- Rearrangement of cytoskeleton and secretory pathways
- Microbe is trapped at this site and cannot penetrate and access nutrients
- If the response is strong, plant suicide will kill the plant cell but also the parasite
- Can see cell wall fortification with EM
- Papilla – cell wall fortifications made up of β-glucan called callose
    - Callose is main plant cell wall component that accumulates at the pathogen penetration site
    - Callose accumulation around pathogen contact sites leads to cell wall fortification and limited penetration
    - Different structure from cellulose
    - Can label separately
    - Specialised microbes can breach the papilla

What happens during cell polarisation? - Summary

- Cytoskeleton is reorganised/reprogrammed to direct the endomembrane system towards the pathogen entry site
- This enables targeted secretion towards the site of pathogen contact
- Can bombard the microbe with antimicrobial proteins
- Especially targeted deployment of callose and phenolics of a highly divergent nature leads to wall thickening at fungal penetration sites termed papillae, which often correlates with failure of fungal ingress

Reprogramming of cellular events during penetration/activation of focal immunity

Accumulation of phenolic compounds can lead to the formation of an effective papilla

- Pathogen contact and penetration leads to accumulation of phenolic compounds
- Effective papilla
    - High callose and AX deposition
    - High polysaccharide-FA cross-linking
    - Penetration peg trapped in papilla
- Effective papilla formation leads to:
    - Decrease in callose deposition
    - High AX deposition continues
    - High cellulose deposition

In the case of susceptibility, there is still accumulation of phenolic compounds

- Ineffective papilla formation allows penetration
    - Low callose and AX deposition
    - Low polysaccharide-FA cross-linking
    - Penetration peg overcomes papilla barrier
- Ineffective papilla leads to:
    - Papilla shrinking to form the collar
    - Haustorial primordia formation
    - Pathogen can invade the plant

- Focal immunity includes cytoplasmic streaming of endomembrane system and nucleus towards pathogen ingress sites, followed by targeted cell wall fortifications and defense related secretion
- Focal immunity is suppressed during susceptibility
- Pathogens penetrating the plant cells are enveloped by host derived membranes with unknown origin

Part II – Surface immune recognition in plants

Most plants are actively resistant to most pathogens

- Disease is the exception, not the rule
- Disease occurs certain environment conditions favouring the pathogen and specialised pathogens that are able to infect the plant
- Plants have an innate ability to recognise potential invade pathogens and to mount effective defences
    - Plants can produce their own food so simply produce a barrier to keep everything out

Local defence

- Occurs at the site of infection – local immune response to prevent spread of parasites
- First line of protection – innate immunity, cell autonomous immunity
- Very early detection of invaders in infected cells through specific or generic interactions
- Restricts pathogen grown and spread (programmed cell death, production of defensive molecules)
- Local response can include hypersensitive response – induced cell death at the point of infection, programmed cell death
    - Can also happen in animals – cell suicide to warn the immune system

Systemic defence

- Protects non-infected tissues from secondary infection
- Signals transmitted throughout plant cell – probably multiple types of signals
- Discovered that plants have an electric singlaling system using glutamate-like receptors
    - Not as fast as our signalling response but similar system
- Systemic activation of plant activation following damage
- SAR – local defence activator alerts the plant
    - Another challenge with a pathogen in a different place – plant is resistant
    - Upregulation of defence systems throughout the plant
- SAR = systemic acquired resistance
- Prioritise this over growth

Surface receptors mediate basal immunity – pattern recognition is key

- Microbes come from outside the cell so makes sense to have receptors on the cell surface
- Extracellular domains to sense pathogens
- Transmembrane domain to transmit signalling
- Cytoplasmic domain to mediate downstream signalling and activate immune response
- PRRs recognise PAMPs (pathogen-associated molecular pathogens)
- PAMPs are conserved molecules essential for lifestyle of pathogen (common molecule) but not necessarily required for infection process; associated with a group of pathogens; recognised by cells of the immune system
    - LPS – protects bacterial cell

Some apoplastic effectors 'trip the wire' and activate immunity in particular plant genotypes

- PAMP activation in plants does not necessarily lead to cell death activation
- Surface receptors in plants that bind to specific molecules secreted by the pathogen to subvert plant processes – virulence factors
    - Required for virulence function of the pathogen (unlike PAMPs)
    - High selective pressure, evolving fast
    - Not conserved like PAMPs
- Plants have strategies to capture these – generally use receptor-like proteins
    - No kinase domain for signalling
- Receptor-like proteins signal through structural change; cytoplasmic tail can interact with signalling molecules
- BAK1 can also pair with this type of receptor to mediate signalling
- Activation through specialised virulence factors can be direct or indirect
    - Factors target guardee, which is recognised by the plant receptor
    - Receptor recognises guardee to trigger basal defences and induce PCD (hypersensitive response)
    - Guardee detection acts as a confirmation signal – no question that there is a real threat present as detects specific virulence factors
- This is effector-triggered immunity
    - Involves cell surface receptors but the majority of signalling is intracellular

Part II summary

- Plants rely on surface immune receptors to sense and respond to PAMPs released by invading pathogens
- PAMPs are conserved to a degree which are able to detect presence of a group of pathogens rather than being specific to certain pathogen strain
- PTI leads to various cellular outputs including ROS production, defense gene induction and cell wall fortifications etc.
- Some apoplastic effectors are recognized by surface immune receptors leading to HR