**Levels of Selection (PSR)**

Martijn A. Schenkel, Kelley Leung, Leo W. Beukeboom



**Outline**

**Activities**

1) Listen to lecture and read background on *Nasonia vitripennis* and Levels of Selection (Paternal Sex Ratio).

2) Answer questions in groups about sexual selection theory, mathematical models, and data simulations.

**Materials**

For each group of 3-4 students:

1-2 work laptops with Microsoft Word and R and R Studio installed

**Objectives**

-Learn basic biology of the haplodiploid *Nasonia vitripennis* system

-Learn how levels of selection under the influence of the Paternal Sex Ratio (PSR) chromosome impacts population dynamics

-Create formulas to represent models of population dynamics

-Learn how to use R/R studio to run and visually represent models and data simulations

**Goal**

To increase students’ knowledge of alternative modes of sexual selection and how to model data

**Feedback**

Instructor will provide feedback on: interpretation of information to answer questions on theory, design formulas, and draw conclusions from running models.

**Levels of Selection**

Start by reading Dalla Benetta et al. 2020 for background understanding, and the Basic Introduction of R to learn how to run analyses needed for this practical.

Paternal Sex Ratio (PSR) is a B chromosome that is found in males of the parasitoid wasp *Nasonia vitripennis*. This species has a haplodiploid sex determination mechanism, in which haploid unfertilized eggs develop into males but diploid fertilized eggs develop into females. PSR is transmitted paternally through sperm. In the developing embryo, it destroys the other chromosomes inherited from the father, resulting in a haploid embryo that develops into a son that carries PSR. In short, PSR converts a diploid female embryo into a haploid male embryo. A mother may maximize reproductive success by altering the sex ratio depending on the size of the population (see lecture slides on Hamiltonian sex ratios). When the population size is sufficiently large, the optimal offspring sex ratio for a mother approaches 50% so that she produces equal numbers of sons and daughters. For now, assume that the entire *N. vitripennis* population consist of one large population with fully random mating (panmixis).

1. Explain how the conversion of diploid (female) eggs into haploid (male) eggs affects the spread of PSR.
2. By converting females into males, the effect of PSR is that there are relatively more males and relatively fewer females in the population (i.e the sex ratio becomes more male-biased). Would this affect the spread of PSR? Explain why or why not.

In *N. vitripennis*, a female may control the sex ratio of her offspring by selectively fertilizing a certain proportion of the eggs that she lays. We can define this proportion as the fertilization rate *x* of a female. Similarly, we can define the proportion of males carrying PSR as *P*.

1. Determine how the frequency of (in males) in the next generation depends on its current frequency and the fertilization rate . To do so, consider that there are two types of sons, i.e. PSR sons and normal sons. PSR sons are derived from fertilized eggs of a female that has mated with a PSR male, whereas normal sons are derived from unfertilized eggs (note that for this it does not matter what mating status the female has).
2. PSR is at its equilibrium frequency when the frequency of PSR in the next generation is identical to its frequency in the current generation, i.e. . We can define this as the equilibrium frequency . Using the equation from the answer to (3), determine how depends on the fertilization rate by solving . Calculate for a variety of values for between 0 and 1 and make a simple graph to illustrate these results. When can PSR spread in the population? Explain why, based on your results so far, PSR will not spread in a natural population.

**PSR in a demic population**

Obviously, the existence of PSR in *N. vitripennis* is not in line with the predictions that you have made so far. In nature, *N. vitripennis* is a parasitoid wasp that lays its eggs inside developing fly pupae. These pupae are found in bird nests and on carcasses and therefore have a patchy distribution. Eggs from *N. vitripennis* develop within the host and ultimately emerge from the pupae as adults. Males and females mate shortly after emergence, after which the mated females disperse to other (“fresh”) patches (or “demes”) for oviposition and males die. Effectively, *N. vitripennis* does not represent one large population with random mating, but a highly structured one (called “metapopulation”) where mating occurs only between individuals emerging within the same deme (i.e. the local subpopulation). Taking into account the population structure of *N. vitripennis* may help us explain why PSR is able to persist. To this end, we will consider the effects of PSR on a smaller scale by looking at its dynamics on the deme level. Relative to the population, a deme contains a much smaller number of individuals. Local mate competition theory predicts that under these conditions, females are selected to produce broods with more daughters than sons (we will discuss this more in-depth a little further on).

1. Explain how having a female-biased sex ratio among the offspring affects the spread of PSR. Compare this to your findings in the previous assignment.

We will now attempt to determine how and when the existence of discrete demes within *N. vitripennis* enables PSR to spread. The difference in effective population size between the different demes and the overall population is essential. To illustrate this, we will first consider a deme consisting of a single female foundress, after which we will explore situations in which demes contain multiple female foundresses. When considering a deme that is founded by a single female, we only have to concern ourselves with two situations: either the female has mated with a PSR male, or she has mated with a regular male.

1. Describe what types of offspring each of these females would produce. What happens to the deme? How does this influence gene flow of PSR? In other words, what happens on a population level with regard to PSR? Note that you do not need to use a formula, answer in descriptive terms.

