.

DISCUSSION

The results of the present study showed that infection types corresponding to nine known and several unidentified *Lr* resistance genes, either singly or in combination, conditioned race-specific seedling resistance in 75 of the 84 investigated wheat cultivars commonly grown in Northern Europe between 1992 and 2002. Genes masked by gene suppression, not expressed in the seedling stage or under the given environmental conditions could remain undetected

Table 4. *Number and percentage of cultivars (cvs) found susceptible, with unidentified resistance (u) and with postulated Lr gene(s) after testing with 12 Puccinia triticina*[1].

				Single *Lr* genes					*Lr* genes in combinations											Single *Lr* genes and combinations								
Country	No cvs	susceptible	unidentified – only	*Lr1*	*Lr10*	*Lr13*	*Lr14a*	*Lr26*	*Lr2a/u*	*Lr3/Lr10/Lr17/u*	*Lr3/Lr17/Lr23/u*	*Lr10/u*	*Lr10/Lr13*	*Lr10/Lr13/u*	*Lr13/u*	*Lr13/Lr26/u*	*Lr14a/u*	*Lr17/Lr26/u*	*Lr26/u*	*Lr1+*	*Lr2a+*	*Lr3+*	*Lr10+*	*Lr13+*	*Lr14a+*	*Lr17+*	*Lr23+*	*Lr26+*
Sweden	54	6	15	2	–	2	5	2	1	1	1	1	1	1	7	–	3	–	6	2	1	2	4	11	8	2	1	8
%		11.1	27.8	3.7	–	3.7	**9.2**	3.7	1.8	1.8	1.8	1.8	1.8	1.8	**13.0**	–	5.5	–	11.1	3.7	1.8	3.7	7.4	**20.4**	14.8	3.7	1.8	14.8
Denmark	31	–	5	–	–	3	2	2	–	1	1	–	1	1	5	1	3	1	5	–	–	2	3	11	5	3	1	9
%		–	16.1	–	–	**9.7**	6.4	6.4	–	3.2	3.2	–	3.2	3.2	**16.1**	3.2	9.7	3.2	**16.1**	–	–	6.4	9.7	**35.5**	16.1	9.7	3.2	29.0
Finland	25	5	12	–	4	–	2	–	–	–	–	1	–	–	–	–	–	–	1	–	–	–	5	–	2	–	–	1
%		20.0	48.0	–	**16.0**	–	8.0	–	–	–	–	**4.0**	–	–	–	–	–	–	**4.0**	–	–	–	**20.0**	–	8.0	–	–	4.0
Norway	16	2	6	2	–	1	3	1	–	–	–	1	–	–	–	–	–	–	–	2	–	–	1	1	3	–	–	1
%		12.5	37.5	12.5	–	6.2	**18.7**	6.2	–	–	–	**6.2**	–	–	–	–	–	–	–	12.5	–	–	6.2	6.2	**18.7**	–	–	6.2
Total	84	9	28	3	4	4	6	4	1	1	1	2	1	1	7	1	4	1	6	3	1	2	9	14	10	3	1	12
%		10.7	33.3	3.6	4.8	4.8	**7.1**	4.8	1.2	1.2	1.2	2.4	1.2	1.2	**8.3**	1.2	4.8	1.2	7.1	3.6	1.2	2.3	10.7	**16.7**	11.9	3.6	1.2	14.3

[1]some cultivars were grown in more than one country, percentages are therefore presented as cultivars/country and total represents the total number/percentage of cultivars within the material. The highest percentage are shown in bold type in different sections of the table.

Table 5. *Infection type*[1] *response patterns of cultivars grown in northern Europe 1992–2002 with only unidentified genes (u) for seedling resistance and/or resistance to more than eight pathotypes after inoculation with twelve pathotypes of Puccinia triticina.*

Cultivar	Pathotype												Postulated
	BBB/BN	BBG/BN	CBJ/QB	CBJ/QL	CCJ/SP	MFB/SP	TBD/TM	TCB/TD	MCJ/QM	MBJ/SP	MCJ/SP	NCJ/BN	*Lr* gene(s)
Anniina	3+	3+	4	2+	3	3+	3+	3+	3+	3+	3+	4	*u*
Aura	3+	3+	3+	2+3	3+	4	3+	3+	3+	3+	3+	4	*u*
Bajas	3+	3+	;23C	*[2]	3+	3	3	3+	3	3+	3+	3+	*u*
Ballad	3+	3+	3+	4	3+	3+	3+	3+	3+	3+	3+	2+	*u*
Bill	;1 −	;1	;	;	3+	12	12	;1 −	;1	3+	3+	1	*Lr3+Lr17+Lr23+u*
Bjørke	3+	;	;	;, 3+	3+	3	3+	4	3+	3+	3+	3+	*u*
Brigadier	;	0;	;1 −	;	X	;12 −	;	;1 −	;11 −	;1	3+	;	*Lr26+37+u*
Dirigent	;	0;	;	0;	3+	2+	;	1+2	;1	12	3C3	12	*u*
Flair	3+	3+	23C	;	3+	3+	3+	3+	3+	3+	3+	3+	*u*
Grommit	;1 −	0;	0;	;23	3+	;	;1 −	0;	;1	;23C	;23	0;	*Lr3+Lr10+Lr17+u*
Hanno	3+	23C	3+	1+3C	3+	3+C	3+	3+4	3+	3+	3C3	2+3C	*u*
Haven	X	0;	0;	0;	3+	2+3	;	;1 −	23C	X	3C3	2+3C	*Lr26+u*
Holme	3+	X	;12	;	3+	4	4	3+	3	3+	3+	3+	*u*
Hurtig	3+	3+	3+	2+3C	3+	2+3C	3+	3+	3+	3+	3+	12 −	*u*
Hussar	;	0;	0;	0;	X+	;1 −	;	;	;1 −	;1	3C3+	;	*Lr26+u*
Kadett	3+	4	23C	;	3+	4	4	3+	3+	3+	3+	3+	*u*
Kartesch	3	1+3C	3+	4	3+	2+3C	3+	3+	3+	3+	3+	;1 −	*u*
Kosack	3+	3C3	;1	0;	3+	3+	4	3+	3+	3+	3+	2+3C	*u*
Kris	;1 −	;1	;	3+	3+	2+3C	12	0;	;12	3+	3+	1+2	*Lr10+Lr13+u*
Laari	3+	3+	3+	2+3C	3+	3+	3+	3+	3+	3+	3+	3+	*u*
Lavett	;1+	;1	1+	1+	1+3C	1 −	;1	;1	1	1+	1+2	12	*u*
Luja	3+	X	4	23C	3+	4	3+	3+	3+	3+	3+	3+	*u*
Lynx	;	0;	0;	0;	23	;1 −	;	;	;	;1	3+	;	*Lr17+Lr26+u*
Manu	3+	X	3+	22+	3+	3+	3+	3+	3+	3+	3+	2+3C	*u*
Marshal	;1 −	0;	;1	;1C	X+	23C	;	1+3C	1+2	23C	3+	12	*Lr26+u*
Otso	3+	X	3+	3+	3+	3+	3+	3+	3+	3+	3+	3+	*u*
Pitko	3+	X	4	4	3+	4	3+	3+	3+	3+	3+	4	*u*
Polkka	3+	3+	3+	23C	3+	3+	;1 −	3+	3+	3+	3+	4	*u*
Rental	3+	3C3+	4	4	3+	3+	4	3+	3+	3+	3+	4	*u*
Rialto	;11+	0;	;	;1 −	X+	23C	;	;, 3+	23C	23C	3+	X, 3+	*Lr13+u*
Runar	3+	4	3+	2+3	3+	3+	3+	3+	3+	3+	3+	4	*u*
Sport	3+	3+	23C	;1 −	3+	3+	3+	3+	3+	3+	3+	4	*u*
Stava	3+	3+	;1	0;	X+3	23C	2+	2+3	3C3	3+	3+	1+	*u*
SW Vals	0;	;	;1 −	0;	0;	;	3+	3+	0;	;	;	X	*Lr2a+u*
Triso	3+	3+	3+	4	3+	3+	3+	3+	3+	2+3C	3+	3+	*u*
Tryggve	3+	3+	13C	;	3+	3+	3+	3+	3+	3+	3+	3+	*u*
Urho	3+	2+3C	3+	2+3C	3+	3+	3+	3+	3+	3+	3+	4	*u*
Vakka	3+	2+3C	4	2+3C	3+	3+	4	3+	3+	3+	3+	4	*u*

[1] 0 = No uredinia or other macroscopic signs of infections;; = no uredinia, but hypersensitive necrotic or chlorotic flecks of varying size; 1 = small uredinia often surrounded by necrosis; 2 = small to medium uredinia often surrounded by chlorosis or necrosis; 3 = medium-sized uredinia that may be associated with chlorosis or rarely necrosis; 4 = large uredinia without surrounding chlorosis; X = random distribution of variable-sized uredinia; + = uredinia somewhat larger than normal for the infection type; − = uredinia somewhat smaller than normal; C = more chlorosis than normal; N = more necrosis than normal. Discrete infection types on different plants of the same line are separated by a comma. A range of infection types on a single leaf is described using more than one infection type with the predominant infection type listed first.

[2] * = missing data.

but may still have an effect on the disease resistance in the field.

Genes *Lr13*, *Lr26*, *Lr14a* and *Lr10* were postulated in more than 10% of the material followed by *Lr1*, *Lr17*, *Lr3*, *Lr23* and *Lr2a*. Twenty-eight cultivars displayed infection type patterns that could not be attributed to known *Lr* genes in the tester lines. Lines with identical infection types to the same pathotypes may or may not have the same unidentified *Lr* gene(s). The unidentified genes could be known *Lr* genes that could not be postulated with the leaf rust pathotypes in the present study, adult-plant resistance genes that are expressed slightly in the seedling stage, undescribed *Lr* genes or alleles of known *Lr* genes. Inclusion of additional testers, pathotypes and adult plant tests would possibly have allowed some of these *Lr* genes to be identified.

In order to understand the usefulness of the genes postulated in the material grown in Northern Europe, it is necessary to compare the presence of postulated *Lr* genes with virulence data for *Puccinia triticina* populations. Pathogenicity surveys carried out in Sweden in 1957 (Gustavsson 1958), 1958 (Björkman 1959), 1959 (Leijerstam 1960) and 1960 (Hermansen 1962) showed five predominating pathotypes often occurring in mixtures. However, the virulence composition of the pathotypes was difficult to determine due to the method of analysis. In the absence of recent virulence data, virulence surveys in neighboring countries and comparisons of the results from the present study with annual disease severity ratings of leaf rust in Sweden and Norway may indicate presence of virulence to the postulated genes in the material grown in Northern Europe.

Surveys in Western and Central Europe 1996–1999 have shown that countries have few pathotypes in common, indicating a great genetic diversity within the pathogen (Mesterházy et al. 2000). Virulence surveys in Western Europe 1995 (Park and Felsenstein 1998) detected 53 pathotypes, including 4 predominating pathotypes with virulence to *Lr3*, *Lr10*, *Lr17b* and *Lr26*. Based on these surveys, cultivars with *Lr9*, *Lr12*, *Lr19*, *Lr22a*, *Lr24*, *Lr25*, *Lr28*, *Lr29*, *Lr34*, *Lr35* and *Lr37* should provide some protection against leaf rust also in Northern Europe, particularly if combined with other efficient resistance genes.

The gene *Lr13* was the most common resistance gene that could be postulated in the material and is probably the most widely distributed *Lr* gene in the world (McIntosh et al. 1995). Winzeler et al. (2000) found that 58% of the European wheat genotypes tested carried *Lr13* alone or in combination. The gene was once considered to confer durable adult plant resistance (Singh et al. 2001) but is now ineffective in several countries including Mexico (Singh 1991). *Lr13* is still considered effective in combinations with other race-specific genes in Australia as the *Lr13*-virulent pathotype was avirulent on many other resistance genes (Singh et al. 2001). However, in Mexico, pathotypes contain virulence to *Lr13* in combination with virulence on several important resistance genes (Singh 1991) and many cultivars that carry *Lr13* alone or in combination with other genes were susceptible in field trials (Singh and Rajaram 1991). In Europe 1996–1999, virulence to cultivars with *Lr13* varied with genotype suggesting that either the frequency of virulence to *Lr13* varied across Europe or that additional genes are present in cultivars displaying higher field resistance (Winzeler et al. 2000). In North America, cultivar 'Era' with *Lr10*, *Lr13* and *Lr34* has remained highly resistant to moderately resistant since the 1970s (Oelke and Kolmer 2004). Effective resistance in the North American cultivars 'Alsen' and 'Norm' were found to be due to the interaction of *Lr13* and *Lr23*, with *Lr34*; and *Lr13*, *Lr16*, *Lr23* with *Lr34* (Oelke and Kolmer 2005). In the present study, *Lr13* was found together with *Lr10*, *Lr26* and/or unidentified genes. According to annual surveys of disease severity in Sweden, cultivar 'Revelj' (*Lr13*+*u*) had an average of 25% diseased leaf area 1997–2001 (Larsson et al. 2002) indicating that *Lr13* and the unidentified gene(s) have been overcome in Sweden. Diseased leaf area for 'Ritmo', 'Konsul' and 'Pagode' was 1% 1993–1997 (Larsson et al. 1997); 'Meridien' 5% and 'Residence' 4% 1997–2001 (Larsson et al. 2002); 'SW Harnesk' 5% 1998–2002 (Larsson et al. 2003) and 'Trintella' 4% leaf rust 1995–1999 (Larsson et al. 2000). It is thus likely that these cultivars contain additional resistance genes more effective than those of 'Revelj'. 'Kris' (*Lr10*+*Lr13*+*u*) had 2% diseased leaf area 1998–2002 (Larsson et al. 2003) while 'Pepital' (*Lr10*+*Lr13*) had 1% diseased leaf area 1993–1997 (Larsson et al. 1997). Although disease severity varies between years and cultivars, it seems that virulence exists to *Lr13* and the combinations of *Lr13* with *Lr10* and unidentified genes in northern Europe.

The resistance gene *Lr26* is present on the rye segment in a T1BL.1RS wheat-rye translocation. The translocation was spread mainly through wheat derivatives produced in Germany during the 1930s (Rabinovich 1998). In the present study, cultivars 'Brigadier', 'Florida', 'Haven', 'Hussar', 'Marabu', 'Rialto', 'Tjelvar', 'Tarso' and 'Toronto' showed infection types corresponding to *Lr26* and carry

the T1BL.1RS translocation (Schlegel et al. 1994; Kazman and Lein 1996). The cultivars 'Kamerat' and 'Marshal' also displayed *Lr26*, most likely derived from 'Disponent' and 'Brigadier' in the pedigrees. Pathotypes virulent to *Lr26* exist in most areas (McIntosh et al. 1995). In annual disease surveys in Sweden, cultivars 'Florida' (*Lr26*) had an average of 3% leaf rust 1986–1995 (Larsson and Magnét 1995); 'Tjelvar' (*Lr26*) 3% 1983–1992 (Carlsson et al. 1992); 'Toronto' (*Lr26*) and 'Tarso' (*Lr26+u*) 5%, 'Haven' (*Lr26+u*) 1% and 'Hussar' (*Lr26+u*) 2% infected leaf area 1993–1997 (Larsson et al. 1997) and 'Marshal' (*Lr26+u*) showed 3% 1998–2002 (Larsson et al. 2003). It is thus clear that virulence to *Lr26* and combinations with unidentified genes exists in Northern Europe.

The gene *Lr14a* originates from *Triticum dicoccum* (McIntosh et al. 1995). The cultivar 'Canon', displaying *Lr14a*, is the likely donor of this gene to 'Curry', 'Avle', 'Zebra' and 'Vinjett'. Seedling tests with 20 Australian leaf rust pathotypes have shown *Lr20* in 'Canon' but not *Lr14a* (Singh et al. 2001). *Lr20* could not be detected with the pathotypes used in the present study because all were virulent to this gene. Expression of *Lr14a* can be variable and difficult to interpret due to host genetic background, virulent pathotypes, epistatic genes or intermediate infection types. The gene *Lr14a* is linked to powdery mildew resistance gene *Pm5* (McIntosh et al. 1995). Adult plant studies and powdery mildew tests may yield additional information on the presence or absence of *Lr14a* and *Lr20* in 'Canon' used in the present study. The difference could also be due to heterogeneity in the material. Annual disease surveys in Sweden have shown 11% diseased leaf area on 'Curry', 8% on 'Dragon' and 3% on 'Vinjett' 1998–2002 (Larsson et al. 2003); 16% on 'Drabant' 1985–1992 (Carlsson et al. 1992); 1% on 'Nova' 1993–1997 (Larsson et al. 1997) and 10% leaf rust on 'Zebra' 2000–2001 (Larsson et al. 2002). This indicates that 'Vinjett' and 'Nova' may have additional adult plant and/or additive minor genes that may have contributed to resistance in the field compared to 'Curry' and 'Dragon'. In a province of Norway in 2002, the cultivar 'Zebra' had 50% diseased leaf area, 'Avle' around 20% and 'Vinjett' about 15% (Abrahamsen et al. 2003). This indicates that resistance gene *Lr14a* has been overcome in Sweden and this region in Norway. These cultivars have been widely used in Northern Europe and it is likely that *Lr14a* has been overcome also in other areas.

Resistance gene *Lr10* originates from bread wheat and may have some effect in combinations with other genes in parts of the world (McIntosh et al. 1995). *Lr10* occurred singly or with unidentified gene(s) in the Finnish cultivars 'Ruso', 'Kruunu', 'Mahti', 'Heta' and 'Tapio'. The source was not apparent from the pedigrees. No information regarding virulence to leaf rust in Finland could be obtained for the present study. In Sweden, annual disease surveys showed 4% diseased leaf area on 'Grommit' and 2% on 'Kris' 1998–2002 (Larsson et al. 2003) compared to 1% on 'Pepital' 1993–1997 (Larsson et al. 1997) that all have *Lr10* in combination with other *Lr* genes. Virulence to *Lr10* thus seems to be present in Northern Europe.

The genes *Lr1*, *Lr2a*, *Lr3*, *Lr17* and *Lr23* occurred in less than 5% of the material. Annual disease surveys in Sweden have shown 3% leaf rust on 'SW Gnejs' (*Lr1*) 1998–2002; 4% leaf rust on 'SW Vals' (*Lr2a+u*); 4% on 'Grommit' (*Lr3+Lr10+Lr17+u*) and 3% on 'Bill' (*Lr3+Lr17+Lr23+u*) 1998–2002 (Larsson et al. 2003). It seems likely that the low incidence of leaf rust on these cultivars could be due to combinations of effective *Lr* genes and adult plant genes and/or additive minor genes.

Several cultivars displayed infection type patterns that did not correspond to known *Lr* genes in the tester lines. In the present material, similar infection types not corresponding to the tester lines were found in 'Hussar', 'Kris', 'Lynx', 'Marshal' and 'Brigadier'. It is possible that these cultivars contain *Lr37*, supported by the presence of 'Rendezvous' in their pedigrees, in addition to unidentified seedling resistance genes. 'Rendezvous' was selected for resistance to eye spot disease and stripe rust from VPM1 and inherited gene *Lr37* from *Triticum ventricosum* (McIntosh et al. 1995). Gene *Lr37* confers mainly adult plant resistance and is difficult to detect in seedling tests. Adult plant tests of 'Hussar', 'Kris', 'Lynx', 'Marshal' and 'Brigadier' could confirm the presence or absence of *Lr37*. Singh et al. (2001) postulated *Lr37* in 'Hussar', 'Lynx' and 'Brigadier'. Winzeler et al. (2000) reported that two cultivars that seemed to carry *Lr37* singly, provided low seedling resistance and full adult plant resistance in western Europe in 1996–1999. Differential tester lines with *Lr37* have shown variable levels of moderate susceptibility to resistance to leaf rust in field trials in Romania, Hungary, Czech Republic, Great Britain and Poland 1998–1999. Virulence to *Lr37* varied between locations although the gene was identified as the most effective of the resistance genes currently used in European wheat cultivars (Mesterhazy et al. 2000). In Sweden, annual disease surveys showed 2% diseased leaf area on 'Hussar' 1993–1997 (Larsson et al. 1997) and 4% on 'Kris' and 'Marshal' 1995–1999 (Larsson et al. 2000). If these cultivars carry *Lr37*, it

appears that virulence exists in Northern Europe to *Lr37* in these cultivars. 'Brigadier' had 0% leaf rust during 1993–1997 (Larsson et al. 1997). It is probable that this cultivar has additional *Lr* resistance genes and/or additive minor genes that are effective in Sweden.

The cultivar 'Kosack' displayed resistance to four pathotypes used in this study but the results did not correspond to any known *Lr* genes in the tester lines. According to the pedigree, the line Mironovskaya 808 contains *Lr3a* (McIntosh et al. 1995) but this does not appear to have been transferred to 'Kosack'. The source of the resistance remains unknown. The unidentified resistance in the Finnish cultivar 'Vakka' is the likely origin of the resistance pattern displayed by 'Aura', 'Otso', 'Pitko' and 'Urho' that contain 'Vakka' directly or indirectly in their pedigrees. The Finnish cultivars 'Laari' and 'Luja' also displayed infection types similar to 'Vakka' although this cultivar is not included in their pedigrees and the source of resistance is unknown. The resistance in 'Anniina' to pathotype CBJ/QL was most likely inherited from 'Polkka'. The source of resistance in 'Polkka', 'Runar' and many other cultivars could not be deduced from their pedigrees. In Sweden, annual disease surveys detected 11% leaf rust on 'Kosack' 1995–1999 (Larsson et al. 2000); 4% on 'Ballad' 1996–2000 (Larsson et al. 2001); 4% on 'Dirigent' and 5% on 'Hurtig' 1998–2002 (Larsson et al. 2002); 9% on 'Flair' 1995–1999 (Larsson et al. 2000); 8% on 'Rental' 1985–1994 (Larsson et al. 1994); 9% on 'Sport' and 7% on 'Tryggve' 1983–1992 (Carlsson et al. 1992); 16% on 'Kadett' 1985–1991 (Bengtsson et al. 1991); 10% on 'Kartesch' 1998–2002 (Larsson et al. 2003); 10% on 'Stava' 1992–1996 (Larsson and Magnét 1996) and 11% on 'Triso' 1998–2002 (Larsson et al. 2003). Virulence seems to exist to the unidentified seedling resistance genes in these cultivars though some genes may be more effective than others, singly or in combination with adult plant or additive minor genes. It is difficult to estimate the virulence on genes that only occur in combinations and further field studies including cultivars or lines with single *Lr* genes are needed to assess the effectiveness of such genes in the present material.

In the material grown in Northern Europe, a total of 9 cultivars were found without seedling resistance to the twelve leaf rust pathotypes used in the study. Winzeler et al. (2000) found that 55% of European wheat cultivars had adult plant resistance, contributed to by quantitative trait loci (QTL) and/or *Lr34* that enhances resistance. In Sweden, diseased leaf area was 3% on 'Urban' 1993–1997 (Larsson et al. 1997); 8% on 'Virke' 1998–2002 (Larsson et al. 2003); 2% on 'Thasos' 1992–1996 (Larsson and Magnét 1996) and 6% on 'Dacke' 1995–1999 (Larsson et al. 2000). Although a completely susceptible tester line was not included in the annual disease surveys, the percentage leaf rust on these cultivars is low compared to for example 16% on 'Drabant' with *Lr14a* (Carlsson et al. 1992). Thus, these cultivars with no postulated *Lr* seedling resistance genes may have additional adult plant resistance or additive minor genes that contribute to low disease pressure in the field and may be interesting for further analysis and use in breeding for leaf rust resistance.

The results of the present study and annual disease surveys in Sweden and Norway indicate that virulence exists to most of the known and unidentified seedling *Lr* genes in the cultivars grown in Northern Europe 1992–2002. Variation in virulence is determined by the genotypes of the pathoypes that were originally introduced to a region; over-wintering infected host plants; migration of urediniospores between regions; sexual recombination, mutation and selection pressure by host resistance genes in the region (Kolmer and Liu 2000). The viability of leaf rust urediniospores has been reported to be 1–5% after 24 h exposure to temperatures of -4 to -6°C (Eversmeyer and Kramer 1995). Although information on the virulence and genetic diversity of *Puccinia triticina* populations in northern Europe is at present limited, the pathogen is likely restricted by sub-freezing winter temperatures and the absence of the principal alternate host, *Thalictrum speciosissimum* (Anikster et al. 1997). The epidemiology of leaf rust populations in northern Europe would largely be influenced by migrating spores from neighboring areas and the presence and distribution of resistance genes in the wheat host.

Comparative studies of resistance genes in the wheat host and pathogenicity surveys have illustrated the effects of host selection pressure on the *Puccinia triticina* pathogen population. In regions of Canada, virulence frequencies in leaf rust collections changed almost annually from 1987–1997 because of the introduction of *Lr* genes in winter wheat cultivars grown in the United States (Kolmer 1999). Pathotypes with virulence to *Lr17* increased in a region of Canada from 1996–1998 due to extensive cultivation of one susceptible wheat cultivar with *Lr17* in Kansas (Kolmer 2001). In the US and Canada, pathotypes with virulence to *Lr16* declined in the late 1980s following decreased cultivation of winter wheat with this gene (Kolmer 1999). In Israel, where cultivars with *Lr26* are absent, pathotypes with virulence to *Lr26* have increased due to annual migration of spores from other regions (Kozman et al. 2004). However,

studies on leaf rust in former Czechoslovakia since the 1960s concluded that changes of virulence in *Puccinia triticina* could only partially be ascribed to changes of resistance genes in wheat cultivars (Bartoš et al. 1996).

The results of the present investigation show that several known and unidentified leaf rust seedling resistance genes have been deployed in Sweden, Norway, Finland and Denmark during 1992–2002 that could have contributed toward host selection on the leaf rust pathogen in the field. The breakdown of the resistance genes present in the material grown in Northern Europe was most likely influenced by the cultivation of a few cultivars on extensive areas. However, information on quantities certified seed was only available as total figures for countries per year and disease severity was available as average percentage diseased leaf area for several years. Area under cultivation and disease severity of a certain cultivar may vary with region and year and from the present information it is thus not possible to conclude how *Lr* genes in wheat hosts may have affected disease severity patterns in Northern Europe. Additional data on disease severity, virulence surveys and adult plant tests in the region are needed to provide evidence of interactions between the *Puccinia triticina* pathogen and *Lr* genes present in the wheat host.

Pyramiding genes, i.e. accumulating several effective resistance genes in the same cultivar, has been suggested as a method to achieve more durable resistance against pathogens with low genetic diversity, high gene flow and an asexual mating system (Mesterházy et al. 2000; McDonald and Linde 2002). The combination of several effective (undefeated) resistance genes into a single cultivar should extend the period of resistance since mutations at several avirulence loci would be required to produce a new virulent pathotype (Pink 2002). Molecular markers that could facilitate gene pyramiding have been developed for several effective resistance genes, including *Lr9*, *Lr19*, *Lr24*, *Lr25*, *Lr35*, *Lr37* and *Lr52* (Helguera et al. 2003; Blaszczyk et al. 2004; Hiebert et al. 2005). Slow-rusting or partial resistance has been reported to be a more durable type of resistance than single seedling resistance genes (Singh et al. 2001). Cultivars with a combination of the adult plant resistance gene *Lr34* and 3–4 additive partially effective genes have been shown to confer high levels of nonspecific resistance in many areas of the world (Singh et al. 2000; Navabi et al. 2005). Quantitative trait loci (QTL) for partial resistance to leaf rust have been identified in several wheat genotypes (Messmer et al. 2000; Xu et al. 2005) that could be used to develop markers necessary for breeding programs. Future host selection pressure on the pathogen could be further decreased by rotating genes through time and space by mixtures or regional deployment of cultivars with different effective resistance genes (McDonald and Linde 2002; Pink 2002). The knowledge of presence of leaf rust seedling resistance genes facilitates future studies and use of adult plant resistance and additive minor genes in these cultivars. A few cultivars have dominated the market and annual disease surveys have shown that many of the most commonly grown cultivars in Northern Europe during 1992–2002 are susceptible to leaf rust (Larsson et al. 1994–2003; Abrahamsen et al. 2003). It is thus important to continue breeding for leaf rust resistance and monitor the pathogen as part of a management control strategy.

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II

Powdery mildew resistance in Nordic bread wheat cultivars and landraces during a century of breeding

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Abstract

The occurrence and distribution of seedling resistance genes and the presence of adult plant resistance to powdery mildew, was investigated in a collection of 155 Nordic bread wheat landraces and cultivars by inoculation with 11 powdery mildew isolates. Eighty-nine accessions were susceptible in the seedling stage, while 66 accessions showed some resistance. Comparisons of response patterns allowed postulation of combinations of genes *Pm1a*, *Pm2*, *Pm4b*, *Pm5*, *Pm6*, *Pm8* and *Pm9* in 21 lines. Seedling resistance was three times more frequent in spring wheat than in winter wheat. The most commonly postulated genes were *Pm1a+Pm2+Pm9* in Sweden, *Pm5* in Denmark and Norway, and *Pm4b* in Finland. Forty-five accessions were postulated to carry only unidentified genes or a combination of identified and unidentified genes that could not be resolved by the 11 isolates. Complete resistance to all 11 isolates was present in 18 cultivars. Adult plant resistance in the greenhouse was assessed for 109 accessions after infection with a mixture of races. In all, 92% of the accessions developed less than 3-5% pathogen coverage on infected leaves while nine lines showed 10-15% infected leaf surface. The characterization of powdery mildew resistance in Scandinavian wheat germplasm will facilitate the combination of resistance genes in plant breeding programmes to promote durability of resistance and disease management.

Introduction

Powdery mildew (*Pm*) of bread wheat (*Triticum aestivum* L.) is caused by the biotrophic parasitic fungus *Blumeria graminis* (DC.) E. O. Speer f.sp. *tritici* that has an economic impact on wheat production in maritime or semicontinental climates (Bennett, 1984) such as the southern and coastal areas of Northern Europe. Powdery mildew infection can cause 45% yield reduction (Johnson *et al.*, 1979) and downgrades grain quality through effects on flour protein (Johnson *et al.*, 1979; Everts *et al.*, 2001). Although fungicides controlling powdery mildew are available, host resistance represents a more effective, economical, sustainable and environmentally safe method of crop protection (Bennett, 1984; Hsam & Zeller, 2002) and the evolution of complete or partial resistance to major fungicides emphasizes an increasing need to rely on varietal resistance (Wolfe, 1984).

To date, 53 powdery mildew (*Pm*) genes and alleles for resistance have been identified at 34 loci in wheat; these generally confer resistance against specific race(s) of the pathogen (Hsam & Zeller, 2002; McIntosh *et al.*,. 2003; Huang & Röder, 2004; McIntosh *et al.*, 2006). The reliance on single or limited combinations of *Pm* genes typically results in a rapid build-up of matching virulence genes, and the subsequent sudden breakdown of cultivar resistance (Wolfe, 1984). An understanding of the identity and the frequency of resistance genes/alleles in breeding germplasm, and of the spectrum of avirulence and virulence in pathogen populations, is a requirement for the methodical substitution and/or supplementation of genes that have lost their effectiveness. It has been argued that the assessment of the potential of new *Pm* genes could facilitate the combination of genes for management control strategies that maximize the durability of resistance (Hsam & Zeller, 2002; Wang *et al.*, 2005).

The identity of known *Pm* genes in a wheat genotype can be postulated according to the gene-for-gene hypothesis by a successive elimination procedure. Infection types displayed on host lines carrying known genes for resistance are compared to those of the lines in question after infection by a range of pathogen isolates with different avirulence and virulence genes (Flor, 1955; Powers & Sando, 1957; Vallavielle-Pope *et al.*, 1990).

The objectives of the present study was to investigate the occurrence and distribution of seedling resistance to 11 European powdery mildew isolates, adult plant resistance (APR) to a mixture of isolates, and postulate the identity of some of the *Pm* genes responsible for the powdery mildew resistance in bread wheat landraces and cultivars grown in Sweden, Norway, Denmark and Finland from the late 19th century until early 21th century.

Materials and Methods

Plant material

The material constituted 26 landraces, one breeding line (J-03, NGB-2146) and 128 bread wheat cultivars of spring or winter type, bred and grown in Sweden, Norway, Denmark and Finland during the 20th century. The material was chosen

on the basis of pedigree information to represent landraces and cultivars that have been used in the Nordic countries during the last century and comprised 54% of the Nordic cultivars and landraces available from The Nordic Gene Bank (NGB, 2006). Seed was obtained from The Nordic Gene Bank and from the plant breeding company Svalöf-Weibull AB (Table 1). Pedigree and year of release information was provided by The Nordic Gene Bank, Svalöf-Weibull AB, or obtained from the Wheat Pedigree and Identified Alleles of Genes On Line (WPIAG, http://genbank.vurv.cz/wheat/pedigree/) database. Near-isogenic lines of cultivar 'Chancellor' and lines carrying identified race-specific genes for resistance to powdery mildew were used as differential tester lines (Table 1). The cultivar 'Kanzler' was used as a universal susceptible control.

