Masculinizing B chromosomes in gynogenetic carp: ultimate selfish genetic elements or facilitators of kin selection?

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In gynogenesis, females mate with males, but produce parthenogenetic. Here I consider the evolution of an exceptional form of gynogenesis, in hexaploid gibel carp. Whereas most gynogenetic species are all-female, relying on mating with heterospecific males, hexaploid gibel carp species include males, some of which carry a masculinizing B chromosome. In addition, in contrast to strict gynogenesis, B-carrying males transmit the B and small amounts of autosomal DNA to offspring. I consider three hypotheses: (i) the B chromosome is selfish; (ii) the B chromosome provides an inclusive fitness advantage by increasing reproductive opportunities for clonal relatives; and (iii) the B chromosome provides a direct fitness benefit, due to increased mating opportunities for males in female-biased populations and/or increased fitness of recombinant offspring. I note that the selfish B hypothesis (i) implies an extreme level of selfish behavior, as B action (nearly) completely blocks transmission of autosomal genes to offspring, comparable to Werren and Stouthamer's "ultimate selfish genetic element," the wasp B chromosome PSR. I suggest that the selfish B hypothesis (i) is likely and suggest a stepwise pathway for the origins of a selfish masculinizing B. I point out that the inclusive benefit hypothesis (ii) depends on high levels of mating between clonal relatives, and thus awaits more population genetic data. I provide rough calculations suggesting that paternal autosomal DNA transmission is insufficient to support the direct fitness hypothesis (iii). In total, I argue that selfish and/or kin selection most likely explain masculinizing B in gibel carp.

Introduction

Selfish genetic elements are elements – transposable elements, genes, chromosomes, etc. – that increase their own transmission to the next generation at a cost to the overall reproductive fitness of the organism in which they reside (Werren 2011; Burt and Trivers 2006). Thus, genes on the X chromosome that produce a toxin that kills Y-bearing sperm can benefit from decreased competition while leading to an overall reduction in sperm number and thus total organismal fitness (Lindholm et al. 2016). Similarly, plant mitochondria that thwart pollen production may increase their transmission through increased female reproductive function, but decrease the overall number of offspring produced by that plant (Chase 2007).

Selfishness of genetic elements arguably reaches its apex in a masculinizing wasp chromosome. Some twenty years ago, Warren and Stouthamer identified the *Nasonia vitripennis* PSR chromosome (for "paternal sex ratio") as the ultimate selfish genetic element (2003). PSR is a male-determining supernumerary (B) chromosome in the haplodiploid wasp *Nasonia vitripennis* and relatives (left of Figure 1). After fertilization by a PSR-bearing sperm, PSR chromosomes block decondensation of the other paternal chromosomes, leading to their eventual loss, thus converting a diploid zygote into a haploid, and thus male, individual (Werren 1991; Werren and Stouthamer 2003). Thus, the action of PSR chromosomes in a male's sperm blocks transmission to the next generation of all of the haploidized male's autosomal genes, with only PSR itself being transmitted.

A superficially very different system involving a masculinizing B chromosome has been developed by a series of papers by Gui and collaborators and others (Figure 1, right). Hexaploid gibel carp reproduce by gynogenesis, a form of reproduction in which females mate with males, but produce offspring that are clonal copies of the mother (Schlupp 2005; Komen and Thorgaard 2007), for instance due to chromosomes inherited through the sperm failing to decondense (Zhao et al. 2011). Gynogenetic reproduction in gibel carp seems to be readily induced by polyploidy, suggesting a potentially complex history of the gynogenetic individuals collectively (Lu et al. 2022; Shao et al. 2018). Unlike many gynogenetic species, which rely on matings with heterospecific males, gynogenetic gibel carp females typically mate with conspecific males, which are typically present at low frequencies (1-26% across populations; Jiang et al. 2013; Li et al. 2018). As with PSR, in some individuals maleness is determined by the presence of a B chromosome (albeit by direct masculinization rather than haploidization), which, unlike other paternal chromosomes, are frequently able to make their way from the sperm into the embryonic genome (Ding et al. 2021; Li et al. 2016, 2017, 2018; Zhao et al. 2021). Genomic characterization of the B chromosome suggests that it harbors a combination of male-specific factors, expanded DNA satellite repeats, and other autosomal-derived sequences (Ding et al. 2021).

