recombination rates. Such experiments could assist future studies of how recombination rates and aging are influenced by genetic background.

One other interesting trend was observed in this study. In almost every run females refrained from laying eggs for two to four days, before resuming oviposition. One explanation for this observation may be that the females needed additional nutrients for the metabolically expensive process of oviposition (Chapman and Partridge, 1996). This gap in oviposition may give females time to build up the necessary nutrients to resume oviposition. This interesting phenomenon should be studied in more depth.

A class discussion of the results of this study might include: 1) Why was recombination and aging only tested in females in this study? There is no recombination in male *D. melanogaster* (Morgan, 1914). 2) Are there genes that are known to directly affect rates of recombination? Yes, including RAD51 in yeast, mice, *Drosophila*, and humans (for a discussion of this topic, see Baker and Hall, 1976; Shinohara *et al*., 1993; Staeva-Vieira *et al.*, 2003).

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Reversion of the *Bar* (*B*) mutation in the Basc X chromosome of *Drosophila melanogaster* by unequal crossing over.

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The dominant, X-linked, *Bar* (*B*) mutation was isolated in 1914 by Sabra Colby Tice as a change in the structure of the eye of *D. melanogaster* from round (wild type) to a narrow bar of eye tissue in homozygous females and hemizygous males, and as less extreme *Bar*-eyes in heterozygous, B/B^{+}, females

(Tice, 1914; for a more detailed description of the *B* mutation, see Lindsley and Zimm, 1992; Wolfner and Miller, 2016).

May (1917) observed that the *B* mutation could revert to wild type (*Bar* eyes to round eyes) and proposed that the reversion mechanism was either "…a reversible chemical reaction between two compounds one of which is more stable than the other." Or "…partial non-disjunction." (May, 1917). Based in part on observations by Zeleny (1919, 1921, 1922) that the *B* to B^+ reversions only occurred in females, Sturtevant (1925) proposed that these reversions were due to unequal crossing-over events that occur in about 1 in 1,000 to 2,000 flies (also see Sturtevant and Morgan, 1923). Bridges (1936) confirmed cytologically that the *B* mutation was associated with a tandem duplication, which we now know arises by crossing over between two *B104* transposable DNA elements (Tsubota *et al*., 1989).

Unequal crossing-over events are important in *Drosophila* and other higher organisms, including humans. For example, red-green color blindness in humans can be caused by an unequal crossing-over event (Nathans *et al*., 1986), and humans with triplications of a segment of chromosome 17 have a more extreme form of Charcot-Marie Tooth disease than those with duplications (Liu *et al*., 2014). Unequal crossing-over events have also given rise to extra copies of genes that can evolve into new functions, including globins (Shen *et al*., 1981), as well as extra and missing DNA base pairs, which can lead to human disorders (Nakamoto *et al*., 2002)

Based on the above reports, we tested the hypothesis that reversions of *B* to B^+ in Basc X chromosomes are caused by a loss of one duplication at the *B* locus due to unequal crossing-over events in homozygous (Basc/Basc) diplo-X females, but not in heterozygous (Basc/+) females or hemizygous males that have a single X chromosome (Basc/Y). To test this hypothesis, we performed the following three sets of crosses.

In these crosses, parental Basc/Basc females and Basc/Y males have *Bar* eyes that are also white-apricot in color (the *a* in Basc stands for the w^a mutation), Basc/+ parental females have a reduced *Bar*-eyed phenotype, and C(1)DX, *y f* parental females are marked with the *y* (*yellow* body color) and *f* (*forked*, short bristles) mutations and contain two X chromosomes attached to a single centromere. In Cross 1, the F1 progeny are Basc / Y patroclinous males (either *B*/Y or B^+/Y) that receive their X chromosome from their fathers and their Y chromosome from their mothers, and C(1)DX, *y f* matroclinous females, that get their attached-X chromosome from their mothers and their Y from their fathers. See Lindsley and Zimm (1992) for a more detailed description of the mutations (*y* and *f*), compound-X chromosome, and Basc balancer chromosome used in this study.

Cross 1: C(1)DX, *y f* / Y females × Basc / Y males

The F1 progeny were scored for *B*/Y *Bar*-eyed males and B^+/Y round-eyed, white-apricot (revertant) males. Each B^+ revertant male, if isolated, would be mated to virgin C(1)DX, *y f* / Y females to make sure the round phenotype bred true into males in the F2 generation. We predict, however, that no B^+ revertant males will be recovered from this cross.

Cross 2: Basc / Basc females × Basc / Y males

In cross 2, since *B* has been reported not to revert in males (Zeleny 1919, 1921, 1922; Sturtevant 1925), any F1 B^+ / Y males should arise by unequal crossing over in parental females. Each of the presumptive B^+ / Y males were mated to C(1)DX, *y f* females to make sure the round phenotype bred true into males in the F2 generation. We predict that B^+ revertant males derived from unequal crossing over in Basc/Basc parental females will be recovered in this cross at a frequency of about 1 in 1,500 F1 males

(Sturtevant 1925). We should also recover some *B/B*$^+$ F1 females that have less extreme *Bar*-eyed phenotypes.

