Sperm modifiers of female drive.

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Abstract:

Introduction

Since all alleles within an individual rely on individual survival and reproduction for evolutionary success, most of life in a diploid eukaryotic genome is harmonious. However, this harmony is incomplete, as in numerous cases an allele can benefit (in the short or long term) from taking advantage of its host individual. A clear opportunity for this conflict arises during gametogenesis where alternative alleles are in direct competition for representation in the next generation. Female meiosis is particularly unfair, only one of the four products of meiosis becomes an egg, while the other threes are disgarded into polar bodies. Alleles that can segregate into the egg more than half the time in heterozygotes, by exploiting asymmetries, can potentially increase in frequency in the population (i.e. experiencing true meiotic drive). If such alleles have fitness consequences they are a source of genetic conflict, and can become balanced in the population if their host in homozygotes out weighs their ability to spread to spread through heterozygotes. Such balanced drive systems can cause the subsequent evolution of linked enhancers of drive and of supressors of drive throughout the genome. Indeed, the known polymorphic female meiotic drive systems (OM, maize knobs, IN, mimulus, others?) habor a great diversity of enhancers and supressors.

Another unusual aspect of animal female meiosis is the fact that it is paused at XXX. In fact in many species of animal the final stages of female meiosis are only completed upon fertlization of the egg by sperm.There's considerable variation in which stage of meiosis requires fertilization. This raises the possibility that an allele in sperm could influence the outcome of meiosis in its favor. For example imagine an allele that when a sperm bearing this allele were to encounter an egg, whose meiotic product was currently heterozygote for another copy of itself, the sperm allele biased meiosis in favor of the other copy of itself. At first sight, it seems as although female meiosis is primed to be exploited by selfish sperm systems.

Why then is the requirement of fertilization to complete female meiosis so unbiquitous?

It is certainly not the case that mechanistically animals are incapable of evolving past this requirement. A number of animal clades (should we try and lower bound how many transitions?) have evolved to allow the completion of female meiosis upon ovulation.

This is not just idle speculation, as there is also direct evidence that sperm components and indeed different sperm genotypes can affect female meiosis.

In C elegans supressing XXX leads to premature deployment of the aster (a vitial component of mitotic mechinary) provided by the sperm, distrupting MII meiotic segregation in the egg, leading to a triploid zygote. This demonstrates that physical components derived from the sperm are are capable of affecting the outcome of female meiosis. Furthermore, there is also more direct genetic evidence that the allele of a sperm can influence the outcome of female meiotic drive in their favor. There are two female meiotic drive systems in mouse, In and Om, both of which are thought to operate through drive at MII. In these systems the outcome of female meiosis seems to depend on the genotype of the fertilizing sperm.

In this article we explore through simple models the consequences of alleles that influence the outcome of female meiosis. We use these models to argue that it is actually surprisingly hard for the influence of sperm on the outcome of meiosis to drive sustained conflict. In fact we find that sperm and egg genomes' interests are often aligned as they are both invested in the fate of the zygote they will form. This suggests that females are unlikely to evolve to prevent the influence of sperm on meiosis, and indeed feature of meiosis may evolve that facilitate the interaction of sperm with female meiosis.

Results

Rough order of rest of text

Lay out Model

We imagine that drive acts in such a way that an allele promote itself into a fraction of d eggs ($d > 1/2$, when it is present in a heterozygote, forcing the other allele to be present in $1 - d$ eggs. In mammals, fertilization takes place at MII, so we imagine this drive must be taking place at MII in order for a sperm to have any influence. For drive to take place at MII there has to be an uneven number of crossovers between the centromere and the drive locus, such that realistically d is bounded to be $< XX$. In other systems where fertilization occurs during MI, sperm could influence either drive at MI or MII, and drivers at MI can have a $d = 1$ if they occur in tight linkage with the centromere.

Cartoon figure of sperms meeting eggs with different genotypes?

Cost of alleles

We'll setup a model where the sperm allele affects outcome of female meiosis. This may not be biologically realistic as there is very little sperm expression. More realistically the male genotype that produced the sperm may be more relevant, as males could place products in their sperm that influenced the outcome of female meoisis. However, in practice models that allow the influence of fertilizing male genotype on female meiosis seem to behave very similiarly to those based on the allele of the fertilizing sperm. For simplicity we'll focus on models based on the allele of the sperm, as it's bloodly complicated enough already.

