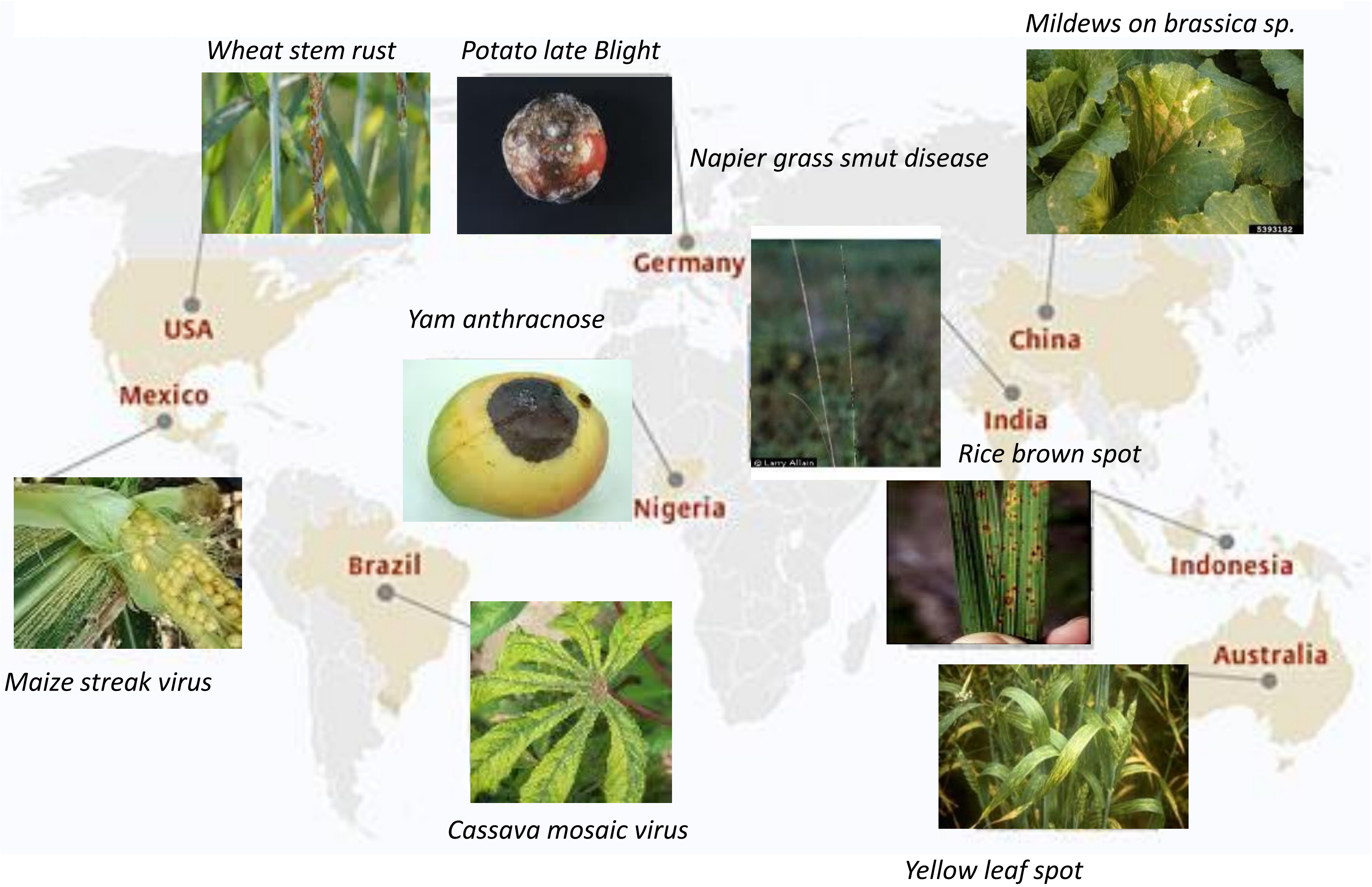
Securing food through pest and disease management under changed climates

Rebecca Ford
Department of Agriculture and Food Systems
The University of Melbourne
rebeccaf@unimelb.edu.au

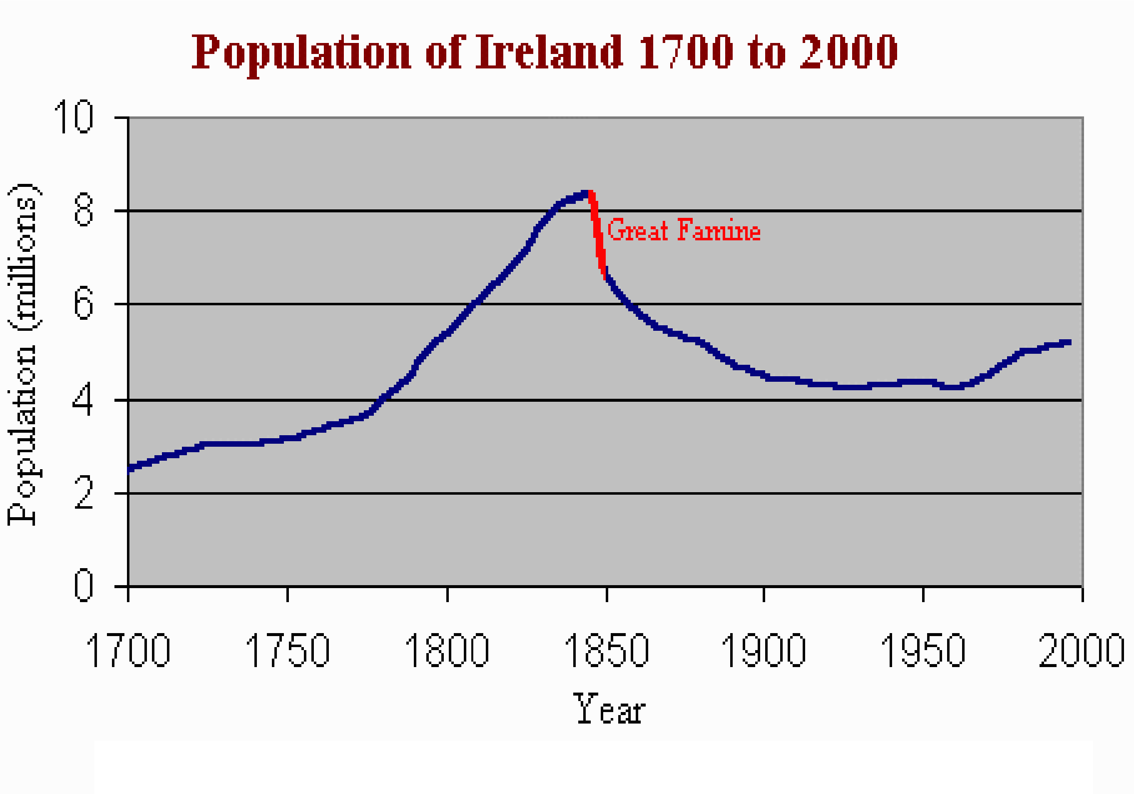
Feeding the World - The major food staples