Pathogen material

The 11 differential isolates of *Blumeria graminis* (Table 2) were collected from different areas in Europe and selected from single-spore progenies (Felsenstein *et al.*, 1991). Isolate nomenclature follows Weihenstephan accession numbers and the isolates are maintained at the Lehrstuhl für Pflanzenzüchtung, Freising-Weihenstephan, Germany. Isolate JIW12, avirulent against *Pm8*, was used to confirm pathotype tests for this gene and was provided by Dr. Rebecca Wyand, John Innes Centre, Norwich, UK.

Inoculation, disease assessment and gene postulation

Disease tests were performed on detached leaf segments maintained in Petri dishes on 6g/l agar, 35 mg/l benzimidazole. Inoculation methods and incubation conditions followed Lutz and co-workers (1995). Each wheat accession was tested using 2 to 4 replications, each consisting of 8 to 10 individual plants. Disease response patterns were recorded ten days after inoculation using a quantitative scale of 0 to 10, where 0 = no visible disease symptoms and 10 = 100% leaf area covered with sporulating colonies (Lutz *et al.*, 1995). Three major classes of host reactions were distinguished: r = resistant (0-20% infection level of 'Kanzler'), i = intermediate (20-50% infection), s = susceptible (> 50% infection). Where accessions were heterogeneous for response, they were recorded for example as 'r,i' with the most common class preceding.

The identity of individual *Pm* genes in a given accession was postulated by first identifying the avirulence/virulence formulas of the pathogen isolates on the tester lines and subsequently determining possible *Pm* genes in the wheat accessions according to the gene-for-gene hypothesis (Flor, 1955; Powers & Sando, 1957; Vallavielle-Pope *et al.*, 1990). The comparison of compatible (susceptible response) and incompatible (resistant response) interactions between the host and pathogen was used to eliminate the hypothesis of presence of a resistance gene. The hypothesis that a certain resistance gene was present in a host was further supported by using pedigree information when available.

Adult plant resistance

In order to screen the material for adult plant resistance to powdery mildew, three seedlings per accession and replication were planted in a randomized greenhouse experiment and exposed to a naturally occurring isolate mixture. Three seedlings of each replication from the near-isogenic lines of cultivar 'Chancellor', tester lines, and susceptible check cultivar 'Kanzler' were used as differential tester lines and controls. Adult plant response to infection was assessed under greenhouse conditions for 109 entries of the accession set (Table 3). The percentage infected leaf area on the penultimate leaf (F-1 leaf) was scored two weeks after flowering, and thereafter every week until maturity.

PCR-based detection for the presence of rye chromosome arm 1RS

A molecular marker was used to detect the presence of the wheat-rye translocation T1BL.1RS, carrying *Pm8*. DNA was extracted from a bulk of 30 seeds per accession using the Qiagen 96DNEasy Plant Kit. A pair of primers directed at the spacer region of *Nor-R1* was used to amplify template DNA, as described by Koebner (1995). DNA from cultivar 'Tarso' (known to contain the T1BL.1RS translocation), triticale line 'Sv876032' and wheat cultivar 'Holme' (lacking rye chromatin) were used as control DNA templates.

Results

Seedling resistance

The disease response patterns of the differential tester lines after inoculation are presented in Table 1. The susceptible and resistant reaction patterns corresponded largely to expected results from literature (Hsam & Zeller, 2002) except for isolate no. 17 that was virulent against *Pm8*. Genes *Pm12*, *Pm16*, *Pm20*, *Pm21* and *Pm24* could not be identified, as all isolates were avirulent against these genes. In all, 85 accessions were susceptible to all of the 11 isolates. 'Extra Squarehead', 'Hunsballe R', 'Stand Tystofte' and 'Storaks Abed' showed susceptible and intermediate responses (Table 2). These 89 lines (57%) were postulated not to carry any of the assayed race-specific seedling resistance genes. The presence of the designated genes *Pm1a*, *Pm2*, *Pm4b*, *Pm5*, *Pm6*, *Pm8* and *Pm9*, was postulated singly or in combination in 13% of the material. Unidentified *Pm* genes were found to be present in 35% of the material, often in combination with known genes. The genes *Pm5* and *Pm4b* were postulated in 4% each of the material, *Pm1a+9*, *Pm2* and *Pm6* in 3% each of the material, while *Pm8* occurred in 1% of the accessions (Tables 2 and 4). For 29% of the material, including 18 lines that were resistant to all the 11 isolates, the *Pm* gene content could not be identified. The 66 accessions (42%) that were resistant to one or more of the isolates were grouped into six Resistance Groups (RGs) according to the dominating combination of response reactions (Table 3).

The reaction patterns in RG-1 (Table 3) corresponded to that of tester line 'Armada' carrying *Pm4b* (Table 1). 'Runar', 'Reno' and 'Manu' most likely inherited *Pm4b* from the German ELS breeding lines known to possess this gene

(Wolfe, 1967; The *et al.*, 1979; Paderina *et al.*, 1995). 'Kosack' indirectly inherited *Pm4b* from ELS through 'Starke M'. The source of the resistance in cultivar 'Harnesk' remains unkown since the pedigree could not be resolved.

The response patterns in RG-2 (Table 3) corresponded to that of tester line 'Hope' carrying *Pm5a*, TP114/2*Starke with *Pm6* and additional tester lines (Table 1). The gene *Pm5a* in 'Rida' was most likely inherited from 'Redcoat' (Limpert et al,. 1994), and later transferred to 'Bjørke'. 'Tjelvar' may have inherited *Pm6* from 'Sture-MB' while the source of *Pm4b* in 'Tjalve' could be 'Reno'. The response pattern of 'Sleipner' corresponded to *Pm2* and *Pm6*, probably derived from C.I. 12633 via 'Maris Huntsman' (McIntosh *et al.*, 1988; Hovmøller, 1989). The line C.I. 12633 is derived from a hybrid between *T. timopheevi* (Zhuk.) Zhuk and bread wheat (Allard & Shands, 1954) and has been shown to possess these two *Pm* genes (Nyquist, 1963). The presence of *Pm5* in 'Kalle', 'Kraka', 'Terra', 'Tjelvar' and 'Nana' could not be predicted from their pedigrees. Hovmøller (1989), using a set of Danish powdery mildew isolates, postulated that 'Kraka' carries both *Pm5* and *Pm6*, and 'Holger' carries *Pm6*. The cultivar 'Holger' has been used as a differential cultivar for *Pm6* in Denmark and the UK. In the present study, the response pattern of 'Holger' corresponded to *Pm6* and an unidentified gene. The contradictory result may be explained by heterogeneity in the seed material as The Nordic Gene Bank has two accessions of 'Holger', NGB-2435 donated by Weibullsholm PBI, and NGB-6398 donated by KVL (The Royal Veterinary and Agricultural University, Denmark), presumably the one used in the study of Hovmøller (1989). Heterogeneous (segregating) response reactions that could not be explained by the inoculation method, were observed for several cultivars e.g. 'Ballad', 'Rida' and 'Safir'. Unfortunately accession NGB-6398 was not included in the present study and therefore further tests using isolates clearly avirulent to *Pm5* and *Pm6* are needed to confirm the hypothesis and the postulations in RG-2.

The accessions in RG-3 all had heterogeneous pedigrees but similar response patterns and were therefore divided into subgroups according to pedigree. Group RG-3a comprised cultivar 'Ring' and the descendents 'Troll', 'Pompe', 'Rang', and 'Saffran'. 'Ring' is known to carry *Pm1* and *Pm9* (McIntosh *et al.*, 1992), also found in the present study. 'Rang' was synthesized by adding a gene for mildew resistance to 'Ring' through repeated backcrosses (Lundin, 1970). The additional *Pm* genes in 'Saffran' may have been inherited from 'Rang' and C.I. 12633. Group RG-3b consisted of Norwegian cultivars with the breeding line J-03 (a mildew resistant breeding line selected from a Norwegian landrace) in the pedigree. The response patterns in 'Fram I' and 'Fram II' appear to originate from the parents J-03 and Mo-07, a *Triticum compactum* (Host) MacKey breeding line (Vik, 1937). The resistance in 'Fram II' was transferred to 'Norrøna' and 'Rollo'. Resistance group RG-3c contained the cultivars 'Fylgia I', 'Fylgia II', 'Brons' and 'Trym', that are descended from the fully susceptible 'Extra Kolben I' (or 'Extra Kolben II') and the Australian cultivar 'Aurore'. The latter was not available to the present study, but could be the source of these response patterns. Group RG-3d comprised Nordic landraces (or selections from landraces) with similar response patterns where the *Pm* gene content could not be predicted from or supported by any pedigree information. Landraces with segregating responses and cultivars that

could not be placed in RG-3 constituted RG-6. The response pattern in RG-4 could not be explained by the pedigrees.

The eighteen cultivars in RG-5 were resistant to all isolates and on the basis of pedigree information were presumed to carry both unidentified and identified *Pm* genes/alleles. The exact combination could not be resolved using the 11 isolates in the present study (Table 3). Cultivar 'Sappo', reported to possess *Pm1+2+4b+9* (Heun & Fischbeck, 1987), features in the pedigrees of 'Sunnan', 'Timmo' and 'William'. Hovmøller (1989) postulated the presence of *Pm1+2+4b+6* in 'Timmo' and *Pm1+4b+6* in 'William'. 'Drabant' may have inherited *Pm1+9* from 'Ring', along with *Pm2+6* from line C.I. 12633. 'Canon', 'Curry' and 'Dragon' may contain *Pm2*, *Pm3d*, *Pm4b*, and/or *Pm6* derived from 'Kadett' (Wiik, 1991). The breeding line M12 in the pedigree of 'Vinjett', was selected from a cross with *T. aestivum* L. var. *pseudomeridionale* (Flaksb.) Mansf., while M14 and M15 in 'Revelj', and 'Avle' are breeding lines incorporating resistance from Ethiopian landraces (Herrera-Foessel, 2001).

A summary of the results for the accessions grouped by country is given in Table 4. Seedling resistance was more frequent in spring wheat (69%) than in winter wheat (21%). This may reflect either a higher sustained disease pressure on the spring wheat populations and/or a faster cultivar turnover from spring wheat breeding programmes, which would encourage a more rapid incorporation of resistance genes. However, the frequencies for the non-Swedish material may be biased due to the disproportionate number of spring and winter wheat accessions. Comparisons of results between countries are difficult as the plant material represents a selection of landraces and cultivars, reflecting a fraction of the possible variation present in Nordic breeding material, and several cultivars have been grown in more than one country.

The results for Sweden were compared over time to identify whether breeding strategy has had any impact on powdery mildew resistance (Table 2). In spring wheat, the five landraces included in the study and five cultivars (31%) released between 1892 and 1955 carried unidentified resistance. Cultivar 'Ring' (*Pm1a+9*) released in 1957, appears to herald a change in breeding strategy as 22 (92%) of the subsequently released cultivars showed seedling resistance, including six cultivars with postulated genes. Seedling mildew resistance was less common in winter wheat than spring wheat during all time periods (Table 2). In winter wheat, four of seven landraces had unidentified powdery mildew resistance, while 45 cultivars released from 1900 to 1981 were susceptible, indicating a decline in resistance during this period. The cultivar 'Holger' (*Pm6+u*) , released in 1981, seems to mark a period of increased focus on seedling resistance where eight of ten released cultivars were resistant. In summary, in Sweden there appears to have been a decline in seedling resistance from landraces until the year 1957 for spring wheat and the year 1981 for winter wheat, as measured using the 11 isolates in the present study.

Adult plant resistance

The tester lines and cultivar 'Kanzler' were completely susceptible (more than 50% infected leaf area) to the powdery mildew isolate mixture in the greenhouse.

The adult plant resistance scores for the 109 cultivars and landraces were consistent across time and therefore means are reported in Table 2. In all, 92% of the accessions developed less than 3-5% infected leaf surface at the adult plant stage thus indicating the widespread presence of adult plant resistance genes, irrespective of growth habit (spring/winter) or country of origin (Table 4). The majority (59 accessions) of these were completely susceptible in the seedling stage. 'Warmland lantvete', 'Diamant I', 'Diamant II', 'Ergo', 'Jarl', 'Progress' and 'Sol IV' developed the highest level of infection (15% leaf coverage), followed by 'Runar' and 'Vårpärl' with 10% infected leaf surface.

Presence of rye chromosome arm 1RS

The PCR amplification profile obtained from 'Tarso' and triticale 'Sv876032' DNA templates consisted of three amplicons of approximately 400, 600 and 700 bp. Cultivar 'Holme', which lacks rye chromatin, did not amplify the 700 bp product. PCR amplification profiles of 154 accessions were generated and compared to those of 'Tarso', 'Sv876032' and 'Holme'. The profiles of 'Tjelvar', 'Sleipner' and 'Saffran' were identical to that of 'Tarso' and 'Sv876032', showing that these accessions are 1RS carriers. The presence of the T1BL.1RS translocation was confirmed by C-badning. The 1RS rye segment, which also contains resistance genes *Lr26* (leaf rust), *Sr31* (stem rust), and *Yr9* (stripe rust) and has a positive effect on yield, occurred spontaneously in wheat/rye hybrid derivatives produced in Germany during the 1930s (Rabinovich, 1998). Inoculation of 'Saffran', 'Sleipner' and 'Tjelvar' with isolate JIW12 showed identical responses as the tester line 'Disponent', indicating that these cultivars share at least one gene (i.e. *Pm8*, present on 1RS) with the tester. Cultivar 'Tjelvar' has been reported to possess a dominant suppressor for *Pm8* (Hanusova *et al.*, 1996) although this does not seem to be present in The Nordic Gene Bank accession of this particular cultivar.

Discussion

A comparison of response patterns derived from the interaction between Nordic bread wheat accessions and a collection of powdery mildew isolates demonstrated the presence of seven known *Pm* genes in addition to unknown mildew resistance. The unidentified genes could be known *Pm* genes, singly or in combination, that could not be postulated with the isolates used in the present study, or undesignated *Pm* genes or alleles of known *Pm* genes. Additional *Pm* isolates and parental accessions may have allowed some of these genes to be identified. Resistance genes for which avirulence did not occur in the isolates used in the study could also be present in any line. It is possible that some *Pm* genes were not expressed under the environmental conditions prevailing in the study, and thereby remained undetected.

The widespread occurrence of adult plant resistance in the material indicated that a breeding strategy incorporating field trials in areas with a climate conducive to powdery mildew, has maintained a high level of APR in most Nordic landraces and cultivars during the last century. The adult plant resistance may be due to race-specific *Pm* genes or quantitative partial resistance. The experiment was

conducted under greenhouse conditions instead of field conditions, as a natural heavy infection pressure was not present in the fields at the test site during that time. Further tests and field trials using pathogen isolates with known virulence and wheat lines known to possess good partial resistance are needed to assess the partial or race-specific resistance components and select suitable candidates for incorporation in breeding programmes.

In order to assess the impact of resistance breeding it would be desirable first to establish what virulence was present in the powdery mildew pathogen populations in different areas and during different time periods. Isolates with the same virulence spectra could then be selected and used for a comparative study. Comprehensive information on the virulence spectra in the Nordic countries is not available, although partial inferences can be made for some areas and time periods. The following sections present a discussion of the results and a brief account of the history of powdery mildew resistance breeding in the individual Nordic countries.

Sweden

The presence of *Pm1a*, *Pm2*, *Pm4b*, *Pm5*, *Pm6*, *Pm8* and *Pm9*, alone or in combinations, was identified in 12 of the 107 Swedish landraces and cultivars (Tables 2 and 4). Thirty-two cultivars possessed unidentified seedling resistance genes. Wheat breeding in Sweden started in the late 19th century and aimed to improve yield through purifying selection from landraces, and later crosses and hybridization. Although powdery mildew was a disease of only minor importance during the early 20th century (Lundin, 1997), 41% of landraces and cultivars from before 1920 had some seedling resistance and 95% had 3-5% mildew infection in adult plant tests. Together, these probably contributed to a low incidence of the disease in the field, although the virulence spectra of the mildew pathotypes at that time are unknown.

Powdery mildew gradually increased in importance after the 1920s due to the use of nitrogen fertilizers. However, cultivars were reported to carry high levels of field resistance (Lundin, 1997). In Sweden, virulence surveys and breeding work to identify and incorporate race-specific genes into cultivars were initiated in 1960. Wolfe & Schwarzbach (1975) reanalyzed the virulence data from Leijerstam (1965) and found that among more than 700 isolates collected in the Nordic countries, 14% carried virulence to *Pm1a*, 20% to *Pm3b*, 3% to *Pm2* and 2% to *Pm5a* (only a limited selection of differentials were available at that time). The most common combination was virulence to *Pm1a+3b* (29%) but complex combinations of *Pm1a+2+3b+5a* (1%) also existed.

In order to assure a selection of the most effective sources of resistance, the research group in Sweden joined the screening programs started at Beltsville, USA and Weihenstephan, Germany. Many of the most resistant lines were thus introduced into the breeding program before race identification was started in Sweden (Leijerstam, 1972a). During the 1960s in Sweden, around 50 wheat accessions in about 10 000 accessions of different ploidy levels were found to be resistant against 39 Nordic races in mildew screening tests. Several of the resistant lines were *T. timopheevi* derivates e.g. C. I. 12633. The resistance derived from

line C.I. 12633 (*Pm2+6*) and 'Khapli' (*Pm4a*) were highly effective in the greenhouse and field from the 1960s to 1970s (Leijerstam, 1965; 1972b). Lundin (1970) notes that 'Ring' (*Pm1a+9*) was susceptible to mildew during the trial period while 'Rang' was almost completely resistant. In the present study, the accessions released after 'Ring' in year 1957 were resistant to an increasing number of pathogen isolates (Table 3). This underlines the impact of a sustained breeding program using race-specific seedling resistance genes against powdery mildew. However, seedling resistance may not ensure field resistance. During the 1980s, powdery mildew spore trapping in southern Sweden showed that the resistance in 'Dragon' (u) was effective, while resistance in cultivars 'Folke' (u), 'Kadett' (*Pm4b+Pm3d+u1+u2*), 'Longbow' (*Pm2*) and 'Sleipner' (*Pm2+6+8*) were ineffective (Wiik, 1991). Thus there appears to have been an evolution in the virulence profile of powdery mildew in Sweden between the 1960s and the 1980s.

In Sweden, a small number of cultivars has dominated the market e.g. 'Eroica I' (*none*) during the 1940s, 'Odin' (*none*) in the period 1950 to 1965, (Donner & Mesdag, 2000), 'Starke' (*none*) during the 1960s (Svensson, 1997), and 'Kosack' (*Pm4b*) during the 1980 to 1990s (Nilsson & Andersson, 1998; Andersson *et al.*, 2002). It seems unlikely that these cultivars could have caused the breakdown of *Pm2+6*. However, several spring wheat cultivars (e.g. 'Sappo', 'Drabant', 'Sunnan', 'Dragon' and 'Canon') that probably carry *Pm2+6* were released from 1970 to 1990 that could have contributed to a change in virulence. The cultivars 'Kosack', 'Stava', 'Dacke', 'Vinjett' and 'Sport' were rated as only marginally susceptible to powdery mildew in recent trials (Gustafsson, 2004). The resistance in 'Kosack' (*Pm 4b*) has largely remained effective since the early 1980s with an average of 2% infected leaf area while 'Kadett' (*Pm4b*) has been more susceptible, with 19% infected leaf area (Bengtsson *et al.*, 1991). Hence, it is likely that the effective resistance of 'Kosack' is due to a combination of several unidentified genes, especially adult plant and/or partial resistance genes. Additional data on area of cultivation, disease severity, virulence surveys and adult plant tests in different regions will be needed to clarify the interactions between the pathogen and the *Pm* genes present in this host.

Norway

Pm4b, Pm5, and unidentified genes were found in 16 of 18 landraces and cultivars from Norway. Ten of eleven tested accessions displayed 3-5% mildew infection in adult plant tests. The plant breeding work in Norway started early in the 20th century and focused on improving earliness, winter hardiness and yield (Wexelsen, 1965). Powdery mildew caused serious yield losses during the 1920s and 1930s in Norway. Breeding for mildew resistance began around 1920 with field tests and crosses. Vik (1937) found only one landrace selection (J-03, NGB-2146) and an unnamed Japanese variety that were resistant to mildew in field trials of thousands of Norwegian and foreign accessions. The cultivars 'Ås', 'Børsum', 'Frøya', Extra Kolben II', 'Rubin'. 'Diamant', 'Fylgia', 'Aurore' and 'Marquis' were reported to be susceptible. The breeding line J-03 was successfully used in breeding for mildew resistance and led to the cultivars 'Fram II', 'Snøgg II' and 'Ås II' (Vik, 1937; Wexelsen, 1965; Donner & Mesdag, 2000). However,

conclusions from the results of the present study regarding the impact of plant breeding on resistance are limited due to the small number of accessions assayed.

The pathogen virulence composition is determined both by migration of inoculum from neighbouring regions and by sexual recombination and/or mutation within the country (Skinnes, 2002). In the southern region around Oslo, powdery mildew often dies during the winter as a result of a lack of snow cover and new inoculum is brought by prevailing winds from Sweden and Denmark. In the northern region around Lake Mjøsa, there is more snow cover and the powdery mildew population can survive in the form of sexual spores. New virulence typically first appears in this region and spreads to the south (Skinnes, 2002).

The cultivars 'Lars', 'Kosack' (*Pm4b*), 'Bjørke' (*Pm5*), 'Mjölner' (*none*), 'Rudolf', 'Portal', 'Kalle' and 'Folke' have been the most widely cultivated winter wheats over the period 1995 to 2003. Cultivars 'Zebra' (*u*), 'Vinjett' (*u*), 'Avle' (*u*), 'Tjalve' *(Pm4b+u*), 'Bastian' and 'Polkka' have dominated the spring wheat market (Åssveen *et al*., 2003). Virulence surveys have shown that the pathogen population around Lake Mjøsa includes virulence to *Pm1*, *Pm2*, *Pm3a-c*, *Pm4a-b*, *Pm5* and other genes in 'Sleipner', 'Avle', 'Dragon', 'Bastian', 'Polkka' and 'Brakar'. In the area around Oslo, the race composition includes virulence to *Pm2*, *Pm4a-b*, *Pm5*, *Pm6* and resistance genes in 'Sleipner' (Skinnes, 2002). In annual disease severity surveys (1999 to 2002), 'Avle' and 'Bastian' were susceptible with 13-17% diseased leaf area while 'Vinjett', 'Zebra' and 'Bjarne' showed 1-5% diseased leaf area. In 2002, 'Bjørke' had 11%, 'Portal' 15%, 'Lars' 12%, 'Kosack 'and 'Mjölner' 8% diseased leaf area. Between 1993 and 2002, the diseased leaf area increased on 'Mjölner' (14%), 'Bjørke' (20%) and 'Portal' (25%) (Åssveen *et al*., 2003). Thus it seems that most known and unidentified resistance genes in wheat cultivars grown during the last decade are no longer effective in Norway.

Denmark

The genes *Pm5*, *Pm6* and unidentified genes were present in five of the 16 accessions assayed from Denmark. The five lines included in the APR test showed 3-5% mildew infection. Identified seedling *Pm* genes were found in cultivars released after 1970, indicating the impact of breeding for race-specific resistance. Most Danish wheats are of winter type, and there is rapid cultivar turnover (Hovmøller, 2001). Therefore the accessions included in the present study is not representative of the actual variation present in the field.

Plant breeding in Denmark was started during the late 19th century by the formation of various associations (Frandsen, 1965). No information was found on the powdery mildew situation in Denmark during the earlier half of the 20th century. Virulence frequencies in barley powdery mildew changed suddenly in the 1970s, and this led to the establishment of virulence surveys in both barley and wheat (Hovmøller, 1987). Virulence frequencies for powdery mildew in wheat 1985 to 1986 were 100% against *Pm5*, 80-90% against *Pm8* and 40-90% against *Pm2*. The combination *Pm4b* + *Pm6* gave a good level of protection. 'Kosack' (*Pm4b+u*), 'Sleipner' (*Pm2+6+8*) and 'Holger' (*Pm6+u*) were the most resistant cultivars. There was also some virulence against *Pm1*, *Pm4a*, *Pm3a*, *Pm3b* and

Pm3c despite the fact that cultivars with these genes have not been widely grown in Denmark (Hovmøller, 1987).

Inoculation experiments using 12 Danish *Pm* isolates on 31 cultivars grown in Denmark and Northwestern Europe in 1988, demonstrated the presence of *Pm1*, *Pm2*, *Pm3d*, *Pm4b*, *Pm5*, *Pm6*, *Pm8* and *Ml-i* (Hovmøller 1989). 'Vitus Sejet', carrying *Ml-k* (*Pm3d*) and an unknown *Pm* gene (Hovmøller, 1989), was the most widely grown winter wheat cultivar in Denmark during the 1980s (Donner & Mesdag, 2000). The winter wheat 'Ritmo' (*Pm2+4+6*; Anonymous, 1997) was common during the 1990s while 'Vinjett' (*u*) and 'Leguan' were the most widely grown spring wheat cultivars from 1999 to 2003 (Pedersen, 2003). According to disease surveys in 2003, 'Ritmo' had 5% diseased leaf area, 'Terra' (*Pm5+u*) 2% and 'Vinjett' 0.1% (Pedersen, 2003). Since 'Kosack', 'Sleipner' and 'Holger' were not included in the disease survey reported by Pedersen (2003), it is unknown whether the resistance of these cultivars has remained effective. The present study has identified several cultivars with *Pm4b* that could be used together with other effective *Pm* genes and adult plant resistance in breeding programmes.

Finland

The gene *Pm4b* was found in one of the 13 Finnish wheats assayed, in agreement with previous studies (Peusha et al., 1996). The single line tested for APR showed 3-5% infection. Karjalainen (1987) reported that powdery mildew occurs in both spring and winter wheat every year, and causes significant yield losses about every third or fourth year. Plant breeding in Finland began around the 1900s and focused on improving yield and winter hardiness (Kivi, 1965). Wheat production during the 1930s was dominated by the Swedish spring wheat cultivar 'Diamant I' (Valle, 1948) which had no seedling resistance to the 11 powdery mildew isolates and somewhat higher percentage infected leaf area (15%) in the adult plant tests. Resistance breeding to control powdery mildew was initiated in the 1960s. Wheat cultivars during the period 1950 to 1975 did not carry any specific resistance genes except *Pm1* (Karjalainen, 1987). In the present study, the majority of landraces and cultivars were susceptible to all 11 *Pm* isolates, although it is possible that field selection has resulted in the accumulation of adult plant resistance to the prevalent pathotypes. A small scale virulence survey in the 1970s found 17 races in 302 isolates, including virulence to *Pm1a, Pm2, Pm3b, Pm4b, Pm5, Pm6* and *Pm8*. Virulence to *Pm5* was most common, while virulence to *Pm4b* was rare (Karjalainen, 1987). This relationship may have changed during the 1980s and 1990s as a result of increased use of cultivars such as 'Reno' and 'Runar' that carry *Pm4b* (Peusha et al., 1996).

Conclusions

The studies of Leijerstam (1962, 1965), Lundin (1970), Wolfe & Schwarzbach (1975) showed that virulence against *Pm1a*, *Pm2*, *Pm3b*, *Pm5a*, *Pm9* and unidentified genes has most likely existed in the Nordic countries from the 1960s onwards. It is striking that virulence to *Pm3b* appears to have been very common during the 1960s although this gene could not be postulated in any of the 155 landraces and cultivars in the present study (Table 2). This result was also noted by Hovmøller (1987) for *Pm3a-c* in Denmark, and indicates that inoculum may arrive from neighbouring areas, or that there is a low fitness penalty to retaining the corresponding virulence gene. Virulence to *Pm1a*, *Pm2*, *Pm3a-d*, *Pm4a-b*, *Pm5a*, *Pm6*, *Pm8*, and *Pm9* have been present during the 1980s and 1990s, perhaps as a result of increased cultivation of wheat material containing these genes (Karjalainen, 1987; Hovmøller, 1987; Bengtsson *et al.*, 1991; Wiik, 1991). Powdery mildew reproduces both sexually and asexually on an annual basis, and shows substantial genotype flow between regions (Wolfe, 1984; McDonald & Linde, 2002). Due to the high genetic variability of the pathogen, new virulent pathogen races can evolve to overcome individual race-specific genes in a short period of time (Wolfe, 1984; Zhu *et al.*, 2004).

Breeding strategies that have been proposed to extend the durability of resistance include the pyramiding of several effective resistance genes, the use of multilines with different resistance genes and breeding cultivars with partial (quantitative) resistance (Yu *et al.*, 2001; McDonald & Linde, 2002; Wang *et al.*, 2005). Molecular markers have been developed for several *Pm* genes and alleles that facilitate selection and pyramiding of effective resistance (Liu *et al.*, 2000; Liu *et al.*, 2002; Huang *et al.*, 2004; Huang & Röder, 2004). Although the presence of several effective resistance genes should result in more durable resistance (Pink, 2002), it can also lead to the evolution of multiple matching virulences if selection pressure on the pathogen is high (Groth, 1976). The breeding of cultivars with partial resistance has been suggested as a more sustainable strategy (Shaner, 1973; Bennett, 1984), and could also be combined with race-specific resistance (Wang *et al.*, 2005). The adult plant resistance in some of the lines in the present study could be useful in combination with effective race-specific *Pm* genes to achieve more durable resistance in wheat.

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Table 1. *Differential reactions of wheat cultivars/lines possessing known powdery mildew resistance genes after inoculation with 11 isolates of* Blumeria graminis *f. sp.* tritici[1]

Cultivar/line	**Isolate**											***Pm* gene**
	2	**5**	**6**	**9**	**10**	**12**	**13**	**14**	**15**	**16**	**17**	
Axminster/8*Cc[2]	r	s	r	i, s	r	s	s	s	r	s	s	*1a*
MocZlatka	r	r	r	r	r	i	r, i	r	r	s	s	*1b*
M1N	r	r	r	r	r	r	r	r	r	r	r	*1c*
Ulka/8*Cc	s	r	r	s	r	s	s	s	s	s	s	*2*
Asosan/8*Cc	r	s	r	r	r	s	r	r	s	r	r	*3a*
Chul/8*Cc	r	s	s	r	r	r	r	r	s	r	i, s	*3b*
Sonora/8*Cc	r	s	s	i	r	s	r	i, s	s	s	s	*3c*
Kolibri	s	s	s	r	s	r	s	r	r	s	r	*3d*
W150	s	i, s	i, s	i	r	i, s	r	r, s	s	s	s	*3e*
Mich.. Amb./8*Cc	r	s	s	i	r	s	r	i, s	s	s	s	*3f*
Khapli/8*Cc	s	r	s	r	i	r	s	s	i	s	i	*4a*
Armada	s	r	s	r	r	r	s	s	r	s	s	*4b*
Hope	i	s	s	s	r	s	s	r	s	s	s	*5a*
Ibis	s	i, r	s	s, i	r	s	s	r	s	s	s	*5b*
TP114/2*Starke[3]	s	r, i	r, i	r	r, i	s	r, i	r, i	r, i	i	s	*6*
Transec	i	i	r, i	s	s	i	s	s	s	s	s	*7*
Disponent	r	s	s	r	s	r	s	s	s	s	s	*8*
N14	s	s	i	r	s	s	s	s	i	s	s	*9*
6BS-6SS/6SL	r	r	r	r	r	r	r	r	r	r	r	*12*
3BL/3SS	s	s	i, r	s, i	s	i	i	i	r	s	i	*13*
BRG 3N	r	r	r	r	r	r	r	r	r	r	r	*16*
Amigo	i	i	i, s	i	i	i	r	s	i	r	r	*17*
XX 186	s	s	r	i	r	r	i	i	s	i	r	*19*
107-41 6RL	r	r	r	r	r	r	r	r	r	r	r	*20*
6AL/6VS	r	r	r	r	r	r	r	r	r	r	r	*21*
Virest	r	i	r	r	r	i	i, r	i	r	i, s	i, s	*22*
81-7241	i	r	s	r	r	r	s	i	i	s	i	*23*
Chiyacao	r	r	r	r	r	r	r	r	r	r	r	*24*
Pova	r	r	r	r	i	r, i	i, s	r	r, i	i, s	s	*29*
Normandie	r	r	r	r	r	s	s	s	r	s	s	*1+2+9*
Maris Huntsman	s	r	r	r	r	s	r, i	r	r	i	s	*2+6*

[1] r = resistant, s = susceptible, i = intermediate; segregating resistance responses are separated by a comma (,).

[2] Seven times backcrossed to ‘Chancellor’.

[3] Once backcrossed to ‘Starke’.

