**Levels of Selection (PSR)**

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**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. *(Note to teachers: answers are in italics)*

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.

*Only males transmit PSR to their offspring via sperm, whereas PSR is not transmitted by females. By converting females into males, PSR increases the proportion of individuals capable of transferring PSR, thereby benefitting its spread.*

1. 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.

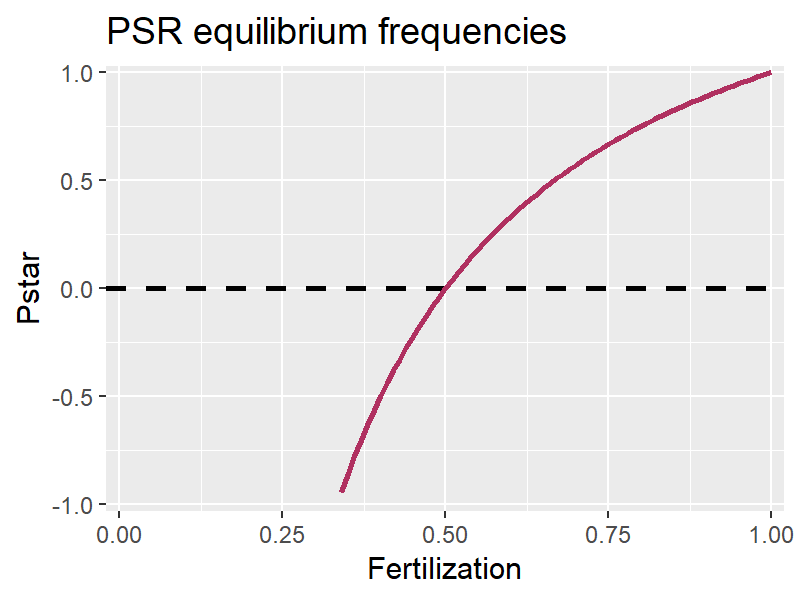
*By increasing the proportion of males, the sex ratio becomes distorted. Sex ratio selection tends to favor the rarer sex, and if there are relatively more males this means that on average they will experience lower fitness because there are fewer females to mate with. This negatively influences the spread of PSR. As an example, we can consider an extreme case where PSR is near fixation; in this case, (almost) all offspring will be males. The lack of females then can cause populations to collapse.*

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).

*PSR males are produced from PSR-mated females; their frequency is equal to the frequency of PSR among males, which we denote P. PSR sons develop from fertilized eggs, and therefore are produced by these females at a rate proportional to their fertilization rate x. The frequency of non-PSR males is proportional to all females, multiplied by , or the proportion of eggs that is not fertilized. Together these form the ‘total’ pool of males. Altogether, we can state that PSR sons / (PSR sons + non-PSR sons) denotes the frequency of PSR among sons. Given the above, the frequency of PSR sons is given by Px, and that of normal males by . The frequency of PSR among the next generation is then:*

1. 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.

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*when the fertilization rate . This means that PSR only spreads when the population would normally consist of more females than males. Since sex ratio selection favors 50:50 ratios and hence a fertilization rate of 0.5, PSR should not normally spread.*

**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.

*When females produce more female-biased broods, this means that they are fertilizing more eggs. Since PSR spreads only to fertilized eggs, this increases the spread of PSR. In a large population, fertilization needs to be for PSR to be able to spread. In this case, this requirement would be met, meaning PSR could indeed spread if the (local) population is small.*

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.

*If the female is mated with a PSR male, she will produce only sons; unfertilized eggs develop into normal non-PSR males, whereas fertilized eggs will develop into PSR males. Females are absent from the deme, so no reproduction can take place. In addition, because males do not disperse, the absence of females means that there is no gene flow from this deme. This means that PSR cannot spread from this deme.*