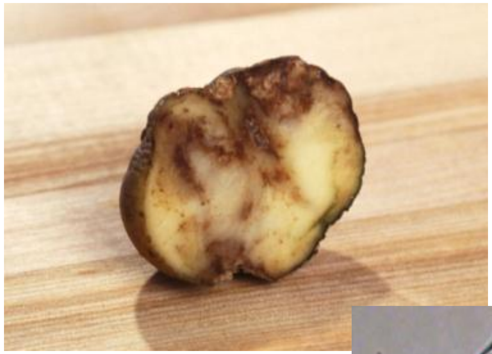
Diseases that have caused famines



Then - Potato Late Blight

- *Phytophthora infestans*
- Oomycete (water mould)
- Wet mild winters
- Sole cause of 1845 irish potato famine - 1M people starved, 2M people migrated





www.physorg.com/news119626269.html

Now - Stem rust fungus of wheat

• Wheat provides 1/3 of the world's calories

• Fungus = *Puccinia* ssp.

• Increasingly occurring in areas with milder winters followed by cool wet springs and dry cool summers

• New highly virulent strain = Ug99

• Sr35 resistance gene just identified – KSU and UC Davis

• 137 identified wheat stripe rust races in the U.S. - 17 new each year

• Average life span of an "all-stage" resistance gene is 3.5 years

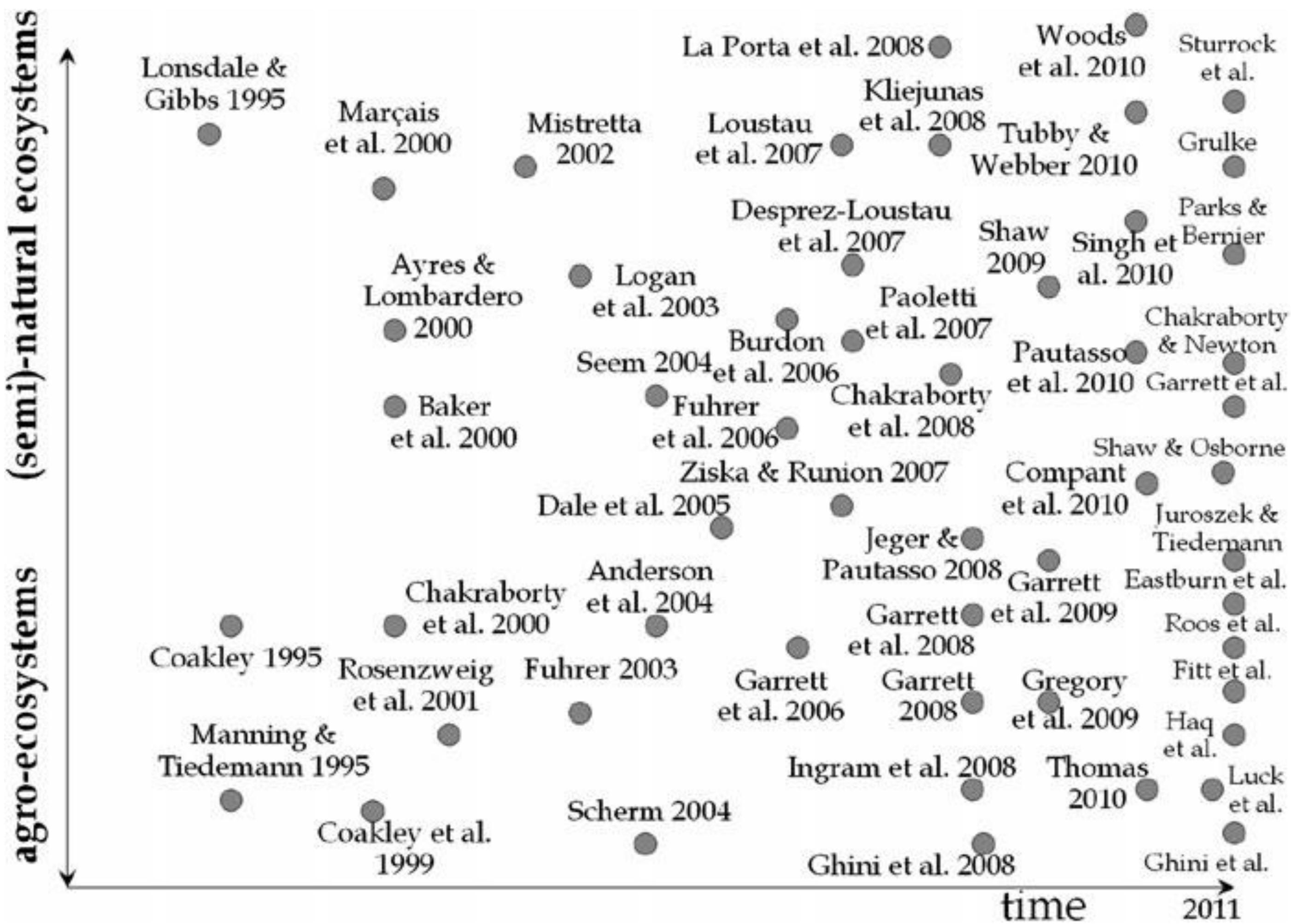
teliospores

Perfect storms

Climate drives the frequency and severity of epidemics

- **Initiation**
  - Source and amount of inoculum
  - Availability of a susceptible host
  - Health of the host
  - Health of the pathogen
  - Recognition of the pathogen
  - **Favourable environment**
- **Development**
  - Primary infection and colonisation
  - **Favourable environment**
- **Spread**
  - Dissemination (vector, wind, water, human machinery)
  - Secondary inoculum
  - **Favourable environment**