A) Invasion by single self promoting allele

show that such alleles:

1. can't be balanced,

2. and homozygous problems are tested out at low freq.

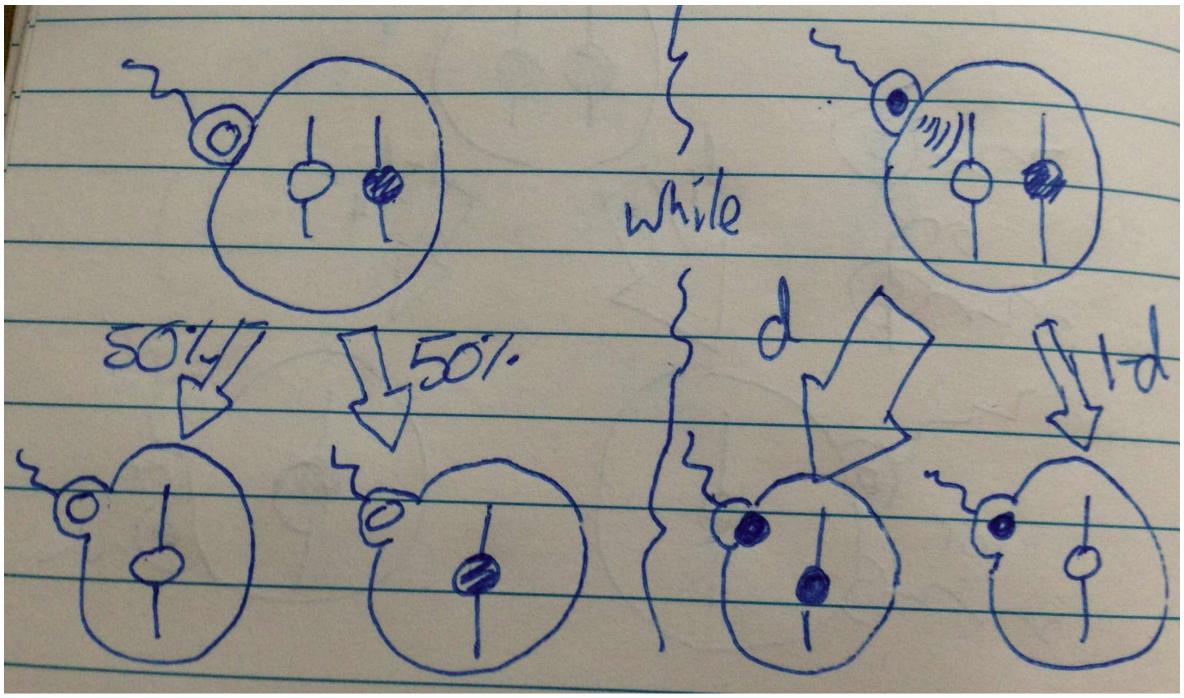

Figure 1: transmission probabilities for alleles through female meiosis depend on sperm genotype. 2 allele models

3. Any heterozygous problems, leads to a bistable allele

4. If these alleles take off they speed through to fixation

5. If allele has any drive ability in absence of sperm effect that is what allows it to enter the population and sperm effect isn't a further cause of conflict. What if anything do we mean by this?

Conclusion, such alleles are unlikely to cause evolution of female supressors, they test hemselves in a homozygous state when they enter the population, and sweep quickly (all the way to fixation) if they enter the pop at all.

B) A more biologically realistic selfish sperm system

Three allele systems. See Figure 1

Perhaps these self promoting alleles are biologically unrealistic as new alleles needs both ability to drive in female meiosis and to influence that drive in sperm. Perhaps more realistic to think that a female drive system evolves, and then a sperm modifier appears. This will only occur if original system is trapped at drive-selection balance. So driver must have homozygous cost.

1. Setup a drive-selection balanced polymorphism in std. drive model. Do this by imagining the sperm-influence allele arising on the background of the driver, so the allele has drive capabilities, and can have had time to evolve new biology. Evolving on the new background means that the allele suffers the fitness consequences of the driver.

