

## Wind-Dispersed Nomadic Diseases: Conclusions for Disease Resistance

E. LIMPERT<sup>1\*</sup> and P. BARTOŠ<sup>2</sup>

<sup>1</sup>Phytopathology, Institute of Plant Sciences, Swiss Federal Institute of Technology Zurich (ETH), 8057 Zurich, Switzerland; <sup>2</sup>Division of Genetics and Plant Breeding – Research Institute of Crop Production,

161 06 Prague-Ruzyně, Czech Republic

\*e-mail: eckhard\_limpert@hotmail.com

**Abstract:** Data on the distribution of some wind-dispersed nomadic plant diseases (powdery mildew, leaf rust) suggest that virulence genes accumulate in the direction of prevailing winds, through the interaction of migration and selection. E.g., for leaf rust of wheat virulence complexity increased from 2–4 on average in France to approximately 7 in Austria and Poland up to 14 and above in Siberia. This situation can be of considerable importance for the use of host resistance. The higher is the virulence in a pathogen population, the more valuable should be the effective resistance genes present in that area for resistance breeding. If virulence complexity increases in the direction of predominant dispersal, the number of unnecessary virulence genes increases as well. The time of efficient use of resistance genes is thought to be prolonged if the area of host culture would shift against the direction of predominant dispersal.

**Keywords:** nomadic pathogens, cereal diseases, resistance genes, avirulence genes, population biology, wind dispersal, gene flow

An efficient and environmentally benign way of plant protection is through the use of genes conferring disease resistance (*R*-genes) in the host (PINK & HAND 2002; POLÁK & BARTOŠ 2002). The economic effect, however, varies according to the level of virulence genes (*V*-genes) enabling the pathogen to overcome the host resistance. For wind-dispersed nomadic diseases a novel pattern of the distribution of *V*-genes and virulence complexity, VC, i.e. the number of *V*-genes per pathogen genotype, became evident recently. The evidence suggests that virulence genes accumulate in the direction of prevailing winds, through the interaction of migration and selection. This is the case and, from the underlying logic, appears to have to be so, for pathogens that depend on dispersal to survive, i.e. for *obligate nomads*, in particular if polycyclic diseases are concerned like the cereal rusts and mildews that are so highly mobile (LIMPERT *et al.* 2002).

For leaf rust on wheat, VC increased from 2–4 on average in France to approximately 7 in Austria and Poland up to 14 and above in Siberia (after PARK & FELSENSTEIN 1998, re-evaluated; MESTERHÁZY *et al.* 2000). These and further results from other data sets confirm consistent evidence obtained with the barley mildew pathogen from where such observations and the concept originate (LIMPERT *et al.* 1999, 2000, and unpublished data).

The concept appears to be significant for any area with prevailing dispersal in certain directions, for continental Europe as well as for Great Britain, where predominant dispersal is from Scotland to England (LIMPERT *et al.* 2000). The example of *Mlo* virulence being most expressed in Japan (LYNGKJAER *et al.* 1995) at the very end of the *Eurasian wind path* suggests that genes acting quantitatively may also be concerned.

As major conditions of host presence and prevailing westerly winds are similar or the same in Europe and Asia, VC is thought to increase along the Eurasian path of population genetics from western Europe up to eastern Asia (LIMPERT *et al.* 2002). According to a more novel, fundamental and less established way of thinking it is the number of avirulence genes decreasing in that direction (DE WIT 1992; ROHE *et al.* 1995; JOOSTEN & DE WIT 1999) but, for convenience, the established terminology is kept here.

### CONCLUSIONS AND OUTLOOK

The hypothesis can be of considerable importance for the use of host resistance. The higher the virulence in a pathogen population, the more valuable should be the effective *R*-genes present in that area. Again, as further

discussed elsewhere (LIMPERT *et al.* 2000, 2002), this has to be regarded as a general trend and further points affecting the potential value of resistance genes of course continue to need careful consideration.

For example, a wild relative of barley, *Hordeum spontaneum*, is known for its resistance diversity that correlates with population size (JAHOR & FISCHBECK 1987; DREISEITL & DINOOR 2003). However if, with other conditions being the same, host populations of similar diversity would be compared, originating in Central Europe or the Near East on one hand or in the Far East on the other, from the above hypothesis there should be no doubt that the population from the Far East should be more valuable for disease resistance. Likewise, resistance from the Far East should be more durable according to reasons discussed elsewhere (LIMPERT *et al.* 1999).

If VC increases in the direction of predominant dispersal, the number of unnecessary virulence genes increases as well. Thus, the hypothesis also casts new light on the discussion of the costs of unnecessary virulence genes, and whether they may or may not be linked with genes for other traits of importance for pathogen fitness.

The present knowledge of the origin of certain *R*-genes of interest against the wheat powdery mildew pathogen (Table 1) is in line with our reasoning. Almost exclusively, the genes described originate from various regions in Asia, from the Hindukush, India and China. Moreover, in Chinese land races of wheat a number of further genes is expected (HSAM – pers. commun.). The concept should be of interest for other host-pathogen systems involving nomadic pathogens and crops of economic importance such as sugar beet, potato and tobacco, for diseases of grassland, and forest diseases.

