Sexual Conflict via Maternal-Effect Genes in ZW Species

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The placenta of eutherian mammals and the endosperm of plants provide an expansive realm for genomic conflict through genetic imprinting (*1*, *2*). Here we show that maternal products in the egg can provide a parallel arena for genetic conflict. The distinction between species in which the heterogamous sex is male (XY) versus female (ZW) has far-reaching consequences for sexual conflict via maternal-effect genes, the products of which can have opposing fitness effects because of the unique developmental trajectories of sons and daughters (Fig. 1). Throughout, the term sex chromosomes refers to their nonrecombining regions in the heterogametic sex.

Maternal effects might lead to sexual conflict in two ways: (i) Incidental harm. Maternal-effect alleles that favor sons but harm daughters through chance pleiotropy accumulate on the Z, and those favoring daughters and harming sons accumulate on the W. (ii) Actively selected harm. When sibling competition occurs, maternal-effect alleles are selected to kill or incapacitate the nontransmitting sex. Z alleles harm daughters and W alleles harm sons [see (*3*) for autosomal examples in the context of selfish elements].

More explicitly, consider a large, randomly mating population with discrete, nonoverlapping generations subject to constant selection. Individual fitness is determined maternally (*4*, *5*). Fitness is defined as viability, mating success (the probability that an adult enters the mating pool), or fertility (a contribution of an individual to the number of offspring produced by the mating pair, assuming that male and female contributions interact multiplicatively). Assume that there are two W-linked alleles: W_0 and W_1. Let Z denote the Z chromosome, and let offspring of ZW_0 mothers have fitness 1. The fitness of daughters and sons of ZW_1 mothers have fitness $1 + B_{\text{daughter}}$ and $1 - C_{\text{sons}}$, respectively; i.e., ZW_1 maternal effects benefit their daughters at a cost to their sons. Let p be the frequency of ZW_1 females in the mating pool. Then, it is straightforward to show that the value of p in the next generation is

$$p' = p + \frac{B_{\text{daughters}}\, p(1 - p)}{1 + pB_{\text{daughters}}}$$

From this equation, one concludes that if $B_{\text{daughters}} > 0$ (that is, daughters of ZW_1 mothers get some fitness advantage relative to daughters of ZW_0 mothers), then allele W_1 invades and gets fixed ($p \to 1$) independently of its effect on sons.

Next assume that there are two Z-linked alleles, Z_0 and Z_1. Let W denote the W chromosome and set the fitness of offspring from Z_0W mothers to be 1. Daughters and sons of Z_1W mothers have fitness $1 - C_{\text{daughters}}$ and $1 + B_{\text{sons}}$, respectively. This fitness scheme implies that Z_1W maternal effects benefit sons at a cost to daughters. Let the frequencies of Z_1W females and of allele Z_1 in males in the mating pool be p_{female} and p_{male}, respectively. Then, it is straightforward to show that in the next generation

$$p'_{\text{females}} = p_{\text{males}} \qquad \text{and}$$

$$p'_{\text{males}} = \frac{p_{\text{males}} + p_{\text{females}}}{2} + B_{\text{sons}} \frac{p_{\text{females}}(1 - p_{\text{females}})}{2(1 + B_{\text{sons}} p_{\text{females}})}$$

This system of equations has only two equilibria: $p_{\text{male}} = p_{\text{female}} = 0$ and $p_{\text{male}} = p_{\text{female}} = 1$. If $B_{\text{sons}} > 0$, the former is unstable whereas the latter is stable. This shows that if sons of Z_1W mothers get a fitness advantage, allele Z_1 invades and gets fixed (so that $p_{\text{male}} \to 1$, $p_{\text{female}} \to 1$) independently of its effect on daughters' fitness.

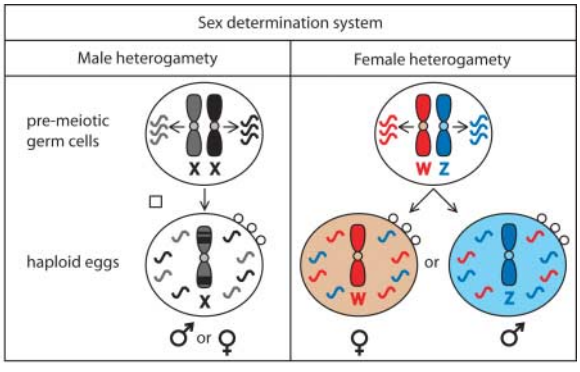

Fig. 1. Maternal-effect alleles on the two X chromosomes (gray/black chromosomes) are transmitted symmetrically to sons and daughters, but in ZW species, the transmission is asymmetrical—Z-linked (blue chromosome) to sons and W-linked (red chromosome) to daughters. Wavy lines depict maternal products coded by the sex chromosomes and packaged in the egg. Blue (sons) and red (daughters) shading of eggs depicts gender-specific differences during subsequent ontogeny of the embryo.

The accumulation of sexually antagonistic maternal-effect genes in ZW species creates the opportunity for an evolutionary arms race. As sexually antagonistic male-benefit maternal-effect alleles accumulate on the Z, and female-benefit maternal-effect alleles on the W, there will be compensatory selection to counteract the harm at genes located on both the other sex chromosome and on the autosomes. Because primitive Z and W chromosomes can carry hundreds of genes, the initial potential for an arms race may be substantial. While the W chromosome decays due to its lack of recombination (*6*), the influence of this chromosome on maternal-effect conflict may diminish. For example, the Z chromosome of the jungle fowl still has at least 516 genes, whereas the W has only 55 (*7*).

Sex-specific gene expression, ontogeny, and physiology provide diverse and simple mechanisms for single-locus maternal-effect genes to harm the sex that is not carrying a maternal Z or W chromosome. The potential for maternal-effect conflict imparts an important distinction between male and female heterogamy. It replaces sexual selection as a source of chronic adaptive evolution that can drive the decay of the nonrecombining W via genetic hitchhiking (*8*). It also increases the scope for genetic conflict in ZW compared with XY species, because the W, unlike the Y, is expressed in both sexes via maternal products in the egg. Species with ZW versus XY sex determination clearly have at least one unique force influencing the evolution of their sex chromosomes.

References and Notes

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