Great strides have been made in understanding the organismal, cellular and molecular biology, and the potential evolutionary consequences of this system. The evolutionary origins and maintenance of the system has received less attention. In this manuscript, I discuss in turn three possible hypotheses for the origin and maintenance of the masculinizing B chromosome: (i) the B chromosome decreases individual fitness, being a selfish element that increases its own transmission while reducing organismal fitness; (ii) the B chromosome increases inclusive fitness by increasing clonal relatives' mating opportunities and thus fitness; and (iii) the B chromosome increases individual fitness due to a combination of some autosomal DNA transmission from B-bearing males, greater mating opportunities for males in female-skewed populations, and increased fitness of recombinant offspring. While generally favoring the first hypothesis, I note that the second hypothesis remains viable pending greater understanding of the incidence of mating between relatives.

I suggest that the current estimates of the degree of autosomal transmission by B-bearing males are too low to support the third hypothesis.

The selfish B hypothesis

As with PSR, the process of B-driven masculinization in gynogenetic gibel carp thwarts transmission of autosomal genes in the genome, a through a different mechanism (Figure 1). In wasps, autosomal transmission by a PSR-carrying male is blocked quite directly by the action of PSR, by the process of masculinization itself (through elimination of paternal autosomes). In gynogenetic gibel carp, transmission of non-B genes is blocked more indirectly. Masculinization does not in itself block transmission of the autosomes, since males likely produce genetically standard sperm containing the male's genes. Nonetheless, because of the lack of sexual females, males lack productive mating opportunities, thus masculinization de facto blocks transmission of an individual's autosomes (with rare exceptions; see below). Despite the differences, the two systems share numerous similarities: in both cases, B chromosomes (but not other genes) are efficiently transmitted from fathers to sons, and lead to masculinization of offspring in ways that block ultimate transmission of non-B genes.

The steps by which the carp masculinizing B could have evolved through selfish processes are possible to envision. Starting with the classic case of gynogenesis, in which only maternal chromosomes are transmitted to offspring, B chromosomes that evolved the ability to make their way from sperm to offspring genomes would increase their overall transmission, favoring their increased frequency in the population. Moreover, if males are rare in the population, males necessarily have more mating opportunities on average than females; thus, paternally-transmitted B chromosomes benefit from being transmitted through males. Notably, this can be so even if B chromosomes are less efficiently transmitted through males than through females because of the greater number of mating opportunities. Because presence in males is beneficial for B chromosome transmission, B chromosomes that lead to masculinization of individuals could benefit. Specifically, the expected number of offspring inheriting a B chromosome through females or male would be proportional to $t_m(1\text{-}R)$ and t_fR, if R is the primary sex ratio (fraction of males) and t_m and t_f are rates of transmission through males and females, respectively. Thus, if males are the rare sex ($R < 0.5$), B chromosomes will have greater reproductive fitness through males so long as $t_m/t_f > R/(1\text{-}R)$. Under these circumstances, B chromosomes will benefit from increasing the probability that embryos develop as males. Therefore, B chromosome-linked alleles that increased the possibility of development as a male would be favored and could spread through the population. These considerations suggest a possible explanation for the male-determining B chromosomes of gibel carp. Note that, despite various particularities introduced by gynogenesis, these thoughts in many clearly resemble previous proposals for selfish sex determining B chromosomes (Clark and Kocher 2019; Beladjal et al. 2002).

This selfish scenario would seem to strongly select for suppressors that block transmission or action of masculinizing B chromosomes. While further work will be necessary to determine whether such suppressors are segregating in the population, Zhao et al.'s data clearly show that suppression is not ubiquitous despite these apparent clear benefits (Zhao et al. 2021). One possibility is that suppressors have not evolved because positive selection is very inefficient in species that lack recombination given ubiquitous selection on linked sites.