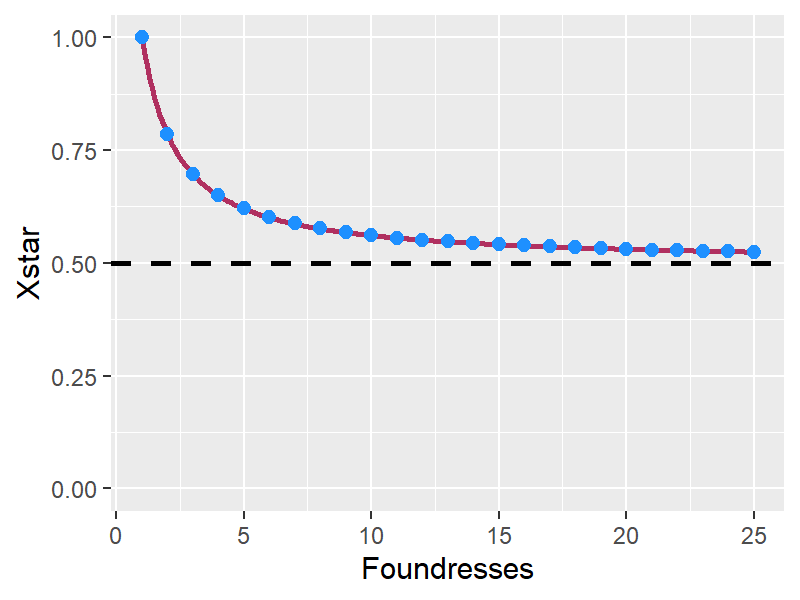
*If a female is mated with a normal male, she will produce normal sons and normal daughters (though note that Hamiltonian SR predicts no sons, so she will likely produce an small proportion of sons). Sons and daughters mate, and daughters will disperse from the deme. Because there are no PSR males in their native deme, these females cannot help spread PSR in the population.*

*Altogether, a deme size of 1 never contributes to the spread of PSR.*

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.

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*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 ()?

*The probability that a single female mates with a PSR male is proportional to the frequency of PSR; the probability that all females mate with a PSR male is then .*

*Related to (8), we can consider how females contribute to spreading PSR in a population, even though PSR causes individuals to develop into males. Daughters from females that mated with a non-PSR male may mate with any other male in their deme, including PSR-carrying sons from females that did mate with a PSR male. These daughters may then disperse to new demes, where they will give birth to non-PSR sons as well as PSR sons.*

1. 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?

*If no males carry PSR, the proportion of daughters produced follows the Hamiltonian sex ratio (). If all males carry PSR, then only sons are produced in that deme. At intermediate frequencies of PSR, some females will still be produced by mothers that have not mated with a PSR male, whereas PSR-mated females will produce only sons.*