Cross 3: Basc / *B* females × Basc / Y males

In cross 3 the revertant F1 progeny from parental females were scored as *B*$^+$ / Y, red-eyed, males (from the *B* containing X chromosome in parental females), or as *B*$^+$ / Y, white-apricot eyed, males from the Basc chromosome in parental females. The non-revertant *B* males were Basc / Y (bar and white-apricot eyes) and *B* / Y (bar and red eyes). We expected no *B*$^+$ / Y, white-apricot, revertants from cross 3, since crossing-over events do not occur in males, and the Basc X chromosome in the parental heterozygous females (Basc / +) contains multiple inversions that cover the entire chromosome. Recombination events in inversion heterozygotes are not recovered in progeny because they contain extra or missing segments of the X chromosome and/or extra or missing centromeres (see references in Woodruff *et al*., 2013).

The results from Crosses 1, 2 and 3 are shown in Table 1. As predicted, *B*$^+$ revertants were only recovered in Cross 2, and the results of Cross 1 and Cross 3 were significantly different from Cross 2. These results support the hypothesis that *B* revertants are caused by loss of a segment of the X chromosome due to unequal crossing-over events.

Table 1. Observed results for the recovery of *B* non-revertants and *B*$^+$ revertants.

Crosses	Number of *B*$^+$ Revertants	Number of *B* Non-Revertants
Cross 1: C(1)DX, *y f* / Y females × Basc / Y males	0	4,976[a,b]
Cross 2: Basc / Basc females × Basc / Y males	6	4,755[a,c]
Cross 3: Basc / *B* females × Basc / Y males	0	5,250[b,c]

[a] Fisher exact test P, one tail = 0.01; [b] Fisher exact test P, two tail = 1.00; [c] Fisher exact test P, one tail = 0.01.

Class discussions of the results of this study might include: 1) *Drosophila* geneticists might look at the results of this study and say of course Morgan, Sturtevant, and Bridges, the founders of *Drosophila* genetics, were right about the mechanism of reversion of the *B* mutation. But they were not always right. For example, Morgan (1912, 1914) reported that *D. melanogaster* males do not undergo recombination. Yet, Bridges and Morgan (1919) recovered male recombination events in a 1912 experiment (see discussion in Woodruff and Thompson, 1977). Maybe these recombination events were associated with transposable DNA elements, such as P elements that are known to cause breakage events that can lead to recombination of markers in *D. melanogaster* males (Henderson, Woodruff and Thompson, 1978). 2) Are there other examples in the literature of unequal recombination events leading to genetic disorders in humans? Yes: see examples in Lupski (1998), including the blood disorder β-thalassemia.

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Measuring narrow-sense heritability in *Drosophila melanogaster* using inbred strains.

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For a trait to evolve by either natural or human selection, the phenotypic variation of the trait must be inherited, *i.e.* be due to genetic variation. The fraction of total variation in a trait due to genetic variation is called the heritability of the trait. In addition, the best measure of whether a trait will evolve or respond to selection is narrow sense heritability (h^2), the fraction of the total variation due to the additive effects of genes. Dominance and gene × environmental interactions also affect quantitative traits and heritability values (for discussions of heritability, see Falconer and Mackay, 1996; Roff 1997; Allendorf and Luikart 2007; Hedrick, 2011).

Three possible ways to estimate the h^2 of a quantitative trait are: 1) trait correlations between parents and their offspring, where h^2 is equal to the regression slope of mid-parent values to offspring values; 2) comparing concordance of traits in monozygotic *versus* dizygotic twins, where h^2 is equal to two times the monozygotic concordance minus dizygotic concordance; 3) and using the results of selection experiments, where h^2 is equal to the response of selection divided by the selection differential (see Falconer and Mackay, 1996).

Everett *et al.* (2016) estimated h^2 for bristle number in *Drosophila melanogaster* by comparing midparent numbers to offspring numbers and observed a h^2 of 0.05 for females and 0.04 for males. In addition, Woodruff and Thompson (2005) estimated h^2 of sternopleural bristle number by selecting for increased bristle numbers over eight generations and observed h^2 values of 0.11 for females and 0.15 for males in non-inbred lines.

In this study, we estimated h^2 for sternopleural bristle numbers using three highly inbred lines of *D. melanogaster* (see sternopleural bristles in Chyb and Gompel, 2013, and in Figure 2 of Everett *et al*., 2016). We used a modified version of the methods of Possidente and McQuade (2015), who estimated h^2 for body size using inbred lines of *D. melanogaster*. The advantage of using such highly inbred, homozygous, lines to measure h^2 is that variation among individuals within the same line is due entirely to non-genetic effects, while dominance effects are eliminated (see discussions of this topic in Falconer and Mackay, 1996; Possidente and McQuade, 2015). With inbred lines, h^2 is equal to the genetic variance (V_G) divided by the sum of genetic variance and environmental variance (V_E) (Possidente and McQuade, 2015), *i.e*.,

$$h^2 = V_G/(V_G + V_E),$$

where V_G can be calculated using half the difference in means squared of the inbred lines examined, divided by 2 ($V_G = 0.5(((Mean^1 – Mean^2)/2)^2)$), and V_E for a given inbred line can be calculated using the standard deviation squared of that line ($V_{E1} = SD_1^2$) (Possidente and McQuade, 2015). To properly estimate h^2 you need to use the pooled estimate of V_E by calculating the average V_E for two populations of the same sample size ($V_E = (V_{E1} + V_{E2}) * 0.5$). To detail this process, we will walk through the calculation of h^2 for sternopleural bristle number using two theoretical inbred lines of *D. melanogaster*, IB_1 and IB_2.

IB_1 males had a mean sternopleural bristle number of 16.00 bristles, with a standard deviation (SD) of ± 2.58, while IB_2 males had a mean of 25.31 bristles with a SD of ± 3.25. Hence,