The kin selection hypothesis

Another possibility is that the autosomal genes that are not transmitted through males nonetheless benefit through kin selection (Figure 2). If females primarily mate with male kin, then families of females that produce males could have more mating opportunities and thus greater reproductive fitness overall (Grosmaire et al. 2019). This possibility is greatly increased by clonal transmission leading to related individuals sharing identical genes (with the exception of new mutations or occasional paternal transmission of genes, see below). Under clonal reproduction, a male's nephew is expected to share 100% of his autosomal genes. Therefore, facilitating a sister's reproduction is as efficient a way of transmitting one's genes to the next generation as is reproducing oneself (i.e., as a female). Thus, males in this species could essentially represent a non-reproductive caste that increase their relatives' reproductive fitness. Notably, this argument would depend on frequent mating between relatives, or perhaps more specifically on male limitation in the absence of male production by a clonal lineage. While such a scenario is easy to imagine for instance in male-producing gynogenetic nematodes, which likely reproduce in gregarious broods (see, e.g., the similar model in Grosmaire et al. 2019), it is somewhat more surprising for a swimming marine organism such as carp. More work on the reproductive biology and population subdivision of gibel carp will be necessary to determine the plausibility of this hypothesis.

Another feature of the gibel carp system also attracts our attention. First, males can be produced by a second mechanism, namely temperature dependent male sex determination (TSD) (Zhao et al. 2021; Zhu et al. 2018). TSD is harder to explain in terms of selfish elements, since strong correspondence between phenotype and genotype is not expected. However, TSD could benefit from kin selection, that is by increasing mating opportunities for close relatives, as described above for masculinizing B chromosomes. An allele that allowed for the possibility of production of occasional males (whether due to temperature, other factors, or simply randomly) could increase its representation in future generations (Figure 2c) due to facilitating reproduction of clonally-related females that also carry the allele. Alternatively, before the advent of masculinizing B chromosomes, populations that lacked TSD males could have frequently gone extinct, leading to an advantage to populations that happened to maintain TSD. While the limits of such group selectionist explanations have been discussed at length (Wade 1978, etc.), it may be that the conditions hold in this case given the potentially rapid rate of extinction when an individually beneficial all-daughter allele spreads in a population.

The direct fitness hypothesis

A remarkable feature of the gibel carp system is the observation of occasional transmission of paternal autosomal DNA. Paternal autosomal DNA transmission occurs specifically from masculinizing B chromosome-containing males, but not TSD males (Figure 3; Yi et al. 2003; Chen et al. 2020; Zhao et al. 2021; Zhu et al. 2018). Restriction of such transfers to masculinizing B chromosome males suggests dependence on B transfer, for instance if B chromosome presence leads to contact between the genomes that facilitates autosomal DNA transfer (Zhao et al. 2021). While paternal autosomal DNA transmission per mating event remains reduced relative to Mendelian expectations, males have more mating opportunities under female-skewed sex ratios. As such, it is theoretically possible that males' total autosomal fitness could rival that of females, particularly if their offspring have increased fitness due to the effects of recombination. In this case, masculinizing B chromosomes' presence could be explained not by selfish behavior nor by inclusive fitness, but as elements that increase the individual fitness of masculinized individuals. Thus, quantitative analysis is necessary.

Scrutiny of available data suggests that such transfers appear to be quite rare. In the largest study of the phenomenon to date, Zhao et al. (2021) used 15 primer pairs to assess transmission. Each primer pair amplifies many different bands within the genome, including some that are found in the father but not the mother. In total, among the 15 primer pairs studied by Zhao et al., there appear to be 70-80 bands that are specific to the father in their crosses (78 by my count; Figure 3 and Supplemental Figure 2 of Zhao et al. 2021). These approximately 78 bands were each assessed in 20 offspring by the original authors, for a total of roughly 1560 observations. Of these, they observed transmission of a paternal band in 11 cases.