Now, consider a deme consisting of multiple females. In this scenario, a few additional things need to be considered.

1. Depending on the number of foundresses within a single deme, the optimal fertilization rate (from the perspective of a foundress) changes. According to Hamilton’s local mate competition theory, this fertilization rate is given by:

in which denotes the number of foundresses. Plot a curve for different values of , then reflect on how the dynamics of PSR might be affected by foundress number.

With increasing number of females, the Hamiltonian fertilization rate drops, and it asymptotically approaches 50% as becomes larger. For PSR to spread, the fertilization rate must be higher than 50%. Therefore, smaller demes will favor the spread of PSR as females produce more fertilized eggs here.

1. A female mating with a PSR male will only produce male offspring. Assuming that mating is random within a single deme, we can determine (given that we know the number of females and proportion of males carrying PSR) how likely it is that all females within a deme mate with a PSR male, and therefore that only males are produced within the deme. How does this depend on the frequency of PSR (i.e. the proportion of males that carry PSR: ) in the deme, and how does this depend on the number of females ()?
2. Consider first a deme without PSR males, then one where all males carry PSR. How do they differ in the proportion of females produced? What happens at intermediate frequencies of PSR?

**Developing a mathematical model of PSR dynamics**

It is hopefully clear that the spread of PSR is affected by a variety of factors. Given that mothers are the ones that ‘spread’ PSR by producing PSR sons, the frequency of PSR in the next generation () is given by the proportion of females that have mated with a male in the current generation.

In this formula, represents the total number of foundresses in a deme; is the number of foundresses that have mated with a PSR male; is the current frequency of PSR; and is the fertilization rate. We can split this formula up into the following parts:

This is the probability of sampling PSR-mated females out of females in a deme.

This is the proportion of PSR males () amongst all males ().

This is the proportion of females produced.

This is the average proportion of females produced in a deme (for a whole population).

is used to iterate over all possible values of , i.e. to consider every possible number of PSR-mated females in a deme between 1 and . Note that what this formula describes is the frequency of PSR in sons. This depends on the probability that the mother has mated with a PSR male, which in turn depends on the grandmother’s status. , , and therefore refer to the conditions in the preceding generation. The formula describes the composition of this generation, depending on which the frequency of PSR in the next generation.

1. Explain for each component [(1), (2), (3), and (4)] whether the rate of PSR spread increases or decreases as this value gets bigger. To do so, assume that only one particular component changes at a given time.

As you hopefully managed to figure out, increased values for some of these components would result in an increased rate at which PSR spreads, whereas others reduce it. So far, we assumed that the components do not affect one another. However, this is not the case as each component shares one or more parameters with the other components, so that when you change the value of a parameter it affects multiple components – and generally does so in different ways. That means that there is no easy way to describe the effect of each parameter on the overall outcome, in this case whether or not PSR will increase or decrease in value. Furthermore, it is not easy to figure out when , i.e. when the frequency of PSR reaches an equilibrium where its frequency in the next generation is equal to its frequency in the current generation. Instead, it might be useful to develop a model (or more accurately, to run iterations of this recursion equation for different values of , , , and ).

Open the R script ”5\_levels\_of\_selection.R” in RStudio. Under “Parameter settings”, you’ll find definitions for variables P, X, and N. In line with our terminology here N represents the different values of deme size , X represents the different fertilization rates , and P represents the different initial frequencies of PSR. We will first focus on N and X to investigate how deme size and fertilization rate influence the spread of PSR. We assume an initial PSR frequency of 0.1.

1. Run the first parts of the script up to and including “Spread of PSR under different fertilization rates”. Once it is done, a PDF file called “different\_fertilization\_rates.pdf” should have been generated in the same folder as where you have the script. The small graphs on the left depict the frequency of PSR over time for different values of (differently-colored lines) and for different values of . The graph of the right shows the frequency of PSR after 200 generations. Describe how PSR is affected by and based on these results.

Hamiltonian sex ratios predict that the value of *x* (fertilization rate) should change depending on (the number of foundresses) and that the evolutionary stable strategy (ESS) is given by:

1. Now, run the section “PSR spread under Hamiltonian sex ratios”, after which a PDF “Hamiltonian\_fertilization\_rates.pdf” should have been generated. The graphs on the left side again shows the frequency of PSR over time for different values of . The right graph shows the PSR frequency after 200 generations. How does the equilibrium frequency of PSR depend on the deme size *N* and the initial frequency ? When is the frequency of PSR maximized, and at what value?

**Synthesis**

Based on your analysis of PSR and its dynamics in the previous assignments, you will have seen that PSR is subject to selection at different levels.

1. Are there selection pressures that affect PSR at the gene, individual and population level? And, are there selection pressures at the gene, individual, and population level that effect PSR? If so, describe them based on the assignments above. At which levels is PSR favored, and at which levels is it not?
2. What mechanistic explanations could there be for selective effects that might influence PSR on the gene level? On the individual level?