**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.

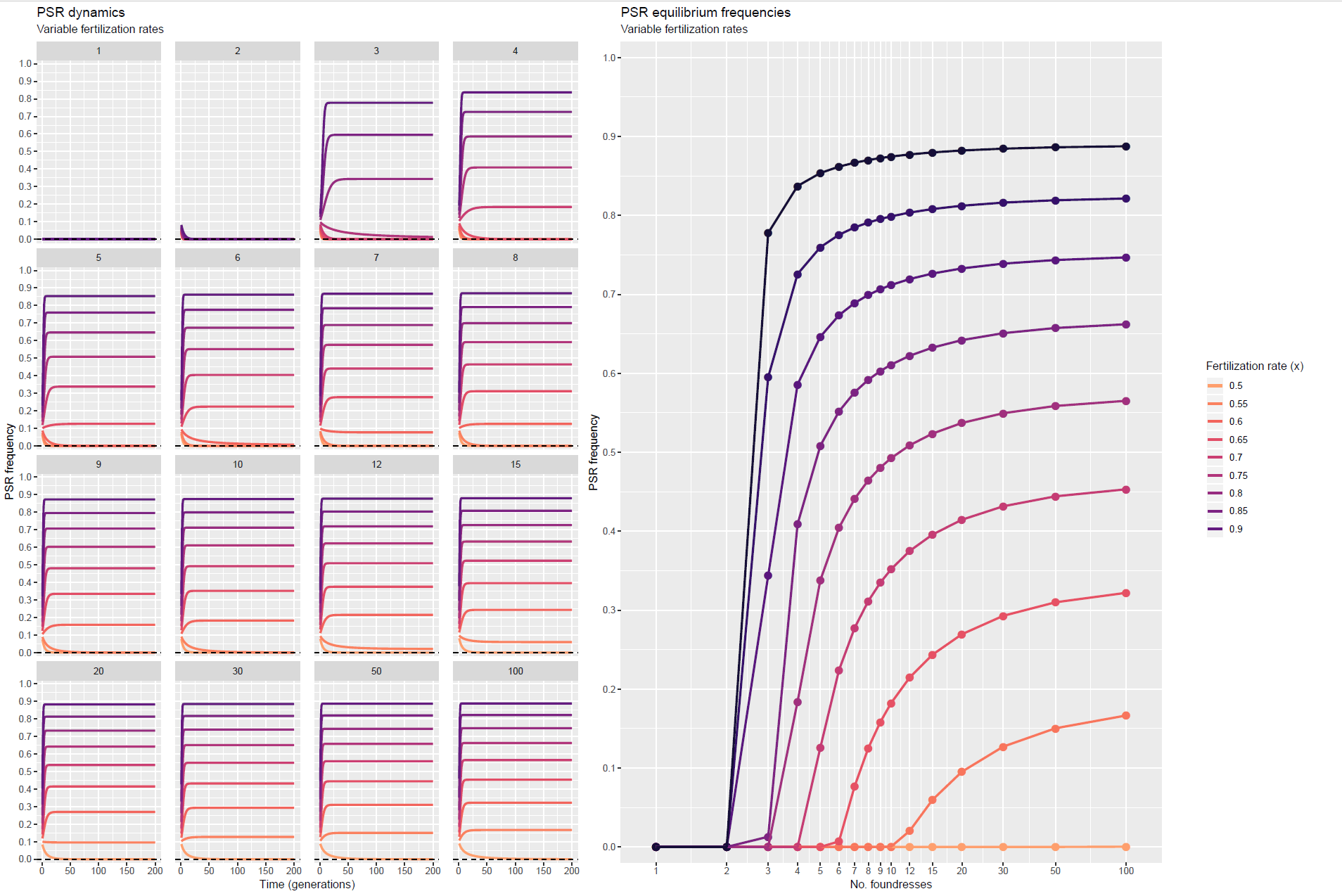
1. Explain for each component [(1), (2), (3), and (4)] whether the rate of PSR spread increases or decreases as this value gets bigger. To do so, assume that only one particular component changes at a given time.
2. *If this increases, it generally increases the spread of PSR as more PSR males will be generated. However, it is maximized when . Therefore, if this is too high, it will limit the rate at which PSR spreads.*
3. *If this increases, PSR spread increases as well, as it means females are more likely to mate with a PSR male rather than a normal male.*
4. *Increasing its value increases PSR spread, as more females will be available to mate with PSR males.*
5. *If this increases, it will decrease the spread of PSR, as it reduces the proportion of females that have mated with PSR males.*

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.

*Increasing is generally associated with increased equilibrium frequencies for PSR, but only when For PSR to spread, must be at least 3. PSR frequency is also positively affected by increasing ; when , PSR can spread. The equilibrium frequency for PSR increases asymptotically with increasing , with different values of being associated with different asymptotes.*



*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?

*The equilibrium value does not depend on the initial PSR frequency assuming that . The equilibrium frequency of PSR depends only on the deme size ; it increases from 0 at to ~0.03 at , after which it decreases again with increasing .*

**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?

*Gene level: PSR is selected to increase its transmission rates at the cost of other genetic elements/at the cost of the individual.*

*Individual: No selective effects considered.*

*Population: PSR has a destabilizing effect on sex ratios, and its spread is inhibited when its frequency becomes too high (e.g. by local extinction of demes).*

1. What mechanistic explanations could there be for selective effects that might influence PSR on the gene level? On the individual level?

*On the gene level, PSR might be selected against when a gene evolves that nullifies its selfish effects. For example, genes might evolve that prevent PSR from destroying the paternal genome in the embryo.*

*On the individual level, PSR might negatively affect sperm performance, or PSR males might be more prone to developmental issues as a result of mitotic anomalies.*

