regards, we can only refer to other results which have found a link between charitable donation and status [12,13] and between blood donation and generosity [14]. Assembling these strands of evidence it is reasonable to speculate that reputational benefits may outweigh donation costs. Interestingly, a model suggests courtship gifts should be costly (and so signal quality or intentions) yet intrinsically worthless to the recipient (to overcome the 'gold-digger' problem) [15], so charitable donations via a fundraiser may be a nice example of this.

Are the results surprising? On the one hand they fit well with existing examples where generosity is displayed publicly [16]. Furthermore, generosity is well known to be a desirable trait in mate choice [17]. A few experimental studies have also found evidence that altruism is used as a display to attractive members of the opposite sex [18,19]. Yet despite all this, sexual selection is rarely invoked in explaining cooperation, and a high profile review does not include it as one of the routes to cooperation [20]. The stimulating work of Raihani and Smith [6] serves to highlight the potentially rather overlooked role of sexual selection in driving displays of altruism. It is well established that aggression may be used in male–male competition over access to females, but this shows that cooperation may also be used in competitive contexts. More generally the results should stimulate further work on how we benefit from being seen to be cooperative, and how explanations for reputation-building extend beyond indirect reciprocity.

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Plant Sex Chromosomes: Lost Genes with Little Compensation

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In many animals, gene loss on Y chromosomes is compensated through altered expression of their X-chromosome homologue. Now, however, a new study in plants finds that even genes deleted from the Y show no dosage compensation.

In species with an XY sex-determination system, such as mammals, genes in the sex-determining region (SDR) on the Y chromosome are never exposed to selection in females, while those on the X will spend twice as much time in females as in males. The same principle applies in species with Z and W chromosomes, such as birds and butterflies, where the

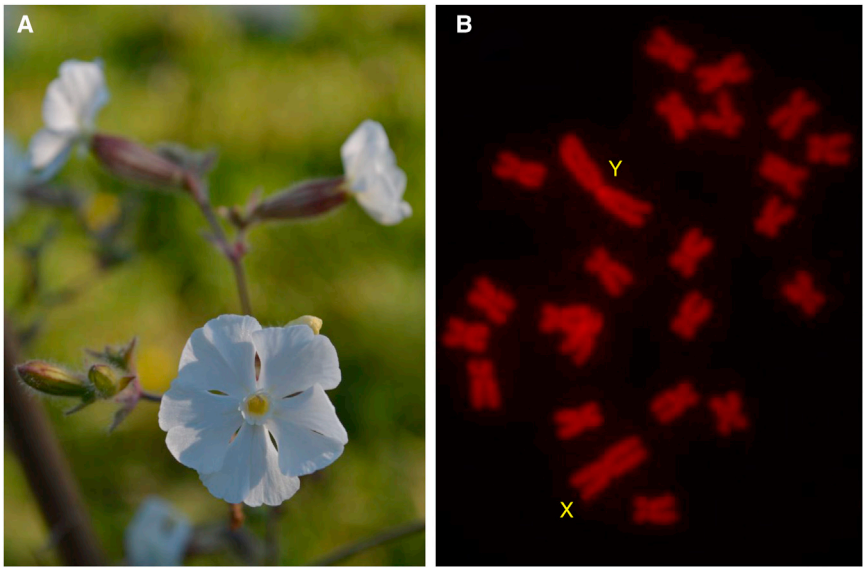

Figure 1. *Silene latifolia.*
(A) Flowers of a male individual of *Silene latifolia*. Males produce smaller but more numerous flowers than females. Image courtesy of Anne-Marie Labouche. (B) Karyotype of a male individual of *S. latifolia*, with DAPI-stained chromosomes, and the two sex chromosomes labelled. The Y chromosome is the largest chromosome and about 40% larger than the X, probably due to the accumulation of repetitive elements [20]. Image courtesy of Boris Vyskot.