Table 2. *Genealogies of Scandinavian wheat cultivars and landraces and results of gene postulations after inoculation with 11 powdery mildew isolates and adult plant resistance (APR) tests after exposure to natural greenhouse powdery mildew mixture*

Year[1]	Accession name[2]	Acc no[3]	Pedigree[4]	Habit[5]	Area[6]	Seedling test Postulated *Pm* genes[7]	Adult test % mildew[8]
Sweden							
n/a	Dala	9708	Landrace	s	S	*u*	3-5%
n/a	Dalarna	6410	Landrace	s	S	*u*	3-5%
n/a	Halland	6409	Landrace	s	S	*u (seg)*	3-5%
n/a	Lantvete from Dalarna	6673	Landrace	s	S	*u*	3-5%
n/a	Lantvete from Halland	6674	Landrace	s	S	*u (seg)*	3-5%
n/a	Borstvete from Gotland	4494	Landrace	w	S	*none*	3-5%
n/a	Gammel svensk landhvete	8199	Landrace	w	S	*none*	3-5%
n/a	Hallandshvete	9057	Landrace	w	S	*none*	3-5%
n/a	Lantvete from Gotland	4496	Landrace	w	S	*u (seg)*	3-5%
n/a	Lantvete from Halland	6691	Landrace	w	S	*u*	3-5%
n/a	Lantvete from Uppsala	6692	Landrace	w	S	*u (seg)*	3-5%
n/a	Warmland lantvete	8198	Landrace	w	S	*u (seg)*	15%
1892	Kolben (Svalöfs Kolben)	6676	(s) from landrace with wide variation	s	S	*none*	3-5%
1900	Extra Squarehead	6694	(s) Leutenritzer Squarehead	w	S	*none*	*
1902	Bore	6695	(s) Squarehead wheat	w	S	*none*	3-5%
1910	Pudel	6698	(s) Shiriff wheat from England	w	S	*none*	3-5%
1910	Renodlat Sammetsvete	6699	(s) Purification of wheat from Ultuna, Sweden	w	S	*none*	3-5%
1911	Iduna	0001	(s) English Squarehead wheat	w	S	*none*	3-5%
1911	Sol	6700	(s) Landrace from Skåne, Sweden	w	S	*none*	3-5%
1916	Sol II	6701	Sol I/Extra Squarehead II	w	S	*none*	3-5%
1917	Thule II	6702	Pudel/Sammetsvete (landrace)	w	S	*none*	3-5%
1919	Extra Kolben	6677	Kolben/unnamed line	s	S	*none*	3-5%
1920	Vårpärl	6675	(s) Emma	s	S	*none*	10%
1921	Rubin	6678	Kolben/Dala	s	S	*none*	3-5%
1921	Rudolf Rubin	14118	WW 25449/Folke	w	S	*none*	3-5%
1923	Pansar III	6707	(s) Pansar I	w	S	*none*	3-5%
1924	Svea I	6704	Pudel/Sammetsvete (landrace)	w	S	*none*	3-5%
1925	Jarl	0003	Iduna/line from Sammetsvete from Uppland	w	S	*none*	15%
1925	Kron	6708	Sol II/Pansar I	w	S	*none*	*
1926	Extra Kolben II	8923	(s) Extra Kolben I	s	S	*none*	3-5%
1927	Stål	6709	Sol II/Pansar I	w	S	*none*	3-5%
1928	Ankar	0004	Iduna/Bore	w	S	*none*	3-5%
1928	Ankar II	0006	Ankar I/Saxo	w	S	*none*	3-5%
1928	Diamant	6679	Kolben/Hallands (landrace)	s	S, D, F	*none*	15%
1929	Saxo	0005	(s) among deviating plants of Tystofte Smaahvede II	w	S	*none*	3-5%
1932	Äring	0007	Ankar I/Saxo	w	S	*none*	3-5%
1933	Fylgia I	6680	Aurore/Extra Kolben I	s	S	*u*	3-5%
1934	Ergo	0008	Ankar I/Jarl	w	S	*none*	15%
1935	Skandia	6383	Kron/Fylgia I	w	S	*none*	3-5%
1936	Thule III	6714	Thule II/Sv 0762	w	S	*none*	3-5%
1937	Sol IV	6715	Kron/Sol II	w	S	*none*	15%
1938	Diamant II	6681	Diamant/Extra Kolben II	s	S, F	*none*	15%

[1] n/a = not available; [2]† = T1BL.1RS translocation line; [3] Numbers denote NGB accession numbers or are missing for cultivars donated by a breeding company; [4] s= selected from cultivar or landrace, n/a = not available; [5] s = spring type, w = winter type; [6] S = Sweden, N = Norway, F = Finland, D = Denmark; The predominant area of cultivation is listed first.; [7] u = undocumented; [8] Percentage infected leaf area; * = not included in adult plant test.

Table 2. ***cont.*** *Genealogies of Scandinavian wheat cultivars and landraces and results of gene postulations…*

Year[1]	Accession name[2]	Acc no[3]	Pedigree[4]	Habit[5]	Area[6]	Seedling test Postulated *Pm* genes[7]	Adult test % mildew[8]
1938	Gyllen II	6716	Kron/Bore II	w	S	*none*	3-5%
1939	Skandia II	6717	(s) Skandia I	w	S	*none*	3-5%
1940	Äring III	0011	(s) Äring I	w	S	*none*	3-5%
1942	Progress	6682	Sv Ä 23-8/Extra Kolben II	s	S	*u*	15%
1943	Eroica	0012	WW 5133/Äring I	w	S	*none*	3-5%
1945	Brons	7456	Aurore/Extra Kolben II	s	S	*u*	3-5%
1945	Walde	0024	Ergo/Svea II	w	S	*none*	3-5%
1945	Virtus	0013	Ergo/Svea II	w	S	*none*	3-5%
1946	Kärn	7457	WW 8244/WW 8388	s	S	*none*	3-5%
1946	Pärl II	6722	Sv 0912/Svea	w	S	*none*	3-5%
1947	Kärn II	7458	(s) Kärn I	s	S	*none*	3-5%
1947	Aros	0014	Äring I/Ergo	w	S	*none*	3-5%
1949	Odin	6723	Gluten/Ergo	w	S	*none*	3-5%
1949	Robur	6724	Skandia II/Sv 36-175	w	S	*none*	3-5%
1950	Blanka	9691	Extra Kolben II/Wilhelmina	s	S	*none*	3-5%
1951	Eroica II	0015	(s) Eroica I	w	S	*none*	3-5%
1952	Fylgia II	6685	Extra Kolben II/Aurore	s	S	*u*	3-5%
1952	Rival	6684	Diamant I/Extra Kolben II	s	S	*none*	3-5%
1953	Svenno	7461	WW 8244/WW 8388	s	S	*none*	3-5%
1953	Ertus	0017	Eroica I/Virtus	w	S	*none*	3-5%
1955	Safir	6687	Extra Kolben I/Brunt Schlanstedt//Svalöfs Kolben/Brunt Schlanstedt	s	S	*u (seg)*	3-5%
1955	Svale	6725	Skandia II/Eroica I	w	S	*none*	3-5%
1957	Ring	7462	Kain/Pondus	s	S	*Pm1a+9*	3-5%
1959	Starke	0018	WW 11556/WW 11376	w	S	*none*	3-5%
1959	Ölve	6727	Eroica I/K 01281 (mother line to Hansa)	w	S	*none*	3-5%
1960	Trond	0019	Virtus/WW 9344	w	S	*none*	3-5%
1961	Thor	0020	WW 11376/WW 11379	w	S	*none*	3-5%
1962	Prins	6688	Diamant II/Kärn II	s	S	*none*	3-5%
1962	Norre	0021	Eroica I/Virtus	w	S	*none*	3-5%
1967	Pompe	7464	Ring/Svenno	s	S	*Pm1a+2+9*	3-5%
1967	Troll	7463	Ring//Pondus/Kärn I	s	S	*Pm1a+2+9*	3-5%
1968	Rang	7466	Ring ^5/Els	s	S	*Pm1a+2+9+u*	3-5%
1968	Snabbe	7465	Svenno/WW 7039 (= Kain/Kimmo)	s	S	*u*	3-5%
1968	Starke II	0022	(s) Starke I	w	S	*none*	3-5%
1968	Virgo	6729	Demeter/Virtus//Odin	w	S	*none*	3-5%
1971	Sappo	7467	WW 177-62/WW 176-62	s	S	*u*	3-5%
1972	Drabant	7469	CItr 12633/Ring ^6	s	S, F	*u*	3-5%
1972	Walter	7473	Starke/WW 14433 (= WW 11376/WW 11418)	s	S	*u*	3-5%
1972	Holme	0023	Starke I//Odin/Banco	w	S	*none*	3-5%
1976	Hildur	4080	Sv 60504/Starke I	w	S	*none*	3-5%
1978	Saffran†	7472	WW 38-68 /WW 11-68	s	S	*Pm1a+2+8+9+u*	3-5%
1979	Timmo	7471	WW-152-65/ Sappo	s	S	*u*	3-5%

[1] n/a = not available; [2] † = T1BL.1RS translocation line; [3] Numbers denote NGB accession numbers or are missing for cultivars donated by a breeding company; [4] s= selected from cultivar or landrace, n/a = not available; [5] s = spring type, w = winter type; [6] S = Sweden, N = Norway, F = Finland, D = Denmark; The predominant area of cultivation is listed first.; [7] u = undocumented; [8] Percentage infected leaf area; * = not included in adult plant test.

Table 2. ***cont.*** *Genealogies of Scandinavian wheat cultivars and landraces and results of gene postulations…*

Year[1]	Accession name[2]	Acc no[3]	Pedigree[4]	Habit[5]	Area[6]	Seedling test Postulated *Pm* genes[7]	Adult test % mildew[8]
Sweden							
1979	William	7474	WW 13 69/WW 41 69	s	S	*u*	3-5%
1981	Folke	2434	Holme/Walde	w	S, N	*none*	3-5%
1981	Holger	2435	WW 2259-68/WW 2250-68	w	S, N	*Pm6+u*	3-5%
1983	Sunnan	7476	Pompe 2r 19/Sappo// Drabant	s	S	*u*	3-5%
1984	Kosack	7482	Mironovskaja 808/Starke M//Holme M	w	S, N, D	*Pm4b*	3-5%
1984	Tjelvar†	9952	Sture3D1/4/StureM3b2M5M7	w	S	*Pm6+8+u*	3-5%
1988	Canon	7481	Sicco/2*WW-12502//2*Sappo/3/Kadett	s	S	*u*	*
1988	Dragon	9954	Sicco/WW-12502//Sappo/5*Kadett	s	S, N	*u*	*
1988	Sleipner†	7483	WW 20102/CB 149//Maris Huntsman///Bilbo	w	S, N, D	*Pm2+6+8*	3-5%
1990	Dacke	9955	P18/17269//19151	s	S	*u*	*
1990	Tjalve	7479	WW-20999/Benno;T-9111/449-73//15432;Reno/WW-16679//WW-15432;(DER) Benno	s	S, N, F	*Pm4b+6+u*	3-5%
1990	Tryggve	9953	Riley/Holme//18614/3/Helge	w	S, F	*none*	3-5%
1991	Sport	9956	Citr 5484/PompeBM///Trippel ^3//WW 17269 ^4 /WW 19151	s	S	*u*	3-5%
1992	Lavett	13041	WW118466/Kadett//Dragon	s	S	*u*	*
1994	Curry	-	Canon s/Nemares//Kadett Mp1	s	S	*u*	*
1996	Avle	-	22279M15/20299M12//Canon	s	S, N	*u*	*
1996	Mjölner	-	TL340/Starke I//W25458	w	S, N	*none*	*
1998	Vinjett	-	Tjalve M14/Tjalve M15//Canon	s	S, N, D, F	*u*	*
1999	Virke	-	n/a	w	S	*u*	*
2000	Revelj	-	Kanzler M15M28	w	S	*u*	*
2001	Ballad	-	Sv85297/Sv85568	w	S	*Pm5+u*	*
2001	Harnesk	-	WW-line/Konsul	w	S	*Pm4b*	*
2001	Zebra	-	Ralle/Dragon	s	S, N, F	*u*	*
Norway							
n/a	Børsum	2125	Landrace	s	N	*u*	3-5%
n/a	J-03	2146	Breeding line for mildew resistance	s	N	*u*	*
n/a	Kr Finset, Eikesdal	2128	Landrace	s	N	*u (seg)*	3-5%
n/a	Landvårkveite	2129	Landrace	s	N	*u*	3-5%
n/a	Enger	8957	Landrace from Aremark, Norway	w	N	*none*	3-5%
1930s	Fram I	2126	J-03/Mo-07	s	N	*u*	*
1938	Fram II	2127	J-03/Mo-07	s	N	*u*	3-5%
1939	Snøgg I	2139	0843/Ås	s	N	*u*	3-5%
1948	Trym	2142	Huron/Fylgia I	s	N	*u*	
1958	Norrøna	2133	Fram II/Sopu	s	N	*u*	3-5%
1963	Rollo	2135	Kärn II/Norrøna	s	N	*u*	3-5%
1970	Lanor	2130	Norrøna/Lade	s	N	*u*	*
1972	Runar	2136	Els/Rollo	s	N, F	*Pm4b*	10%
1975	Reno	2134	Els/T-110-21-41;Tammi/Kärn-II//Els	s	N, F	*Pm4b*	3-5%
1976	Rida	11317	Mo-0944-15/Redcoat//Trond	w	N	*Pm5+u (seg)*	3-5%

[1] n/a = not available; [2]† = T1BL.1RS translocation line; [3] Numbers denote NGB accession numbers or are missing for cultivars donated by a breeding company; [4] s= selected from cultivar or landrace, n/a = not available; [5] s = spring type, w = winter type; [6] S = Sweden, N = Norway, F = Finland, D = Denmark; The predominant area of cultivation is listed first.; [7] u = undocumented; [8] Percentage infected leaf area; * = not included in adult plant test.

Table 2. ***cont.*** *Genealogies of Scandinavian wheat cultivars and landraces and results of gene postulations…*

Year[1]	Accession name[2]	Acc no[3]	Pedigree[4]	Habit[5]	Area[6]	Seedling test Postulated *Pm* genes[7]	Adult test % mildew[8]
Norway							
1983	Tautra	2141	n/a	s	N	*none*	*
1990	Kalle	11316	n/a	w	N	*Pm5+u*	*
1998	Bjørke	13659	SvU75630/Rida	w	N	*Pm5*	*
Finland							
n/a	Haarajärvi ME0102	0121	Landrace	s	F	*none*	*
n/a	Jokikylä ME0505	0040	Landrace	s	F	*none*	*
n/a	Järvenkylä ME0302	0131	Landrace	s	F	*none*	*
n/a	Laitiala AP0103	4406	Landrace	s	F	*none*	*
n/a	Sarkalahti ME0101	0120	Landrace	s	F	*none*	*
n/a	Haukiala Pirola	8968	Landrace	w	F	*none*	*
n/a	Storvik sjundeå	4783	Landrace	w	F	*none*	*
1933	Varma Tammisto	9020	Svea I/Landrace from Finland	w	F	*none*	*
1936	Hopea	13345	Canadian Marquis/Ruskea	s	F	*none*	*
1941	Kimmo	13347	(s) Population of Pisarev, Russian wheat	s	F	*none*	3-5%
1965	Jyvä	0348	(s) Vakka	w	F	*none*	*
1975	Hankkijan Ilves	6773	Hja-B-356/Vakka	w	F	*none*	*
1993	Manu	11709	Ruso/Runar	s	F	*Pm4b*	*
Denmark							
n/a	Brödtorp pajo	8946	Landrace	w	D	*none*	3-5%
n/a	Lading skaeghvede	6388	Landrace	w	D	*none*	*
n/a	Østby	8922	Landrace	s	N	*u*	*
1907	Tystofte Stand	8197	(s) Squarehead	w	D	*none*	*
1915	Sma II Tystofte	7183	(s) Tystofte smaahvede	w	D	*none*	*
1929	Dania Abed	7027	n/a	w	D	*none*	*
1937	Als	4770	(s) Landrace from Als	w	D	*none*	3-5%
1939	Konge II	8194	(s) Konge (= Ideal /spontaneous cross)	w	D	*none*	3-5%
1955	Hunsballe R	5153	(s) Jubilee	w	D	*none*	*
1967	Borg Abed	8933	Trifolium 14/Abed 92	w	D	*none*	*
1967	Storaks Abed	7184	n/a	w	D	*none*	*
1967	Tystofte Stakket	9017	(s) English Squarehead	w	D	*none*	*
1975	Nana	9118	Ibis/Stella	w	D	*Pm5*	*
1980	Kraka	9123	Kranich/Caribo	w	D	*Pm5*	3-5%
1981	Anja	9122	Kranich/Caribo	w	D	*none*	*
1990	Hanno	-	Banjo/Hermes	s	D, N	*u*	*
1992	Terra	-	Kraka/TJB730-3637	w	D	*Pm5+6+u*	3-5%

[1] n/a = not available; [2]† = T1BL.1RS translocation line; [3] Numbers denote NGB accession numbers or are missing for cultivars donated by a breeding company; [4] s= selected from cultivar or landrace, n/a = not available; [5] s = spring type, w = winter type; [6] S = Sweden, N = Norway, F = Finland, D = Denmark; The predominant area of cultivation is listed first.; [7] u = undocumented; [8] Percentage infected leaf area; * = not included in adult plant test.

Table 3. *Reactions[1] of wheat accessions showing resistance to one or more isolates after inoculation with 11 isolates of* Blumeria graminis *f. sp.* tritici

Name of Accession	Acc	Isolate											Postulated seedling
	No[2]	2	5	6	9	10	12	13	14	15	16	17	*Pm* gene(s)[3]
RG 1													
Harnesk	-	s	r	s	r	r	r	s	s	r	s	s	*4b*
Kosack	7482	s	r	s	r	r	r	s	s	r	s	s	*4b*
Manu	11709	s	r	s	r	r	r	s	s	r	s	s	*4b*
Runar	2136	s	r	s	r	r	r	s	s	r	s	s	*4b*
Reno	2134	s	r	s	r	r	r	s	s	r	s	s	*4b*
RG 2													
Ballad	-	s	r	s	s	r	s,r	s	r	s	s	s	*5+u (seg)*
Kraka	9123	s	r	s	s	r	s	s	r	s	s	s	*5*
Nana	9118	s	r	s	s	r	s	s	r	s	s	s	*5*
Bjørke	13659	s	r	s	s	r	s	s	r	s	s	s	*5*
Kalle	11316	s	r	i	r	r	s	s	r	s	s	s	*5+u*
Rida	11317	r,s	r	s	r	r	s	s	r	s	s	s	*5+u (seg)*
Holger	2435	s	r	s	r	r	s	r	r	r	r	r	*6+u*
Terra	-	i	r	s	r	r	s	r	r	r	r	r	*5+6+u*
Tjelvar	9952	s	r	s	r	r	s	r	r	r	r	r	*6+(8)+u*
Tjalve	7479	s	r	s	r	r	r	r	r	r	r	r	*4b+6+u*
Sleipner	7483	r	r	r	r	r	r	i,s	r	r	i	r,s	*2+6+8*
RG 3a													
Ring	7462	r	s	r	r	r	s	s	s	r	s	s	*1a+9*
Troll	7463	r	r	r	r	r	s	s	s	r	s	s	*1a+2+9*
Pompe	7464	r	r	r	r	r	s	s	s	r	s	s	*1a+2+9*
Rang	7466	r	r	r	r	r	r	r	s	r	s	s	*1a+2+9+u*
Saffran	7472	r	r	r	r	r	r	r	s	r	s	s	*1a+2+(8)+9+u*
RG 3b													
J-03	2146	r	r	r	r	r	s	s	s	r	s	s	*u*
Fram I	2126	r	r	r	r	r	i	r	r	r	i	r	*u*
Fram II	2127	r	r	r	r	r	s	r	r	r	s	s	*u*
Snøgg I	2139	r	r	r	r	r	r	r	r	r	s	s	*u*
Norrøna	2133	r	r	r	r	r	s	r	r	r	s	s	*u*
Rollo	2135	r	r	r	r	r	s	r	i,r	r	s	s	*u*
RG 3c													
Brons	7456	r	r	r	r	r	s	s	s	r	s	s	*u*
Fylgia I	6680	r	r	r	r	r	s	s	s	r	s	s	*u*
Fylgia II	6685	r	r	r	r	r	s	s	s	r	s	s	*u*
Trym	2142	r	r	r	r	r	r	r	s	r	r,i	s,i	*u*

[1] r = resistant, s = susceptible, i = intermediate; r,s etc. indicates that lines segregated for reaction response patterns

[2] Numbers denote NGB accession numbers or are missing for cultivars donated by a breeding company.

[3] u= unidentified gene(s).

Table 3. *cont. Reactions[1] of wheat accessions showing resistance…*

Name of Accession	Acc No[2]	Isolate											Postulated seedling *Pm* gene(s)[3]
		2	5	6	9	10	12	13	14	15	16	17	
RG 3d													
Østby	8922	r	r	r	r	r	s	i	s	r	s	i	*u*
Børsum	2125	r	r	r	r	r	r	r	s	r	s	s	*u*
Landvårkveite	2129	r	r	r	r	r	s	r	r	r	s	s	*u*
Lr from Dalarna	6673	r	r	r	r	r	s	i	s	r	s	s	*u*
Dalarna	6410	r	r	r	r	r	s	i	s	r	s	s	*u*
Dala	9708	r	r	r	r	r	s	s	s	r	s	s	*u*
Lr from Halland	6674	r	r	r	r	r	r,i	r	r	r	i,s	s	*u (seg)*
Halland	6409	r	r	r	r	r	i,s	i,r	s,r	r	s	s	*u (seg)*
RG 4													
Progress	6682	s	s	s	r	s	s	s	s	s	s	s	*u*
Snabbe	7465	s	s	s	r	s	s	s	s	s	s	s	*u*
Safir	6687	r,s	s	s	r	s	s	s	s	s	s	s	*u (seg)*
RG 5													
Sappo	7467	r	r	r	r	r	r	r	r	r	r	r	*u*
Avle	-	r	r	r	r	r	r	r	r	r	r	r	*u*
Canon	7481	r	r	r	r	r	r	r	r	r	r	r	*u*
Curry	-	r	r	r	r	r	r	r	r	r	r	r	*u*
Dacke	9955	r	r	r	r	r	r	r	r	r	r	r	*u*
Drabant	7469	r	r	r	r	r	r	r	r	r	r	r	*u*
Dragon	9954	r	r	r	r	r	r	r	r	r	r	r	*u*
Hanno	-	r	r	r	r	r	r	r	r	r	r	r	*u*
Lavett	13041	r	r	r	r	r	r	r	r	r	r	r	*u*
Revelj	-	r	r	r	r	r	r	r	r	r	r	r	*u*
Sport	9956	r	r	r	r	r	r	r	r	r	r	r	*u*
Sunnan	7476	r	r	r	r	r	r	r	r	r	r	r	*u*
Timmo	7471	r	r	r	r	r	r	r	r	r	r	r	*u*
Vinjett	-	r	r	r	r	r	r	r	r	r	r	r	*u*
Virke	-	r	r	r	r	r	r	r	r	r	r	r	*u*
Walter	7473	r	r	r	r	r	r	r	r	r	r	r	*u*
William	7474	r	r	r	r	r	r	r	r	r	r	r	*u*
Zebra	-	r	r	r	r	r	r	r	r	r	r	r	*u*
RG 6													
Lanor	2130	s	s	s	s	s	s	i	i	s	s	r	*u*
Kr Finset, Eikesdal	2128	i,s	s,r	s,r	r	s,r	s,i	i	s	s	s	s	*u (seg)*
Lr from Gotland	4496	r,i	r	i,r	r,i	r,s	r,s	r,s	s,i	r,s	s,r	s,r	*u (seg)*
Lr from Uppsala	6692	s,r	r,s	s,r	s,r	s,r	s,r	s,r	s,r	s,r	s	r,s	*u (seg)*
Warmland Lr	8198	s,i	r,s	r,s	s,r	r,s	r,s	s,i	s,r	s,r	s,r	r,s	*u (seg)*
Lr from Halland	6691	s	s	i,r	s	i,s	s	s	s	s	s	s	*u*

[1] r = resistant, s = susceptible, i = intermediate; r,s etc. indicates that lines segregated for reaction response patterns

[2] Numbers denote NGB accession numbers or are missing for cultivars donated by a breeding company.

[3] u= unidentified gene(s).

Table 4. *Number and percentage of accessions originating in different countries and found susceptible, resistant, with unidentified resistance genes, postulated* Pm *gene(s) and adult plant resistance (APR)*[1]

Country	Material			Seedling				Adult	Single *Pm* genes and Combinations of *Pm* genes					
	Accessions total	**Landraces**	**Cultivars**	**Susceptible**	**Resistant**	**Unidentified (only)**	**Postulated *Pm* genes**	**Less than 5% infection**	*Pm1a+9+*	*Pm2+*	*Pm4b+*	*Pm5+*	*Pm6+*	*Pm8+*
Sweden														
Total ***(%)***	107	12	95	63 *(59)*	44 *(41)*	32 *(30)*	12 *(11)*	84 *(91)*	5	5	3	1	4	3
Spring	45	5	40	13 *(29)*	32 *(71)*	26 *(58)*	6 *(13)*	33 *(89)*	5	4	1	-	1	1
Winter	62	7	55	50 *(81)*	12 *(19)*	6 *(10)*	6 *(10)*	51 *(93)*	-	1	2	1	3	2
Norway														
Total ***(%)***	18	4	14	2 *(11)*	17 *(94)*	12 *(67)*	5 *(28)*	10 *(91)*	-	-	2	3	-	-
Spring	14	3	11	1 *(7)*	13 *(93)*	11 *(78)*	2 *(14)*	7 *(87)*	-	-	2	-	-	-
Winter	4	1	3	1 *(25)*	3 *(75)*	-	3 *(75)*	2 *(100)*	-	-	-	3	-	-
Denmark														
Total acc. ***(%)***	17	2	15	12 *(71)*	5 *(29)*	2 *(12)*	3 *(19)*	5 *(100)*	-	-	-	3	1	-
Spring	2	-	2	-	2 *(100)*	2 *(100)*	-	-	-	-	-	-	-	-
Winter	15	2	13	12 *(80)*	3 *(20)*	-	3 *(20)*	5 *(100)*	-	-	-	3	1	-

[1] percentages are presented as accessions/country and All countries represent the total number/percentage of accessions within the material; figures and percentage for APR have been adjusted according to the number of lines tested for APR in each country (SWE=92, NOR=11, DEN=5, FIN=1)

Table 4. *cont. Number and percentage of accessions originating in different countries…*

Country	Material			Seedling				Adult	Single *Pm* genes and Combinations of *Pm* genes					
	Accessions total	Landraces	Cultivars	Susceptible	Resistant	Unidentified (only)	Postulated *Pm* genes	Less than 5% infection	*Pm1a+9+*	*Pm2+*	*Pm4b+*	*Pm5+*	*Pm6+*	*Pm8+*
Finland														
Total ***(%)***	13	7	6	12 *(92)*	1 *(8)*	-	1 *(8)*	1 *(100)*	-	-	1	-	-	-
Spring	8	5	3	7 *(87)*	1 *(12)*	-	1 *(12)*	1 *(100)*	-	-	1	-	-	-
Winter	5	2	3	5 *(100)*	-	-	-	-	-	-	-	-	-	-
All countries														
Total ***(%)***	155	25	130	89 *(57)*	66 *(42)*	45 *(29)*	21 *(13)*	100 *(92)*	5	5	6	7	5	3
Spring	69	13	56	21 *(30)*	48 *(69)*	39 *(56)*	9 *(13)*	43 *(89)*	5	4	5	-	1	1
Winter	86	12	74	68 *(79)*	18 *(21)*	6 *(7)*	12 *(14)*	57 *(93)*	-	1	1	7	4	2

[1] percentages are presented as accessions/country and All countries represent the total number/percentage of accessions within the material; figures and percentage for APR have been adjusted according to the number of lines tested for APR in each country (SWE=92, NOR=11, DEN=5, FIN=1)

III

Temporal diversity changes among 198 Nordic bread wheat landraces and cultivars detected by retrotransposon-based S-SAP analysis

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Abstract

The sequence-specific amplified polymorphism (S-SAP) method was used to genotype 198 Nordic bread wheat landraces and cultivars from the 19th to 21th century. All except five putatively identical accessions were discriminated, showing that the *Sukkula*-9900-LARD retrotransposon primer was highly suitable for resolving closely related wheat materials. Cluster analysis was generally consistent with pedigree information and there was a clear separation for growth habit but not for countries. These findings were supported by a principal coordinates analysis (PCoA), which in addition revealed a separation into different time periods (before 1910, 1910-1969 and 1970-2003). These results are consistent with the breeding history and pedigree information, indicating that little hybridization has occurred between winter and spring wheat, but there has been a frequent exchange of germplasm between the Nordic countries. Estimates of gene diversity, the PCoA results, and changes in band frequencies across time indicate that plant breeding has led to substantial genetic shifts in Nordic wheat. Diversity was reduced through selections from landraces during the early 20th century, followed by a period of relatively lower genetic diversity, and a subsequent increase and net gains in diversity from the late 1960s onwards through the use of exotic germplasm. Thus, an anticipated loss of overall genetic diversity was found to be negligible, although allele losses have occurred at specific loci. Analyses of gene diversity were undertaken on subsets of the accessions selected by a Maximum diversity algorithm. The results show that a carefully selected core collection with 15 accessions would retain 90% of the variability.

Introduction

The identification and characterization of cultivars and breeding lines is important for the documentation and utilization of genetic resources. Several morphological, physiological and molecular methods have been developed for this purpose (Liu *et al.*, 1992). Marker systems that show high levels of DNA polymorphism are a prerequisite for distinguishing closely related genotypes of self-pollinating crops such as bread wheat (*Triticum aestivum* L.).

Retrotransposons are among the most prevalent class of eukaryotic transposable elements, characterized by their ability to transpose via an RNA intermediate, which they convert to DNA by reverse transcription prior to insertion (Waugh *et al.*, 1997). Monocot retrotransposons expressed sequence tags (ESTs) tend to match across multiple genera (Vicient *et al.*, 2001) and sequences isolated from one species can thus be used as markers in other genera. Molecular markers derived from barley retrotransposons have been shown to reveal genetic diversity also in wheat (Gribbon *et al.*, 1999). The sequence-specific amplified polymorphism (S-SAP) method (Waugh *et al.*, 1997) for the detection of retrotransposons, utilizes polymerase chain reaction (PCR) with outward facing primers matching retroelements in combination with primers corresponding to a restriction site adaptor. In the S-SAP method, selective bases added to the primers reduce the complexity of the the amplified retrotransposon DNA targets (Waugh *et al.*, 1997; Gribbon *et al.*, 1999).

The long terminal repeat (LTR) retrotransposon superfamily is the most abundant in crop species (Sabot *et al.*, 2004) and several groups have been identified including the transpositionally active *l*arge *r*etrotransposon *d*erivative (LARD) *Sukkula* elements in barley (Kalendar *et al.*, 2004). LTRs do not excise as part of the retrotransposition mechanism, thus marker band polymorphisms represent the integration of new retrotransposon copies and irreversible biological events (Vicient et al., 2001). Retrotransposon markers are therefore highly suitable for studies of genetic diversity and have been applied to pea (Ellis *et al.*, 1998; Pearce *et al.*, 2000), the genera *Aegilops* and *Triticum* (Queen et al., 2004), tomato and pepper (Tam *et al.*, 2005). Retrotransposon markers generate data that is more consistent with geographical and morphological criteria than amplified fragment length polymorphism (AFLP) based markers (Ellis *et al.*, 1998). In a comparative study of genetic diversity in tomato and pepper, estimates of genetic relationships were significantly correlated between S-SAP, AFLP and simple sequence repeat (SSR) generated datasets. The S-SAP method showed the highest number of polymorphic bands and marker index, and was thus found to be more informative than AFLP or SSR (Tam et al., 2005).

Investigations of genetic diversity in wheat using molecular markers have demonstrated a set of different scenarios. Donini and co-workers (2000) analyzed 55 UK wheat accessions from 1934 to 1995 at six AFLP and 14 microsatellite (SSR) loci, and reported that no significant decrease in overall genetic diversity had occurred. Plant breeding had resulted in a qualitative rather than quantitative shift in the temporal diversity. Huang and co-workers (2002) used 24 microsatellite markers to assess genetic diversity in 998 wheat accessions from 68

countries and found genetic diversity to be highest in the Near East and Middle East, regions which represent the origination of wheat. Wheat accessions from Europe could be divided into three regions where genetic diversity was higher in Southeast Europe compared to the North and Southwest Europe. Roussel and co-workers (2004) studied 559 French bread wheat accessions from 1800 to 2000 using 42 SSRs and demonstrated a significant decrease in allelic diversity at the end of the 1960s. In a wider study using 39 SSRs in 480 European wheat cultivars from 1840 to 2000, Roussel and co-workers (2005) discerned a decrease in allelic diversity from the 1960s onwards. Genetic diversity changes could be explained both by temporal and geographical variation in different countries. Genetic diversity in 75 Nordic spring wheat cultivars as measured by allelic variation in 47 microsatellites was found to have increased from 1900 to 1940 and again from the 1960s due to breeding efforts (Christiansen *et al.*, 2002). Similarly, genetic diversity measured using 19 microsatellites was found to increase after 1960 in 91 Bulgarian accessions from 1925 to 2003 (Landjeva *et al.*, 2005). In a study of 90 SSR markers in 253 modern wheat cultivars bred at CIMMYT, in addition to landraces and *Aegilops tauschii* Coss. accessions, genetic diversity was found to decrease from 1950 to 1989 and then increase from 1990 to 1997 through introgression of novel material (Reif *et al.*, 2005). The landrace and *Aegilops tauschii* accessions contained numerous unique alleles that were absent in the modern wheat cultivars and thus represent potential sources for useful variation in wheat breeding programmes.