What does this imply for the overall rate of transmission of paternal autosomal DNA? The 11 observed cases are unlikely to represent the total number of true paternal DNA transmission events at these loci, since in addition to the paternal-specific band, the father may share alleles with the mother that do not give rise to the discrepant band: such alleles are "invisible" to the assay, since their transmission is not distinguishable from maternal transmission. Gibel carp are hexaploid, which potentially complicates the calculation since it is not clear which of two possibilities are more appropriate to this particular case: first, what may be called "true" hexaploidy, in which there is allele sharing between the six homologous chromosomes (i.e., a transmitted paternal allele could be integrated in to the genome at any of 6 loci); and second, de facto diploidy, in which alleles do not move between the three different homologous diploid loci. Whereas in cases of sexual hexaploids, this question could be addressed by looking at patterns of meiotic segregation, asexuality prohibits resolution by these methods.

Consideration suggests that such ploidy distinctions may not matter much for our estimation of the maximum rate of paternal DNA transfer. Assuming de facto

diploidy, the father may contain one or two total allelic copies of the paternal-specific sequence. If the father contains one copy (i.e., is heterozygous), then only one-half of transmission events would be detected. If the father were heterozygous at each locus, the 11 observed events would suggest roughly 11 additional non-observed events in which the shared allele was transmitted from the father. This suggests that out of 1560 loci times two alleles per locus, the paternal allele was transmitted roughly 22 times, implying that 0.71% of transmitted alleles were paternal, and 92.3% maternal. If instead the father is homozygous for each locus, this would imply no unobserved transmissions, for a rate of 11/(1560 x 2) = 0.35%.

Assume true hexaploidy changes the calculation slightly but with similar results. In this case the father's genome could contain 1-6 copies of the paternal-specific sequence, with a total of 6 copies per locus in the offspring genome. If the paternal-specific sequence is present in only a single copy, then this would suggest that along with the 11 observed transmission events there would also be roughly 55 unobserved events, for a total of roughly 6 X 11 events. Because in this case there are a total of 6 copies per locus, these 66 transfer events are out of a total of some 1560 X 6 events, which again yields a value of 0.71%. At the other extreme, if the paternal genome has six alleles yielding the paternal-specific band, then this would imply no unobserved transmissions, for 11/(1560 x 6) = 1.2% paternal DNA transmission. Probably a common case would involve the paternal genome containing 2/6 alleles yielding the father-specific band (e.g., if the band arises from a sequence specific to one of the three progenitor genomes that gave rise to the hexaploid paternal genome), in which case we would expect roughly 22 total transfer events, for an estimated transmission rate of 22/(1560 x 6) = 0.24%.

The final possibility worthy of consideration is that a paternal-specific band arises from a shared multi-locus sequence, for instance an active transposable element within the father's genome. In this case, transfer of only one or a subset of the transposable element copies could lead to detection in the offspring. This would mean that the observed number of 11 transmission events is observed across a larger number of loci than calculated above, implying that a lower rate of transmission per locus than calculated, by roughly a factor of the number of loci per genome giving rise to the band.

In total, then, the above suggests a paternal contribution of roughly 0.71% or less of total offspring autosomal DNA. This much reduced contribution of males to future generations relative to females needs to be weighted by the fact that males have more opportunities to mate because of skewed sex ratios. Under the estimate of 0.71% DNA transmission for B chromosome-containing males relative to females, total transmission for B chromosome-containing males will equal that of females when males represent 0.71% of the population or less. However, for all 21 populations with substantial population samples across two studies (Jiang et al. 2013; Li et al. 2018) (e.g., with at least 70 individuals, which would translate to an expectation of 0.5 or more males under 0.71% males), the estimated frequency of males was higher than 0.71% (median 8.0%; mean 10.2%). At 8% male frequency,

males are expected to have 92%/8% = 11.5 times as many mating opportunities on average as females, translating to an average total direct genetic contribution to the next generation of less than or equal to 0.71% x 11.5 = 8.2% , relative to females.