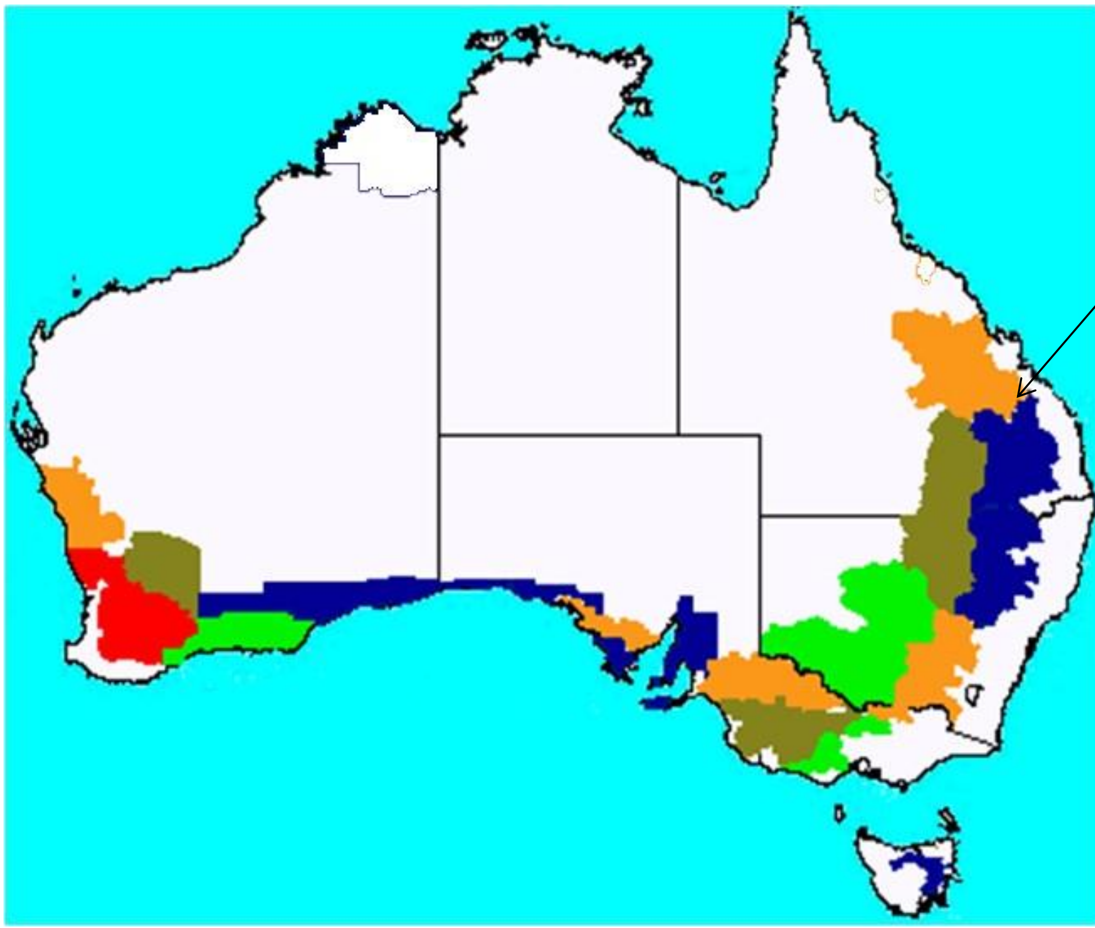
Impact studies of climate change on plant health (from Pautasso et al 2012)