W chromosome is restricted to females (for brevity we will henceforth refer only to the X and Y). This simple fact has three significant implications that help to explain why, in so many species with genetic sex determination, the sex chromosomes responsible differ in their size and content (i.e., they are 'heteromorphic').

First, we should expect Y chromosomes to become enriched for alleles that enhance the fitness of males (and not females), and X chromosomes to accumulate alleles that are beneficial to females (and not males) [1] — there has been little empirical support for such 'sexually antagonistic loci', but evidence is beginning to accumulate [2]. Second, selection should favour the suppression of recombination between the sex-determining locus and sexually antagonistic loci [1] — suppressed recombination between homologues is indeed one of the major hallmarks of heteromorphic sex chromosomes [3]. And third, because of recombination suppression, Y chromosomes lose the genetic benefits of sexual reproduction and degenerate: they accumulate repetitive elements, other deleterious mutations, and ultimately begin to lose their genes [3]. Gene loss, which in humans accounts for the fact that the Y has only ~45 functional genes in comparison to ~1000 on the X [4], implies that males and females end up with a different dosage for many sex-linked genes.

Variation in gene dosage among loci usually has deleterious effects. This is perhaps most clearly demonstrated by the strong phenotypic effects of aneuploidies such as trisomy 21, which causes Down's syndrome in humans [5]. Referring to the reduced gene dosage in males with a degenerated Y as 'the peril of hemizygosity', Susumu Ohno [6] proposed that such males would upregulate alleles on their X in response to the lower expression from the Y, in a process termed dosage compensation. Initial work in XY model organisms, such as humans, *Drosophila*, and *Caenorhabditis elegans*, supported this hypothesis (reviewed in [7]). Remarkably, dosage compensation has evolved independently and differently in various lineages [7]. In mammals it involves the inactivation of one entire copy of the X chromosome in females, while in *Drosophila* it involves the doubling of X expression in males [7]. In other taxa, including those with ancient sex chromosome systems, dosage compensation occurs on a gene-by-gene basis [7]. Although it is clear that dosage compensation varies widely among animal lineages [7], almost nothing is known about it in plants. But now, in a report recently published in *Current Biology*, Bergero and colleagues [8] throw new light on genomic responses to Y degeneration in the European plant *Silene latifolia* (Figure 1).

Plants, in fact, provide ideal material for studying sex-chromosome evolution. Separate sexes have evolved independently from hermaphroditism on numerous occasions [9], so many plant sex chromosomes are probably relatively young. *Silene latifolia* is a case-in-point — whereas mammalian sex chromosomes have a history dating back to more than 170 million years ago [4], the XY system in *S. latifolia* is between 5 and 10 million years old [8]. *S. latifolia* sex chromosomes already show some of the typical hallmarks of sex-chromosome evolution known from animals. For instance, recombination has been suppressed in two strata that can be detected in terms of step-like changes in genetic divergence between the X and Y [10]. As in mammals, Y-linked sequences in *S. latifolia* display very low genetic diversity as a result of processes that act to sweep out genetic diversity in non-recombining parts of the genome [11]. However, in contrast to the mammalian Y chromosome, which has lost almost all of its genes [4], the *S. latifolia* Y still has a large number of genes with clear homology to their X-linked counterparts [12,13]. This might be attributed to the young age of the *S. latifolia* Y, but we might also expect selection to maintain functional genes on plant Y chromosomes [12], because approximately 60% of plant genes are expressed during the growth of haploid pollen tubes [14].