The aim of the present study was to use S-SAP to fingerprint 198 bread wheat accessions from the Nordic countries Sweden, Norway, Finland and Denmark and to (i) estimate levels of genetic diversity within and between growth habit, countries and time periods; (ii) assess the relationships among accessions; and (iii) determine the number of accessions needed for an *ex situ* core collection of Nordic bread wheat.

Materials and Methods

Plant materials and S-SAP reactions

The material comprised 32 landraces and 166 bread wheat cultivars of spring or winter type from the 19th to the 21th century and Sweden, Norway, Denmark or Finland. Seed was donated by The Nordic Gene Bank (NGB), The John Innes Centre (JIC) and plant breeding companies Svalöf-Weibull AB (SW), Pajbjerg Fonden and Abed Fonden (Table 2). Information on pedigrees and year of release was provided by Svalöf-Weibull AB, The Nordic Gene Bank or obtained from the internet at Wheat Pedigree and Identified Alleles of Genes On Line database (http://genbank.vurv.cz/wheat/pedigree/) (WPIAG 2007-01-10).

Genomic DNA was extracted from a bulk of 30 seeds of each accession using a DNeasy96 Plant Kit (Qiagen). The S-SAP method as described by Waugh and co-workers (1999) and Lee and co-workers (2003) was used with the following modifications. Genomic DNA (400 ng) was incubated with 5 U *Taq*I (New England BioLabs) in 5xRL buffer (50 mM TRIS-acetate pH 7.5, 50 mM magnesium acetate, 250 mM potassium acetate, 25 mM DTT and 25 ng/µl BSA)

in a total volume of 40 µl per reaction at 65°C for 3 hours. Template DNA was prepared by adding 10 µl of a ligation mixture [50 pmol *Taq*I (5'-ATG AGT CCT GAA-3' plus 5'-CGT TCA GGA CTC AT-3'), 5xRL buffer, 0.1 mM ATP, 1 U T4 DNA ligase (Invitrogen)] and incubating at 37 °C overnight. The samples were then diluted with 100 µl T0.1E (10 mM TRIS-HCl pH 8.0, 0.1 mM EDTA) and stored at –20°C.

The *Sukkula*-9900-LARD retrotransposon has been found to be highly polymorphic in wheat (Hysing, 2004 unpublished results). The relative activity of *Sukkula* elements in barley has been estimated to be less than *BARE-1* while the order of copy number is similar to that of *BARE-1* (Leigh *et al.*, 2003), which is present in 14,000 full-length copies (Vicient *et al.*, 1999) and more than $1x10^{-5}$ solo LTRs (Shirasu *et al.*, 2000). The *Sukkula*-9900-LARD oligonucleotide primer (5'-GAT AGG GTC GCA TCT TGG GCG TGA C-3') was end-labelled by incubating 0.134 µl *Sukkula*-9900-LARD (50 ng/µl stock) with 0.1 µl γ-[^{33}P]-ATP (3000 Ci/mmol), 0.1 µl 10xT4 buffer, 0.25 U T4 polynucleotide kinase (0.025 µl) (Invitrogen) in a total volume of 0.668 µl per subsequent reaction at 37 ºC for 2 to 3 hours. Each selective amplification reaction contained 0.66 µl [^{33}P]-labelled *Sukkula*-9900-LARD, 2 µl unlabelled *Sukkula*-9900-LARD adaptor primer (5'-ATG AGT CCT GAA CGA - 3') (50 ng/µl stock) with one of five different combinations of selective bases at the 3' end (-AAT, -TAA, -ATT, -CAT, -CAA), 1 µl 10xPCR buffer, 1 µl dNTP mix (2 mM stocks, Amersham Biosciences), 3 µl digested template, 0.08 µl (0.4 U) *Taq* DNA polymerase (Qiagen) and 2.26 µl sterile distilled water. The touchdown PCR protocol of Vos and co-workers (1995) was followed.

The PCR products were mixed with an equal volume of loading buffer (94% de-ionized formamide, 10 mM EDTA, 0.5 mg/ml bromophenol-blue, 0.5 mg/ml xylene cyanol FF) and denatured at 95°C for 5 minutes. Amplification products were resolved by loading an aliquot of 4 µl from each sample onto 6% denaturing polyacrylamide gels (Sequa Gel 6, National Diagnostics) and electrophoresed on BIO RAD vertical gel apparatus for 2 hours at 80 W constant power. Gels were transferred to Whatman chromatography 3 mm paper (Fischer), dried and exposed to Kodak XO-Mat Imaging film for three days at room temperature. DNA fingerprints were evaluated and scored manually.

Statistical analyses

Each S-SAP band was treated as an independent locus with two alleles, presence or (1) or absence (0) of a band. Gene variation was quantified through $1-p^2-q^2$ where p is the frequency of band-presence and q is the frequency of band-absence. This measure has been referred to as *gene diversity* (Weir, 1996), *expected heterozygosity H* (Nei, 1973), or *polymorphic index content* PIC (Ghislain *et al.*, 1999). The latter term is commonly used for comparisons of primers or markers and it is customary to calculate the sum in the case of a multiplex marker, e.g. when several loci are scored for the same primer pair. It should be mentioned that originally PIC was defined as the probability to deduct which allele an offspring had received from a specific parent if the genotypes of both parents and the offspring are known (Botstein *et al.*, 1980). In the following we will use the term

gene diversity and the symbol *H* when applied to categories of accessions and PIC when applied to primer extensions.

The first application of this measure was to evaluate the informativeness and genetic diversity of each S-SAP primer extension. The sum of PIC values for all bands generated by the same primer extension constituted the S-SAP primer extension index. Gene diversity (*H*) was then calculated for the entire material, and for categories based on growth habit (spring/winter), country of origin (Sweden, Norway, Denmark, Finland) and time period of release (decade).

Genetic differentiation among categories was calculated using Wright's fixation index (F_{ST}). The significance values for differentiation among categories were obtained through a bootstrap randomization procedure using 10,000 simulations in the SAS statistical package (Statistical Analyses System, Version 9.1.3, SAS Institute, Cary, NC, USA). The relationships between categories and accessions were then visualized by using principal coordinates analysis (PCoA) based on Eigen vector values of the primary matrix (Flury, 1984). In addition, pairwise comparisons were calculated among all accessions using the Jaccard index (Weising *et al.*, 2005). The resulting matrix was employed in a cluster analysis performed with the NTSYS-pc statistical package (Rohlf, 1998) and using the unweighted pair/group method with arithmetic averages (UPGMA) (Sneath & Sokal, 1973).

Suggestions for an *ex situ* core collection were generated by a program written in Dev-Pascal 1.9.2 based on the Maximum genetic diversity algorithm (Marita *et al*., 2000). The initial accession was chosen randomly. A mean index (I) describing the proportion of loci including both presence (1) and absence (0) of the alleles was calculated for core collections based on 10, 15, 20, 30, 50 and 100 accessions (20 replications each).

Results

S-SAP amplification

A binary matrix based on 142 polymorphic S-SAP bands was generated by scoring the presence (1) and absence (0) of bands. Values obtained for the S-SAP primer extension indices were PIC_{TAA}=9.3, PIC_{AAT}=8.6, PIC_{ATT}=6.2, PIC_{CAA}=5.6 and PIC_{CAT}=3.5, indicating that the TAA extension yielded the highest number of polymorphic bands.

Gene diversity within categories

Comparisons of the amount of gene diversity (*H*) within categories (Table 2) revealed higher average gene diversity for the spring growth habit than winter habit in Sweden and the entire material, in contrast to Norway, Denmark and Finland. Winter wheat is more common than spring wheat in Denmark and the majority of the Danish accessions were of winter habit. The results for Norway and Finland could not be explained except for the lower number of winter accessions compared to that in Sweden. Gene diversity partitioned between countries showed a decrease in *H* from Sweden>Denmark>Norway≈Finland, and with the highest figure (0.234) for the entire material. It is possible that the gene

diversity estimates could have been biased by the disproportionate number of accessions in each category; however the results indicate a slight difference in *H* between countries.

Gene diversity in winter and spring wheat (Table 2 and Fig. 3) appears to have declined from the turn of the last century until the late 1960s and subsequently increased to the original level and above. The net gain in gene diversity was 12% in winter wheat, 8% in spring wheat and 19% in total (Table 2). The changes in gene diversity were also apparent as changes in band frequencies over time, where some bands were lost and others gained (Fig. 4). In all, eight S-SAP bands present in the landraces and cultivars before 1910 were not found in the modern material released during 1989 to 2003, conversely eleven bands not found among the landraces were present in the modern material. These results seem to reflect the impact of plant breeding on gene diversity where pure line selections in the early 1900s led to a decrease in diversity similar to a bottleneck, while the incorporation of exotic material beginning in the 1950s has contributed to an increase in diversity.

Gene diversity between categories

Comparisons of the amount of genetic differentiation (F_{ST}) between categories (Table 3) showed that the values were significantly higher for growth habit than for countries (p=0.012). The means were adjusted according to the number of accessions in each population (Norway had only 5 winter wheat accessions and Denmark 2 spring wheat accessions). The genetic differentiation was highest between growth habits in Finland, followed by Norway, Sweden and Denmark, indicating a greater separation between spring and winter germplasm in Finland than in other Nordic countries. Comparisons of F_{ST} between time periods in Sweden were higher for spring than winter wheat. In all, the differentiation between time period categories was lower than differentiation due to growth habit or country.

Genetic relationships among categories and accessions

The principal coordinates analysis (PCoA) showed a clear separation between growth habits but not countries (Fig. 1). The first component explained only 11% of the variation and the second component 8% of the variation. Such a low explanatory power indicates that there has been a high turnover within the material. Observation of the PCoA diagram suggests that changes in the wheat germplasm can be grouped into three different time periods (before 1910, 1910 to 1969 and 1970 to 2003) for both winter and spring wheat (Fig. 1). The time periods seem to reflect a genetic shift from a horizontal distribution of genetic variation during the first time period, to a narrowing and clustering during the second period, and a subsequent horizontal and vertical broadening during the third period. These results are in agreement with the UPGMA dendrogram and the breeding history of wheat in the Nordic countries.

The dendrogram from the UPGMA cluster analysis (Fig. 2) also clearly showed an association between genetic relatedness and growth habit with few exceptions, namely the spring accessions ‘Hallandshvete’, ‘Brons’, ‘Safir’, ‘Lavett’-NGB and

‘Lavett’-SW that were located among the winter accessions. There was no clear clustering of accessions based on geographic origin and the results therefore likely reflect a frequent exchange of germplasm between the Nordic countries. A separate cluster consisted of the four winter cultivars ‘Sleipner’, ‘Tjelvar’, ‘Galicia’ and ‘Abika’, carrying the T1BL.1RS wheat-rye chromosome translocation. The presence of the translocation was verified by C-banding (Hysing 2005, unpublished results). One or several of 17 rare bands (present in less than 5% of the population) were observed in 27% of the material. Two bands were present only in the four cultivars that possess the T1BL.1RS translocation, and presumably correlated with the 1RS chromosome segment. The cultivars ‘Abika’, ‘Diamant II’ and landrace NGB-4496 possessed one unique band each.

The dendrogram was largely in agreement with available pedigree information (Table 1). Fingerprints generated by combinations of primer extensions could distinguish all accessions except two clusters of Finnish landraces (Horsmanaho, Tiimantii-Paavo and Järvenkylä; Jokikylä and Larinsaari). Presumably these accessions are very closely related or even genetically identical. This is in contrast to the two pairs of accessions from different germplasm collections: ‘Lavett’-NGB and ‘Lavett’-SW, and ‘Kimmo’-NGB and ‘Kimmo’-JIC that could be separated in spite of being supposedly the same cultivar. The accessions ‘Kimmo’-NGB and ‘Kimmo’-JIC clustered closer with other cultivars than with one another, and it could be questioned if these are indeed the same cultivar.

Selection of samples for ex situ core collection

Potential core collections comprising 10-100 accessions (5-50%) were sampled from the material using the Maximum diversity algorithm. The results showed that the mean index (I) describing the proportion of loci with both presence and absence of the allele, increased with increasing number of accessions in the collection. Based on the Maximum diversity algorithm, a core collection comprising 15 accessions had an index of about 0.9. Thus 90% of the loci showed both presence and absence of alleles while 10% of the loci showed either presence or absence (Fig. 5).

Discussion

The present study utilized polymorphisms from the *Sukkula*-9900-LARD retrotransposon to study genetic diversity and relationships in wheat. Retrotransposon integration sites are stably inherited, and therefore integration sites shared between accessions are likely to have been present in their common ancestor(s). S-SAP based polymorphism may be the result of transpositional activity of retroelements, a restriction site polymorphism, or both (Soleimani *et al.*, 2005).

This S-SAP based retrotransposon study in 198 Nordic bread wheat accessions revealed several findings regarding the genetic relationships and diversity. The polymorphism patterns generated by the *Sukkula*-9900-LARD retrotransposon primer and four primer extensions allowed the discrimination of 97% of the accessions, showing that the primer was highly suitable for resolving closely

related accessions. The five accessions that could not be separated based on the S-SAP polymorphisms may be identical. Soleimani and co-workers (2005) detected intra-cultivar retrotransposon genetic heterogeneity (biotypes) in 84% of modern Canadian barley cultivars, and concluded that selection in inbreeding crops results in a heterogeneous population of homozygous plants. An analysis of banding patterns in families showed that the source of variation was likely due to residual variation from the parents and retrotransposon activity. In the present study, bulked samples from 30 seeds per accession were used to capture the intra-cultivar diversity. However, the UPGMA dendrogram (Fig. 1) showed that seven landrace accessions could not be separated, while duplicate accessions were placed in different clusters. Missing or incomplete passport data, and imprecise characterization have been identified as limiting factors in the use of landrace and cultivar *ex situ* collections (Dreisigacker *et al.*, 2005). In this respect, the retrotransposon S-SAP method could provide useful information for the description, optimization and use of accessions in seed bank collections. However, as wheat is particularly suitable for seed storage, the conservation of accessions in perpetuity is currently more cost-effective than DNA fingerprinting and thus the identification and removal of suspected duplicates should not be a priority (Dreisigacker *et al.*, 2005).

During the evolution, domestication and breeding of wheat, genetic variation created by mutation and recombination has been reduced by genetic drift and selection, both natural and that of early farmers, which eventually resulted in landraces adapted to specific conditions of their habitats (Reif *et al.*, 2005). It has been postulated that modern wheat cultivars, bred with a limited number of landraces in their pedigree, contain less genetic diversity than landraces (Frankel, 1970). Studies on allelic diversity in landraces and modern wheat cultivars have shown that modern breeding has had a strong selection pressure in the D genome (Hao *et al.*, 2006), which implies that the D genome in landraces may be a rich source for widening the genetic diversity of the D genome in modern varieties (Andolfatto, 2001). Reduction in diversity caused by intensive selection could also be counterbalanced by introgression of novel germplasm. An allelic reduction and genetic shift was detected by SSR markers in Canadian hard red spring wheat germplasm from 1845 to 2004 and partially related to different breeding efforts for e.g. stem rust resistance (Fu *et al.*, 2005). Reif and co-workers (2005) found a significant decrease in relative SSR gene diversity from *Ae. tauschii* accessions to landraces and modern wheat cultivars. The decrease in genetic diversity from 1950 to the late 1970s was ascribed to the "Early Green Revolution" where breeding was characterized by the production of high yielding semi-dwarf wheats that were based on a limited number of parents. There was also an increase in diversity after the late 1970s, explained by a change in breeding strategy aimed at increasing genetic diversity through the use of landraces, spring and winter wheat from different regions, and wild relatives of wheat.

In the present study, estimates of gene diversity, the PCoA results, and changes in band frequencies across time together indicate that plant breeding has led to substantial genetic shifts in Nordic wheat. Genetic variation was reduced during the early 20th century, followed by a period of relatively lower genetic diversity, and a subsequent increase and net gain in diversity from the late 1960s onwards.

These results are supported by the history of wheat breeding in the region. Plant breeding was initiated during the late 19th century in the Nordic countries and practiced through mass and pure line selection in landraces. This was followed by pedigree selection until around the 1950s to 1960s when breeding objectives and choice of germplasm changed to include exotic material. In Sweden around 1915, landraces were substituted by cultivars produced through pedigree selection that combined high yielding Squarehead wheat with good winter hardiness from Swedish landraces. This breeding work was expanded to include quality aspects and continued until the 1950s, when there was an increased focus on e.g. resistance breeding against cereal rust diseases and powdery mildew, the use of distant relatives of wheat and mutation breeding (Lundin, 1997; Olsson, 1997; Svensson, 1997). The decrease in gene diversity in Norway (Table 2) coincides with the period 1945 to 1965 when wheat production decreased because the old varieties were unsuitable for combine harvesting. The gene diversity increased after 1970, which is consistent with the initiation of a breeding program in 1959 emphasizing resistance to sprouting, shattering, lodging and various diseases in combination with earliness and high yield and the introduction of semidwarf wheats during the late 1960s (Donner & Mesdag, 2000). In a study of SSR genetic diversity in 75 Nordic spring wheat cultivars, Christiansen and co-workers (2002) found a general distinction between accessions from different countries and time periods. There was an increase in genetic diversity from 1900 to 1940 followed by a period of genetic loss from 1940 to 1960, and a subsequent increase from 1960 onwards. Our results are in agreement with those of other marker-based studies of diversity changes in wheat, where a general loss of genetic diversity has been found to be negligible, but narrowing has been observed during some periods of time and significant allele loss has occurred at specific loci (Donini *et al.*, 2000; Christiansen *et al.*, 2002; Koebner *et al.*, 2003; Roussel *et al.*, 2004, 2005; Landjeva *et al.*, 2005).

To facilitate the conservation, evaluation, management and efficient utilization of plant genetic collections, Frankel (1984) put forward the concept of the 'core collection' with a limited size, maximized genetic diversity and a minimum of repetitions. Opinions regarding the relative sizes of the total and core collections differ. Brown and co-workers (1987) recommended that the number of accessions in the core collection should account for 5-10% of the accessions and at least 70% of the genetic variation in the base collection while van Hintum and co-workers (2000) suggested 10 to 20% of accessions to represent 70 to 90% of the genetic diversity in the base collection depending on the objective of the core collection. Hao and co-workers (2006) found that sampling 13% of a wheat base collection was sufficient for retaining 98.5% of the SSR alleles. In the present study, it was found that a core collection comprising only 15 accessions selected by a Maximum diversity algorithm program (Marita *et al.*, 2000) would have both presence and absence of alleles at 90% of the loci, but all alleles would not be represented at the remaining 10%. This is in contrast to the results by Garkava-Gustavsson and co-workers (2005) on RAPD diversity in the outcrossing lingonberry *Vaccinium vitis-idaea* L. where no bands were lost in the subset (core), although the band frequencies in the core relative to the total population were affected.

Different retrotransposons in wheat and barley have been shown to have different transpositional activity (Gribbon *et al.*, 1999; Shirasu *et al.*, 2000; Leigh *et al.*, 2003; Queen *et al.*, 2004). The overall resolution of the genetic structure revealed by the *Sukkula*-9900-LARD retrotransposon could be improved further by analyzing the population with a variety of retrotransposons that show differing transposition histories or a combination of different molecular markers. Association studies using the combination of molecular marker data and phenotypic data could yield potentially useful information on alleles at loci of interest (Dreisigacker *et al.*, 2005). S-SAP markers based on *BARE-1*/ *Wis-2-1A* barley retrotransposons were found to be broadly distributed among all wheat chromosomes and on a wheat RFLP linkage map (Queen *et al.*, 2004) although a tendency for S-SAP markers to cluster has been observed for several retrotransposon primers including *Sukkula* (Rodriguez *et al.*, 2006). The S-SAP markers used in this study were not mapped and therefore no conclusions can be made regarding their genomic location and possible linkage to agronomically significant traits. However, the results of the PCoA and UPGMA dendrogram showed that at least some of these markers could potentially be linked to agronomic traits e.g. growth habit and the presence of the rye chromosome segment 1RS. To maximize the utility of gene bank germplasm for breeding purposes, it is essential to characterize the material for agronomically important traits and resistance to abiotic and biotic stresses.

Wheat breeding aims to improve cultivars through crossings and selections of the genetic diversity available in the gene pools of wheat and ultimately the production of new useful allele combinations. In conclusion, the results of the present study show that the retrotransposon *Sukkula*-9900-LARD is highly suitable for S-SAP diversity studies in wheat; and that the extent and nature of genetic variation in Nordic bread wheat is heavily affected by plant breeding strategies, which may have implications for future wheat breeding and for the conservation of wheat genetic resources.

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Table 1. *Material used in sequence-specific amplification polymorphism (S-SAP) analysis of Nordic wheat landraces and cultivars. R region of origin (D Denmark, F Finland, N Norway, S Sweden), G growth habit (s spring, w winter), Year of release or approval*

Acc.no[a]	Name	Pedigree[b]	G	R	Year
AF	Abba	A 0336.19/Catamaran	w	D	2002
AF	Abika	Brigadier/A 91295.16	w	D	2003
PF	Alrø	PF 97227-2/Parade	w	D	1990s
NGB-4770	Als	(s) landrace from Als	w	D	1937
NGB-9122	Anja	Kranich/Caribo	w	D	1980
NGB-0004	Ankar	Iduna/Bore	w	S	1928
NGB-0006	Ankar II	Ankar/Saxo	w	S	1928
JIC-0113	Apu	Garnet/Pika	s	F	1949
NGB-0007	Äring	Ankar/Saxo	w	S	1932
NGB-0011	Äring III	(s) Äring I	w	S	1940
NGB-0014	Aros	Äring/Ergo	w	S	1947
NGB-2143	Ås	(s) Lv from Norway	s	N	1926
JIC-3196	Aura	Ertus/Vakka	w	F	1976
SW	Avle	22279M15/20299M12//Canon	s	S	1996
SW	Ballad	Sv85297/Sv85568	w	S	2001
JIC-1208	Banco	WW6518/WW6431//Ankar II	w	S	1953
NGB-11315	Bastian	Bajio-66/Runar/4/Yaktana/Norin10/Brevor/3/ Moystad/5/Rollo/Magnif/4/Sonora/ Tezanos-Pintos-Precoz//Nainari/3/Moystad	s	N	1989
NGB-13659	Bjørke	SvU75630/Rida	w	N	1998
NGB-9691	Blanka	Extra Kolben II/Wilhelmina	s	S	1950
NGB-6695	Bore	(s) from English wheat	w	S	1902
NGB-8933	Borg Abed	Trifolium 14/Abed 92	w	D	1967
NGB-4494	Borstvete fra Gotland	Landrace	w	S	-
NGB-2125	Børsum	Landrace	s	N	-
NGB-8946	Brødtorp pajo	Landrace	w	D	-
NGB-7456	Brons	Aurore/Extra Kolben II	s	S	1945
NGB-7481	Canon	Sicco/2*WW-12502//2*Sappo/3/Kadett	s	S	1988
SW	Curry	Canon s/Nemares//Kadett Mp1	s	S	1994
NGB-9955	Dacke	P18/17269//19151	s	S	1990
NGB-9708	Dala	Landrace	s	S	-
NGB-6410	Dalarna	Landrace	s	S	-
NGB-7027	Dania	Landrace	w	D	-
NGB-6679	Diamant	Kolben/Hallands (landrace)	s	S	1928
NGB-6681	Diamant II	Diamant/Extra Kolben II	s	S	1938
NGB-7469	Drabant	CItr 12633/Ring ^6	s	S	1972
SW	Dragon	Sicco/1250^2/3/Sappo^2/5/Kadett	s	S	1988
NGB-8957	Enger	Landrace	w	N	-
NGB-0008	Ergo	Ankar I/Jarl	w	S	1934
NGB-0012	Eroica	WW 5133/Äring	w	S	1943
NGB-0015	Eroica II	(s) Eroica I	w	S	1951
NGB-0017	Ertus	Eroica/Virtus	w	S	1953
NGB-6677	Extra Kolben	Kolben/unnamed line	s	S	1919
NGB-8923	Extra Kolben II	(s) Extra Kolben	s	S	1926
NGB-6694	Extra Squarehead (SWE)	(s) Leutenritzer Squarehead	w	S	1900
NGB-2434	Folke	Holme/Walde	w	S	1981
NGB-2126	Fram I	J-03/Mo-07	s	N	1930s
NGB-2127	Fram II	J-03/Mo-07	s	N	1938
NGB-6680	Fylgia I	Aurore/Extra Kolben	s	S	1933
NGB-6685	Fylgia II	Extra Kolben II/Aurore	s	S	1952
AF	Galicia	n/a	w	D	2000
NGB-8199	Gammel svensk landhvete	Landrace	w	S	-
SW	Gnejs	KosackMB/3*Kraka/4/Kurier	w	S	2001
NGB-6716	Gyllen II	Kron/Bore II	w	S	1938
NGB-0121	Haarajärvi ME0102; Apu	Landrace	s	F	-
NGB-6409	Halland	Landrace	s	S	-
NGB-9057	Hallandshvete	Landrace	s	S	-
NGB-6773	Hankkijan Ilves	Hja B 356/Vakka	w	F	1984
SW	Harnesk	WD-linje/Konsul	w	S	2001
NGB-8968	Haukiala Pirola	Landrace	w	F	-
NGB-4080	Hildur	Sv 60504/Starke	w	S	1976
NGB-2435	Holger	WW 2259-68/WW 2250-68	w	S	1981
NGB-0023	Holme	Starke//Odin/Banco	w	S	1972
NGB-13345	Hopea	Canadian Ruskea/Marquis	s	F	1936
NGB-0042	Horsmanaho ME201 Timantti	Landrace	s	F	-
SW	Hugin	Dragon (sib)/Nemares	s	S	1996
NGB-5153	Hunsballe R	(s) Jubile	w	D	1955
NGB-8973	Ideal	Trifolium 14/spontaneous cross	w	D	1929
NGB-0001	Iduna	(s) Squarehead	w	S	1911

Table 1. cont.

Acc.no[a]	Name	Pedigree[b]	G	R	Year
JIC-7535	Ilves	Hja B 356/Vakka	w	F	1987
NGB-0003	Jarl	Iduna/line from Sammetsvete from Uppland	w	S	1925
NGB-0131	Järvenkylä ME0302 Sep A	Landrace	s	F	-
NGB-0040	Jokikylä ME0505;Apu	Landrace	s	F	-
NGB-0348	Jyvä	(s) Vakka	w	F	1965
SW	Kadett	Kolibri/WW 439-66/Pompe-M	s	S	1981
NGB-11316	Kalle	n/s	w	N	1990
NGB-7457	Kärn	WW 8244/WW 8388	s	S	1946
NGB-7458	Kärn II	(s) Kärn	s	S	1947
JIC-0114	Kimmo-JIC	(s) Russian wheat	s	F	1949
NGB-13347	Kimmo-NGB	(s) Population of Pisarev, Russian wheat	s	F	1941
JIC-0800	Kiuru	Aurore/Sopu	s	F	1951
NGB-6676	Kolben	(s) landrace with wide variation or Heines Kolben	s	S	1892
NGB-8194	Konge II	(s) Konge (= Ideal/spontaneous cross)	w	D	1939
NGB-7482	Kosack	Mironovskaja 808/Starke M//Holme M	w	S	1984
NGB-2128	Kr Finset, Eikesdal	Landrace	s	N	-
NGB-9123	Kraka	Kranich/Caribo	w	D	1980
NGB-6708	Kron	Sol II/Pansar	w	S	1925
NGB-6388	Lading Skæghvede	Landrace	w	D	-
NGB-4406	Laitiala AP0103	Landrace	s	F	-
NGB-2129	Landvårkveite	Landrace	s	N	-
NGB-2130	Lanor	Norrøna/Lade	s	N	1970
NGB-6673	Lantvete från Dalarna	Landrace	s	S	-
NGB-4496	Lantvete från Gotland	Landrace	w	S	-
NGB-6674	Lantvete från Halland	Landrace	s	S	-
NGB-6691	Lantvete från Halland	Landrace	w	S	-
NGB-6692	Lantvete från Uppsala	Landrace	w	S	-
NGB-0122	Larinsaari ME0101; Apu	Landrace	s	F	-
NGB-13041	Lavett-NGB	WW118466/Kadett//Dragon	s	S	1992
SW	Lavett-SW	WW118466/Kadett//Dragon	s	S	1992
JIC-7542	Linna	TA A 2701/Virtus	w	F	1965
JIC-8372	Luja	Svenno//Hopea /Tammi	s	F	1981
NGB-11709	Manu	Ruso/Runar	s	F	1993
JIC-1159	Mendel	Standard/Trifolium 14	w	S	1926
SW	Mjölner	TL340/Starke//W25458	w	S	1996
NGB-0043	Monola ME1301	Landrace	s	F	-
JIC-0766	Møystad	(Mo 042-40)/Kärn II	s	N	1971
NGB-9118	Nana	Ibis/Stella	w	D	1975
JIC-7545	Nisu	(s) Vakka	w	F	1966
JIC-1319	Nora	Fram II/Sopu	s	N	1973
NGB-0021	Norre	Eroica/Virtus	w	S	1962
NGB-2133	Norrøna	Fram-II/Sopu	s	N	1958
NGB-6723	Odin	Gluten/Ergo	w	S	1949
NGB-6727	Ölve	Eroica I/K 01281 (mother line to Hansa)	w	S	1959
NGB-8922	Østby	Landrace	s	D	-
NGB-6707	Pansar III	(s) Pansar I	w	S	1923
NGB-6722	Pärl II	Sv 0912/Svea	w	S	1946
NGB-7464	Pompe	Ring/Svenno	s	S	1967
NGB-6688	Prins	Diamant II/Kärn II	s	S	1962
NGB-6682	Progress	Sv Ä 23-8/Extra Kolben II	s	S	1942
NGB-6698	Pudel	(s) Shiriff wheat from England	w	S	1910
NGB-7466	Rang	Ring ^5/Els	s	S	1968
NGB-2134	Reno	Els/T-110-21-41;Tammi/Kärn-II//Els	s	N	1975
NGB-6699	Renodlat Sammetsvete	Selection through purification of wheat from Ulltuna, Uppland	w	S	1910
SW	Revelj	Kanzler M15M28	w	S	2000
NGB-11317	Rida	MO-0944-15/Redcoat//Trond	w	N	1976
NGB-7462	Ring	Kain/Pondus	s	S	1957
NGB-6684	Rival	Diamant/Extra Kolben II	s	S	1952
NGB-6724	Robur	Skandia II/Sv 36-175	w	S	1949
NGB-2135	Rollo	Kärn-II/Norrøna	s	N	1963
NGB-6678	Rubin	Kolben/Dala (landrace)	s	S	1921
NGB-14118	Rudolf Rubin	WW 25449/Folke	w	S	1921
NGB-2136	Runar	Els/Rollo	s	N	1972
JIC-7551	Ruso	Reward/Pika	s	F	1967
NGB-7472	Saffran	WW 38-68/WW 11-68	s	S	1978
NGB-6687	Safir	Sv 1015/A 24-585	s	S	1955
NGB-7467	Sappo	WW 177-62/WW 176-62	s	S	1971
NGB-0120	Sarkalahti ME0101	Landrace	s	F	-
SW	Satu	Snabbe/Drabant//15962	s	S	1990
PF	Saxild	Britta//Pepital/Gawain	w	D	
NGB-0005	Saxo	(s) deviating plants of Tystofte Smaahvede II	w	S	1929

Table 1. cont.

