Transmission of Ash Dieback

A European Epidemic
Common and narrow leaf ash trees are now threatened on a continental scale by an emerging disease, ‘ash dieback’, caused by the ascomycete fungus *Chalara fraxinea*. The process of an emerging infectious disease can be understood in three steps:
- Introduction into a new habitat
- Initial colonisation and establishment in the habitat
- Subsequent dispersal and secondary spread into additional habitats.

Introduction into a new habitat is typically due directly or indirectly to human activities. Genetic evidence suggests that the fungus was introduced into Europe from East Asia, possibly Japan, and multiple introductions may have occurred. The transmission potential of the pathogen within the host population of ash trees has determined the extent and dynamics of the epidemic. Transmission potential is influenced by a complex array of factors, such as the pathogen (the fungus) and host ecology (ash trees), host distribution, host genetics, pathogen genetics and environmental disturbance. Invasive pathogens usually have a number of common features, such as efficient dispersal through spores spread by wind, water or via an insect vector.

Life Cycle and Long Range Dispersal
Both the sexual phase and asexual phase of some pathogenic fungi can produce spores. The spores produced by the sexual phase, ascospores, are known to play a major role in transmission of *C. fraxinea*. Windborne ascospores are released during the summer by the sexual phase growing on leaf stalks infected the previous year and start new infections on healthy ash leaves. The fungus colonises different parts of the tree, including shoots, buds, bark, wood and roots, where it causes wilts, lesions, cankers and discolours the wood. Although asexual spores (conidia) have been observed on infected material, their role in dispersal remains unclear.

It is likely that ascospores are the main means of long range dispersal of the pathogen. Monitoring in Norway has shown high numbers of ascospores in a group of diseased ash, with approximately 2.8 million spores discharged per hour at the peak time in the morning in July (the period for sporulation is June to September). It is not known whether the number of spores released correlates with particular weather conditions such as temperature, precipitation and relative humidity.

Ascospores are viable (infectious) for only a limited period. Movement of diseased seedlings by the nursery trade appear to have accelerated the spread of the pathogen, by creating new disease sources long distances away from woodland infection sources. Both natural windborne and nursery trade transmission are implicated by the pattern of outbreaks of ash dieback across Europe. However, transmission by private movement of seedlings or disease material cannot be discounted.

Spread in the UK
The Epidemiology and Modelling Group in the Department of Plant Sciences at Cambridge University used meteorological models to assess the relative risk to different UK regions from airborne ascospores from the whole of continental Europe. The results suggest that between 2008 and 2011, there were at least 100 days on which environmental conditions including wind direction, rainfall and humidity meant that infection could have occurred, predominantly in east and south-east England. Given the difficulties in modelling transmission when the initial wider environment outbreak site in the UK remains unknown, this remains only a probable path of transmission, and further modelling work is underway to assess the comparative probability of the trade network as a path of transmission. Epidemiological modelling to predict the spread within the UK is also being undertaken to inform where mitigation and sampling strategies would be cost-effective. This will also allow the development of risk maps that show the probability of the disease spreading to different parts of the country over time, and hazard maps that show the relative impact in terms of loss of biodiversity, public amenity, and timber value if a particular region becomes infected.

Host Resistance
All ash trees are affected by *C. fraxinea*, but a minority appear to be resistant to the pathogen and may survive the epidemic. The resistance is likely to be from multiple genes and so is unlikely to be rapidly overcome by genetic changes in the pathogen. However, the pathogen has a much shorter generation time and can evolve faster than the ash tree. Without sufficient understanding of the host and pathogen genetics and ecology to develop effective tree breeding strategies, the outcome could be a long term epidemic.

References
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8. Personal Communication, Professor Chris Gilligan and Dr Matt Castle

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