**Introduction**

Long-term evolution of speciation events leads to the differentiation of ancestrally related species. These differences are easily observed through the phenotypic changes species undergo including morphology and behaviour. Recent advancements in science now allows us to study genotypic changes and further our understanding of phenotype-genotype relationship. Phenotypic evolution is assisted with genotypic evolution. Phenotype of species are influenced by genotype and environment. Collective expression of certain genes through pathways assist in the morphology and behaviour we observe in species. Hereditary genome alterations through random changes molecular mechanisms coupled with evolutionary effects including selection and drift allow species to evolve over time (Chandrasekan). These molecular changes induced by mutation and recombination include duplication, lateral gene transfer, gene fusion and fission which lead to the variation of descending species (Chandrasekan; Ohno 1970; Genome 2nd edition). Over time, drift and selection acts on these polymorphisms and those best suited passes their variant genome and as a result phenotype, to future generations.

Orr showed that there is variation with respect to genetic differences or gene influence on phenotype with regards to reproductively isolated species. The effects of adaptive and non-adaptive processes vary among species where there is no common set of genes involved, nor is the effects and interactions of the genes similar for species (Orr 2001). Although generalizations cannot be made of genetic function and interactions, what can be considered is the pattern at which these genetic processes develop over time. Genetic network simulations can be used to understand these patterns of evolution and the effect on phenotype- genotype relationships. We know that over time, genetic interaction within the network develop and lead to stability, robustness and redundancy (Omholt et al. 2000; Lynch 2007). This is influenced by both adaptive and non-adaptive mechanisms. Robustness can evolve from the effects of epistasis, additivity and dominance, all of which are connected (Omholt et al. 2000). Lynch highlighted the importance of non-adaptive processes as well in shaping genetic networks. His study showed that networks can still evolve its architecture and become redundant even without the influence of natural selection (Lynch 2007).

Species variation and evolution is non-linear, descending with modification and constant splitting from lineages of a common ancestor. This continuous process over long temporal periods results in the accumulation of optimal genetic adaptations that results in a robust network structure. Even once this structure is reached, how does the structure resist change and maintain its network despite perturbations and evolutionary processes. What I am concerned with in this study, are the patterns of change that a genetic network undergoes with adaptive and non-adaptive evolution. Especially with gene flow through migration in addition to selection, drift, mutation, recombination effects. The concern is on how a network development changes when these evolutionary forces come into play and seeing the evolving genetic interactions, and if the new structure is robust to perturbations. We know that over time, the variance in dominance decreases and dominance plateaus to a specific value, with small deviations around that value. Using quantitative trait loci (QTL) we are able to numerically interpret and visualise the patterns of change. Previous research looked at the effects of gene flow, selection and mutation at generating local adaptation at the phenotypic level, showing how maintenance of alleles and linkage is important in adaptation (Yeaman and Whitlock 2011). It was shown that with random perturbations and aid of genetic modifiers, there are bounds for which selection for canalization can act on, leading to evolution of robustness. They also showed that under migration selection balance, selection for robustness increases with the migration rates (Proulx et al. 2004). The purpose of the research will be looking at the change in genetic architecture dynamics and structure with the interactions of the systems that maintains this robustness. Furthermore, ﻿as the network evolves, it has been shown that there exists a threshold which is actively regulating these homeostatic genes (Gjusvland et al. 2007). As selection for robustness occurs within the local population, it can give insight into the change in architecture and statistically significant interaction (Gjusvland et al. 2006).

Using a multi-locus system, we can simulate the effects over many generations and see how the output of the network changes, specifically looking at allelic interactions and tracking the dominance and variance over time. Variance decreases as a genetic network becomes robust, making it resistant to perturbations. Fitness is represented as offspring contribution and the quantitative values generated from the alleles signify trait values. Phenotypic values are calculated from these trait values and used as probabilities for fitness.

**Methods**

The multi-locus model used is a di-allelic interlocus model from the research of Omholt where all genes represent the regulatory region, contributing to the protein production, which contributes to phenotype. Similar to the work of Omholt et al., this model structure evolves dominance through epistatic interactions and regulatory effects. Using a system of equilibrium solutions and ordinary differential equations (ODE), protein concentrations are measured over time. Here I consider genes as quantitative factors of protein function, and trait value representing total protein concentrations. To limit the trait value from exceeding to infinity, the first two locus X1 and X2 acts to through a negatively feedback loop which then positively act on X3, which is the trait value. Thus increasing expression of X2 decreases X1 expression, negatively autoregulating the system and maintaining a specified value. [alpha] is the production rate and [gamma] is the degradation rate. R(1j) is the regulatory Hill Function which represents a Michaelis-Menten mechanism, where S([Theta],[P]) = Xp/Xp + 0P. Depending whether the locus is positively or negatively regulated, it leads to the following equations:

﻿Rj(y) ⫽ 1 ⫺ S(y, ?j , pj), j ⫽ 1, 2, (2) if negative

﻿Rj(y) ⫽ S(y, ?j, pj), j ⫽ 1, 2, if positive

[mu] is the ratio of alpha and gamma per locus and using the equilibrium solutions, total protein concentration is calculated by the following equations:

﻿y1 ⫽?11(1 ⫺ S(y2, ?211, p211)) ⫹?12(1 ⫺ S(y2, ?212, p212))

Y2

Y3

The exact same model is used to represent the migratory population.

For the effect of genetic drift and to account for the large deviations of values, a Cauchy distribution is then used with the y3 values to generate fitness probabilities per generation. These values determine parental contribution to offspring for the next generation.

**Results**

**Conclusion**

**Looking forward**

In just a few centuries, societal development has caused many anthropogenic changes, many of which will have harmful long-term effects to many species. This coupled with geological changes, will lead to future shifts in the distribution of land as well as alteration of habitat states. One effect of this change can be migration. Whether it is forced migration or new route opportunities, the distribution and movement of species will also change in generations to come. These migrations can cause the introduction of novel genes into local habitats, implicating future evolutionary effects in these local habitats. Evolution of both the local species and migratory species. The presence of these novel suboptimal genes, along with the interaction of species in the environment will lead to evolutionary change. After many generations, this change will be seen in a phenotypic level as well as genotypic.

**Assumptions:**

* Population size is always constant even with migration
* Both populations undergo stabilising selection toward specified value
* All individuals begin with same allele values
* Migrant population is a related species but y\_2 is negatively autoregulated by y\_1 compared to main population where it is vice versa. Both y\_3 however represent trait values
* All locus are quantitative trait values
  + Each locus can mutate with a defined probability and mutation can be beneficial, neutral or deleterious all equally
  + Mutation is constant
* Fitness is represented as offspring contribution and set the probabilities each individual is the parent of the next generation offspring
  + No limit as to how many times an individual can be a parent
* Migration probability is constant
* Recombination is equal chance at any locus