Acc.no[a]	Name	Pedigree[b]	G	R	Year
NGB-0473	Sigyn II	Heid/Labors-Elite-05	w	N	1972
NGB-6383	Skandia	Kron/SV-0860-D	w	S	1935
NGB-6717	Skandia II	(s) Skandia	w	S	1939
NGB-2138	Skirne	Gelchsheimer/Särimner	s	N	1937
NGB-7483	Sleipner	WW 20102/CB 149//Maris Huntsman///Bilbo	w	S	1988
NGB-7183	Små II Tystofte	(s) Tystofte Smaahvede	w	D	1915
NGB-7465	Snabbe	Svenno/WW 7039 (= Kain/Kimmo)	s	S	1968
NGB-2139	Snøgg I	0843/Ås	s	N	1939
NGB-6700	Sol	(s) Landrace from Skåne, Sweden	w	S	1911
NGB-6701	Sol II	Sol I/Extra Squarehead II	w	S	1916
NGB-6715	Sol IV	Kron/Sol II	w	S	1937
NGB-13346	Sopu	Canadian Marquis/Ruskea	s	F	1935
NGB-9956	Sport	Citr 5484/PompeBM///Trippel ^3//WW 17269 ^4/WW 19151	s	S	1991
AF	Stakado	AD 7020/AO 7021	w	D	1994
NGB-6709	Stål	Sol II/Pansar	w	S	1927
NGB-8197	Stand Tystofte	(s) Squarehead	w	D	1907
NGB-0018	Starke	WW 11556/WW 11376	w	S	1959
NGB-0022	Starke II	(s) Starke I	w	S	1968
NGB-13479	Stava	Helge-M7D1/Helge-M7D2// WW-31254	w	S	1995
NGB-7184	Storaks Abed	n/a	w	D	1967
NGB-4783	Storvik sjundea	Landrace	w	F	
NGB-7476	Sunnan	Pompe 2r 19/Sappo//Drabant	s	S	1983
NGB-6725	Svale	Skandia II/Eroica I	w	S	1955
NGB-6704	Svea I	Pudel/Sammetsvete (landrace)	w	S	1924
NGB-7461	Svenno	WW 8244/WW 8388	s	S	1953
NGB-0355	Tähti	Kärn-I/JO-0172	s	F	1972
NGB-2141	Tautra	n/a	s	N	1983
NGB-0020	Thor	WW 11376/WW 11379	w	S	1961
NGB-6702	Thule II	Pudel/Sammetsvete (landrace)	w	S	1917
NGB-6714	Thule III	Thule II/Sv 0762	w	S	1936
NGB-0130	Timantti Paavo	Landrace	s	F	-
NGB-7471	Timmo	WW-152-65/Sappo	s	S	1979
NGB-7479	Tjalve	WW-20999/Benno;T-9111/449-73//15432;Reno/WW-16679//WW-15432;(DER)Benno	s	S	1990
NGB-9952	Tjelvar	Sture3D1/4/StureM3b2M5M7	w	S	1984
NGB-0359	Touko	Timantti/Hopea	s	F	1950
NGB-9016	Trifolium 14	(s)Wilhelmina	w	D	1925
NGB-7463	Troll	Ring//Pondus/Kärn	s	S	1967
NGB-0019	Trond	Virtus/WW 9344	w	S	1960
NGB-9953	Tryggve	Riley/Holme//18614/3/Helge	w	S	1990
NGB-2142	Trym	Huron/Fylgia-I	s	N	1948
NGB-9017	Tystofte Stakket	(s) Squarehead	w	D	1967
NGB-0351	Ulla	Tammi/TA-C-4431	s	F	1975
JIC-0858	Vakka	n/a	w	F	1959
SW	Vals	Can.M12 M14 M18 B9 B10/Can.M14 M15 B9	s	S	2001
JIC-0526	Varma	Svea/Lv-Orimattila,S.E.Finland	w	F	1933
NGB-9020	Varma Tammisto	Landrace	w	F	-
NGB-6675	Vårpärl	(s) Emma	s	S	1920
NGB-9109	Viking	Starke I/WW 14433	w	D	1962
SW	Vinjett	Tjalve M14/Tjalve M15//Canon	s	S	1998
AF	Vip	A 0336.19/Yacht	w	D	2001
NGB-6729	Virgo	Demeter/Virtus//Odin	w	S	1968
SW	Virke	n/a	w	S	1999
NGB-0013	Virtus	Ergo/Svea II	w	S	1945
NGB-10867	Vitus	Kleiber//Transec-7/2*Capa-2	s	D	1981
NGB-0024	Walde	Ergo/Svea II	w	S	1945
NGB-7473	Walter	Starke I/WW 14433	s	S	1972
NGB-8198	Warmland lantvete	Landrace	w	S	-
AF	Wasmo	Britta/Nova	w	D	1999
NGB-7474	William	WW-13-69/WW-41-69	s	S	1979
SW	Zebra	Ralle/Dragon	s	S	2001

[a] Accessions from AF Abed Fonden, NGB Nordic Gene Bank, JIC John Innes Centre, PF Pajbjerg Fonden, SW Svalöf-Weibull AB

[b] / Primary cross, // secondary cross, raised number preceding number of backcrosses, (s) selection ; n/a not available,

Table 2. *Number of accessions, S-SAP bands and gene diversity (*H*) for different categories in Sweden, Norway, Denmark and Finland partitioned by habit (w=winter, s=spring) and time period (1=before 1910, 2=1910-1969, 3=1970-2003)*

Country	Habit	Time	*H**	N acc.
Sweden	**w**		**0.173**	**65**
		1	*0.187*	*8*
		2	*0.140*	*42*
		3	*0.175*	*15*
	s		**0.212**	**50**
		1	*0.189*	*7*
		2	*0.152*	*22*
		3	*0.160*	*21*
	total **w+s**		**0.226**	**115**
		1	*0.207*	*15*
		2	*0.193*	*64*
		3	*0.217*	*36*
Norway	**w**		**0.165**	**5**
		1	-	*1*
		2	-	-
		3	*0.144*	*4*
	s		**0.153**	**19**
		1	*0.125*	*3*
		2	*0.114*	*9*
		3	*0.159*	*7*
	total **w+s**		**0.186**	**24**
		1	*0.142*	*4*
		2	*0.114*	*9*
		3	*0.206*	*11*
Denmark	**w**		**0.182**	**25**
		1	*0.129*	*4*
		2	*0.100*	*10*
		3	*0.208*	*11*
	s		**0.137**	**2**
		1	-	*1*
		2	-	-
		3	-	*1*
	total **w+s**		**0.195**	**27**
		1	*0.170*	*5*
		2	*0.096*	*10*
		3	*0.218*	*12*
Finland	**w**		**0.148**	**11**
		1	*0.100*	*3*
		2	*0.113*	*5*
		3	*0.103*	*3*
	s		**0.143**	**21**
		1	*0.104*	*9*
		2	*0.103*	*7*
		3	*0.136*	*5*
	total **w+s**		**0.185**	**32**
		1	*0.143*	*12*
		2	*0.169*	*12*
		3	*0.191*	*8*
All	**w**		**0.192**	**106**
		1	*0.139*	*16*
		2	*0.118*	*57*
		3	*0.158*	*33*
	s		**0.214**	**92**
		1	*0.139*	*20*
		2	*0.123*	*38*
		3	*0.152*	*34*
	total **w+s**		**0.234**	**198**
		1	*0.204*	*36*
		2	*0.201*	*95*
		3	*0.252*	*67*

*Differences in allele frequencies between categories result in a higher *H* for total than the individual categories.

Table 3. *Pairwise comparison of genetic differentiation (F_{ST}, averaged over pairs of population categories)**

Category	F_{ST}
Between countries	
within winter (w)	0.103
within spring (s)	0.149
within w without Norway	0.090
within s without Denmark	0.138
within w+s Sweden and Finland	0.113
within w+s All countries	0.124
Between growth habit	
within Sweden	0.159
within Norway	0.164
within Denmark	0.082
within Finland	0.220
within Sweden and Finland	0.172
within All countries	0.159
Between time period (per 2 decades)	
within Sweden	0.148
within Sweden w	0.176
within Sweden s	0.309
within All countries w+s	0.087

*Figures were calculated also without Norway or Denmark as Norway had only 5 winter wheat accessions and Denmark 2 spring wheat accessions.

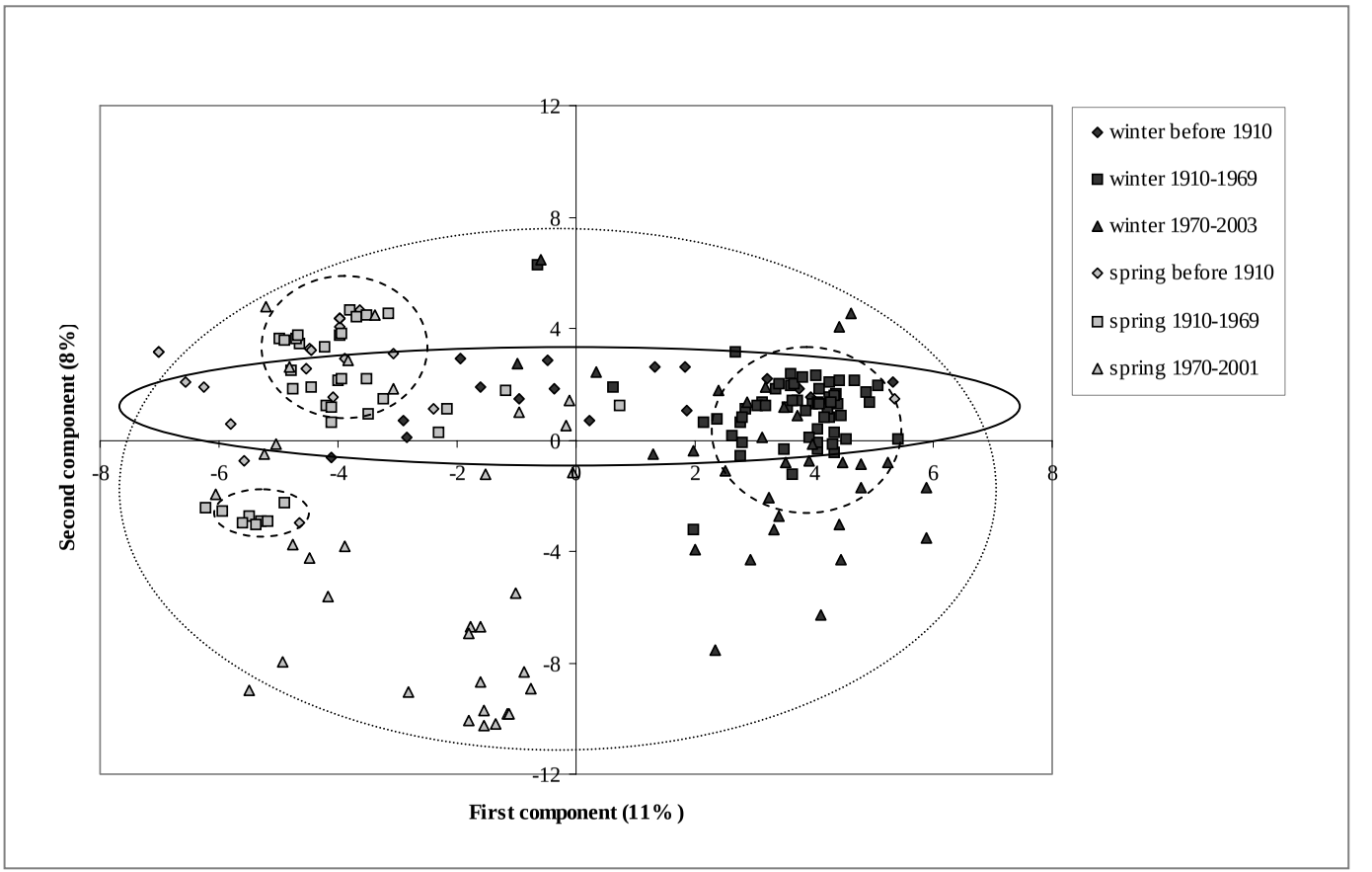

Figure 1. PCoA of Nordic bread wheat separated by growth habit and three time periods (temporal groups are indicated by (1) solid line = before 1910, (2) dashed line = 1910-1969, (3) dotted line = 1970-2003).

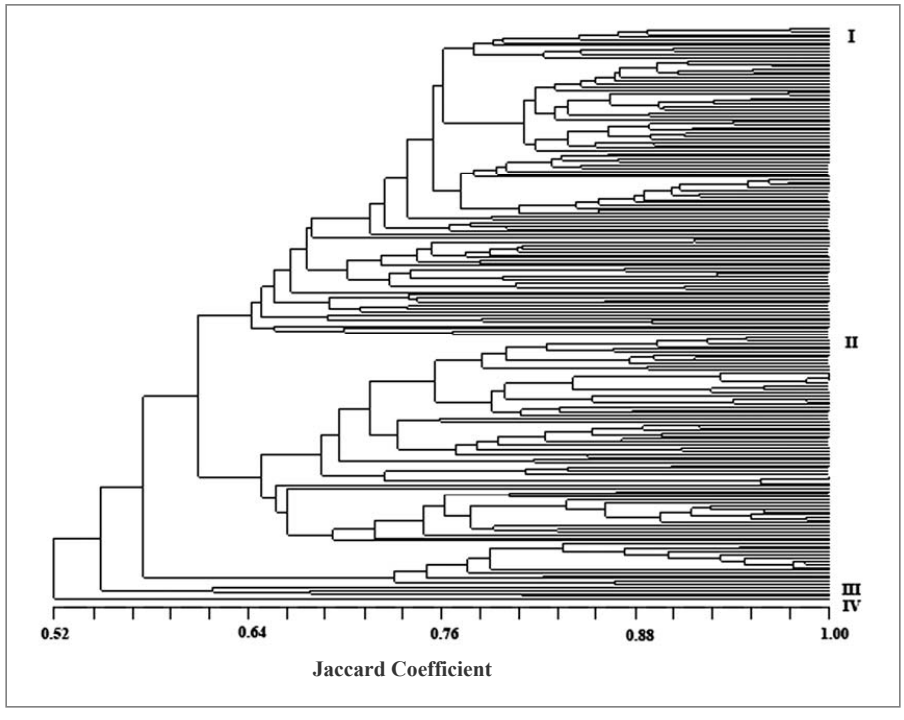

Figure 2. UPGMA dendrogram based on S-SAP markers in Nordic bread wheat (I = winter wheat and spring wheat acc. 'Hallandshvete', 'Brons', 'Safir', 'Lavett'-NGB, 'Lavett'-SW ; II = spring wheat; III = T1BL.1RS cvs. 'Sleipner', 'Tjelvar', 'Galicia', 'Abika'; IV = acc. NGB-4496 Lv from Gotland).

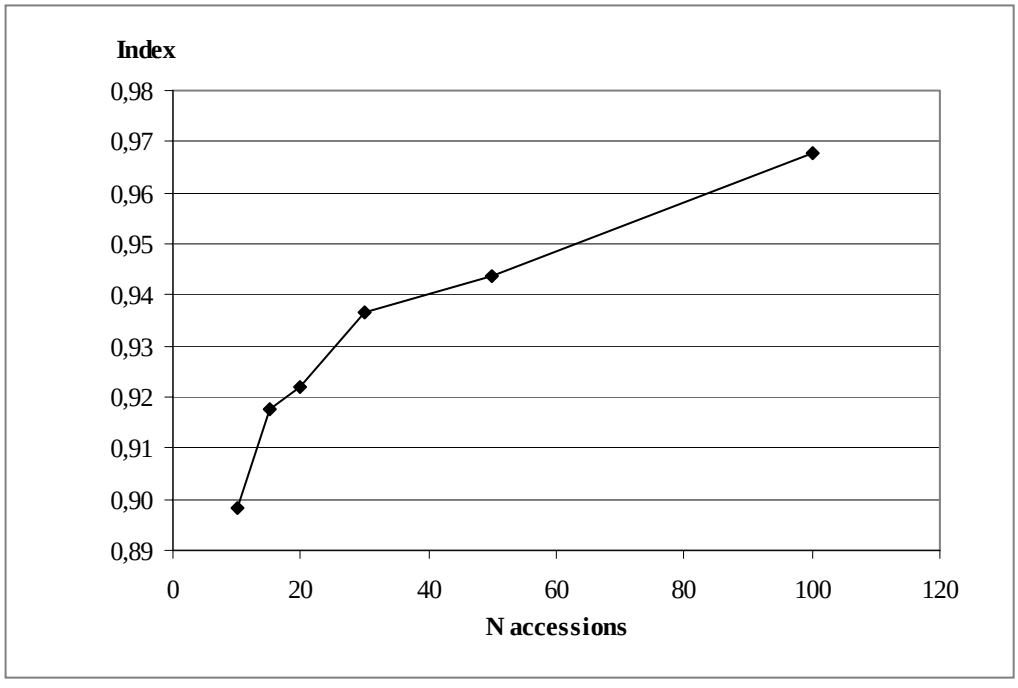

Figure 5. Proportion of loci with both presence and absence of alleles in core collections based on N accessions.

IV

Agronomic Performance and Multiple Disease Resistance in T2BS.2RL Wheat-Rye Translocation Lines

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ABSTRACT

Wheat (*Triticum aestivum* L.)-Rye (*Secale cereale* L.) T2BS.2RL translocations were shown to increase grain yield, resistance to biotic and abiotic stresses, and had minor effects on baking quality. The objective of this study was to test agronomic performance and resistance of a new wheat-rye translocation (T2BS.2RL, SLU) to powdery mildew (*Blumeria graminis* f. sp. *tritici* (DC.) E. O. Speer), leaf rust (*Puccinia triticina* Eriks.), stem rust (*Puccinia graminis* f. sp. *tritici* Eriks. & Henn.), stripe rust (*Puccinia striiformis* f. sp. *tritici* Westend.) and Hessian fly [*Mayetiola destructor* (Say)]. F_2 derived F_4–F_6 T2BS.2RL lines, non-translocation lines, and the wheat cultivar Holme were compared using intact seedlings or leaf segments. T2BS.2RL conferred seedling resistance to 17 powdery mildew isolates, 14 leaf rust and one stem rust pathotype. The T2BS.2RL lines were susceptible to 3 stripe rust pathotypes as seedlings, while showing adult plant resistance under natural conditions. Agronomic characters were compared in a 2-yr hill-plot field trial in Sweden. T2BS.2RL lines flowered 2 to 3 d later and had an increased number of spikelets per spike. The T2BS.2RL had no significant effect on yield, straw length, lodging, volume weight, 1000-kernel weight, fertility, α-amylase activity, or starch or protein content. The multiple disease resistance and the minor negative effects on agronomic performance of the T2BS.2RL, SLU translocation encourage its use in wheat breeding.

WHEAT-RYE CHROMOSOME translocations have been extensively used in wheat breeding, primarily as sources of disease resistance, but also to raise yield potential (Rabinovich, 1998). Genes for resistance against Russian wheat aphid (*Diuraphis noxia* Mordvilko) and green bug (*Schizaphis graminum* Rond.) infestation, and against powdery mildew (*Pm8*, *Pm17*), leaf rust (*Lr26*), stem rust (*Sr31*) and stripe rust (*Yr9*) infection have been transferred from chromosome 1RS into wheat in the form of T1RS.1BL translocation lines (Singh et al., 1990; Friebe et al., 1996; Hsam and Zeller, 2002). The short arm of chromosome 1R also has a positive effect on the yield, stress tolerance, and adaption of wheat (Merker, 1982; Carver and Rayburn, 1994; Moreno-Sevilla et al., 1995; McKendry et al., 1996; Villareal et al., 1998; Kim et al., 2004). The expression of rye genes for resistance, protein content and grain yield in T1RS.1AL and T1RS.1BL translocation lines is dependent on both the origin of the rye and the genetic background of the wheat (Kim et al., 2004). However, most of the major disease resistance genes on 1RS have now been overcome by virulent pathotypes and the absence of wheat chromosome arm 1BS is detrimental to bread-making quality (Martin and Stewart, 1986; Dhaliwal et al., 1987; Koebner and Shepherd, 1988).

In contrast, wheat chromosome 2B appears highly suitable for alien gene transfers since it lacks any of the storage protein genes (gliadins or glutenins) that affect baking quality (Knackstedt et al., 1994). Genetic and physical mapping has shown that 2B and 2R are largely homeologous to one another, but homeologous recombination is restricted to the distal regions of the chromosomes (Lukaszewski et al., 2004). In particular, 2RL is genetically equivalent to 2BL, while 2RS shares homeology with both wheat 2BS and 6BS, and cannot fully compensate for the absence of either of them (Naranjo et al., 1987; Devos et al., 1993). The presence of 2R in wheat increases water-use efficiency and rooting characteristics (Lahsaiezadeh et al., 1983; Ehdaie et al., 2003), while the addition of 2RL significantly increases the content of kernel arabinoxylans, which are important for both baking and nutritional quality of cereals (Vinkx and Delcour, 1996; Boros et al., 2002). Genes for resistance to powdery mildew (Heun and Friebe, 1990), leaf and stem rust (Brunell et al., 1999), Hessian fly (Friebe et al., 1990; Sears et al., 1992; Lee et al., 1996), and tolerance to barley yellow dwarf virus (Nkongolo and Comeau, 1998) have all been located to chromosome 2R. The presence of resistance genes, promising quality characters and the good agronomic performance associated with 2RL make this chromosome arm an attractive target for wheat improvement.

The T2BS.2RL translocation from Chaupon rye in wheat cultivar Hamlet (PI 549276) was identified in a regenerant from tissue culture and found to carry a single dominant gene (*H21*) for Hessian fly resistance (Sears et al., 1992; Hatchett et al., 1993). Investigations of the milling and baking properties of the Hamlet translocation line found a slight reduction in test weight, flour yield, kernel hardness, mixograph-mixing time, and bake-

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Abbreviations: GISH, genomic in situ hybridization.

mixing time; and a slight increase in flour color and water absorption that could be overcome by selecting for earliness (Knackstedt et al., 1994). A T2AS.2RL (Imperial rye) translocation in the wheat cultivar Chinese Spring was superior to normal Chinese Spring with respect to grain yield, shoot biomass at maturity, root biomass, and water use efficiency (Lahsaiezadeh et al., 1983; Ehdaie et al., 1991, 1998). The presence of the Hamlet T2BS.2RL in cv. Pavon and Karl wheat backgrounds delayed maturity, while the effects on number of seeds per spike, grain yield and harvest index were background-dependent (Fritz and Sears, 1991; Ehdaie et al., 2003).

The objective of the present study was to investigate the effects of a new wheat-rye T2BS.2RL translocation (T2BS.2RL, SLU). We report the outcomes of experiments testing the resistance to powdery mildew, leaf rust, stem rust, stripe rust, Hessian fly, and agronomic performance, and discuss its potential for wheat improvement.

MATERIALS AND METHODS

Plant Materials

A T2BS.2RL, SLU translocation line was isolated from crosses between normal winter wheat cultivars Holme and Kraka and a double disomic substitution line in which 1R and 2R replaced, respectively, 1B and 2B (Merker and Forsström, 2000). The homozygous T2BS.2RL, SLU translocation carrier was crossed with cv. Holme. Lines homozygous for either the presence or absence of the T2BS.2RL, SLU translocation were isolated in F_2 and F_3 generations, by means of mildew resistance response tests and chromosome C-banding (Merker and Forsström, 2000). Each line described in the present study is descended from a different F_2 plant. F_2 derived F_4–F_6 lines were used to compare translocation lines (i.e., lines homozygous for the T2BS.2RL translocation), and non-translocation lines (i.e., lines without the translocation, homozygous for chromosome 2B). All the lines are fully fertile and have normal seed-set. Chromosome C-banding and genomic in situ hybridization (GISH) were used to detect the presence or absence of T2BS.2RL in the lines used for determining disease and pest resistances. Cultivar Holme was used as a universal control.

International sets of differential tester lines carrying specific genes for disease resistance were used to compare the infection type patterns of translocation and non-translocation lines with identified genes. The cv. Chancellor powdery mildew (*Pm*) near-isogenic lines, and TP114/2*Starke for *Pm6*, are maintained at Lehrstuhl für Planzenbau, Freising-Weihenstephan, Germany. The North American and Mexican leaf rust (*Lr*) resistance tester lines and International, North American, and Australian stem rust differentials (McIntosh et al., 1995) are maintained at CIMMYT, Mexico. The International, European, Australian, and North American stripe rust (*Yr*) differentials (McIntosh et al., 1995) are maintained at the John Innes Centre, UK. The check lines H3, H5, H6, H7H8, and cv. Hamlet used in the Hessian fly experiment are maintained at USDA/ARS Dep. of Entomology, Purdue University, West Lafayette, U.S.A.

Powdery Mildew Experiments

Detached seedling leaf segment tests of translocation, non-translocation lines and cv. Holme were conducted with 17 European powdery mildew single-spore isolates (No. 2, 5, 6, 9, 10, 12, 13, 14, 15, 16, 17, 70, 72, 76, 90, 98, and 117) with virulence against combinations of the major resistance genes *Pm1a, Pm1b, Pm1c, Pm2, Pm3a, Pm3b, Pm3c, Pm3d, Pm3e, Pm3f, Pm4a, Pm4b, Pm5a, Pm5b, Pm6, Pm7, Pm8, Pm9, Pm17, Pm19, Pm22,* and *Pm29* (Hsam and Zeller, 2002). The isolates are maintained at the Lehrstuhl für Planzenbau, Freising-Weihenstephan, Germany. Seedling tests were performed on primary leaf segments maintained on 6 g/L agar with 35 mg/L benzimidazole. Each set of leaf segments was inoculated with one single-spore isolate at a time. The methods of inoculation and conditions of incubation followed Lutz et al. (1995). Ten plants of each line were used in two-three replications for each isolate. Infection types were recorded 10 d after inoculation using a scale of 0 to 9, where 0 = no visible disease symptoms and 9 = 50 to 100% leaf area covered with sporulating colonies (Heun and Friebe, 1990). Greenhouse tests were performed to determine the effectiveness of resistance at the adult plant stage. Three seedlings from each line and replication were grown to maturity and subjected to the highly virulent, naturally occurring powdery mildew pathotypes present in the greenhouse at Lehrstuhl für Planzenbau, Freising-Weihenstephan, Germany. The percentage infected leaf area on the penultimate leaf (F-1 leaf) was first assessed 2 wk after flowering, and thereafter every week until maturity.

Leaf Rust Experiments

The reaction to leaf rust infection was tested on intact seedlings of translocation, non-translocation lines, and cv. Holme using six Mexican pathotypes, and on detached seedlings using eight European single-spore isolates. The Mexican pathotypes CCJ/SP, MBJ/SP, MCJ/QM, MCJ/SP, TBD/TM, and TCB/TD carrying different combinations of virulence against *Lr1, Lr2a, Lr2b, Lr2c, Lr3, Lr3bg, Lr10, Lr11, Lr12, Lr13, Lr14a, Lr14b, Lr15, Lr17, Lr18, Lr20, Lr22a, Lr22b, Lr23, Lr26, Lr27+31, Lr28, Lr34, Lr35, Lr37,* and *LrB*, are maintained at CIMMYT, Mexico. The European single-spore isolates S-12, S-28, S-29, S-48, S-71, Pt-8, Pt-9, and Pt-60 with combinations of virulence against *Lr1, Lr2a, Lr2b, Lr2c, Lr3, Lr3bg, Lr3ka, Lr10, Lr11, Lr12, Lr13, Lr14a, Lr14b, Lr15, Lr16, Lr17, Lr18, Lr20, Lr21, Lr22a, Lr22b, Lr23, Lr26, Lr27+31, Lr28*, and *Lr34*, are maintained at the Lehrstuhl für Planzenbau, Freising-Weihenstephan, Germany. Ten intact 10-d-old seedlings per line were challenged with each pathotype, by spraying with urediniospores suspended in the light-weight mineral oil Soltrol 170 (Phillips 66, Bartlesville, OK) (2–3 mg/ml). Inoculated plants were incubated in a dew chamber overnight at 18 to 20°C and then transferred to greenhouse chambers at 18 to 22°C, with 16 h of daylight (Singh and Rajaram, 1991). The detached leaf segment tests followed the methods of Lutz et al. (1995), except that after inoculation, the material was held in a dark, humid chamber overnight. The tests were conducted with 10 to 12 plants per line in two replications. Infection types were recorded 9 to 12 d after inoculation, using a scale of 0 to 4 similar to that described by Stakman et al. (1962) where 0 to 2 is considered resistant to moderately resistant, and 3 to 4 moderately susceptible to susceptible.

Stem Rust Experiment

The Mexican stem rust pathotype RTR with virulence against *Sr5, Sr6, Sr7a, Sr7b, Sr8a, Sr8b, Sr9a, Sr9b, Sr9d, Sr9f, Sr9 g, Sr11, Sr12, Sr15, Sr16, Sr19, Sr20, Sr21, Sr23, Sr28, Sr34, Sr36, SrP1,* is maintained at CIMMYT, Mexico. A set of 10 8-d-old seedlings of translocation, non-translocation lines, cv. Holme, and tester lines were inoculated by spraying with urediniospores suspended in Soltrol 170, placed in a dew chamber overnight at 18 to 20°C, and transferred to a greenhouse at 18 to 22°C. Infection types were scored after 12 to 14 d based on a scale of 0 to 4 for stem rust (Singh, 1991) where 0 to 2 is con-

sidered resistant to moderately resistant, and 3 to 4 moderately susceptible to susceptible.

Stripe Rust Experiments

An equal parts mixture of the European stripe rust isolates WYR 94–519, WYR 96–17 and WYR 96–502 with virulence against *Yr1, Yr2, Yr3, Yr4, Yr6, Yr9*, and *Yr17* were used to evaluate resistance in the translocation, non-translocation lines, and cv. Holme. To break the dormancy of the urediniospores stored over liquid nitrogen, the inoculum was held at 40°C for 5 min. The urediniospores were mixed with an equal volume of talcum powder and air sprayed on to 10 10-d-old seedlings per line and tester line, which had been pre-sprayed with ddH_2O containing a few drops of Tween 20 (Sigma Chemical Company, St. Louis, MO) as a wetting agent. The inoculated plants were placed in a dark incubator room at 10°C and 95% humidity for 24 h to optimize spore germination and then transferred to a containment greenhouse at 15/18°C and 16/8 h day/night cycle. Infection types were recorded 12 to 14 d after inoculation, using a scale of 0 to 4 (McIntosh et al., 1995) where 0 to 1 is resistant, 2 moderately resistant, and 3 to 4 susceptible (Boyd and Minchin, 2001).

Hessian Fly Experiment

Translocation, non-translocation, cv. Holme, and tester lines were evaluated for reaction to Hessian fly biotype L and biotype E maintained at USDA/ARS Dep. of Entomology, Purdue University, West Lafayette, U.S.A. The tests were performed using a set of 10 to 20 individuals per line according to procedures described by Hatchett et al. (1981) and Foster et al. (1988). Individual plants were scored for reaction to larval feeding 10 to 14 d after infestation. Susceptible plants were stunted, blue-green and had broad second and third leaves. Resistant plants retained their normal height and color.

Field Experiments

Field trials were conducted in Alnarp, southern Sweden, over 3 yr (2001, 2002, and 2003). Hill plots consisting of 15 plants per plot were sown in trays in the greenhouse, vernalized for 10 wk at 2 to 4°C and planted in a randomized complete-block design in the field in spring. In all, 85 T2BS.2RL line hill plots, 72–73 non-translocation line hill plots, and 21 cv. Holme hill plots were assessed. The spacing between hill plots was 110 cm and the trial was surrounded by a row of winter wheat cv. Kosack. The field was fertilized with NPK and micronutrients (75 kg N/ha) immediately after planting. In 2001 and 2003, the trial was treated with 1 L/ha of Tilt Top 500 EC (cis-4-[3-(4-tert-butylfenyl)-2-metylpropyl]-2,6-dimetylmorfolin and 1-[2-(2,4-diklorfenyl)-4-propyl-1,3-dioxolan-2-yl-metyl]-1H-1,2,4-triazol) (Makhteshim-Agan, Leusden, Holland) to control fungal infection. During 2002, no fungicide treatment was given, exposing the trial to a natural stripe rust epidemic. Because of severe damage to non-translocation carrier lines and cv. Holme, only the data from years 2001 and 2003 were analyzed for agronomic performance.

The following parameters were recorded for each hill plot: grain yield per hill plot (g); heading time (days after half the spikes of the first hill plot had emerged); straw length (cm); lodging (100% = fully upright); grain volume weight (kg/hl); 1000-kernel weight (g); spike size (number of spikelets per spike) and fertility (number of seeds per spikelet of one well developed spike). Grain α-amylase activity was measured according to Bernfeld (1951) for the 2003 trial. Starch and protein concentration (%) was determined for the 2001 trial on a dry weight basis, according to ICC methods, by the Swedish Cereal Laboratory, Svalöf Weibull AB, Svalöv, Sweden.

Analyses of variance were performed for each character measured for each year and combined across years using General Linear Modeling (Minitab Release 14 statistical program). The datasets were normally distributed, except for lodging, as there was no variation in this character. *t* tests were used for mean comparisons.

Coleoptile Color Experiment

During vernalization, variation for seedling (red/green) coleoptile color was observed, and an experiment was conducted to study this trait. Ten seeds per each of 26 translocation, 21 non-translocation, and 6 cv. Holme hill plots harvested during the year 2003, were germinated on Wettex paper (FHP Vileda Professional, Liege, Belgium) for 3 d in a growth chamber at 19°C and transferred to an unheated greenhouse chamber 2 to 5/0°C and 10/14 h day/night cycle. After 11 d, the coleoptiles were 1–2 cm high and the color was recorded.