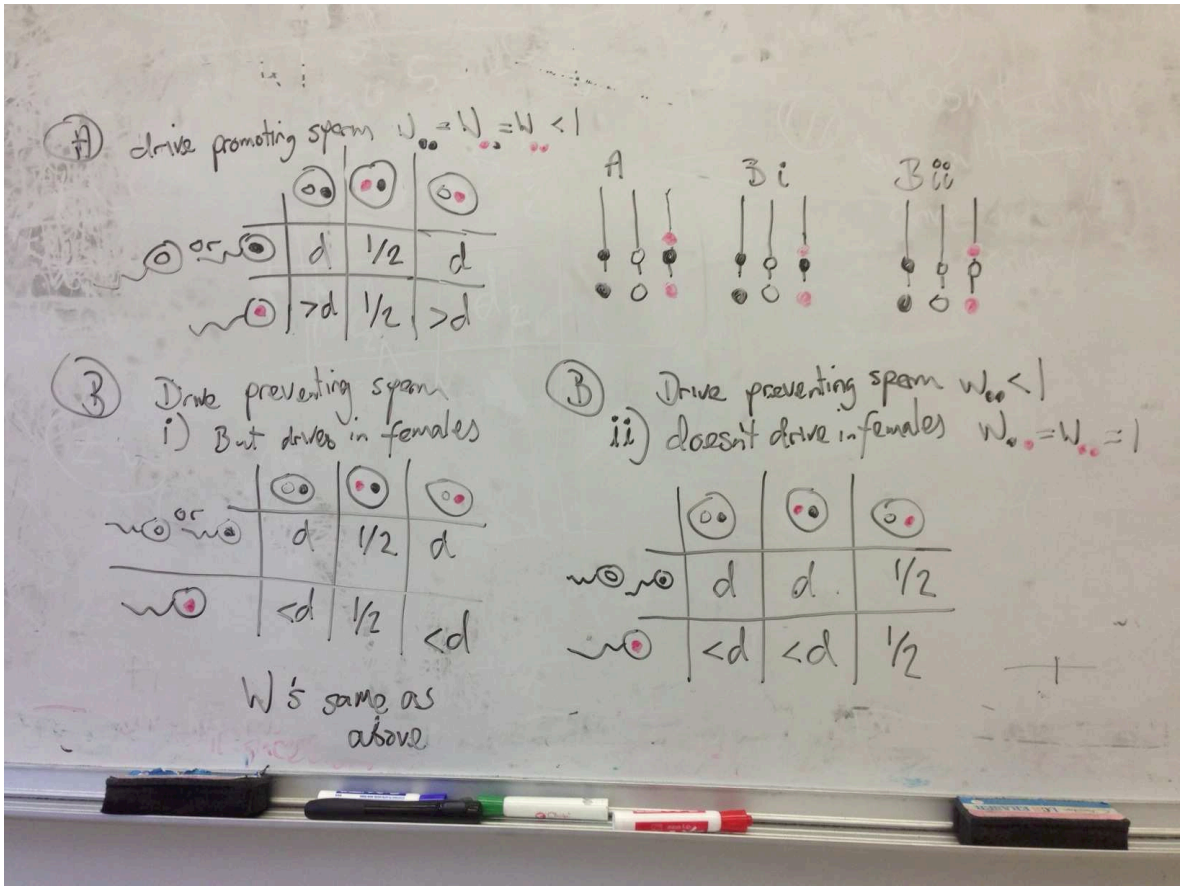

Figure 2: transmission probabilities righthand allele through female meiosis

See A in Figure 1

2. Sperm-based enhancers of drive can't invade (can they in some situations?).

3. Intuition is that the driver has already driven to a frequency where it is held in check by its cost in homozygotes. The sperm allele thus can't really help as it creates zygotes which suffer the homozygous fitness consequences.

0.1 So what can evolve?

So what can happen?

1. Alleles that arise in linkage with drive systems, which when in sperm switch off drive, can spread. They benefit from drive, but avoid some of the consequences ((Bi) in Figure 1). Overall as a side product they are benefiting all in pop.

2. Presumably alleles that actually switch the allele that drives may do even better? As they'd end up in hets. Although they'd not drive, so hard to say.

3. Alleles that cause sperm to switch off drive that arise on other background or unlinked to the drive system are selected, and spread as fast as female supressors of drive ((Bii) in Figure 1)..

4. Alleles that in females facilitate the action of sperm supressors of drive (or vis versa) can spread. Haven't actually checked this.

Conclusions.

Discussion of general conclusion that females have little reason to evolve supression mechanisms to prevent sperm influence on meiosis. General logic that sperm genome has to live in a zygote with consequences of its effect on female meiosis, so it can not generate too dire a consequence.

This logic may not hold for sex chromosomes. In ZW systems Male modification of recombination rates POssibility that this could happen in plants if pollen emit signals to "egg" Discussion of OM and IN.