Against this background, Bergero *et al.*'s [8] study yields two surprises. First, the *S. latifolia* Y chromosome *has* lost genes — indeed 14.5% of them. Previous work hinted at this possibility, with a substantial number of missing Y-linked gene transcripts [12]. However, Bergero *et al.* [8] have convincingly shown that the missing transcripts are not just the

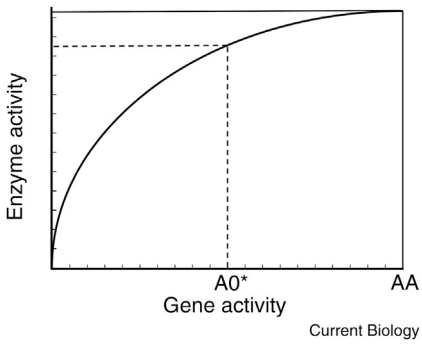

Figure 2. Wright's model of dominance.
Wright's [19] model of dominance, as it might be interpreted in the context of gene dosage. Enzyme activity exhibits diminishing returns when compared to gene dose. When a previously autosomal allele becomes sex-linked, the X allele is retained (A) and the Y allele may be lost (0*). The halved gene dosage in hemizygous males (A0*) should thus not result in halving of gene product activity, even in the absence of dosage compensation.

result of a lack of gene expression but are properly missing from the Y. Their data also reject the possibility that hemizygous genes on the X chromosome might simply have moved there from elsewhere (rather than being lost on the Y), because the same genes are found on a chromosome homologous to the X and Y in the related hermaphroditic species *Silene vulgaris*. The observed gene loss on the *S. latifolia* Y immediately poses the question as to whether the missing genes are restricted to those not expressed in haploid pollen tubes during their race down the style to fertilize ovules. This hypothesis would be interesting to test, although Y-chromosome-bearing pollen tubes can sometimes be less competitive than their X-bearing counterparts (e.g., [15]), indicating that haploid selection will not always be sufficient to maintain optimal gene function.

The second surprise is that loci at which genes have been lost do not show straightforward evidence of dosage compensation. In particular, the level of expression of most genes lost from the Y was twice as high in females as in males, in accordance with the expectation of no chromosome-wide compensation. Some individual genes did show evidence for dosage compensation, but here there was no clear pattern with respect to the evolutionary stratum in which they were sampled, nor their overall level of expression. Genes lost from the Y were interspersed among retained genes, effectively ruling out gene loss as a result of large deletions. A previous study of *S. latifolia* [16] found evidence for dosage compensation for lowly expressed Y-linked alleles, consistent with recent work on the dioecious plant *Rumex hastatulus* [17].

The results on studies of plant sex chromosomes reinforce the emerging view [7] that chromosome-wide dosage compensation is not universal. The contrasting responses to gene loss on Y chromosomes draw attention to important general questions in population genomics. On one hand, chromosome-wide imbalance in gene dosage can be deleterious, as shown by Down's syndrome in humans [5]. On the other hand, most species harbour mutations that cause effective hemizygosity for individuals at the corresponding loci, yet such individuals would generally seem to be none the worse for it because gene knock-out mutations are typically recessive. How might we reconcile these two views? The founding population geneticists Fisher and Wright famously locked horns over why deleterious mutations should tend to be recessive [18]. Wright's [19] explanation was intuitive and has stood the test of time — because of the flattening curve of gene-dose effects on physiological performance, a single functional copy of a gene should be much better than half as good as two copies (Figure 2). We might be tempted from this idea to conclude that dosage compensation should not be greatly needed. But an important difference between deleterious recessive mutations that segregate in population and the genes that have been lost from Y chromosomes is that the former tend to fluctuate under genetic drift at low frequencies, whereas effects of the latter are ultimately felt in half the population (all of the males).

Another important difference is, of course, the fact that the loss of Y-linked genes amounts to an accumulated product of potentially small selective effects that, together, may weigh heavily upon affected males. Should the implementation of chromosome-wide dosage compensation wait until a sufficient burden of gene loss has occurred? But then how much gene loss should be enough to warrant compensation? And what occurs in populations as they approach that point? Alternatively, how might populations evolve from gene-by-gene dosage compensation to a mechanism that enacts chromosome-wide compensation? The surprising results of Bergero *et al.*'s [8] study throw further new light on variation among taxa in dosage compensation, but many fundamental questions remain.

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