RESULTS AND DISCUSSION

Disease and Pest Resistance

The infection types of the differential tester lines corresponded to expected results (Singh, 1991; Singh and Rajaram, 1991; McIntosh et al., 1995; Hsam and Zeller, 2002) except for powdery mildew isolate no. 17 which was virulent against *Pm8*. The results from all the disease and pest resistance experiments are summarized in Table 1. The lines carrying the T2BS.2RL, SLU translocation were completely resistant to all 17 powdery mildew isolates at the seedling stage and to the mixture of isolates at the adult plant stage. The leaf rust and stem rust experiments showed similar results, although infection types in the translocation carriers were consistently higher when challenged with the Mexican leaf rust pathotypes than with the European leaf rust single-spore isolates, most likely because of different experimental methods (intact seedling vs. leaf-segment method). The non-translocation carrier lines and cv. Holme were susceptible to all the powdery mildew and rust pathotypes used in the study. Thus the genetic basis of the resistance in the T2BS.2RL, SLU translocation is almost certainly ascribable to gene(s) located on the 2RL rye segment. The highly consistent infection types produced by the different translocation lines showed that the resistance to powdery mildew, leaf rust, and stem rust was independent of genetic background.

In contrast, the T2BS.2RL, SLU translocation did not confer seedling resistance to any of the European stripe rust isolates, showing the same infection types as the non-translocation lines and cv. Holme. Preliminary results from adult plant experiments using the same isolate mixture does however suggest that the T2BS.2RL, SLU confers adult plant resistance to stripe rust (resistant to moderately resistant) compared to the non-translocation lines and cv. Holme (moderately resistant to susceptible) (unpublished results, 2004). This result is consistent with our 2002 field observations, when no fungicide was applied. In the presence of a heavy natural stripe rust epidemic, the translocation carriers were resistant to mod-

Table 1. Comparison of seedling and adult plant resistance (APR) to powdery mildew (Pm), leaf rust (Lr), stem rust (Sr), stripe rust (Yr) and Hessian fly (HF) in T2BS.2RL translocation and non-translocation lines, and wheat cultivar Holme.†

	T2BS.2RL translocation			Non-translocation			Holme	
Character	Mean	Range	No	Mean	Range	No	Mean	No
Pm isolates from Europe (17 isolates) seedling	0	0–1	10	8	7–9	10	8	1
Pm pathotypes from Europe (mix) APR	0	0	10	15%	5–25%	10	5	1
Lr isolates from Europe (8)	; 1	; – 1	10	4	3–4	10	4	1
Lr pathotypes from Mexico (6)	X	; 1 – X	5	3+	3C3–3+	5	3+	1
Sr pathotype from Mexico (1)	X	X	5	3+	3+	5	3+	1
Yr pathotypes from Europe (mix of 3) seedling	4	3–4	4	4	4	2	4	1
HF North American Biotype L	4/10	0–8/9–15	7	0/15	0/12–18	5	0/18	1
HF North American Biotype E	0/18	0–1/16–19	7	0/16	0/10–18	5	0/19	1

† No = number of lines (10–20 plants per line) used in the experiments. Powdery mildew seedling experiments were scored on an infection type scale from 0–9 where 0 = no visible signs of infection, 1 = small pustules and necrotic flecks, 7–9 = 50–100% leaf area covered with mycelium; Powdery mildew APR was scored as percentage infected leaf area; Leaf rust and stem rust experiments were scored on a scale from 0–4 where 0 = no uredinia or other macroscopic signs of infections, ; = no uredinia, but hypersensitive necrotic or chlorotic flecks of varying size present, 1 = small uredinia often surrounded by necrosis, 3 = medium-sized uredinia that may be associated with chlorosis or rarely necrosis; 4 = large uredinia without surrounding chlorosis; X = random distribution of variable-sized uredia on single leaf, + = uredinia somewhat larger than normal for the infection type, C = more chlorosis than normal; Stripe rust experiment was scored on a scale from 0–4 where 0 = no uredinia or other macroscopic signs of infections, 3 = sporulation with chlorosis; 4 = abundant sporulation without chlorosis; Hessian fly resistance was scored as number of resistant plants/number of susceptible plants using 10 to 20 plants per line.

erately resistant, while non-translocation carriers were heavily infected (Merker and Hysing, 2003). The stripe rust infection damaged agronomic performance to such an extent in the susceptible lines that this field trial had to be excluded from the present analysis. Cultivar Holme appears to carry some adult plant resistance to stripe rust, and the putative adult plant resistance located on the 2RL segment must therefore be further studied in a stripe rust susceptible wheat background.

Carriers of the T2BS.2RL, SLU translocation were susceptible to the two Hessian fly biotypes used in the study. This differs from the T2BS.2RL, Hamlet translocation which confers complete resistance to biotype L, which has the widest virulence range of the known biotypes (Hatchett et al., 1993), and presumably reflects variation in allelic content of the two independent rye sources.

Powdery mildew and rusts are economically destructive diseases of common wheat in many areas of the world (Roelfs et al., 1992; Hsam and Zeller, 2002). Resistance is one of the most effective, environmentally sound, and economic means of control (Pink, 2002). However, new pathogen races rapidly overcome most race-specific resistance genes and there is a continuous need to identify and incorporate effective resistance into new wheat cultivars. Although no race of powdery mildew, leaf rust, or stem rust was identified to be virulent on T2BS.2RL, SLU translocation carriers, any widescale use of the translocation would likely result in the breakdown of the resistance, as has occurred for the 1RS resistance complex (*Pm8-Lr26-Sr31-Yr9*) in the T1BS.1RL translocation. A breeding strategy combining the T2BS.2RL, SLU translocation with several effective resistance genes (pyramiding) in cultivars with high levels of partial (quantitative) resistance (McDonald and Linde, 2002; Wang et al., 2005) should promote the durability of resistance.

Agronomic Performance

The combined ANOVA (data not shown) indicated significant differences in means for heading date, straw length, lodging, grain volume weight, spike size, and fertility between years. However, because no significant change in the rank order of groups (translocation, non-translocation and cv. Holme) was observed in any year, the means across years are reported in Table 2. Significant differences within lines seen for the agronomic traits are common in hill-plot designs, and were probably caused by climatic factors, different genetic backgrounds, and interactions of genetic backgrounds with the translocation chromosome. The consistent infection types in the disease resistance experiments, however, indicated no, or at most minor, effects due to differences in genetic background within translocation and non-translocation lines.

No significant difference was found between non-translocation lines and control cv. Holme for any of the characters examined, showing that there was no effect due to the wheat-background. Therefore, any significant difference between the translocation and non-translocation groups could be ascribed to the presence of the T2BS.2RL translocation. The presence of the T2BS.2RL, SLU significantly increased the number of spikelets per spike (+12%), possibly because of the significantly delayed heading by 2 to 3 d compared to non-translocation lines and cv. Holme. There was no significant difference between the translocation, non-translocation lines, and cv. Holme for grain yield, straw length, lodging, grain volume weight, 1000-kernel weight, fertility, grain α-amylase activity, or grain starch and protein content (Table 2).

The Hamlet T2BS.2RL translocation delays maturity and increases the number of seeds per spike (Fritz and Sears, 1991; Ehdaie et al., 2003). Similarly, the T2BS.2RL, SLU translocation delays heading and increases the number of seeds per spike via a positive effect on the number of spikelets per spike (Table 2). The Hamlet T2BS.2RL translocation in wheat cv. Karl increases grain yield, aerial biomass at maturity, seeds per spike, and seeds per plant, but delays maturity, and reduces grain weight and harvest index (Fritz and Sears, 1991). Its presence in wheat cv. Pavon reduces the number of spikes and delays maturity, but has no affect on either harvest index or grain yield (Ehdaie et al., 2003). This inconsistency shows that the effect of the Hamlet T2BS.2RL is background dependent and operates via a genetic interaction between genes on the 2RL segment

Table 2. Comparisons of means and standard deviations (SD) of agronomic performance for T2BS.2RL translocation and non-translocation lines, and wheat cultivar Holme grown at Alnarp, Sweden 2001 and 2003.

	T2BS.2RL translocation			Non-translocation			Holme		
Character	**Mean**	**SD**	**No**	**Mean**	**SD**	**No**	**Mean**	**SD**	**No**
Yield per hill (g)	61.3	29.7	85	61.1	30.7	73	56.6	31.5	21
Heading (days after first hill)†	8.2 a	2.9	85	5.6 b	2.8	73	5.8 b	3.0	21
Straw length (cm)	93.1	10.2	85	92.3	9.6	73	88.3	9.1	21
Lodging (%)	98.4	4.6	85	97.5	4.6	73	100.0	0.0	21
Grain volume weight (kg/hl)	75.5	4.4	85	73.7	5.1	73	72.9	5.2	21
Thousand kernel weight (g)	37.4	4.5	85	37.3	5.4	72	36.6	5.4	21
Spike size (spikelets/spike)†	24.0 a	2.7	85	21.4 b	2.5	73	20.1 b	2.8	21
Fertility (seeds per spikelet)	3.1	0.5	85	3.1	0.3	73	3.1	0.3	21
Grain alpha amylase activity (μmol/mg)	1.0	0.3	37	0.9	0.3	48	0.9	0.3	12
Grain starch content (%)	66.8	1.4	38	67.0	1.0	34	67.1	2.0	10
Grain protein content (%)	16.5	1.4	38	15.0	1.2	34	16.0	1.8	10
Coleoptile color	Red	–	26	Green	–	21	Green	–	6

† Significantly different from the mean of the non-translocation lines at $p = 0.05$ based on t test; values with the same letter in the same row do not differ significantly at $P < 0.05$.

and those elsewhere in the wheat genome. In contrast, the agronomic effects of 1RS on cv. Pavon 76 reflected differences between rye origins rather than an interaction between rye and wheat genes (Kim et al., 2004).

Homologous recombination between alien chromosome segments transferred to wheat represents a strategy for increasing variation and for incorporating new genes for disease and pest resistance. Thus, for example, Mater et al. (2004) combined the powdery mildew resistance from the 1RS segment of a T1RS.1BL translocation with green bug resistance from the 1RS segment in a T1RS.1AL translocation. A further strategy lies in shortening the segment by de novo translocation and/or recombination. Thus, Friebe et al. (1994) were able to stabilize the powdery mildew resistance present in a monosomic 6RL(6D) substitution line by selecting a T6BS.6RL translocation chromosome. Similarly, the usefulness of the present T2BS.2RL, SLU and other T2BS.2RL translocations could be enhanced by recombination between different 2RL segments to combine pest and disease resistance in an agronomically desirable wheat background.

Molecular markers for 2RL segments from various rye sources, based on Random Amplified Polymorphic DNA (RAPD) (Seo et al., 1997; Brunell et al., 1999), moderately repetitive rye DNA (Lee et al., 1996), Amplified Fragment Length Polymorphism (AFLP) (Seo et al., 2001) and conversion of Random Fragment Length Polymorphism (RFLP) probes to Sequence Tagged Site (STS) markers (Forsström et al., 2003) have been described. These can be used to efficiently identify and track the rye segment in breeding programs. In this study, the result of the coleoptile color experiment showed that the translocation lines develop a deep red coleoptile color compared to the green or occasionally pink color of non-translocation lines and cv. Holme, allowing this trait to be used as a simple morphological marker. The presence of a coleoptile color gene(s) on chromosome 2R was proposed by Melz and Thiele (1990), but the red phenotype could also be caused by the absence of suppressors of anthocyanin pigments on the long arm of wheat chromosome 2B (Sutka, 1977). However, the stability of the marker has yet to be investigated, and its expression may be modulated by temperature and/or be dependent on the wheat background. The ability to genetically map 2RL using microsatellites and other marker types (Khlestkina et al., 2004; Camacho et al., 2005) may facilitate further studies and mapping of valuable genes in wheat-rye translocations.

In conclusion, the results of the present study on the T2BS.2RL, SLU translocation and studies on the Hamlet T2BS.2RL translocation (Hatchett et al., 1993; Knackstedt et al., 1994; Ehdaie et al., 2003) show that these translocations have a positive effect on yield, little influence on baking quality, and variable useful disease and pest resistances that could contribute to wheat improvement. The use of molecular markers in conjunction with genetic recombination between different rye 2RL segments in a wheat background would open the way to genetically define the useful genes present in this rye chromosome arm.

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Mapping of powdery mildew and leaf rust resistance on rye chromosome 2RL in wheat-rye translocation lines

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Abstract

Rye chromosome arm 2RL is homoeologous to wheat chromosome arm 2BL and highly suitable for transfer of disease and pest resistance through wheat-rye translocations. The characterization of resistance from different rye sources is important to effectively utilize translocations for wheat improvement. In the present study, the triticale derived T2BS.2RL wheat-rye translocation line HT02-9, was found to be completely resistant to eleven powdery mildew and eight leaf rust isolates, in contrast to the completely susceptible 'Imperial' rye derived T2AS.2RL translocation line P66. In order to map the resistance, an HT02-9 × P66 F_2 mapping population of 133 plants was analyzed using 117 microsatellite markers. Seven markers were found to be specific for 2RL, including three markers polymorphic between the two parental 2RL segments. A major quantitative trait locus (QTL) for resistance to one powdery mildew isolate and a minor QTL for resistance to one leaf rust isolate was significantly associated with marker *Xrms46*. Molecular markers could facilitate the identification and combination of effective disease resistance through homologous recombination of alien chromosome segments in translocation lines.

Introduction

Powdery mildew (*Blumeria graminis* (D. C.) E. O. Speer f. sp. *tritici*) and leaf rust (*Puccinia triticina* Eriks.) are parasitic fungi on bread wheat (*Triticum aestivum* L.) of economic importance for wheat production in many areas of the world (Sayre *et al.*, 1998). Genetic disease resistance is the most economical and environmentally sound method to reduce crop losses in agriculture (Bennett, 1984). Over 30 major genes conferring resistance to powdery mildew and over 50 genes for leaf rust resistance have been identified (McIntosh *et al.*, 2005). However, due to the high genetic variability of powdery mildew and leaf rust, new virulent pathogen race mutants can overcome individual race-specific genes in a short period of time and there is therefore a continued need for the identification and incorporation of new resistance genes.

Rye (*Secale cereale* L.) has been used as a source of insect and disease resistance in wheat breeding (Rabinovich, 1998). Several genes for resistance have been transferred from rye chromosome 1R into wheat in the form of T1BL.1RS translocations (Friebe *et al.*, 1996), including resistance against Russian wheat aphid (*Diuraphis noxia* Mordvilko) and green bug (*Schizaphis graminum* Rond.) infestation, and against powdery mildew, leaf rust, stem rust (*Puccinia graminis* Pers.) and stripe rust (*Puccinia striiformis* Westend.) infection (Singh *et al.*, 1990; Friebe *et al.*, 1996; Hsam & Zeller, 2002). However, most of the resistance genes have been rendered ineffective by virulent pathogen races, and the absence of wheat chromosome arm 1BS is detrimental to bread-making quality (Martin & Stewart 1986; Koebner & Shepherd 1988).

In contrast to wheat chromosome 1B, wheat chromosome 2B lacks any of the storage protein genes (gliadins or glutenins) that affect baking quality (Knackstedt *et al.*, 1994) and is highly suitable for bread wheat improvement through chromosome engineering. The rye chromosome arm 2RL is genetically equivalent to wheat 2BL, while 2RS shares homoeology with both wheat 2BS and 6BS, and cannot fully compensate for the absence of either arm (Naranjo *et al.*, 1987; Devos *et al.*, 1993). Resistance against powdery mildew (genes *Pm7, Pm8* and *Pm17*) (Lind, 1982; Heun & Friebe 1990; Hsam & Zeller 1997), leaf rust (*Lr25* and *Lr45*) (Driscoll & Jensen, 1965; McIntosh *et al.*, 1995a), stem rust (Brunell *et al.*, 1999), Hessian fly [*Mayetiola destructor* (Say)] (*H21, H25*) (Friebe *et al.*, 1990; Sears *et al.*, 1992; Lee *et al.*, 1996) and tolerance to barley yellow dwarf virus (Nkongolo & Comeau, 1998) have been located to chromosome 2R. The presence of 2R in wheat has a positive effect on yield (Fritz & Sears, 1991), water-use efficiency and rooting characteristics (Lahsaiezadeh *et al.*, 1983; Ehdaie *et al.*, 2003). The 2R chromosome also carries genes for kernel arabinoxylans, that are important for both the baking and nutritional quality of cereals (Vinkx & Delcour, 1996; Boros *et al*., 2002). The combination of resistance genes, promising quality characters and the good agronomic performance associated with 2RL make this chromosome arm an attractive target for wheat improvement.

Wheat-rye translocation lines involving rye chromosome 2RL have not been extensively used in breeding despite many positive characters. A T2AS.2RL 'Imperial' rye translocation in the wheat cultivar 'Chinese Spring' was found to be

superior to normal 'Chinese Spring' with respect to grain yield, shoot biomass at maturity, root biomass and water use efficiency (Lahsaiezadeh *et al.*, 1983; Ehdaie *et al.*, 1991, 1998). A T2BS.2RL translocation from octoploid triticale in a 'Holme' and 'Kraka' wheat backgound was found to carry powdery mildew and leaf rust resistance, and had no negative effects on major agronomic characteristics except for delayed maturity compared to non-translocation sister lines (Merker & Rogalska, 1984; Merker & Forsström, 2000; Merker & Hysing, 2003; Hysing et al., 2007). The 'Hamlet' T2BS.2RL translocation line from 'Chaupon' rye was identified in a regenerant from tissue culture and found to carry a single dominant gene (*H21*) for Hessian fly resistance (Sears *et al.*, 1992; Hatchett *et al.*, 1993). Investigations of the agronomic and quality characters of the 'Hamlet' translocation line revealed that it delays maturity, and confers a slight reduction in test weight, flour yield, kernel hardness, mixograph-mixing time and bake-mixing time, and a slight increase in flour colour and water absorption that could be overcome by selecting for earliness (Fritz & Sears, 1991; Knackstedt *et al.*, 1994; Ehdaie *et al.*, 2003).

Molecular markers are important to facilitate the identification and monitoring of introgressed alien chromosome segments. Several molecular markers for 2RL segments from various rye sources, based on random amplified polymorphic DNA (RAPD) (Seo *et al.*, 1997; Brunell *et al.*, 1999), moderately repetitive rye DNA (Lee et al., 1996), amplified fragment length polymorphism (AFLP) (Seo *et al.*, 2001) and conversion of random fragment length polymorphism (RFLP) probes to sequence tagged sites (STS) (Forsström *et al.*, 2003) have been described. However, no genes or quantitative trait loci (QTLs) for disease resistance have been mapped on rye chromosome 2RL.

Microsatellites or simple sequence repeats (SSRs) are highly polymorphic, co-dominant and easily detected in PCR-based assays. Microsatellites have been used to identify several *Pm* genes and alleles through bulked segregant analysis (BSA) of e.g. F_2 populations and F_3 families (Huang *et al.*, 2004; Zhu *et al.*, 2004; Röder *et al.*, 2004). Several microsatellite markers have been mapped to chromosome 2R of the genetic map of rye that could facilitate the mapping of single genes, dissection of QTLs and analysis of genetic diversity from rye (Schlegel *et al.*, 1998; Saal & Wricke, 1999; Khlestkina *et al.*, 2004; Schlegel & Korzun, 2006).

The objectives of the present study were to investigate powdery mildew and leaf rust resistance from two different rye sources in wheat-rye 2RL translocation lines, and to map the gene(s) responsible for resistance by using microsatellite molecular markers.

Materials and Methods

Plant materials

The T2AS.2RL 'Chinese spring' wheat - 'Imperial' rye translocation line P66 was originally devloped by. Dr. H. L. Shands at the University of Wisconcin (Sears, 1968) and provided by the late Professor E. R. Sears, Missouri, USA. The T2BS.2RL translocation line HT02-9 and non-translocation sister line HT02-8 were developed at the Swedish University of Agricultural Sciences. The HT-lines

were selected from F_2 derived F_4-F_6 crosses between winter wheat cultivars 'Holme' and 'Kraka' and a double disomic substitution line in which 1R and 2R from octoploid triticale replaced 1B and 2B, respectively (Merker & Rogalska, 1984; Merker & Forsström, 2000). The presence or absence of T2BS.2RL in HT02-9 and HT02-8 was confirmed by chromosome C-banding. A mapping population of 133 F_2 lines from one selfed F_1 hybrid was developed for chromosome 2RL from a cross between the T2BS.2RL HT02-9 and the TAS.2RL P66 chromosome translocation lines.

Disease assessments

The HT02-9, HT02-8 and P66 translocation lines were challenged with eleven powdery mildew and eight leaf rust isolates maintained at Lehrstuhl für Pflanzenzüchtung, Freising-Weihenstephan, Germany. The wheat cultivars 'Holme', 'Kraka', 'Chinese Spring', 'Kanzler' and 'Vuka' were used as controls. The 'Chancellor' powdery mildew near-isogenic lines, the North American and Mexican leaf rust resistance tester lines, and the wheat-rye translocation lines 'Transec' with *Pm7* and *Lr25,* 'Disponent' with *Pm8* and *Lr26,* and 'Amigo' with *Pm17* from rye and *Lr24* from *Thinopyrum ponticum* Host D. R. Dewey *(syn Agropyron elongatum)* were included to compare the infection type patterns with those of identified genes (McIntosh *et al.*, 1995b; Hsam & Zeller, 1997; Hsam & Zeller, 2002), and are maintained at Lehrstuhl für Pflanzenzüchtung, Freising-Weihenstephan, Germany.

The European powdery mildew single-spore isolates used in the study (no. P-2, P-5, P-6, P-9, P-10, P-12, P-13, P-14, P-15, P-16, and P-17) carry virulence against combinations of the major resistance genes *Pm1a, Pm1b, Pm1c, Pm2, Pm3a, Pm3b, Pm3c, Pm3d, Pm3e, Pm3f, Pm4a, Pm4b, Pm5a, Pm5b, Pm6, Pm7, Pm9, Pm17, Pm19, Pm22* and *Pm29* (Hsam and Zeller 2002). The European leaf rust single-spore isolates (no. S-12, S-28, S-29, S-48, S-71, Pt-8, Pt-9, and Pt-60) carry virulence against different combinations of *Lr1, Lr2a, Lr2b, Lr2c, Lr3, Lr3bg, Lr3ka, Lr10, Lr11, Lr12, Lr13, Lr14a, Lr14b, Lr15, Lr16, Lr17, Lr18, Lr20, Lr21, Lr22a, Lr22b, Lr23, Lr26, Lr27+31,* and *Lr28* (Hsam 2006, unpublished results).

Detached seedling tests were performed on primary leaf segments from ten seedlings per parental line and controls, maintained on 6g/l agar with 35 mg/l benzimidazole. The methods of inoculation and conditions of incubation followed Lutz and co-workers (1995), except for the leaf rust tests where the material was held in a dark, humid chamber overnight after inoculation, according to the method of Felsenstein and co-workers (1998). Each set of leaf segments from ten seedlings per line was inoculated with one single-spore isolate at a time in three replications. The disease responses for powdery mildew were recorded 10 days after inoculation using a quantitative scale of 0 to 10, where 0 = no visible disease symptoms and 10 = 100% leaf area covered with sporulating colonies (Lutz *et al.*, 1995). Infection types for leaf rust were scored 10-12 days after inoculation using a modified scale (Stakman *et al.*, 1962) from 0 to 4 as described by Felsenstein and co-workers (1998).

The HT02-9 × P66 F_2 mapping population was analysed with the powdery mildew isolate no. P-13 (virulence/avirulence formulae *Pm1a, 2, 3d, 4a, 4b, 4b, 5a, 5b, 7, 8, 9, 23, 29/1b, 1c, 3a, 3b, 3c, 3e, 3f, 6, 12, 16, 17, (19), 20, 21, 22, 24*) using two leaf segments per plant, and leaf rust isolates no. S-12 (virulence/avirulence formulae *Lr(2b), 2c, (3), (3bg), (3ka), 11, 12, 13, 14a, 14b, 15, 16, 17, 18, 20, 21, 22a, 22b, (23), 26, 27, 28, 29, 30, 31, 33, 34/Lr1a, 2a, 9, 10, 19, 24, 25*) and no. S-71 (virulence/avirulence formulae *Lr3bg, 3ka, (10), (12), (13), 14a, 14b, 15, (17), 18, 20,(22b), (23), 25, (28), 29, 30, 31, 33/Lr1a, 2a, 2b, 2c, 3, 9, 11, 16, 19, 21, 22a, 24, 26, 27*) using one leaf segment per plant. The isolates were chosen to include virulence against the designated powdery mildew genes *Pm7* and *Pm8*, and leaf rust genes *Lr25* and *Lr26* from rye.

Microsatellite marker analysis

Wheat and rye microsatellite markers located on chromosome arm 2RL published by Saal and Wricke (1999), Khlestkina and co-workers (2004), and unmapped rye microsatellites developed by Lochow-Petkus GmbH were selected as potential polymorphic markers. Information on the unmapped markers can be obtained from Dr. Viktor Korzun (v.korzun@kws.com) upon request.

Parental lines HT02-9 and P66, non-translocation line HT02-8, 'Holme', 'Kraka', and a subset of the mapping population were used to screen for polymorphism in the 2RL segment. The 'Chinese Spring'-'Imperial' rye disomic and ditelosomic wheat-rye addition lines CS-1R, CS-2R, CS-2RL, CS-3R, CS-4R, CS-5R, CS-6R, CS-7R, as well as 'Imperial' rye, and wheat cultivar 'Chinese Spring' were used to verify that microsatellite markers polymorphic between the parental lines were specific for rye chromosome arm 2RL.

DNA was extracted from one single leaf of each plant in the F_2 population according to a modified CTAB-protocol, and from 5-10 bulked individuals of the controls according to Doyle and Doyle (1990). Polymerase chain reaction was performed according to Röder and co-workers (1995) using 50-100 ng DNA and 0.05 units *Taq* DNA polymerase (Bioline GmbH) in a 12 µl reaction. Amplified fragments were analyzed in an automated laser fluorescence (ALF) express sequencer (Amersham-Biosciences, Freiburg, Germany). Fragment sizes were calculated using the computer programme FRAGMENT ANALYSER 1.02 and by comparing with internal size standards.

The map was constructed using MAPMAKER v. 3.0 (Lander *et al.*, 1987) applying the Kosambi (1944) mapping functions to calculate distances. The software QGENE v. 3.06 (Nelson, 1997) was used for QTL analysis. The association between phenotype and marker genotype was investigated using single-marker regression. The positions of identified QTLs were determined using interval mapping. QTLs with a maximum LOD (logarithm of odds ratio) score greater or equal to 3.0 were considered significant.

Results

Disease assessments

The disease responses of the differential tester lines corresponded to expected results (McIntosh *et al.*, 1995b, Hsam & Zeller, 2002) except for powdery mildew isolate no. P-17 which was virulent against *Pm8*. The results of the disease assessments showed that the T2BS.2RL HT02-9 translocation line was resistant to all the powdery mildew and leaf rust isolates tested, while the non-translocation line HT02-8, 'Holme', 'Kraka', 'Chinese spring' and T2AS.2RL P66 were mostly susceptible (Table 1). The powdery mildew resistance response pattern and leaf rust infection types in line HT02-9 differed from that of the differential tester 'Transec' carrying *Pm7* and *Lr25* in a T4BS.4BL-2RL translocation, differential tester 'Disponent' carrying *Pm8* and *Lr26* in a T1BL.1RS translocation, and differential tester 'Amigo' carrying *Pm17* in a T1AL.1RS translocation (Table 1). The leaf rust resistance in tester 'Amigo' is ascribed to gene *Lr24* located on the wheat-*Thinopyrum ponticum* translocation also present in this cultivar (Hsam & Zeller, 1997).

The phenotypic distribution of resistance responses to powdery mildew isolate no. P-13 in the HT02-9 × P66 F_2 population (Fig. 1) showed a clear distribution of 94 resistant plants (disease response 0-7) and 39 susceptible plants (disease response 9-10) that was not significantly different from an expected 3:1 segregation ratio using the chi-square goodness-of-fit analysis ($\chi^2 = 1.22$) and one degree of freedom. The phenotypic segregation ratio for leaf rust isolate S-12 (Fig. 2) was 68 resistant plants (IT=0-1) to 65 susceptible plants (IT=2-4), not significantly different from an expected segregation ratio of 1:1 ($\chi^2 = 0$). For leaf rust isolate S-71 (Fig. 2), 75 plants were resistant (IT=0-X) and 58 plants susceptible (IT=3-4), not significantly different from an expected ratio of 9:7 for two segregating genes ($\chi^2 = 0$). As no clear cut deviations allowing a qualitative approach could be determined for the powdery mildew and leaf rust infection phenotypes, the QTL method was chosen to analyze the results.

Microsatellite marker analysis

A total of 117 microsatellites were screened for presence on 2RL and polymorphism between 2RL in HT02-9 and P66. Seven microsatellite markers were found to be specific for 2RL and the amplification products from three rye derived microsatellites (RMS15, RMS46 and RMS47) displayed polymorphism between the parents (Table 2). The three markers formed one linkage group for chromosome arm 2RL with 1.6 cM between loci *Xrms15* and *Xrms46*, and 7.1 cM between *Xrms46* and *Xrms47* (Fig. 3).

The results of QTL analysis by interval mapping in QGENE (Table 3 and Fig. 3) showed that one QTL was detected closely linked to RMS46 for powdery mildew resistance to isolate P-13, that explained 52% of the phenotypic variation (LOD 20.52). One minor QTL closely linked to RMS46 was detected for resistance to leaf rust isolate S-12, that explained 11% of the phenotypic variation (LOD 3.35). The analysis for leaf rust isolate S-71 found no significant QTL.

The observed allelic distributions (Fig. 1 and 2) for microsatellite marker RMS46 and powdery mildew isolate P-13 (87:30), RMS46 and leaf rust isolate S-12 (54:26), and RMS15 and leaf rust isolate S-71 (60:20) were calculated and compared with expected distributions using chi-square goodness-of-fit analysis and one degree of freedom. No significant difference was found from the expected 3:1 segregation ratio for powdery mildew and RMS46 (χ^2 = 0.01). The allelic distribution of marker RMS46 for leaf rust isolate S-12 corresponded to a 2:1 ratio, supported by chi-square analysis that showed no significant difference (χ^2 = 0.01). The allelic distribution of marker RMS15 for leaf rust isolate S-71 was not significantly different from a 3:1 expected ratio (χ^2 = 0).

Discussion

Disease and pest resistance from different rye sources could be used in wheat breeding through wheat-rye translocation lines, provided that the substitution of the wheat segment does not adversely affect agronomic performance. Rye chromosome arm 2RL has been identified as suitable for gene transfers as it is homoeologous to wheat chromosome arm 2BL (Naranjo *et al.*, 1987; Devos *et al.*, 1993) that lacks major genes affecting baking quality (Knackstedt et al. 1994). In the present study, broad powdery mildew and leaf rust resistance was unambiguously correlated to a 2RL segment from an octoploid triticale in the T2BS.2RL wheat-rye translocation line HT02-9, in contrast to a 2RL segment from 'Imperial' rye in the T2AS.2RL translocation line P66 that was found to be susceptible to all the isolates tested (Table 1). The difference in resistance reflects the different origin of the rye segments.

The phenotypic distributions of resistance responses (Fig. 1 and 2) strongly indicated that resistance to powdery mildew isolate no. P-13 corresponded to one dominant gene, while the resistance to the two leaf rust isolates most likely involved more than one gene and/or genes segegating in the wheat background. QTL analysis and interval mapping allowed the identification of a major QTL for powdery mildew resistance to isolate no. P-13 and a minor QTL for leaf rust resistance to isolate no. S-12. Twenty-three quantitative trait loci have been reported in rye (Schlegel & Korzun, 2006) for several agronomic traits including flowering time, spike number, plant height, peduncle length, yield, thousand grain weight (Börner *et al.*, 2000), drought tolerance (Mohammadi *et al.*, 2003), chlorophyll content in leaves (Milczarski & Masojc, 2002) and restorer cytoplasmic male sterility (Stojalowski *et al.*, 2004). The present study is the first report of QTLs for powdery mildew and leaf rust resistance in rye.

The analysis of a population derived from a cross between two wheat-rye translocations possessing the same rye chromosome segment from different sources could be used also to find molecular markers for other traits and in other wheat-rye translocation lines. However, the feasability and success in finding polymorphic markers depends on the distance in relationship between the parents and the degree of homoeology between translocated wheat and rye segments. Kantety and co-workers (2002) reported that nearly 70% of EST-derived SSRs amplified PCR products both in rye and bread wheat. Khlestkina and co-workers

(2004) found that 69% of 65 rye expressed microsatellite sites (REMS) amplified good PCR products also in bread wheat, allowing 60 wheat microsatellite markers to be mapped onto the genetic map of rye. In the present study, the close homoeologous relationship between wheat and rye, especially between chromosome arms 2BL and 2RL, made it necessary to screen a large number of microsatellites to find comparatively few (2.5 %) microsatellite markers specific for the 2RL segment and polymorphic between the two parental lines. The inclusion of the 'Chinese Spring'-'Imperial' wheat-rye addition lines is essential in similar studies to verify that the microsatellite primers amplify PCR products only from a specific rye segment and not elsewhere from the wheat or rye genomes.

