

# Lecture 1

## The basics of host : pathogen genetics

This is the DNA era – our scientists are increasingly well trained in DNA and DNA marker technologies, but are decreasingly knowledgeable about basic (Mendelian) genetic principles of host : pathogen genetics and the biology of disease systems.

The rust pathogens of cereals are obligate fungal pathogens belonging to the Basidiomycetes. They are dikaryotic and behave as functional diploids. *Puccinia graminis* and *P. triticina* and *P. recondita* are heteroecious, having alternate hosts on which the sexual cycle occurs. They are described as macrocyclic pathogens with five different spore types. *P. striiformis* is microcyclic in having only functional urediniospores.

In addition to **alternate hosts**, the rust pathogens have a range of **alternative or ancillary** hosts on which the uredinial stage may occur. For example, barley is an alternative host for the wheat stem rust pathogen. The range of alternative hosts suggested in the literature may be larger than normally occurs under natural conditions.

### Some concepts

The disease triangle

The disease cycle

Measurement of disease: infection type, area of infection/tissue damage, co-efficient of infection, disease progress curves, AUDPC

### Terminology

Response or reaction – resistance and susceptibility.

Pathogenicity – avirulence and virulence

Infection type – the symptoms that we observe. These are used to make inferences about the host, the pathogen, or in some cases, the environment.

### The laws of host pathogen genetics

These have similarities to the Mendelian laws of genetics: LIT = low infection type; HIT = High infection type

Law 1: LIT = Low reaction / Low pathogenicity

Law 2: LIT<sub>1,2</sub> = or < LIT<sub>1</sub>, where LIT<sub>1</sub> < LIT<sub>2</sub>

### Basic experiments in host : pathogen genetics

Four types of experiments are performed in H:P genetics:

- gene postulation without any knowledge of the genetics of host or pathogen
- sub-division of pathogen phenotypes into races (or strains or pathotypes) based on their responses on host lines with different genes for resistance
- identification of host variation in resistance based on responses to different races of the pathogen

- using characterised host lines and pathogen lines in laboratory experiments; for example, to study the effects of temperature on a particular h:p interaction.

### Gene postulation

Based on analyses of matrices of pathogen isolate and host line interaction data; that is, row and column arrays are sorted into similar pathotypes and resistance groups

Infection types are much more informative than simple incompatible / compatible (resistance or susceptible in host terminology, or avirulent or virulent in pathogen terminology) arrays.

Control host lines or pathogen races enable the arrays to be classified relative to what is known. Because many of the resistance genes in a local host population will be known, these can be targeted by selected pathotypes (or markers) to allow us to focus on what might be new. Work on gene discovery can then concentrate on lines with potentially new genes for resistance.

### Variability in rust pathogens

Rust pathogens are subject to the laws that affect gene frequency in all living organisms.

- **Migration.** The threat of spread of race Ug99 to new areas in Iran and to countries such as Pakistan and India is an excellent example. The occurrences of stripe rust in Australia in 1979 and in 2002, and a clearly different group of races in the USA in 2000 are other examples. The introduction of barley leaf rust to Central America and its subsequent spread throughout the Americas over a 25 year period is another.

- **Mutation.** Single virulence changes within local races are due to mutation. In order to be a selected mutant such a change presumably occurs in a heterozygous avirulent race, either as an SNP mutation in an avirulence gene, or a deletion of the entire locus. Mutation pathways have been followed in Australia. These can also be simulated by induced mutation in the laboratory. A predominant pathotype in South Africa appears to be identical with Ug99, except that it is avirulent for *Sr31*.

- **Recombination.** Although recombination is more important in sexually reproducing organisms, the rusts are capable of undergoing asexual recombination by **somatic hybridisation**, a process that is not fully understood. Good examples of somatic hybridization leading to important variation in *P. graminis* were documented in Australia.

- **Selection.** New variants in a population, irrespective of origin, may increase by selection (otherwise they might never be detected). Their long term significance and survival will depend on their selective survival relative to other genotypes in the population.

- **Chance.** Chance effects are more likely to occur in small populations. Although we usually think that rust pathogens occur as very large populations, but they must also survive between cropping seasons, and through prolonged drought periods, and thus can easily be affected by chance effects.

### Pathogenicity surveys

Pathogen populations are monitored by surveys of disease incidence and severity in farmers' fields. Pathogen variability is monitored by knowing the varieties and their past responses, by the use of trap plots of key varieties, or by systematic sampling and identification of races in the laboratory.

Molecular biologists have a vision of being able to monitor these predominantly clonal populations by molecular methods. It may be possible to differentiate clonal groups, but identifying variants differing by single mutation events will depend on being able to monitor and differentiate the key avirulence allele products or effectors. In rust pathogens potential effectors have been isolated from haustoria.

## Resistance

### **Methods of genetic analysis**

- Conventional hybrid analysis of selfed or testcrossed populations, SSD and DH lines.
- Chromosome location using aneuploids and deletion lines (telocentric mapping)
- Gene location and mapping with markers – morphological & physiological markers, protein markers, DNA markers - RFLP, RAPD, SSR, RGA, SNP
- Tests of allelism
- Resistance gene naming

### **Types of resistance**

Resistance may be classified in two groups:

- Seedling or all-stage resistance. These are mainly major gene resistances.

Post-seedling or adult plant resistance, including high temperature adult plant resistance (HTAPR). These can include major gene resistances, such as *Lr12* and *Lr35*, predominantly single gene resistances, such as *Sr2*, or minor additive slow rusting resistances such as those involving *Lr34/Yr18* and *Lr46*.

### **Cloned resistance genes**

Some major resistance genes in wheat have been cloned. Like many other cloned resistance genes, the structures of *Lr1*, *Lr10* and probably *Yr10* are NBS-LRR. The LRR regions of such genes are probably involved in specificity and the NBS components, in signalling. It is interesting that the two slow rusting genes that have been cloned have different structures; *Lr34/Yr18* is an ABC (ATP-binding cassette) transporter, and *Yr36* has a kinase and putative START lipid-binding domain. Most importantly, these genes have structures that are different from the NBS-LRR type.