# Rose’s Thesis…

*Student number: 2417024*

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# Chapter 1

# Introduction

Whole-genome duplication (polyploidisation) occurs due to errors during cell division, such as non-disjuntion. The result is either single cells or–when that cell is a gamete–whole organisms that become polyploid, and so posses more than two complete sets of chromosomes. In many taxa this mutation is not well tolerated; for instance, in humans whole-genome duplication accounts for around 5% of miscarriages (**?**), and only one polyploid mammal has been recorded (**?**). However, the same is not so true of amphibians, fish, fungi and plants.

Image here about Whole-genome duplication / levels of ploidy?

Polyploidy is exceptionally well tolerated in plants, being a ubiquitous feature in the lineages of almost all angiosperms (**?**), and having occured recently (post genera formation) for 35% of all vascular plants (Wood et al. 2009). Furthermore, **?** also showed that whole-genome duplication could be linked with major innovation. Their phylogenetic analysis brought to light two ancient groups of duplication events (around 319 and 192 MYA). These events lead to the diversification of regulatory genes that were integral to seed development and later to genes that enabled flower development. Therefore, these duplications contributed to the appearance and success of all seed plants and angiosperms.

However, despite having been studied for over a century, the factors that drive the success of polyploid establishment in the face of reproductive disadvantages and high extinction rates are still unclear. What is it that allows this extreme mutation to persist and become fixated within plant communities?

The process of whole-genome duplication is thought to be fundamental in the diversification of plant species; having been found to coincide with around 15% of angiosperm speciation events, and 31% in ferns (Wood et al. 2009). However, the situation was later found to be less clear-cut; according to **?** polyploidisation is critical in increasing speciation rates of diploids, but new polyploid lines don't further speciate by that same mechanism, and so their speciation rates are smaller in comparison. Furthermore, their extinction rates are greater than those of diploids. This Liklihood-based analysis of vascualr plants provided the first quantitative support for the traditionally popular view that polyploidy most often leads to evolutionary dead ends.

## 1.1 Costs

Arrigo and Barker (2012) conclude that polyploids tend to become extinct at the establishment phase due to reproductive disadvantages such as triploid sterility, or limited mate-choice; the latter occuring via diploid pollen-swamping, or delayed flowering. By exploring each mechanism in more detail, we can start to get a feel for how they work, the conditions that will cause them to be important and, ultimately, whether or not they are realistic.

> Are they realistic? How do they work? Why/when are they important? consider the conditions.

### Diploid Pollen-Swamping

### Delayed Flowering

### Triploid Sterility

## 1.2 Benefits

Benefits associated with polyploidy may offset these costs: Polyploids are frequently linked with distinct traits such as “gigas effects”, which include increases in plant organs, reversal of selfing inhibition, enhanced capabilities for buffering of deleterious mutation (due to increased heterozygosity), and hybrid vigour (heterosis) (Woodhouse et al. 2009; Ramsey and Ramsey 2014). These traits are thought to overcome the reproductive disadvantages of polyploidy and instead make this mutation key to the invasive and adaptive potential of plants, ultimately shaping broader patterns of plant diversification.

### Gigas-effects

### Genetic buffering

### Hybrid Vigour

So what are the core mechanisms?

Or the most suspicious?

## 1.3 How do these mechanisms link?

Does limited mate-choice set the scene for the evolution of selfing vs out-crossing?

Delayed flowering (cost) is associated with gigas-effects of increased size (benefit). TRADE-OFF.

# References


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