Transformational changes within industries

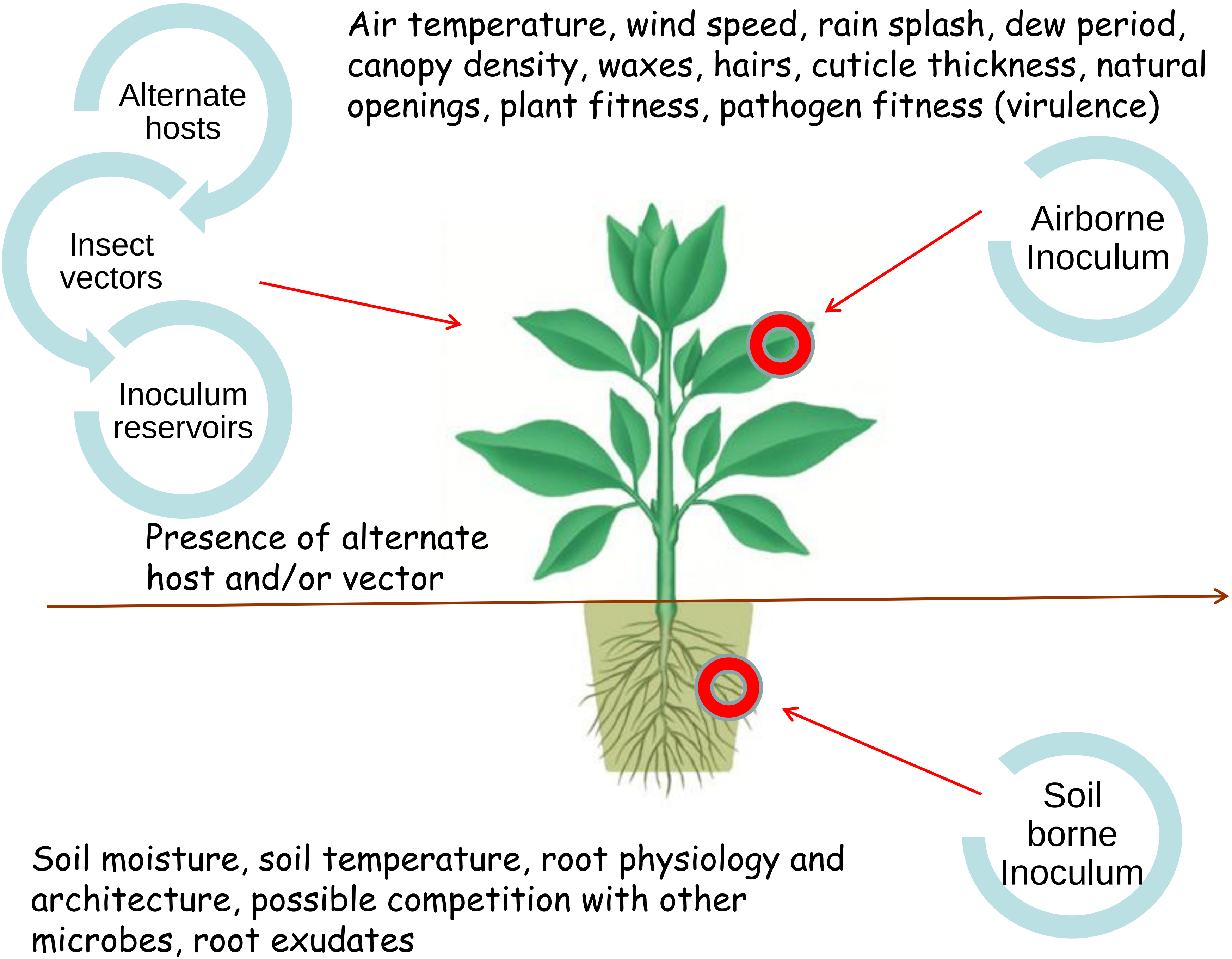
The chickpea story

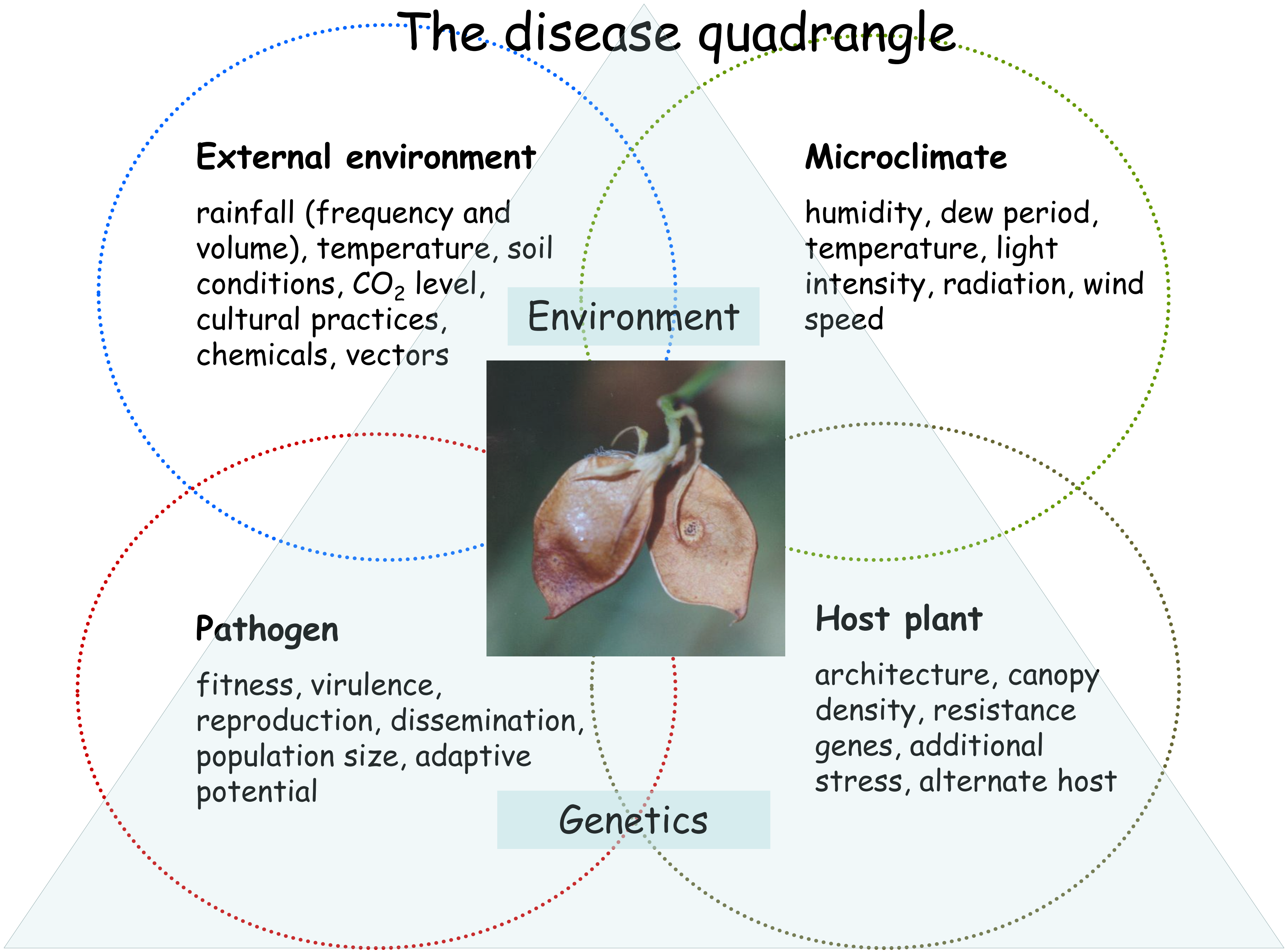
"Moving north to where the water is" – consequences



Severe Ascochyta Blight epidemics in 2010

Source: Murray and Brennan (2011). Current and potential costs from diseases of pulse crops in Australia. GRDC report


Air temperature, wind speed, rain splash, dew period, canopy density, waxes, hairs, cuticle thickness, natural openings, plant fitness, pathogen fitness (virulence)
Alternate hosts
Insect vectors
Inoculum reservoirs
Airborne Inoculum
Presence of alternate host and/or vector
Soil borne Inoculum
Soil moisture, soil temperature, root physiology and architecture, possible competition with other microbes, root exudates

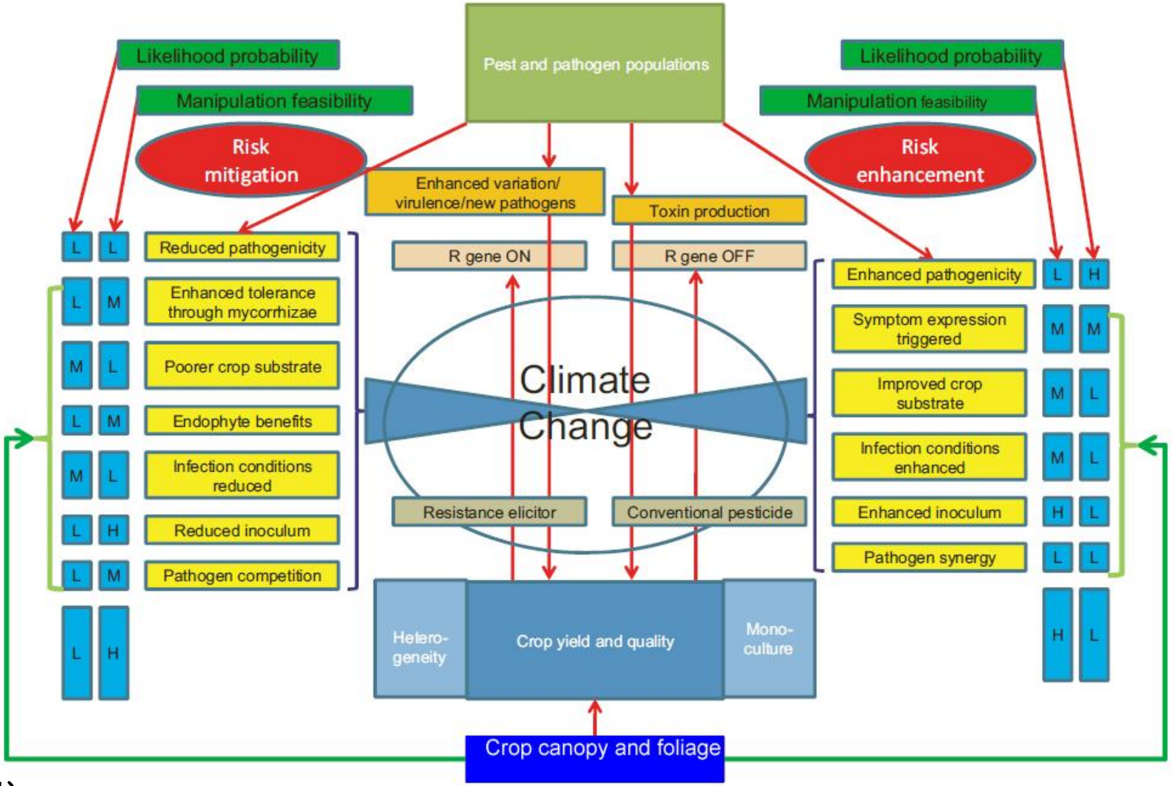

The disease quadrangle
External environment
rainfall (frequency and volume), temperature, soil conditions, CO_2 level, cultural practices, chemicals, vectors
Microclimate
humidity, dew period, temperature, light intensity, radiation, wind speed
Environment
Pathogen
fitness, virulence, reproduction, dissemination, population size, adaptive potential
Host plant
architecture, canopy density, resistance genes, additional stress, alternate host
Genetics

Predictability, modelling and extrapolation

Based on

- Already observed effects of climate change on plant diseases
- Extrapolation from expert knowledge on host and pathogen behaviours
- Multifactorial models.