Despite the considerable evidence and logic in favour of the hypothesis, it is but too obvious that we only recognised the tip of a novel iceberg so far, where the entire volume would be worth discovering. But one consequence of practical importance for disease resistance relates to the use of host resistance in space. The time of efficient use of *R*-genes is thought to be prolonged if the

area of host culture would shift *against* the direction of predominant dispersal, rather than *in* that direction (LIMPERT & BARTOŠ 1997).

For discovering the hypothesis, the cereal pathogens obviously had a pilot function. The hypothesis should be of similar importance for human and animal health, and for further populations affected by wind such as species of insects, spiders, and even some crustaceae living in temporary waters like certain Ostracodae the eggs of which appear to be as easily wind-dispersed as are mildew spores. Therefore, we recently submitted an Expression of Interest for the 6<sup>th</sup> EU Framework Programme entitled Effects of Wind and Bio-Aerosols on Health and Growth Across Europe, to which further thoughts are always welcome.

**Acknowledgements:** We are grateful to COST at Berne and Brussels for financial support.

## References

- DE WIT P.J.G.M. (1992): Molecular characterization of gene-for-gene systems in plant-fungus interactions and the application of avirulence genes in control of plant pathogens. *Ann. Rev. Phytopath.*, **30**: 391–418.
- DREISEITL A., DINOOR A. (2003): Phenotypic diversity of promising barley powdery mildew resistance sources. *Genet. Resour. & Crop Evol.* (accepted).
- HSAM S.L.K., HUANG Q.X., ZELLER F.J. (2001): Chromosomal location of genes for resistance to powdery mildew in common wheat (*Triticum aestivum* L. em Thell) 6. Alleles at the Pm5 locus. *Theor. Appl. Genet.*, **102**: 127–133.
- JAHOR A., FISCHBECK G. (1987): Genetical studies of resistance of powdery mildew in barley lines derived from *Hordeum spontaneum* collected in Israel. *Plant Breeding*, **99**: 265–273.
- JOOSTEN M.H.A.J., DE WIT P.J.G.M. (1999): The tomato-*Cladosporium fulvum*-interaction: A versatile experimental system to study plant pathogen-interactions. *Annu. Rev. Phytopath.*, **37**: 335–367.
- LIMPERT E., BARTOŠ P. (1997): Analysis of pathogen virulence as decision support for breeding and cultivar choice. In: HARTLEB H., HEITEFUSS H., HOPPE H.-H. (eds): Resistance of Crop Plants Against Fungi. Fischer, Jena: 401–424.
- LIMPERT E., GODET F., MÜLLER K. (1999): Dispersal of cereal mildews across Europe. *Agric. Forest. Met.*, **97**: 293–308.
- LIMPERT E., BARTOŠ P., GRABER W.K., MÜLLER K., FUCHS J.G. (2000): Increase of virulence complexity of nomadic airborne pathogens from west to east across Europe. *Acta Phytopath. Entomol. Hung.*, **35**: 261–272.
- LIMPERT E., BARTOŠ P., BUCHENAUER H., GRABER W.K., MÜLLER K., ŠEBESTA J., FUCHS J.G. (2002): Airborne nomadic pathogens: Does virulence genes accumulate along the way from Paris to Beijing? Proc. 6<sup>th</sup> EFPP Conf., Prague, *Plant Protect. Sci.*, **38** (Special Issue 1): 66–64.

Table 1. Origin of *R*-genes against wheat powdery mildew (HSAM *et al.* 2001 for further references, and pers. commun.)

Gene	Alleles/Origin	Geographic origin
<i>Pm3</i>	10 alleles	Hindukush (mostly)
<i>Pm5</i>	a Hope (Australia)	Israel?
	b Mli (Germany)	Hindukush?
	c India	
	d spelt?	Asia
	e China	
<i>Pm24</i>	China	

- LYNGKJAER M.F., JENSEN H.P., OSTERGARD H. (1995): A Japanese powdery mildew isolate with exceptionally large infection efficiency on *Mlo*-resistant barley. *Plant Pathol.*, **45**: 786–790.
- MESTERHÁZY Á., ANDERSEN O., BARTOŠ P., CASULLI F., CSÓSZ M., GOYEAU H., ITTU M., JONES E., MANISTERSKI J., MANNINGER K., PASQUINI M., RUBIALES D., SCHACHERMAYER G., STRZEMBICKA A., SZUNICS L., TODOROVA M., UNGER O., VANCO B., VIDA G., WALTHER U. (2000): European virulence survey for leaf rust in wheat. *Agronomie*, **20**: 793–804.
- ROHE M., GIERLICH A., HERMANN H., HAHN M., SCHMIDT B., ROSAHL A., KNOGGE W. (1995): The race-specific elicitor, NIP1, from the barley pathogen, *Rhynchosporium secalis*, determines avirulence on host plants of the *Rrs1* resistance genotype. *EMBO*, **14**: 4168–4177.
- PARK R.F., FELSENSTEIN F.G. (1998): Physiological specialization and pathotype distribution of *Puccinia recondita* in western Europe, 1995. *Plant Pathol.*, **47**, 157–164.
- PINK D.A.C., HAND P. (2002): Plant resistance and strategies for breeding resistant varieties. Proc. 6<sup>th</sup> EFPP Conf., Prague, *Plant Protect. Sci.*, **38** (Special Issue 1): 9–14.
- POLÁK J., BARTOŠ P. (2002): Natural sources of resistance to plant diseases and their importance in plant breeding. *Czech J. Genet. Plant Breed.*, **38**: 146–149.