While greater fitness of offspring due to the fitness-increasing effects of recombination could help to compensate or this deficit, it is hard to imagine that the average offspring would have the requisite 100%/8.2% = 12.2-fold increase in fitness relative to non-recombinant offspring. These arguments thus suggest that, while recombination may indeed help the population to overcome the long-term effects of mutational meltdown, that it cannot account for the presence of the recombinogenic masculinizing B chromosome in the population, since B-carrying individuals are expected to have much lower direct fitness. Instead, it seems more likely that the selfish or kin selection dynamics discussed above can explain the maintenance of the B chromosome in the population.

Concluding remarks

In total, while similarities of gynogenetic gibel carp's masculinizing B chromosome to the PSR chromosomes of wasps suggest that this chromosome to represents an "ultimate selfish genetic element", the very high degree of relatedness between relatives raises the possibility that masculinizing B chromosomes benefit non-B genes through kin selection. Estimation of the rate of mating between relatives, as well as the extent to which conspecific male presence increases fitness of females due to limited availability of heterospecific males, will be crucial to resolving this issue. The results of Zhao et al. build our understanding of the curious male-producing gynogenetic system of carp, and provide a rare opportunity to address the larger questions of diversity, maintenance, and evolutionary forces shaping gynogenetic systems. It will be fascinating to learn more about the mating structures and possible existence of suppressors, as well as the molecular mechanisms driving genetic sex determination in these remarkable fish.

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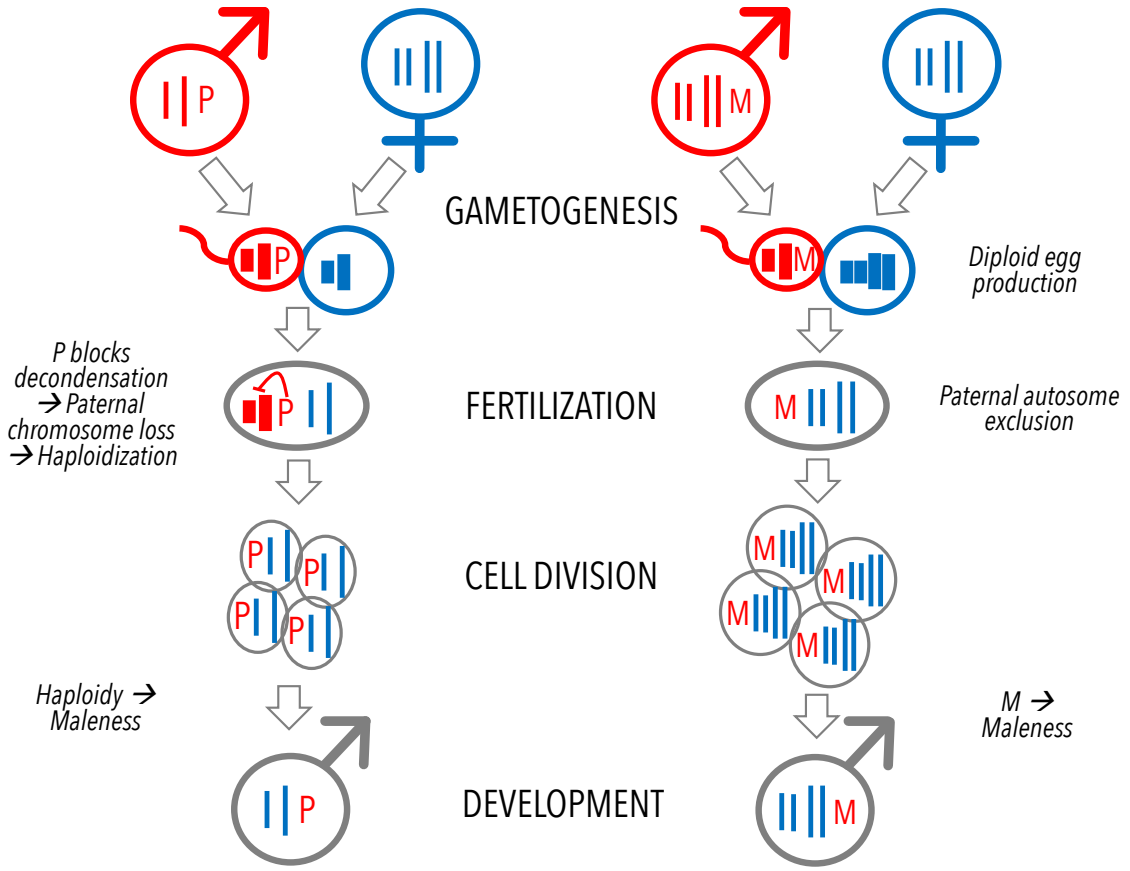