The use of F_2 populations for mapping has the advantage that the development of the populations is rapid compared to using recombinant inbred lines or F_3 populations, but has the disadvantages that the plant material is ephemeral and the next generation is not genetically identical to the the individual F_2 parents (Winther & Kahl, 1995). The difference in results for powdery mildew and leaf rust resistance may reflect different numbers of genes involved in resistance, and may also have been influenced by the limited number of polymorphic microsatellite markers found for 2RL and most likely not covering the entire 2RL chromosome arm. Genomic *in-situ* hybridization (GISH) of mitotic chromosome preparations using the microsatellite markers as specific probes could yield more information on the location of the markers on a physical map. The disease resistance assessment method allows the continued development of the plants and bulked F_3 populations could be used to further investigate the stability and inheritance of the QTLs, and their isolate specific or isolate non-specific correlation.

Molecular markers are essential for gene pyramiding through marker-assisted selection (MAS) and germplasm diagnostics. Pyramiding genes has been suggested as a method of achieving durable resistance (Wolfe, 1984; McDonald & Linde, 2002) and the combination of several undefeated resistance genes into a single cultivar should extend the period of resistance since genetic change at several avirulence loci would be required to produce a new virulent mutant race (Wolfe, 1984; Pink, 2002). Homologous recombination between alien chromosome segments transferred to wheat also represents a strategy for increasing variation and for incorporating new genes for disease and pest resistance. For example, Mater and co-workers (2004) combined the powdery mildew resistance from the 1RS segment of a T1BL.1RS translocation with green bug resistance from the 1RS segment in a T1AL.1RS translocation. Similarly, the usefulness of the present HT02-9 and other 2RL translocations could be enhanced by recombination between different 2RL segments to pyramid pest and disease resistance in an agronomically desirable wheat background. The identification of molecular markers, as described in the present study, will facilitate the combination and monitoring of effective race-specific genes and quantitative trait loci for resistance in plant breeding programs.

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Table 1. *Disease responses of wheat cultivars/lines with rye translocations after inoculation with 11 isolates of* Blumeria graminis *f. sp.* tritici *(Bgt) and 8 isolates of* Puccinia triticina *(Prt)*

Wheat Cultivar/line	Wheat-Rye Translocation	Rye Source	Bgt Isolate[1]											Prt Isolate[2]							
			P 2	P 5	P 6	P 9	P 10	P 12	P 13	P 14	P 15	P 16	P 17	S 12	S 28	S 29	S 48	S 71	Pt 8	Pt 9	Pt 60
HT02-9	T2BS.2RL	Triticale	0,1	0	1	0	0	0	01	0	0	0	0	0	0;	0;	0;	0;	0;	0;	0;
HT02-8	-	-	8	8	8	8	8	8	8	8	8	8	8	4	4	4	4	4	4	4	4
Holme	-	-	8	8	8	8	8	8	8	8	8	8	8	3	4	4	4	3	1	4	4
Kraka	-	-	9	0,1	8	8	2,5	8	8	1,2	8	8	8	3	4	4	3	3	12	3	3
P66	T2AS.2RL	'Imperial'	8	8	8	8	8	8	8	8	8	8	8	4	4	4	4	4	4	4	4
Chinese spring	-	-	8	8	8	8	8	8	8	8	8	8	8	4	4	4	4	4	4	4	4
Transec	T4BS- 4BL.2RL	'Rosen'	3	4	0,3	7	7	4	8	8	7	8	8	;1	1	3	3	4	0;	;	;1
Disponent	T1BL.1RS	'Petkus'	0,1	8	8	0	8	0	7	7	7	7	8	4	1	0;	2	0	3	4	X
Amigo[3]	T1AL.1RS	'Insave'	5	5	2,6	4	4	4	0	8	4	0	0	0	0	0	0	0;	0	0	1

[1] 0 = no visible disease symptoms; 10 = 100% of leaf area covered with mycelium. Segregating resistance responses are separated by a comma (,).

[2] 0= no macroscopic signs of infection, ; = hypersensitive necrotic or chlorotic flecks of varying size present, 1 = small uredinia often surrounded by necrosis, 3 = medium-sized uredinia that may be associated with chlorosis or rarely necrosis, X = random distribution of variable-sized uredinia on a single leaf segment , 4 = large uredinia without surrounding chlorosis. A range of infection types on a single leaf segment is described using more than one infection type with the predominant infection type listed first.

[3] 'Amigo' also carries a translocated chromosome segment from *Thinopyrum ponticum* (syn. *Agropyron elongatum).*

Table 2. *Annealing temperatures (AT) and fragment sizes of the rye (rms) and* Secale cereale *(scm) microsatellites specific for rye chromosome arm 2RL in three wheat-rye translocation lines*

Locus	AT (°C)	Fragment size (bp) in		
		HT02-9 Triticale T2BS.2RL line	**P66** 'Imperial' rye T2AS.2RL line	**CS-2RL** 'Imperial' rye addition line
Xrms5	60	135	135	135
Xrms15	55	130	135	135
Xrms42	60	163	163	163
Xrms46	60	212	218	216
Xrms47	60	null	289	289
Xscm43	60	103	102	102
Xscm75	60	191	191	191

Table 3. *Results from QTL analysis in QGENE of disease resistance from rye 2RL in wheat against powdery mildew isolate P-13, and leaf rust isolates S-12 and S-71*

Trait	Marker	Distance (cM)	N plants	Source	F-value	RSquare	LOD	P-value
P-13	*RMS46*	7.1	127	P66	68.48	0.5248	20.52	0.000
	RMS15	8.7	131	P66	53.81	0.4568	17.36	0.000
	RMS47	0.0	129	P66	14.82	0.1904	5.92	0.000
S-12	*RMS46*	7.1	127	P66	7.54	0.1084	3.17	0.001
	RMS15	8.7	131	P66	7.54	0.1054	3.17	0.001
	RMS47	0.0	129	P66	1.32	0.0205	0.58	0.271
S-71	*RMS15*	8.7	131	P66	3.75	0.055	1.62	0.026
	RMS46	7.1	127	P66	3.25	0.050	1.41	0.042
	RMS47	0.0	129	HT02-9	0.11	0.002	0.05	0.860

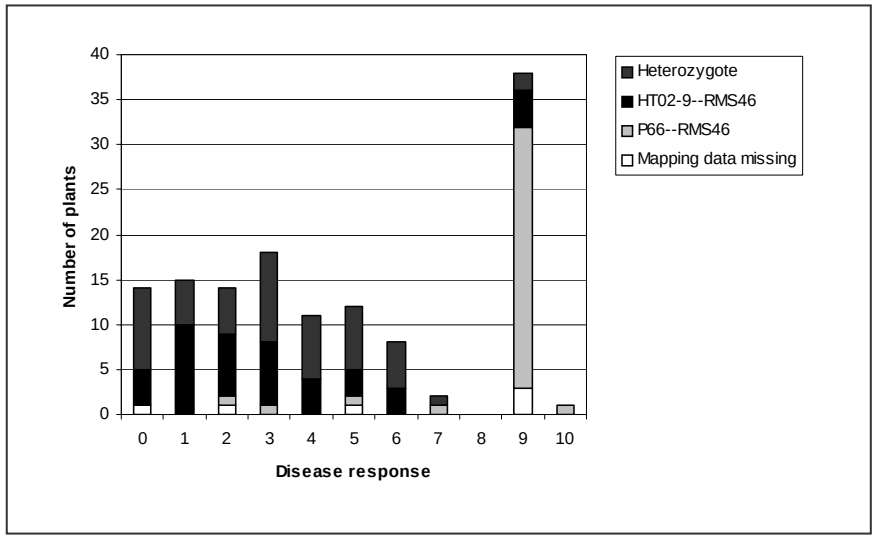
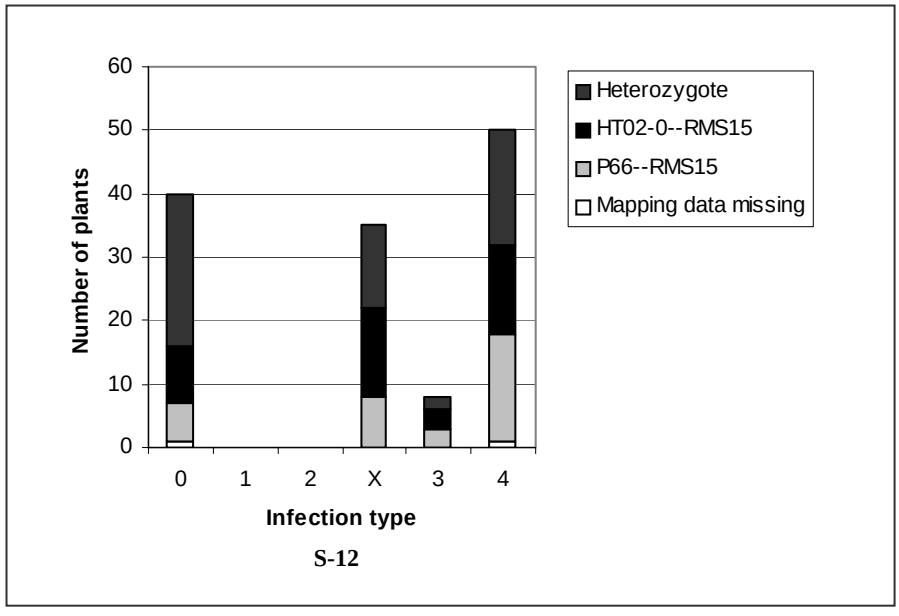


Figure 1. Phenotypic and allelic distribution of marker RMS46 in the F_2 plants infected with powdery mildew isolate P-13. Disease response 0 = no visible disease symptoms and 10 = 100% leaf area covered with sporulating colonies.



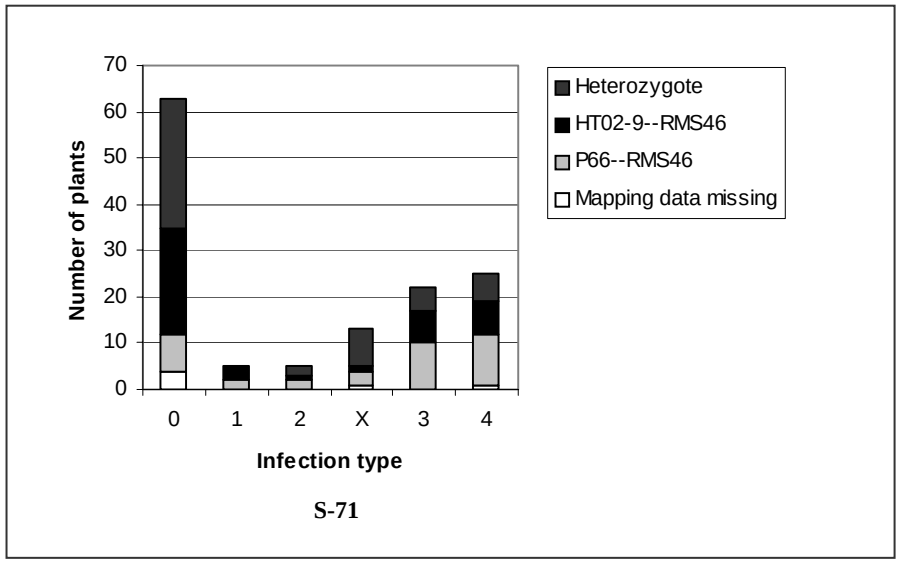


Figure 2. Phenotypic and allelic distribution of the F_2 plants infected with leaf rust isolate S-12 and marker RMS15; and isolate S-71 and marker RMS46. Infection type 0 = no uredinia or other macroscopic signs of infections, 1 = small uredinia often surrounded by necrosis, 2 = small to medium uredinia often surrounded by chlorosis or necrosis, X = random distribution of variable-sized uredinia on a single leaf segment, 3 = medium-sized uredinia that may be associated with chlorosis or rarely necrosis, 4 = large uredinia without surrounding chlorosis.

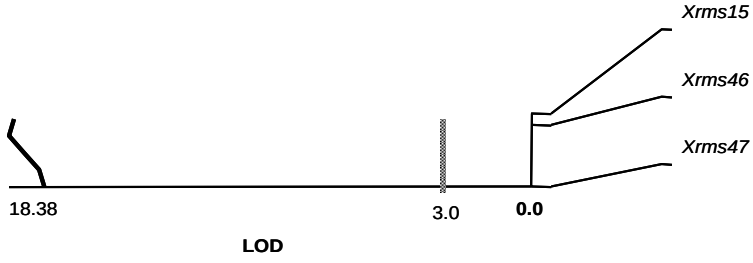


Figure 3. Linkage map of rye chromosome 2RL showing the location of quantitative trait loci (QTL) determining resistance to powdery mildew isolate P-13. The position of the centromeric region is unknown. The distance between *Xrms15* and *Xrms46* is 1.6 cM, and 8.7 cM between *Xrms46* and *Xrms47*.

A

Appendix A. Summary of information on wheat accessions in Papers I-III

Donor/Acc.nr[1]	Name	Pedigree[2]	G[3]	R[4]	Year[5]	Postulated *Lr genes*	Postulated *Pm genes*	Pm APR[6]	Paper
AF	Abba	A 0336.19/Catamaran	w	D	2002				III
AF	Abika	Brigadier/A 91295.16	w	D	2003				III
PF	Alrø	PF 97227-2/Parade	w	D	1990s				III
NGB-4770	Als	(s) landrace from Als	w	D	1937		*none*	3-5%	II, III
NGB-9122	Anja	Kranich/Caribo	w	D	1980		*none*		II, III
NGB-0004	Ankar	Iduna/Bore	w	S	1928		*none*	3-5%	II, III
NGB-0006	Ankar II	Ankar/Saxo	w	S	1928		*none*	3-5%	II, III
B	Anniina	Satu/Polkka	s	F	2001	*u*			I
JIC-0113	Apu	Garnet/Pika	s	F	1949				III
NGB-0007	Äring	Ankar/Saxo	w	S	1932		*none*	3-5%	II, III
NGB-0011	Äring III	(s) Äring I	w	S	1940		*none*	3-5%	II, III
NGB-0014	Aros	Äring/Ergo	w	S	1947		*none*	3-5%	II, III
NGB-2143	Ås	(s) Lv from Norway	s	N	1926				III
JIC-3196	Aura	Ertus/Vakka	w	F	1975				III
B	Aura	Ertus/Vakka	w	F	1975	*u*			I
SW	Avle	22279M15/20299M12//Canon	s	S	1996	*Lr14a*	*u*		I, II, III
G	Bajas	Bastian/Sport	s	N	n/a	*u*			I
SW	Ballad	Sv85297/Sv85568	w	S	2000	*u*	*Pm5+u*		I, II, III
JIC-1208	Banco	WW6518/WW6431//Ankar II	w	S	1953				III
G	Bastian	Bajio-66/Runar/4/Yaktana/Norin10/Brevor/3/Moystad/5/Rollo/Magnif/4/Sonora/Tezanos-Pintos-Precoz//Nainari/3/Moystad	s	N	1989	*none*			I
NGB-11315	Bastian	Bajio-66/Runar/4/Yaktana/Norin10/Brevor/3/Moystad/5/Rollo/Magnif/4/Sonora/Tezanos-Pintos-Precoz//Nainari/3/Moystad	s	N	1989				III

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Appendix A. *cont.* Summary of information on wheat accessions in Papers I-III

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SW	Bill	(DH) Multicross	w	DEU	n/a	*Lr3+17+23+u*			I
G	Bjarne	SvB82293/Bastian	s	N	2002	*Lr1*			I
G	Bjørke	SvU75630/Rida	w	N	1998	*u*			I
NGB-13659	Bjørke	SvU75630/Rida	w	N	1998		*Pm5*		II, III
NGB-9691	Blanka	Extra Kolben II/Wilhelmina	s	S	1950		*none*	3-5%	II, III
NGB-6695	Bore	(s) from English wheat	w	S	1902		*none*	3-5%	II, III
NGB-8933	Borg Abed	Trifolium 14/Abed 92	w	D	1967		*none*		II, III
NGB-4494	Borstvete från Gotland	Landrace	w	S	-		*u*	none	II, III
NGB-2125	Børsum	Landrace	s	N	-		*u*	3-5%	II, III
SW	Brigadier	Squadron/Rendezvous	w	GBR	1992	*Lr26+u*			I
NGB-8946	Brødtorp pajo	Landrace	w	D	-		*none*	3-5%	II, III
NGB-7456	Brons	Aurore/Extra Kolben II	s	S	1945		*u*	3-5%	II, III
SW	Canon	Sicco/2*WW-12502//2*Sappo/3/Kadett	s	S	1988	*Lr14a*			I
NGB-7481	Canon	Sicco/2*WW-12502//2*Sappo/3/Kadett	s	S	1988		*u*		II, III
SW	Citadel	Composite cross of 24 cvs with main cv Tadorna	w	NLD	1983	*Lr13*			I
SW	Curry	Canon s/Nemares//Kadett Mp1	s	S	1994	*Lr14a+u*	*u*		I, II, III
SW	Dacke	P18/17269//19151	s	S	1990	*none*			I
NGB-9955	Dacke	P18/17269//19151	s	S	1990		*u*		II, III
NGB-9708	Dala	Landrace	s	S	-		*u*	3-5%	II, III
NGB-6410	Dalarna	Landrace	s	S	-		*u*	3-5%	II, III
NGB-7027	Dania	Landrace	w	D	-		*none*		II, III
NGB-6679	Diamant	Kolben/Hallands (landrace)	s	S	1928		*none*	15%	II, III
NGB-6681	Diamant II	Diamant/Extra Kolben II	s	S	1938		*none*	15%	II, III
SW	Dirigent	Ritmo/Reaper	w	D	n/a	*u*			I
SW	Drabant	CItr 12633/Ring ^6	s	S	1972	*Lr14a*			I
NGB-7469	Drabant	CItr 12633/Ring ^6	s	S	1972		*u*	3-5%	II, III
SW	Dragon	Sicco/1250[2]/3/Sappo[2]/5/Kadett	s	S	1988	*Lr14a+u*			I
NGB-9954	Dragon	Sicco/1250[2]/3/Sappo[2]/5/Kadett	s	S	1988		*u*		II, III
NGB-8957	Enger	Landrace	w	N	-		*none*	3-5%	II, III

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Appendix A. *cont.* Summary of information on wheat accessions in Papers I-III

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NGB-0008	Ergo	Ankar I/Jarl	w	S	1934		*none*	15%	II, III
NGB-0012	Eroica	WW 5133/Äring	w	S	1943		*none*	3-5%	II, III
NGB-0015	Eroica II	(s) Eroica I	w	S	1951		*none*	3-5%	II, III
NGB-0017	Ertus	Eroica/Virtus	w	S	1953		*none*	3-5%	II, III
NGB-6677	Extra Kolben	Kolben/unnamed line	s	S	1919		*none*	3-5%	II, III
NGB-8923	Extra Kolben II	(s) Extra Kolben	s	S	1926		*none*	3-5%	II, III
NGB-6694	Extra Squarehead (SWE)	(s) Leutenritzer Squarehead	w	S	1900		*none*		II, III
SW	Flair	Ares/Marabu; Ares/3/Rabe/Jubilar//Armada	w	DEU	1996	*u*			I
SW	Florida	Caribo/Disponent	w	DEU	1985	*Lr26*			I
NGB-2434	Folke	Holme/Walde	w	S	1981		*none*	3-5%	II, III
NGB-2126	Fram I	J-03/Mo-07	s	N	1930s		*u*		II, III
NGB-2127	Fram II	J-03/Mo-07	s	N	1938		*u*	3-5%	II, III
NGB-6680	Fylgia I	Aurore/Extra Kolben	s	S	1933		*u*	3-5%	II, III
NGB-6685	Fylgia II	Extra Kolben II/Aurore	s	S	1952		*u*	3-5%	II, III
AF	Galicia	n/a	w	D	2000				III
NGB-8199	Gammel svensk landhvete	Landrace	w	S	-		*none*	3-5%	II, III
SW	Gnejs (SW Gnejs)	KosackMB/3*Kraka/4/Kurier	w	S	2001	*Lr1*			I, III
SW	Grommit	Apostle/Torfrida//Hereward	w	GBR	n/a	*Lr3+10+17+u*			I
NGB-6716	Gyllen II	Kron/Bore II	w	S	1938		*none*	3-5%	II, III
NGB-0121	Haarajärvi ME0102; Apu	Landrace	s	F	-		*none*		II, III
NGB-6409	Halland	Landrace	s	S	-		*u*	3-5%	II, III
NGB-9057	Hallandshvete	Landrace	s	S	-		*none*	3-5%	II, III
NGB-6773	Hankkijan Ilves	Hja B 356/Vakka	w	F	1984		*none*		II, III
SW	Hanno	Banjo/Hermes	s	GBR	n/a	*u*	*u*		I
SW	Harnesk (SW Harnesk)	WD-linje/Konsul	w	S	2001	*Lr13+u*	*Pm4b*		I, II, III
NGB-8968	Haukiala Pirola	Landrace	w	F	-		*none*		II, III
SW	Haven	Hedgehog/Norman//Moulin	w	GBR	1988	*Lr26+u*			I
B	Heta	Hja a 1105/Hja a 1099	s	F	1988	*Lr10*			I
NGB-4080	Hildur	Sv 60504/Starke	w	S	1976		*none*	3-5%	II, III

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NGB-2435	Holger	WW 2259-68/WW 2250-68	w	S	1981		*Pm6+u*	3-5%	II, III
SW	Holme	Starke//Odin/Banco	w	S	1972	*u*			I
NGB-0023	Holme	Starke//Odin/Banco	w	S	1972		*none*	3-5%	II, III
NGB-13345	Hopea	Canadian Marquis/Ruskea	s	F	1936		*none*		II, III
NGB-0042	Horsmanaho ME201 Timantti	Landrace	s	F	-				III
SW	Hugin	Dragon (sib)/Nemares	s	D	1996	*Lr13+u*			I
NGB-5153	Hunsballe R	(s) Jubile	w	D	1955		*none*		II, III
SW	Hurtig	Severin M8B8/Sperber//Urban/3/Konsul	w	S	2003	*u*			I, II, III
SW	Hussar	Squadron/Rendezvous	w	GBR	1991	*Lr26+u*			I
NGB-8973	Ideal	Trifolium 14/spontaneous cross	w	D	1929				III
NGB-0001	Iduna	(s) Squarehead	w	S	1911		*none*	3-5%	II, III
B	Ilves	Hja B 356/Vakka	w	F	1984	*none*			I
JIC-7535	Ilves	Hja B 356/Vakka	w	F	1984				III
NGB-2146	J-03	Breeding line for mildew resistance	s	N	-		*u*		II
NGB-0003	Jarl	Iduna/line from Sammetsvete from Uppland	w	S	1925		*none*	15%	II, III
NGB-0131	Järvenkylä ME0302 Sep A	Landrace	s	F	-		*none*		II, III
NGB-0040	Jokikylä ME0505;Apu	Landrace	s	F	-		*none*		II, III
SW	Jondolar	Sicco/Tilly//VDH-1166-76-M	s	S	1990	*Lr14a*			I
NGB-0348	Jyvä	(s) Vakka	w	F	1965		*none*		II, III
SW	Kadett	Kolibri/WW 439-66/Pompe-M	s	S	1981	*u*			I, III
NGB-11316	Kalle	n/a	w	N	1990		*Pm5+u*		II, III
SW	Kamerat	Rida/Moulin//Disponent/2*Rida	w	N	n/a	*Lr26*			I
NGB-7457	Kärn	WW 8244/WW 8388	s	S	1946		*none*	3-5%	II, III
NGB-7458	Kärn II	(s) Kärn	s	S	1947		*none*	3-5%	II, III
SW	Kartesch	Severin M8B8/Sperber//Urban/3/Konsul	w	S	2003	*u*			I
NGB-13347	Kimmo	(s) Population from Pisarev in Moscow	s	F	1941		*none*	3-5%	II, III
JIC-0114	Kimmo	(s) Russian wheat	s	F	1949				III
JIC-0800	Kiuru	Aurore/Sopu	s	F	1951				III

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NGB-6676	Kolben (Svalöfs Kolben)	(s) landrace or Heines Kolben	s	S	1892		*none*	3-5%	II, III
NGB-8194	Konge II	(s) Konge (= Ideal/spontaneous cross)	w	D	1939		*none*	3-5%	II, III
SW	Konsul	Ertus/Norre//Holme-M/3/Cerc.Res	w	S	1990	*Lr13+u*			I
SW	Kosack	Mironovskaja 808/Starke M//Holme M	w	S	1984	*u*			I
NGB-7482	Kosack	Mironovskaja 808/Starke M//Holme M	w	S	1984				II, III
NGB-2128	Kr Finset, Eikesdal	Landrace	s	N	-		*u*	3-5%	II, III
SW	Kraka	Kranich/Caribo	w	D	1980				I
NGB-9123	Kraka	Kranich/Caribo	w	D	1980		*Pm5*	3-5%	II, III
SW	Kris	Hereward/Rendezvous//Torfrida	w	DEU	1999	*Lr10+13+u*			I
NGB-6708	Kron	Sol II/Pansar	w	S	1925		*none*		II, III
B	Kruunu	Mahti/Hja23471	s	F	2001	*Lr10*			I
B	Laari	Villa Glofi/Tuoko	s	F	1990	*u*			I
NGB-6388	Lading Skæghvede	Landrace	w	D	-		*none*		II, III
NGB-4406	Laitiala AP0103	Landrace	s	F	-		*none*		II, III
NGB-2129	Landvårkveite	Landrace	s	N	-		*u*	3-5%	II, III
NGB-2130	Lanor	Norrøna/Lade	s	N	1970		*u*		II, III
NGB-6673	Lantvete från Dalarna	Landrace	s	S	-		*u*	3-5%	II, III
NGB-4496	Lantvete från Gotland	Landrace	w	S	-		*u*	3-5%	II, III
NGB-6674	Lantvete från Halland	Landrace	s	S	-		*u*	3-5%	II, III
NGB-6691	Lantvete från Halland	Landrace	w	S	-		*u*	3-5%	II, III
NGB-6692	Lantvete från Uppsala	Landrace	w	S	-		*u*	3-5%	II, III
NGB-0122	Larinsaari ME0101; Apu	Landrace	s	F	-				III
SW	Lavett	WW118466/Kadett//Dragon	s	S	1992	*u*			I
NGB-13041	Lavett	WW118466/Kadett//Dragon	s	S	1992		*u*		II, III
SW	Leguan	ST-324-84/ST-174-83	s	CZE	1997	*Lr14+u*			I
B	Linna	Panu/Hja04519/Virtus	w	F	1976	*none*			I
JIC-7542	Linna	TA A 2701/Virtus	w	F	1976				III
B	Luja	Svenno//Hopea/Tammi	s	F	1981	*u*			I
JIC-8372	Luja	Svenno//Hopea /Tammi	s	F	1981				III
SW	Lynx	Rendezvous/CWW-4442-64	w	GBR	1992	*Lr17+26+u*			I

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Appendix A. *cont.* Summary of information on wheat accessions in Papers I-III

Donor/Acc.nr[1]	Name	Pedigree[2]	G[3]	R[4]	Year[5]	Postulated *Lr genes*	Postulated *Pm genes*	Pm APR[6]	Paper
SW	Magnifik	Composite cross of German and Swedish lines	w	S/N	2004	*Lr1*			I
B	Mahti	Cebeco 1036/Hja a 20519	s	F	1995	*Lr10*			I
B	Manu	Ruso/Runar	s	F	1993	*u*	*none*		I
NGB-11709	Manu	Ruso/Runar	s	F	1993				II, III
SW	Marabu	LP-6077-71/Monopol//Kronjuwel	w	DEU	1988	*Lr26+u*			I
SW	Marshal	Kontiki/Brigadier	w	GBR	2001	*Lr26+u*			I
JIC-1159	Mendel	Standard/Trifolium 14	w	S	1926				III
SW	Meridien	Starke/Norre/3/2*Ertus/Norre//Holme/4/ Wampum/5/Moisson	w	S	1993	*Lr13+u*			I
SW	Mjölner (Mjølner)	TL340/Starke//W25458	w	S	1996	*Lr10+u*	*none*		I, II, III
NGB-0043	Monola ME1301	Landrace	s	F	-				III
JIC-0766	Møystad	(Mo 042-40)/Kärn II	s	N	1971				III
NGB-9118	Nana	Ibis/Stella	w	D	1975		*Pm5*		II, III
JIC-7545	Nisu	(s) Vakka	w	F	1966				III
JIC-1319	Nora	Fram II/Sopu	s	N	1973				III
NGB-0021	Norre	Eroica/Virtus	w	S	1962		*none*	3-5%	II, III
NGB-2133	Norrøna	Fram-II/Sopu	s	N	1958		*u*	3-5%	II, III
SW	Nova	Angela/TJB-330-1491//Arminda	w	DEU	1993	*Lr14a+u*			I
NGB-6723	Odin	Gluten/Ergo	w	S	1949		*none*	3-5%	II, III
NGB-6727	Ölve	Eroica I/K 01281 (mother line to Hansa)	w	S	1959		*none*	3-5%	II, III
NGB-8922	Østby	Landrace	s	D	-		*u*		II, III
B	Otso	Elo/Vakka	w	F	1995	*u*			I
SW	Pagode	Composite cross of 36 cultivars	w	NLD	1986	*Lr13*			I
NGB-6707	Pansar III	(s) Pansar I	w	S	1923		*none*	3-5%	II, III
NGB-6722	Pärl II	Sv 0912/Svea	w	S	1946		*none*	3-5%	II, III
SW	Pepital	ROC/VDH-040-71-B; ROC-109-751/VDH-040-71-B; ROC/VDH-1040-71-B	w	NLD	1989	*Lr10+Lr13*			I
B	Pitko	Ta 05901/Vakka	w	F	1985	*u*			I

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Appendix A. *cont.* Summary of information on wheat accessions in Papers I-III

Donor/Acc.nr[1]	Name	Pedigree[2]	G[3]	R[4]	Year[5]	Postulated *Lr genes*	Postulated *Pm genes*	Pm APR[6]	Paper
SW	Polkka	Sv70415/Snabbe//Norrena/Karn-2/3/Snabbe	s	S	1992	*u*			I
NGB-7464	Pompe	Ring/Svenno	s	S	1967		*Pm1a+2+9*	3-5%	II, III
NGB-6688	Prins	Diamant II/Kärn II	s	S	1962		*none*	3-5%	II, III
NGB-6682	Progress	Sv Ä 23-8/Extra Kolben II	s	S	1942		*u*	15%	II, III
NGB-6698	Pudel	(s) Shiriff wheat from England	w	S	1910		*none*	3-5%	II, III
NGB-7466	Rang	Ring ^5/Els	s	S	1968		*Pm1a+2+9*	3-5%	II, III
NGB-2134	Reno	Els/T-110-21-41;Tammi/Kärn-II//Els	s	N	1975		*u*	3-5%	II, III
NGB-6699	Renodlat Sammetsvete	Selection through purification of wheat from Ulltuna, Uppland	w	S	1910		*none*	3-5%	II, III
SW	Rental	Sv70355/M. Huntsman	w	S	1993	*u*			I
SW	Residence	Obelisk//Cebeco-8451/Arminda	w	NLD	1998	*Lr13+u*			I
SW	Revelj	Kanzler M15M28	w	S	2000	*Lr13+u*	*u*		I, II, III
SW	Rialto	Haven (sib)/Fresco (sib)	w	GBR	1993	*Lr13+26+u*			I
NGB-11317	Rida	MO-0944-15/Redcoat//Trond	w	N	1976		*Pm5+u*	3-5%	II, III
NGB-7462	Ring	Kain/Pondus	s	S	1957		*Pm1a+9*	3-5%	II, III
SW	Ritmo	Hobbit//Line-1320/Wizard/3/Marksman/4/Virtue	w	NLD	1990	*Lr13+u*			I
NGB-6684	Rival	Diamant/Extra Kolben II	s	S	1952		*none*	3-5%	II, III
NGB-6724	Robur	Skandia II/Sv 36-175	w	S	1949		*none*	3-5%	II, III
NGB-2135	Rollo	Kärn-II/Norrøna	s	N	1963		*u*	3-5%	II, III
NGB-6678	Rubin	Kolben/Dala (landrace)	s	S	1921		*none*	3-5%	II, III
NGB-14118	Rudolf Rubin	WW 25449/Folke	w	S	1921		*none*	3-5%	II, III
G	Runar	Els/Rollo	s	N	1972	*u*			I
NGB-2136	Runar	Els/Rollo	s	N	1972		*Pm4b*	10%	II, III
B	Ruso	Reward/Pika	s	F	1967	*Lr10*			I
JIC-7551	Ruso	Reward/Pika	s	F	1967				III
NGB-7472	Saffran	WW 38-68/WW 11-68	s	S	1978		*Pm1a+2+8+9+u*	3-5%	II, III