Much uncertainty in modelling risk



Adapted from Chakraborty and Newton (2011)

Risk of increased loss from wheat rusts (from Chakraborty et al, 2010)

- Changes in host-pathogen interaction
- Changes in host morphology, anatomy, physiology
- Changes in fungal pathogenicity and populations

1. Risk of new race evolving faster

- Larger pathogen population size from increased crop biomass
- Greater pathogen fitness
- Increased number of infection cycles from higher temperature

2. Risk of more severe rust infection from environmental stress impacts on the host

- Changed geographical distribution of wheat growing areas
- New pathotypes/strains, adaptive potential

3. Risk of resistance breakdown

- Temperature effects on rust resistance gene expression
- Fungal diversity

Predicted pathogenicity changes

Crop	Disease and pathogen	Predicted influence of climate change on disease	Reference
Barley	Powdery mildew – *Blumeria graminis*	Decrease at higher CO_2	Hibberd *et al*, 1996
Rice	Leaf blast – *Magnaportha oryzae*	Increase at higher CO_2	Kobayashi *et al*, 2006
Soybean	Brown spot – *Septoria glycines*	Increase at higher CO_2	Eastburn *et al*, 2010
Soybean	Sudden death syndrome – *Fusarium virguliforme*	No effect at higher CO_2	Eastburn *et al*, 2010
Wheat	Stripe rust – *Puccinia striiformis*	Increase with higher temperature	Coakley, 1979; Chakraborty *et al*, 1998; Milus *et al*, 2006
Wheat	Crown rot – *Fusarium pseudograminearum*	Increase at higher CO_2, cultivar and soil water dependant	Chakraborty *et al*, 1998 ; Mulloy *et al*, 2010

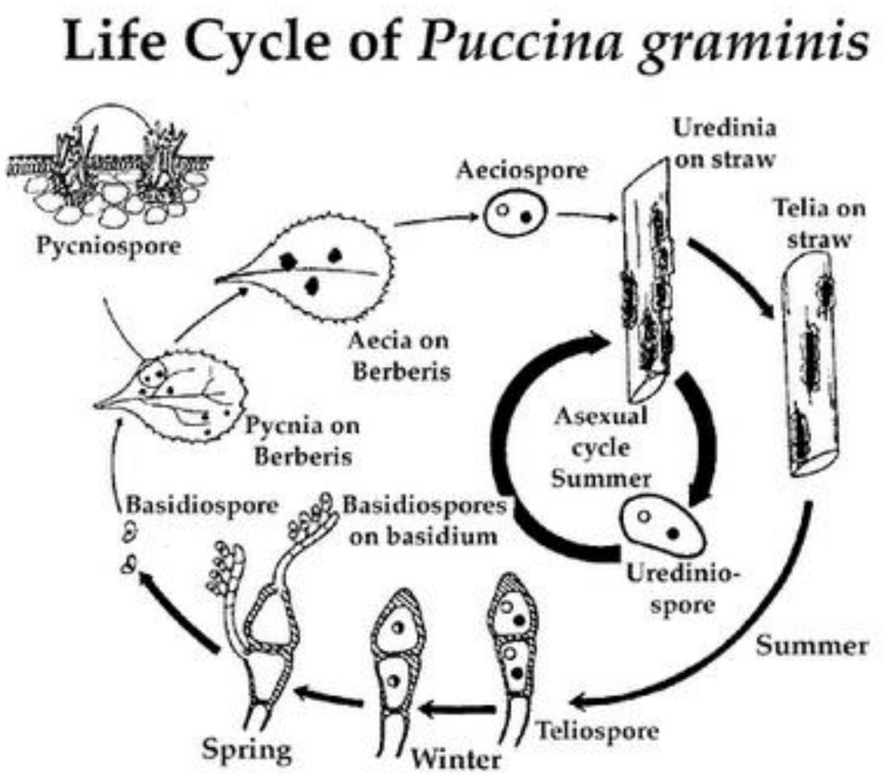
Adapted from Luck *et al*, (2011) Plant Pathology 60: 113-121

Pathogen behaviour

Effect of climatic conditions on……

- Spatial and temporal distribution
  - Dispersal - seed, rain splash, wind, vector
- Fitness and survival
  - Virulence – ability to produce pathogenic factors/elicitor compounds
  - Over-season - survival structures
- Evolutionary potential (selective adaptation)
  - Population diversity and structure
  - Reproductive system (recombination verses clonal)
  - Effect of host genotype changes
  - Effect of other management changes – chemical and cultural

