**Minor comments on Jaquiéry et al., "Masculinization of the X-chromosome in aphid soma and gonads"**

Sexual antagonism (SA), wherein the fitness interests of the sexes do not align, is inherent to organisms with two (or more) sexes, because sperm are cheap and eggs (and parental care) are expensive. SA leads to intra-locus sexual conflict, where an allele that confers higher fitness in one sex reduces fitness in the other. This situation leads to what has been referred to as "gender load" (why not "sex load" is unclear, but try typing those keywords into Google and see what you get), resulting from the segregation of SA alleles in the population. Gender load can be reduced by the evolution of sex-specific (or sex-biased) gene expression, in which the expression of the deleterious allele is suppressed in the sex in which it is deleterious. A specific prediction is that gene-duplication can lead to sub- or neo-functionalization, in which the two duplicates partition the function in the different sexes. The conditions for invasion by a SA allele differ between sex-chromosomes and autosomes, leading to the prediction that (in XY or XO systems) the X should accumulate recessive male-favored alleles and dominant female-favored alleles; similar considerations apply in ZW systems.

 Aphids present an interesting special case, for several reasons: they have XO sex-determination, and three distinct reproductive morphs (sexual females, parthenogenetic females, and males). Previous theoretical work by the lead author predict that the X should be optimized for male function, which was borne out by whole-animal transcriptome analysis.

 Here, the authors extend that work to investigate tissue-specific, sex-specific gene expression. They argue that, if intra-locus SA is the primary driver of sex-biased gene expression, it should be generally true in all tissues. They set up as an alternative the possibility that sex-biased gene expression could also be driven by dosage compensation. They cite references supporting their argument that "dosage compensation (could be) stronger in the brain", although the underlying motivation for that argument appears to be based on empirical evidence rather than theoretical predictions.

 At any rate, the results are clear: all tissues investigated show masculinization of the X. Further, X-linked copies of gene duplicates were more frequently male-biased than duplicated autosomal genes or X-linked single-copy genes.

 To sum up, this is a nice empirical study with clearly interpretable (and interpreted) results. The prediction that sex-biased gene expression resulting from some selective force other than SA should lead to variation among tissues, whereas SA should lead to uniform variation is not justified on clear theoretical principles; if such principles exist, they should be explicitly-stated. For example, I could imagine a situation in which all of the sex-biased gene expression was the outcome of sexual antagonism, but for some reason only some tissues experienced SA selection. If such principles are not forthcoming, the stated motivation for the study has the feel of a straw man.