Figure 1. Two potentially "ultimate selfish elements," in which masculinization by B chromosomes bars transmission of autosomal genes. Left, PSR (paternal sex ratio) chromosome in haplodiploid wasps. Following fertilization, presence of the PSR ("P") blocks decondensation of paternally-inherited autosomes, leading to their loss during cell division. Thus haploidized, the individual develops as a male, consistent with haplodiploidy in wasps, containing only maternally-inherited autosomes (blue). Presence of PSR will similarly block transmission of this individual's autosomes to the next generation. Right. Masculinizing B chromosome ("M") in gynogenetic hexaploid gibel carp. Fertilization of nonreduced diploid eggs produces diploid offspring carrying only maternal autosomes along with the paternal M. The M causes male development. Because females in this species do not use paternal DNA to produce offspring (as shown), masculinization by M will lead this offspring not to transmit autosomal genes to the next generation. Note that this scenario is slightly simplified given small amounts of paternal transmission of autosomal DNA (e.g., Figure 4).

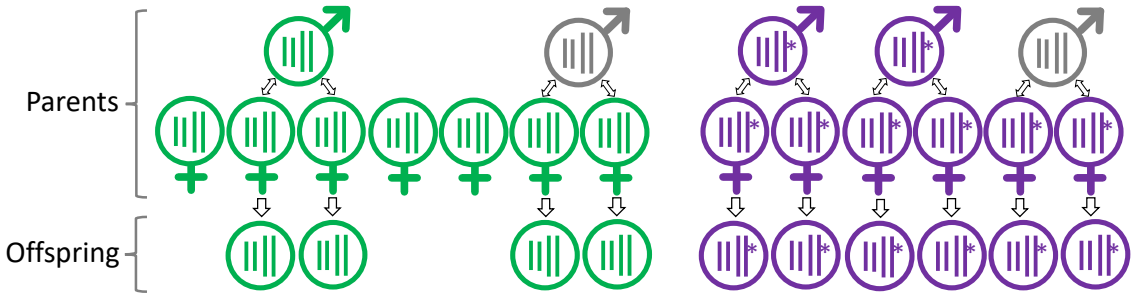

Figure 2. The inclusive fitness/kinship model for male production. Green and purple represent different clonal lineages/families, grey unrelated males. If in some clonal lineages (green), rarity of males leads to reduced (or no) mating/offspring for some females, then an allele that increases the probability of developing as a male (asterisk, purple lineage/family) can be inherited by a larger number of total offspring (bottom of diagram; 6 versus 4) despite that a greater fraction of individuals carrying the allele develop as males that do not transmit autosomal genes. This can be the case so long as many mating are among clonal relatives (green/green, purple/purple) even if not all (grey represents unrelated males).

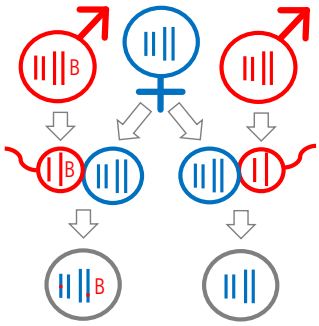

Figure 3. Masculinizing B-containing males contribute small amounts of paternal autosomal DNA to offspring. Cartoon of a single female mating with a B-containing male (left) and a non-B-containing male. A small fraction of autosomal DNA is transmitted to offspring from the father (red) for B-containing but not non-B-containing males.