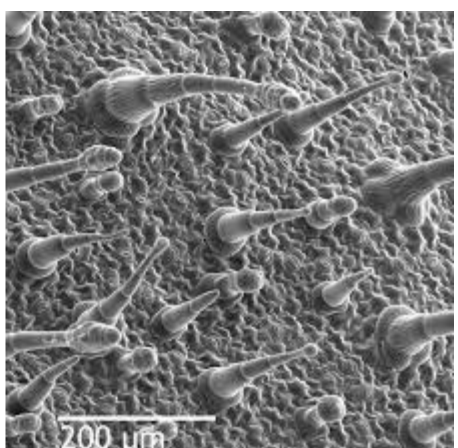
Challenges for disease management

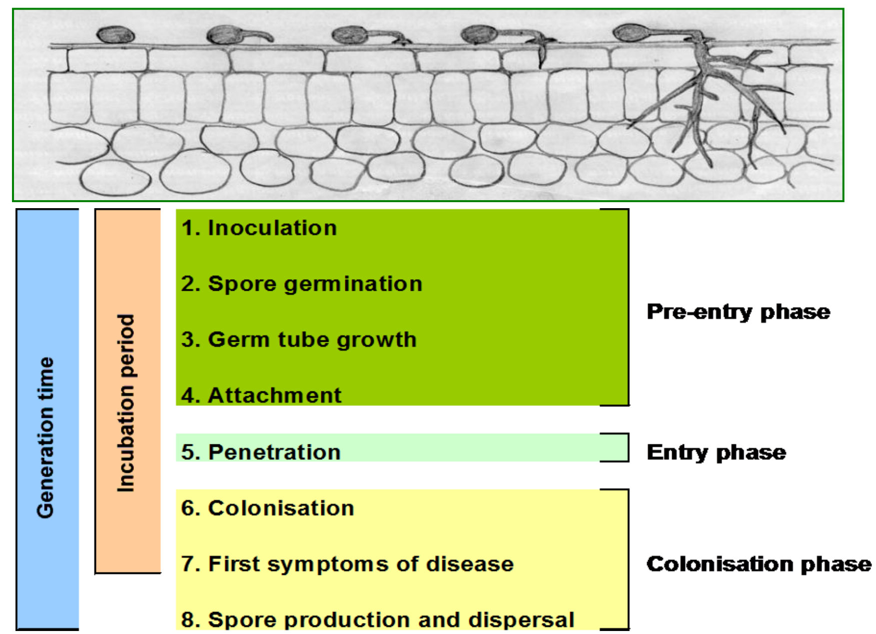


- Several growth stage (spore types)
- Able to over-winter on straw/stubble
- Spores fly a long way
- Multiple epidemics in one season

Host behaviour

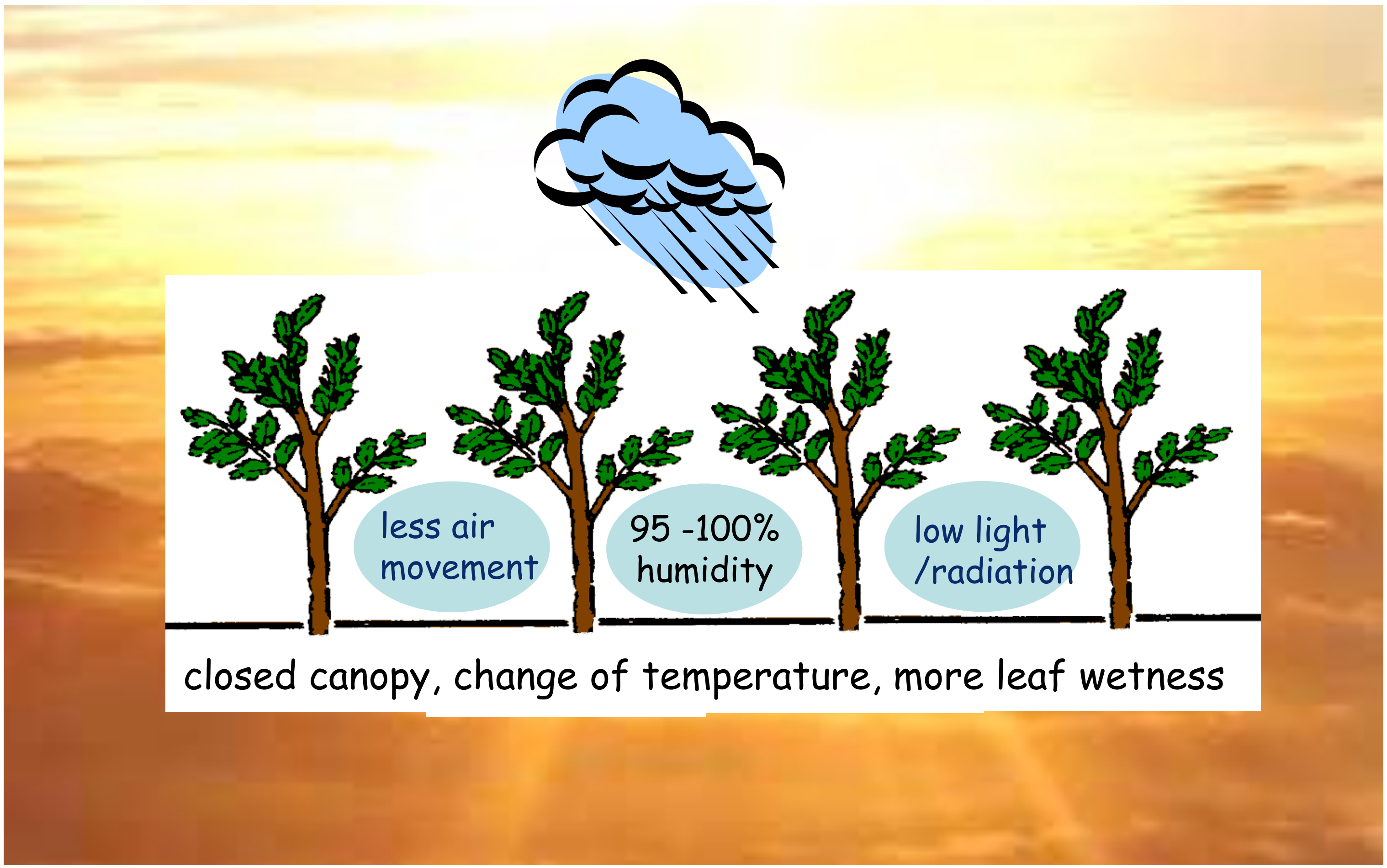
- **Physiological**
  - Canopy density and subsequent microclimate
  - Wax and thickness
  - Hairs and density
  - Cuticle and epidermal cell wall thickness
  - Stomata and other natural openings (eCO2 effect)
  - Concurrent abiotic/biotic stresses





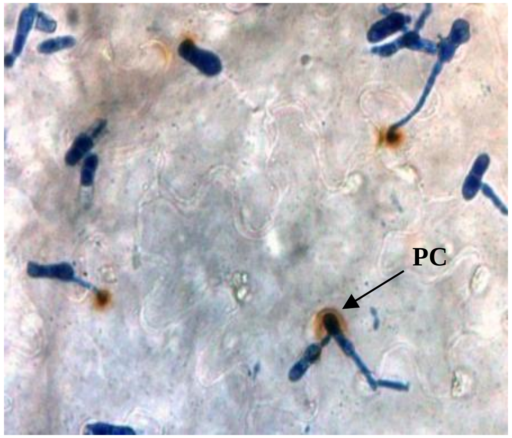
Canopy density affects the microclimate

(eCo_2 increases biomass)

