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**The spread of Dutch Elm Disease in elm populations with resistance**

**Background**

Plant pathogens and their host species have been co-evolving for hundreds of millions of years (Han, 2018). It is a perpetual arms race, with pathogens continually overcoming host defenses. Although plants are lacking the adaptive immune system of vertebrate species, plants species have developed several innate immune responses to pathogens (Han, 2018). Genetically based disease resistance provides species with an intrinsic mechanism to respond and adapt to pathogens—several species of elm trees provide an example into this mechanism.

Millions of elm trees worldwide are threatened by Dutch Elm Disease (DED), which results in wilt, dieback, and death in elms (Brunet and Gurles, 2016). Depending on the age and susceptibility, the tree can die in as little as 2 months to a few years following infection (Grabowski, 2019). DED is caused by a group of fungal pathogens, *Ophiostoma ulmi,* and is vectored by bark beetles, which feed on the bark of elms (Brasier, 2001). DED can also spread from tree to tree through root graphs (Grabowski, 2019). While some species of elm are very susceptible to the disease, particularly those native to Europe and North America, other species, mostly those found in Asia, are completely resistant and can overcome infection of DED (Gibbs, 1978). Resistance phenotypes range from completely susceptible, to tolerant (survives with infection), to resistant (avoids infection). Since DED resistance is genetically based, resistance can spread between populations and species through hybridization and introgression. Introducing resistant genotypes in a population of susceptible trees should allow the resistance genotype to spread over time, allowing the population to survive DED infection. However, not much is known about the gene or genes governing resistance to DED. Additionally, due to the long generation times of trees (at least 15 years to reach reproductive age), resistance spread will be slow.

**Research goals and questions:**

Here, I aim to explore the dynamics of DED spread in populations of elms with varying levels of resistance using population simulation models and disease models. In particular, how do different starting levels of resistance within a population impact DED spread?

I will model resistance in two different ways, since the genetic basis of resistance is not well studied. First, I will model resistance in the form of major gene resistance (resistance is caused by a single gene), then as quantitative resistance (resistance is controlled by multiple genes acting together). In the first case, full resistance will be represented by a homozygous dominant genotype (AA), tolerance will be represented by a heterozygous genotype (Aa), and susceptibility will be represented by a homozygous recessive genotype (aa). Most elms are diploid, so I will use a diploid model. Here, resistance ranges from 0 to 1, with possible values of 0, 0.5 and 1, with 0 being fully susceptible, and 1 being fully resistant. In the second case, resistance will range on a continuous scale from 0 to 1. The continuous nature of resistance here is used as a proxy for multiple genes interacting to produce the resistance phenotype. During mating, seedlings will have a resistance value averaged from both parents.

Selective pressure will select against individuals with low resistance values. The rate of disease spread will be a variable that varies with time. As there are more susceptible individuals, the rate will be higher, and it will decrease as resistance spreads.

I aim to answer the following questions for both models of resistance (major gene resistance and quantitative resistance): **1) at what levels of resistance within a population will DED cease to spread 2) how long it will take for resistance to spread throughout a population of trees?**

**Anticipated findings**

I aim to explore how the population of elm trees changes over time. Before DED is introduced in the population, I would expect there to be some population growth, unless the population is at carrying capacity. Once DED is introduced, the population will start to decline. When resistant individuals are introduced, there will be some time lag before the population will either start to grow again or go extinct. I hypothesize that resistance will take longer to become fixed in a population when the starting proportion of resistant individuals within the population is smaller. I aim to find the optimal number of resistant individuals to introduce in a population.

I will also explore the implications of different timings of introducing resistant individuals in the population in relation to DED introduction. For example, what will happen if DED infects a population before resistance is fixed? Further, will resistance become fixed without selection pressure from DED? *Ophiostoma ulmi* is not found in Asia; however, the elms there are resistant to DED. Either *Ophiostoma* coevolved with elms in Asia in the past, or resistance can become fixed without selection pressure, perhaps through linkage with another trait associated with fitness. From this I will determine the approximate number of generations required to create an effectively resistant population (herd immunity) based on the model assumptions. I hypothesize that resistance will take longer to spread in the model of quantitative disease resistance compared to major gene resistance since there are more 'genes' contributing to resistance. Lastly, I hypothesize that the proportion of resistant and tolerant individuals in the population will need to be quite high (over 80%) to stop the spread of DED.

**Connections to course material**

- Models of selection for resistant genotypes (diploid models of selection)
- Population dynamics of the elm trees (limited by carrying capacity, impacted by disease states)
- Disease models, herd immunity

**References**

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