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Appendix A. *cont.* Summary of information on wheat accessions in Papers I-III

Donor/Acc.nr[1]	Name	Pedigree[2]	G[3]	R[4]	Year[5]	Postulated *Lr genes*	Postulated *Pm genes*	Pm APR[6]	Paper
NGB-6687	Safir	Sv 1015/A 24-585	s	S	1955		*u*	3-5%	II, III
NGB-7467	Sappo	WW 177-62/WW 176-62	s	S	1971		*u*	3-5%	II, III
NGB-0120	Sarkalahti ME0101	Landrace	s	F	-		*none*		II, III
SW	Satu	Snabbe/Drabant//15962	s	S	1990	*none*			I
PF	Saxild	Britta//Pepital/Gawain	w	D	n/a				III
NGB-0005	Saxo	(s) Tystofte Smaahvede II	w	S	1929		*none*	3-5%	II, III
NGB-0473	Sigyn II	Heid/Labors-Elite-05	w	N	1972				III
NGB-6383	Skandia	Kron/SV-0860-D	w	S	1935		*none*	3-5%	II, III
NGB-6717	Skandia II	(s) Skandia	w	S	1939		*none*	3-5%	II, III
NGB-2138	Skirne	Gelchsheimer/Särimner	s	N	1937				III
SW	Sleipner (syn. Slejpner)	WW 20102/CB 149//Maris Huntsman///Bilbo	w	S	1988				I
NGB-7483	Sleipner (syn. Slejpner)	WW 20102/CB 149//Maris Huntsman///Bilbo	w	S	1988		*Pm2+6+8*	3-5%	II, III
NGB-7183	Små II Tystofte	(s) Tystofte Smaahvede	w	D	1915		*none*		II, III
NGB-7465	Snabbe	Svenno/WW 7039 (= Kain/Kimmo)	s	S	1968		*none*	3-5%	II, III
NGB-2139	Snøgg I	0843/Ås	s	N	1939		*u*	3-5%	II, III
NGB-6700	Sol	(s) Landrace from Skåne, Sweden	w	S	1911		*none*	3-5%	II, III
NGB-6701	Sol II	Sol I/Extra Squarehead II	w	S	1916		*none*	3-5%	II, III
NGB-6715	Sol IV	Kron/Sol II	w	S	1937		*none*	15%	II, III
NGB-13346	Sopu	Canadian Marquis/Ruskea	s	F	1935				III
SW	Sport	P18/17269//19151	s	S	1991	*u*			I
NGB-9956	Sport	Citr 5484/PompeBM///Trippel ^3//WW 17269 ^4/WW 19151	s	S	1991		*u*	3-5%	II, III
AF	Stakado	AD 7020/AO 7021	w	D	1994				III
NGB-6709	Stål	Sol II/Pansar	w	S	1927		*none*	3-5%	II, III
NGB-8197	Stand Tystofte	(s) Squarehead	w	D	1907		*none*		II, III
SW	Starke	WW 11556/WW 11376	w	S	1959				I
NGB-0018	Starke	WW 11556/WW 11376	w	S	1959		*none*	3-5%	II, III
NGB-0022	Starke II	(s) Starke I	w	S	1968		*none*	3-5%	II, III

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Appendix A. *cont.* Summary of information on wheat accessions in Papers I-III

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SW	Stava	Helge-M7D1/Helge-M7D2// WW-31254	w	S	1995	*u*			I
NGB-13479	Stava	Helge-M7D1/Helge-M7D2// WW-31254	w	S	1995				III
NGB-7184	Storaks Abed	n/a	w	D	1967		*none*		II, III
NGB-4783	Storvik sjundeå	Landrace	w	F	-		*none*		II, III
NGB-7476	Sunnan	Pompe 2r 19/Sappo//Drabant	s	S	1983		*Pm4b*		II, III
NGB-6725	Svale	Skandia II/Eroica I	w	S	1955		*none*	3-5%	II, III
NGB-6704	Svea I	Pudel/Sammetsvete (landrace)	w	S	1924		*none*	3-5%	II, III
NGB-7461	Svenno	WW 8244/WW 8388	s	S	1953		*none*	3-5%	II, III
NGB-0355	Tähti	Kärn-I/JO-0172; Kärn//Aurore/Pika	s	F	1972				III
B	Tapio	Hja 33929/Kolibri	s	F	1980	*Lr10+u*			I
SW	Tarso	Taras/Hadmerslebener 13313-80	w	DEU	1992	*Lr26+u*			I
NGB-2141	Tautra	n/a	s	N	1983	*none*	*Pm5+6+u*	3-5%	II, III
SW	Terra	Kraka/TJB-730/3637	w	D	1992	*Lr13*			I, II
SW	Thasos	Miron.808/ Bastion// Minaret; Max /Ze73.1331.2/Minaret	s	DEU	1994	*none*			I
NGB-0020	Thor	WW 11376/WW 11379	w	S	1961		*none*	3-5%	II, III
NGB-6702	Thule II	Pudel/Sammetsvete (landrace)	w	S	1917		*none*	3-5%	II, III
NGB-6714	Thule III	Thule II/Sv 0762	w	S	1936		*none*	3-5%	II, III
NGB-0130	Timantti Paavo	Landrace	s	F	-				III
NGB-7471	Timmo	WW-152-65/Sappo	s	S	1979		*u*	3-5%	II, III
SW	Tjalve	Reno/KolibriM//15432	s	S	1981	*none*			I
NGB-7479	Tjalve	Reno/KolibriM//15432	s	S	1990		*Pm4b+5+u*	3-5%	II, III
SW	Tjelvar	Sture D/4/StureM3bM5M7	w	S	1984	*Lr26*			I
NGB-9952	Tjelvar	Sture3D1/4/StureM3b2M5M7	w	S	1984		*Pm6+8+u*	3-5%	II, III
SW	Toronto	Disponent/Weihenstephaner 616-67//Kronjuwel	w	DEU	1990	*Lr26*			I
NGB-0359	Touko	Timantti/Hopea; Diamant/Hope	s	F	1950				III
NGB-9016	Trifolium 14	(s)Wilhelmina	w	D	1925				III

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Appendix A. *cont.* Summary of information on wheat accessions in Papers I-III

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SW	Trintella	CB-239/VDH-256-81//RPB-48-75-A/Moulin	w	NLD	1994	*Lr13*			I
SW	Triso	SaxoArgan/Kadett	s	DEU	1996	*u*			I
NGB-7463	Troll	Ring//Pondus/Kärn	s	S	1967		*Pm1a+2+9*	3-5%	II, III
NGB-0019	Trond	Virtus/WW 9344	w	S	1960		*none*	3-5%	II, III
SW	Tryggve	Riley/Holme//18614/3/Helge	w	S	1990	*u*			I
NGB-9953	Tryggve	Riley/Holme//18614/3/Helge	w	S	1990		*none*	3-5%	II, III
NGB-2142	Trym	Huron/Fylgia-I	s	N	1948		*u*		II, III
NGB-9017	Tystofte Stakket	(s) Squarehead	w	D	1967		*none*		II, III
NGB-0351	Ulla	Tammi/TA-C-4431	s	F	1975				III
SW	Urban	Kranich/Diplomat	w	DEU	1981	*none*			I
SW	Urho	Nisu/Tsitsin	w	F	1999	*u*			I
JIC-0858	Vakka	Varma/Kehra	w	F	1959				III
B	Vakka	Varma/Kehra	w	F	1960	*u*			I
SW	Vals (SW Vals)	Can.M12 M14 M18 B9 B10/ Can.M14 M15 B9	s	S	2001	*Lr2a*			I, III
JIC-0526	Varma	Svea/Lv-Orimattila,S.E.Finland	w	F	1933				III
NGB-9020	Varma Tammisto	Landrace	w	F	-		*none*		II, III
NGB-6675	Vårpärl	(s) Emma	s	S	1920		*none*	10%	II, III
NGB-9109	Viking	Starke I/WW 14433	w	S	1962				II, III
SW	Vinjett	Tjalve M14/Tjalve M15//Canon	s	S	1998	*Lr14a*	*u*		I, II, III
AF	Vip	A 0336.19/Yacht	w	D	2001				III
NGB-6729	Virgo	Demeter/Virtus//Odin	w	S	1968		*none*	3-5%	II, III
SW	Virke	n/a	w	S	1999	*none*	*u*		I, II, III
NGB-0013	Virtus	Ergo/Svea II	w	S	1945		*none*	3-5%	II, III
NGB-10867	Vitus	Kleiber//Transec-7/2*Capa-2	s	S	1981				III
NGB-0024	Walde	Ergo/Svea II	w	S	1945		*none*	3-5%	II, III
NGB-7473	Walter	Starke I/WW 14433	s	S	1972		*u*	3-5%	II, III
NGB-8198	Warmland lantvete	Landrace	w	S	-		*u*	15%	II, III
AF	Wasmo	Britta/Nova	w	D	1999				III
NGB-7474	William	WW-13-69/WW-41-69	s	S	1979		*u*	3-5%	II, III
SW	Zebra	Ralle/Dragon	s	S	2001	*Lr14a*	*u*		I, II, III

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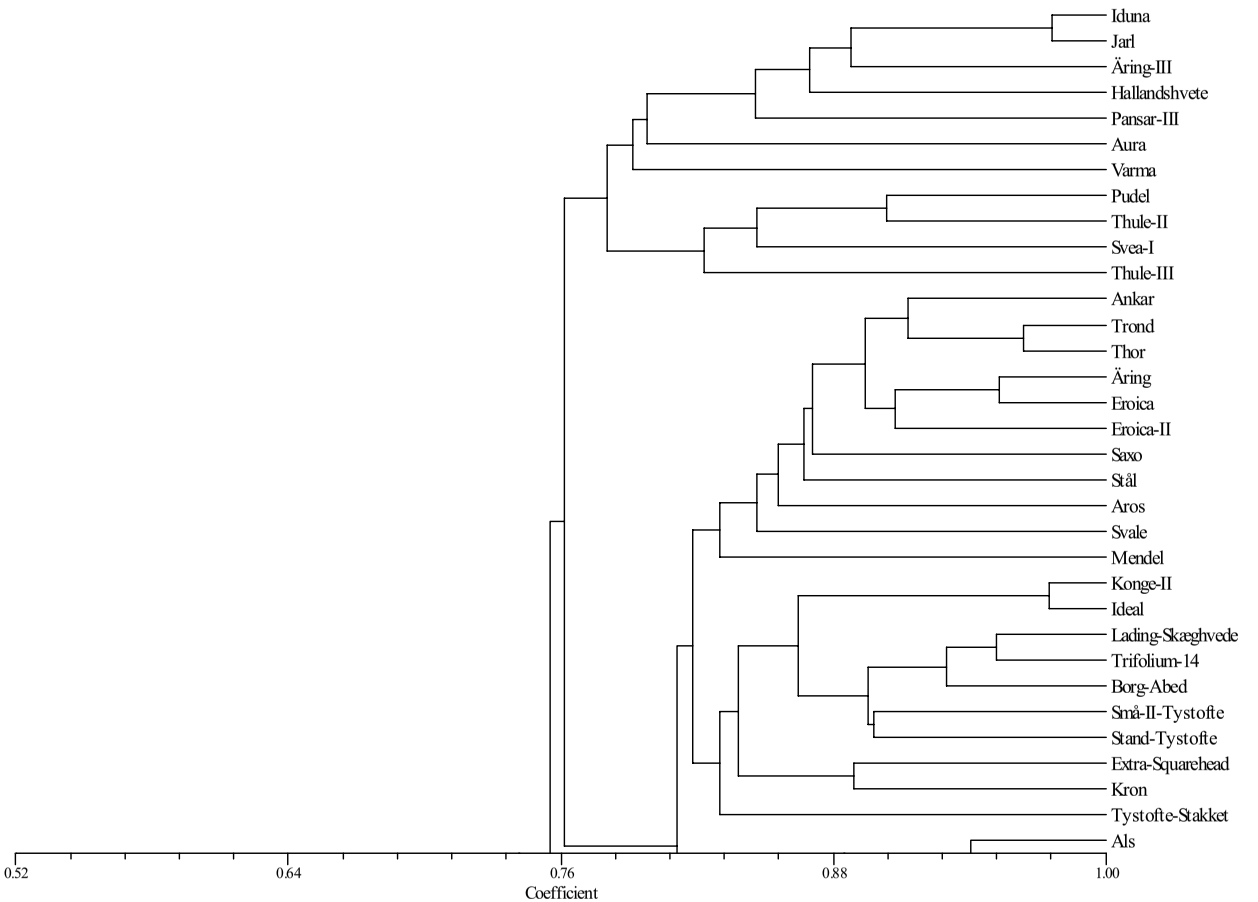
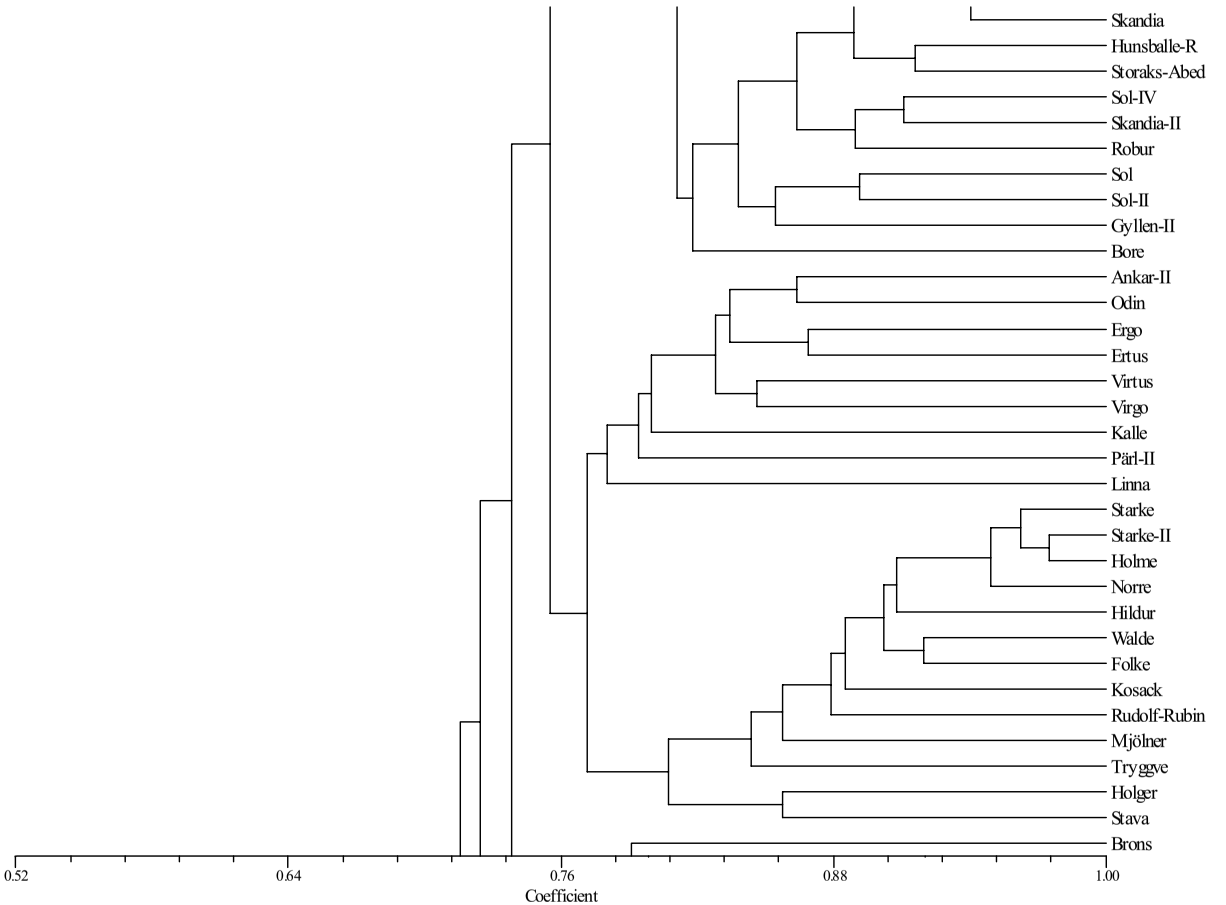
B

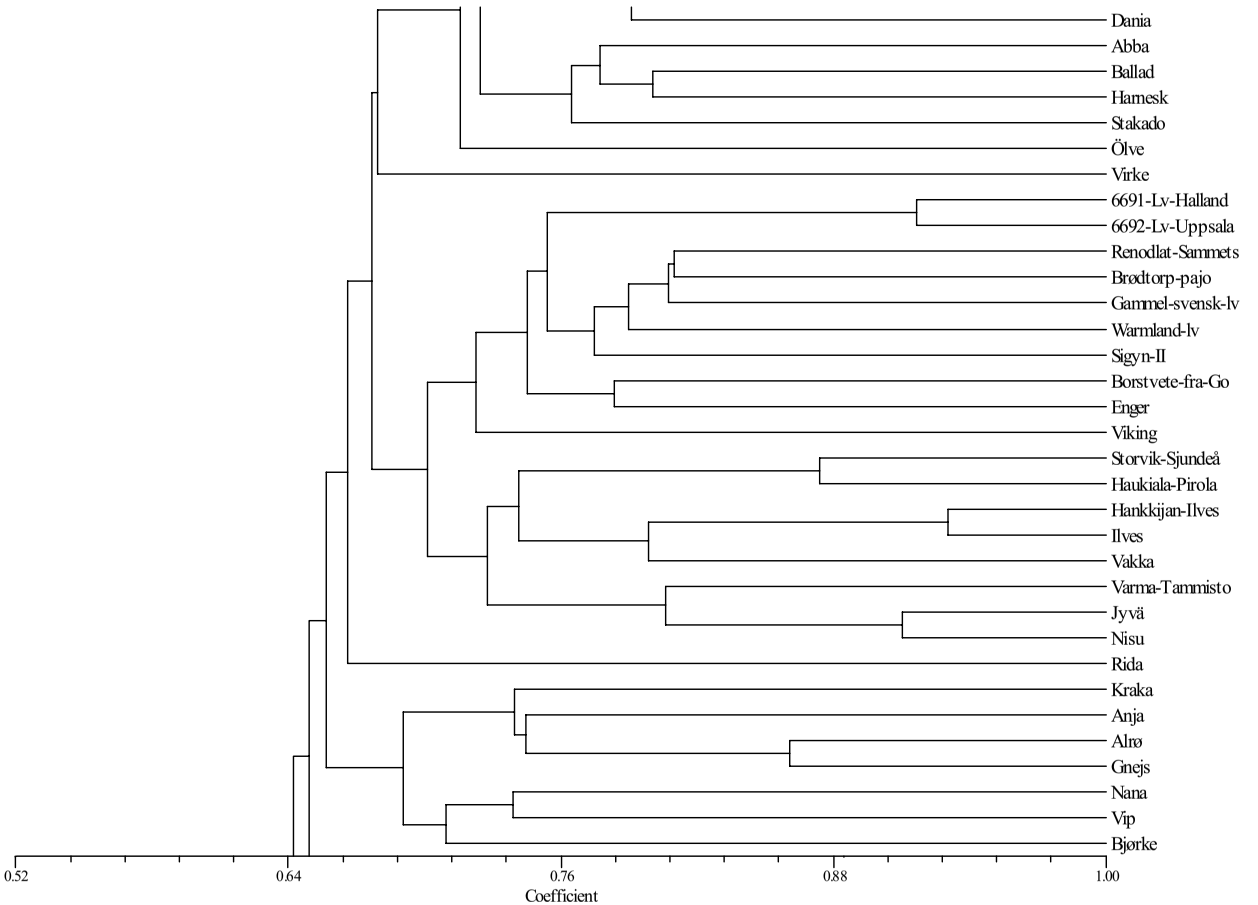
Appendix B. UPGMA dendrogram based on a Jaccard coefficient similarity matrix from S-SAP generated banding polymorphisms in Nordic bread wheat (Paper III)



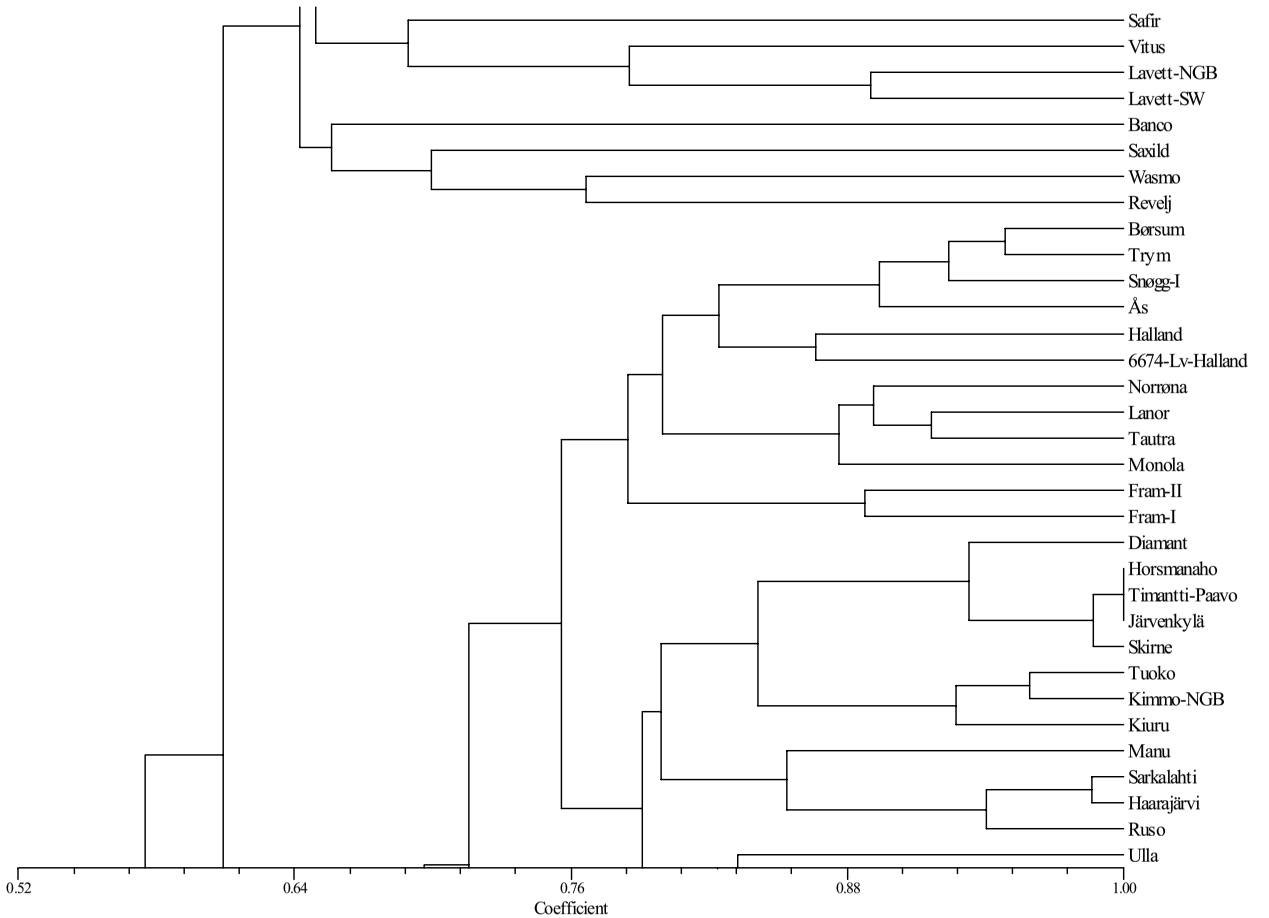
Appendix B. *cont.* UPGMA dendrogram based on a Jaccard coefficient similarity matrix from S-SAP generated banding polymorphisms in Nordic bread wheat (Paper III)



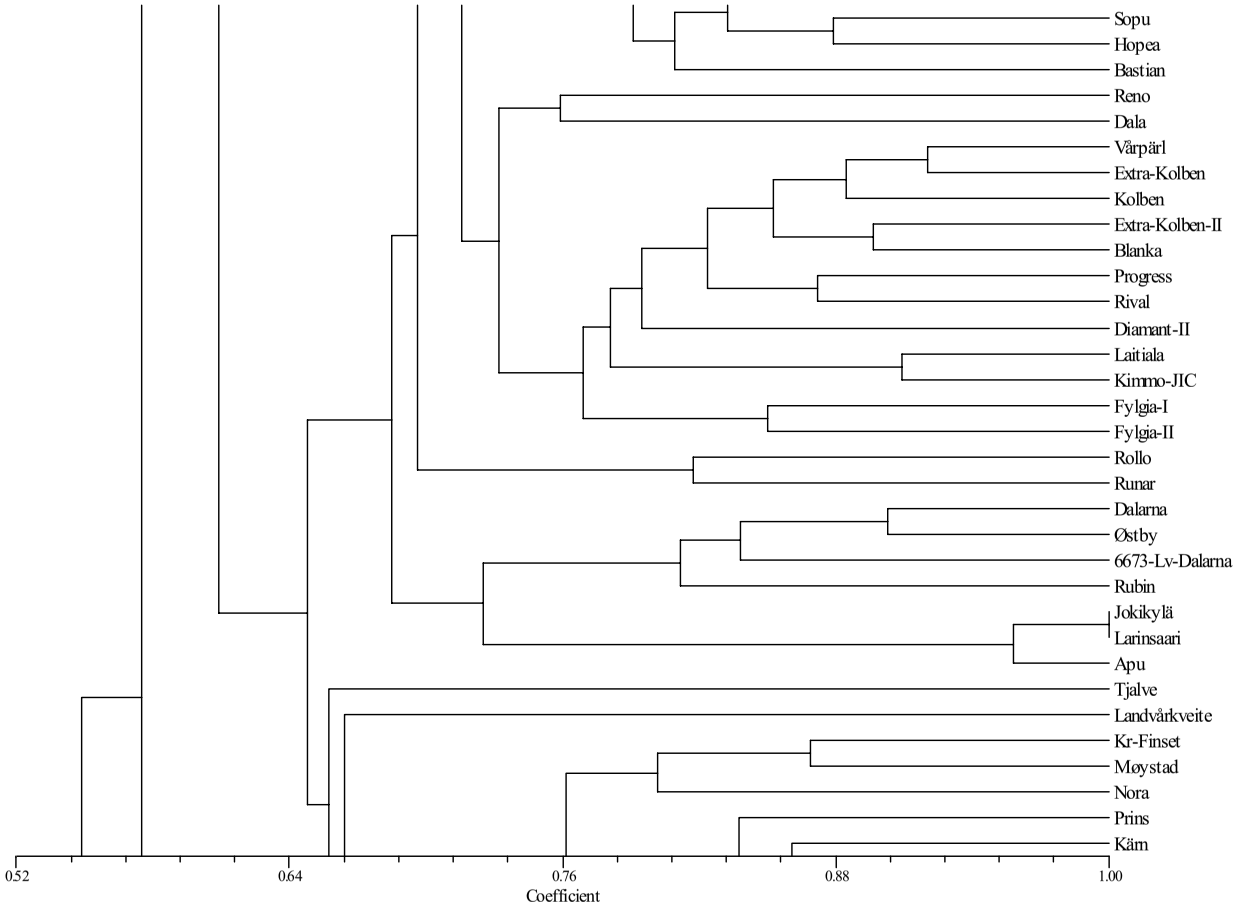
Appendix B. *cont.* UPGMA dendrogram based on a Jaccard coefficient similarity matrix from S-SAP generated banding polymorphisms in Nordic bread wheat (Paper III)



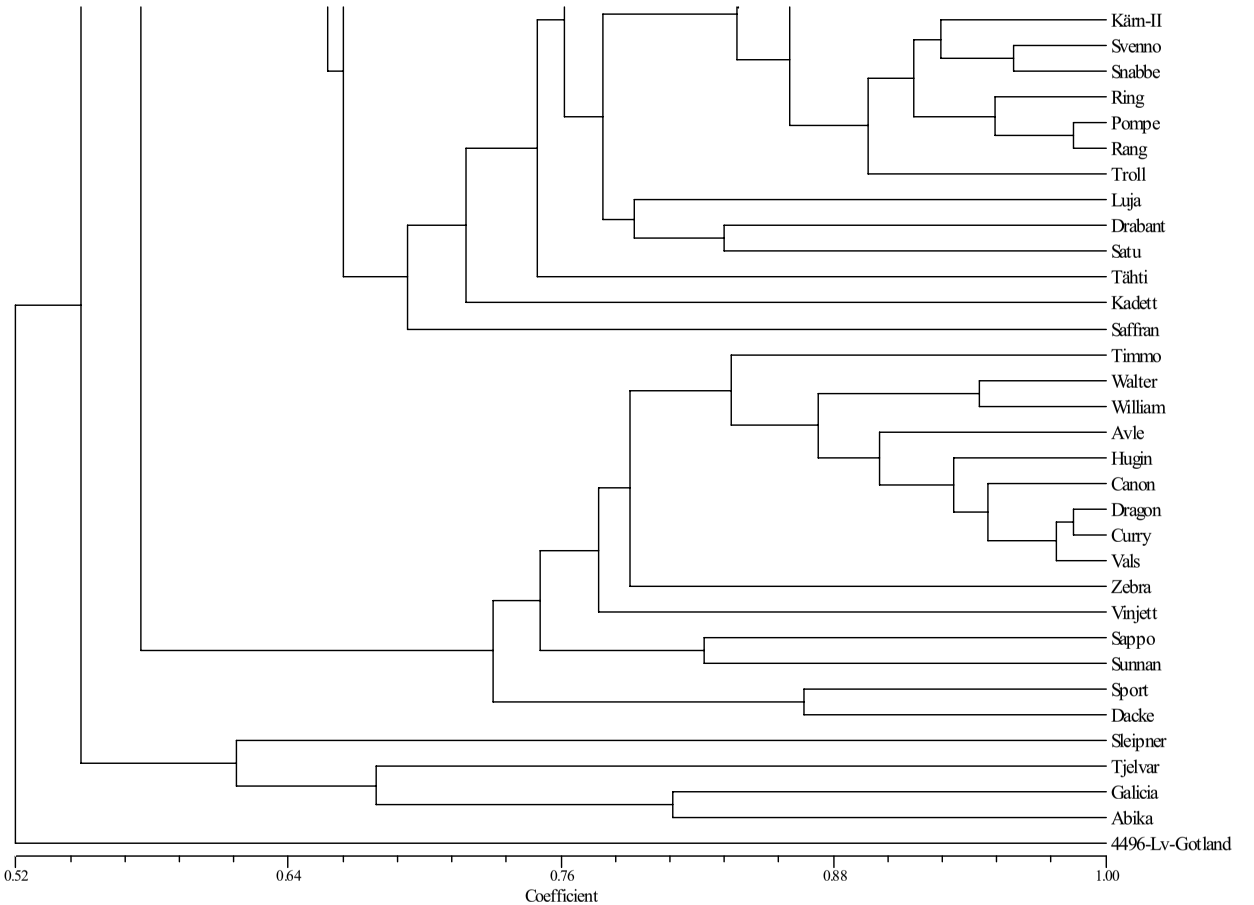
Appendix B. *cont.* UPGMA dendrogram based on a Jaccard coefficient similarity matrix from S-SAP generated banding polymorphisms in Nordic bread wheat (Paper III)



Appendix B. *cont.* UPGMA dendrogram based on a Jaccard coefficient similarity matrix from S-SAP generated banding polymorphisms in Nordic bread wheat (Paper III)



Appendix B. *cont.* UPGMA dendrogram based on a Jaccard coefficient similarity matrix from S-SAP generated banding polymorphisms in Nordic bread wheat (Paper III)



C

Appendix C. Abbreviations

AFLP	amplified fragment length polymorphism
BSA	bulk segregant analysis
DNA	deoxyribonucleic acid
EST	expressed sequence tag
F_{ST}	Wright's fixation index (genetic differentiation) for a subpopulation (S) relative to the total population (T)
H	Nei's gene diversity
H#	Hessian fly resistance gene # (number)
HF	Hessian fly
GISH	genomic *in situ* hybridisation
LARD	large retrotransposon derivative
Lr#	leaf rust resistance gene # (number)
LRR	leucine rich repeat
LTR	long terminal repeat
MAS	marker-assisted selection
NB	nucleotide binding
Nor	nucleolar organising region
PCR	polymerase chain reaction
Pm	powdery mildew
Pm#	powdery mildew resistance gene # (number)
QTL	quantitative trait loci
s	spring type habit
SLU	Swedish University of Agricultural Sciences
Sr#	stem rust resistance gene # (number)
S-SAP	sequence-specific amplified polymorphism
SSR	simple sequence repeat (microsatellite)
T	translocation
TILLING	targeting induced local lesions in genomes
w	winter type habit
Yr#	yellow (stripe) rust resistance gene # (number)
<	less than
>	more than
≈	almost equal to