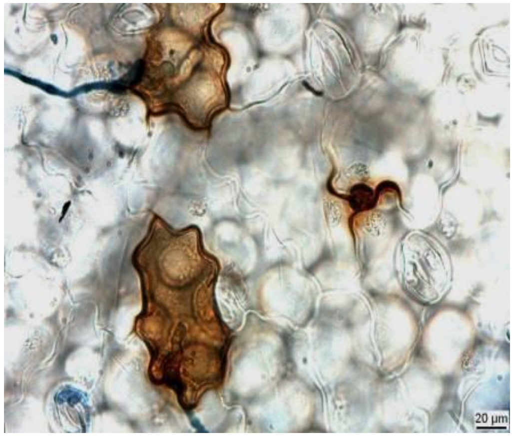


Host changes affect disease development

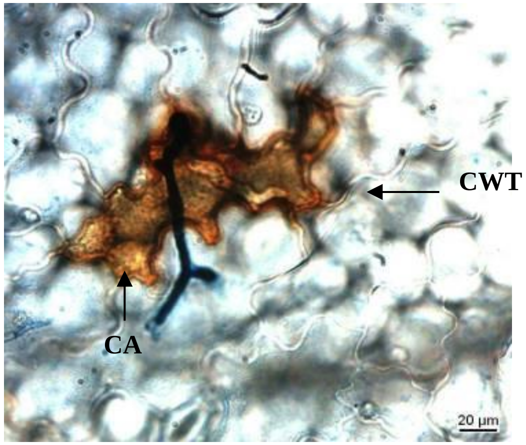
- **Biochemical and molecular**
  - Volatile gas production – recognition for entry points (stomata opening)
  - Antimicrobial compound production (ie chitinase) – primary defence molecules
  - Receptor molecule production (pathogen-associated molecular patterns)



20 hai = visualise phenolic compound accumulation

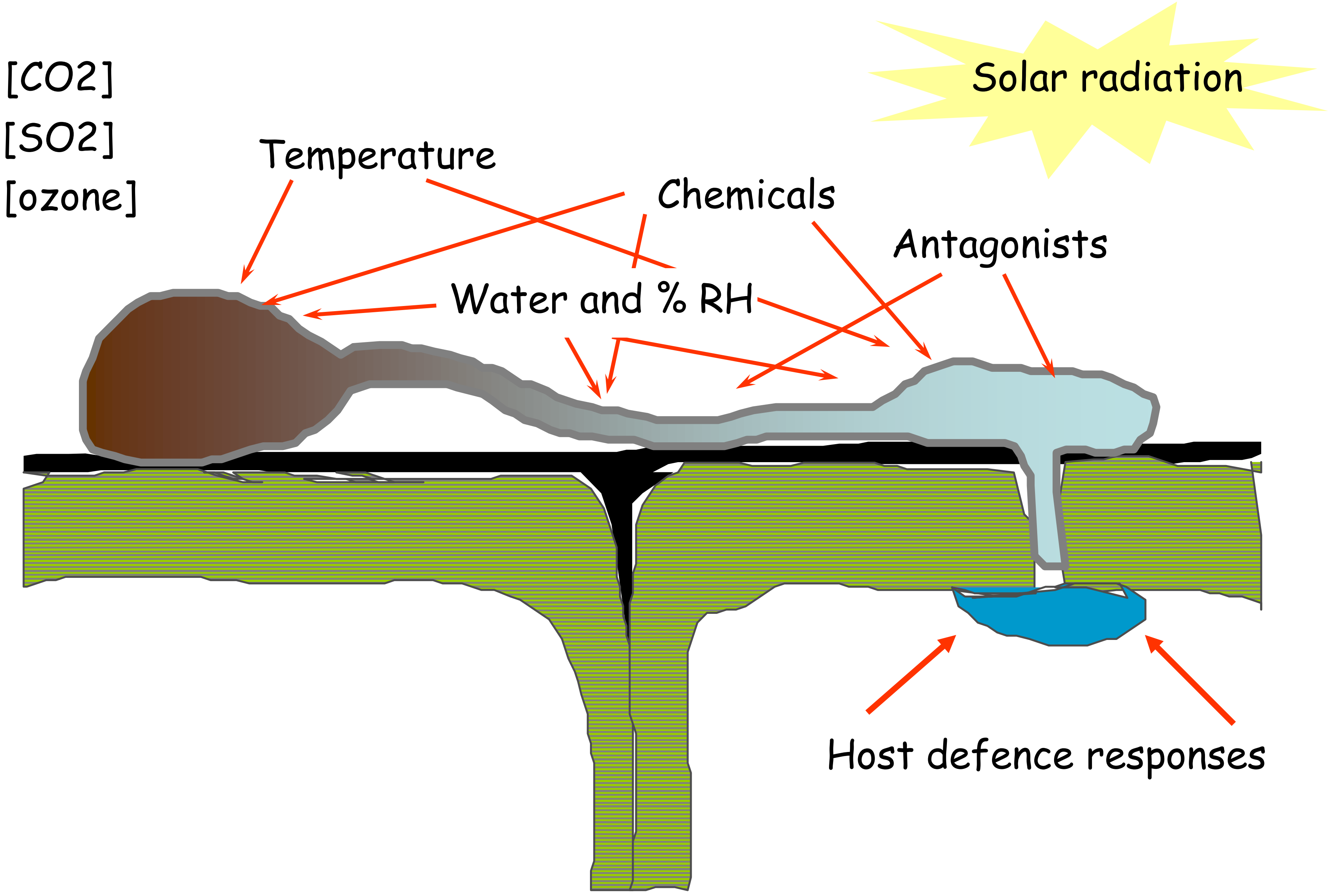


28 hai = containment of PC



36 hai = cell wall thickening (CWT) and cytoplasmic aggregation (CA)

Pathogen-host interaction



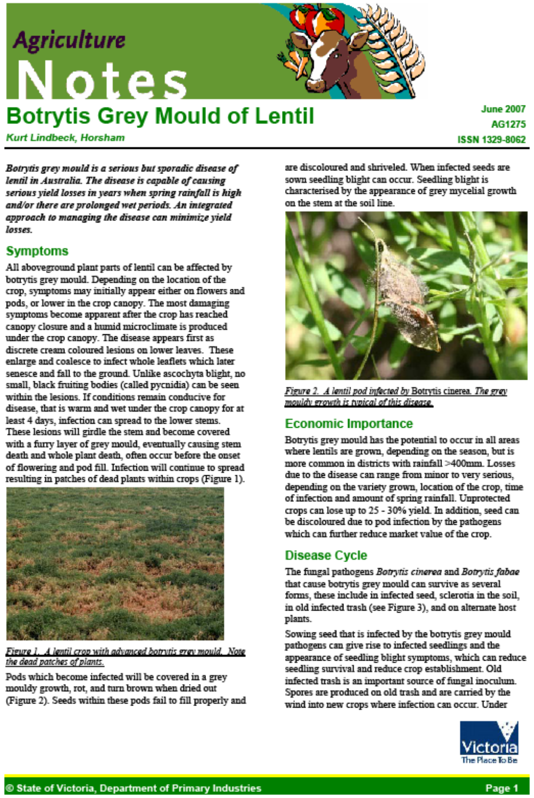
Where the research is required…..

Studies on individual pathosystems – broad conclusions are misleading

Multiple agroecological studies of climate change effects on
- Pathogen behaviour (population dynamics, pathogenicity, toxic compounds)
- Host behavior (physiological, molecular)
- Pathogen-host interaction changes (disease reaction R/S)

Revised disease management plans based on
- Existing knowledge
- New cultivars
- Adapted cultural strategies to
    - Alter microclimates (planting density)
    - Manage pathogens (chemical timing, biosecurity)

Smarter resistance breeding based on
- Molecular knowledge
    - Targeted R-genes
    - Pathogen recognition
    - Non-recognition


Agriculture Notes

Botrytis Grey Mould of Lentil

Kurt Lindbeck, Horsham

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Botrytis grey mould is a serious but sporadic disease of lentil in Australia. The disease is capable of causing serious yield losses in years when spring rainfall is high and/or there are prolonged wet periods. An integrated approach to managing the disease can minimize yield losses.

Symptoms

All aboveground plant parts of lentil can be affected by botrytis grey mould. Depending on the location of the crop, symptoms may initially appear either on flowers and pods, or lower in the crop canopy. The most damaging symptoms become apparent after the crop has reached canopy closure and a humid microclimate is produced under the crop canopy. The disease appears first as discrete cream coloured lesions on lower leaves. These enlarge and coalesce to infect whole leaflets which later senesce and fall to the ground. Unlike ascochyta blight, no small, black fruiting bodies (called pycnidia) can be seen within the lesions. If conditions remain conducive for disease, that is warm and wet under the crop canopy for at least 4 days, infection can spread to the lower stems. These lesions will girdle the stem and become covered with a furry layer of grey mould, eventually causing stem death and whole plant death, often occur before the onset of flowering and pod fill. Infection will continue to spread resulting in patches of dead plants within crops (Figure 1).

Figure 1. A lentil crop with advanced botrytis grey mould. Note the dead patches of plants.

Pods which become infected will be covered in a grey mouldy growth, rot, and turn brown when dried out (Figure 2). Seeds within these pods fail to fill properly and are discoloured and shriveled. When infected seeds are sown seedling blight can occur. Seedling blight is characterised by the appearance of grey mycelial growth on the stem at the soil line.

Figure 2. A lentil pod infected by Botrytis cinerea. *The grey mouldy growth is typical of this disease.*

Economic Importance

Botrytis grey mould has the potential to occur in all areas where lentils are grown, depending on the season, but is more common in districts with rainfall >400mm. Losses due to the disease can range from minor to very serious, depending on the variety grown, location of the crop, time of infection and amount of spring rainfall. Unprotected crops can lose up to 25 - 30% yield. In addition, seed can be discoloured due to pod infection by the pathogens which can further reduce market value of the crop.

Disease Cycle

The fungal pathogens *Botrytis cinerea* and *Botrytis fabae* that cause botrytis grey mould can survive as several forms, these include in infected seed, sclerotia in the soil, in old infected trash (see Figure 3), and on alternate host plants.

Sowing seed that is infected by the botrytis grey mould pathogens can give rise to infected seedlings and the appearance of seedling blight symptoms, which can reduce seedling survival and reduce crop establishment. Old infected trash is an important source of fungal inoculum. Spores are produced on old trash and are carried by the wind into new crops where infection can occur. Under

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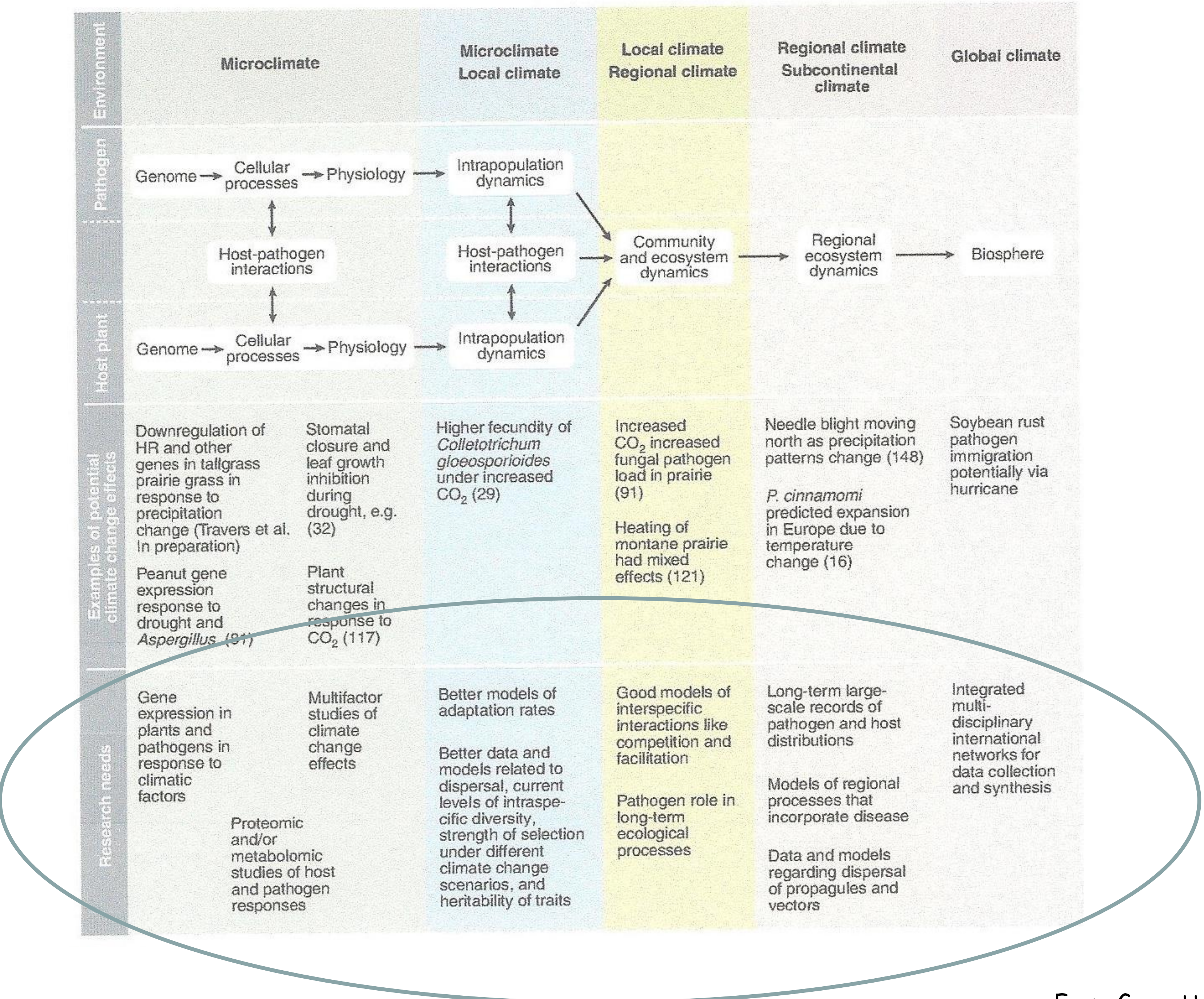

From Garrett et al (2006).