"I do not think anything in my scientific life has given me so much satisfaction as making out the meaning of the structure of heterostylos flowers".-Darwin
The height of the anthers is controlled by alleles $a$ and $A$.

The length of the style is controlled by alleles $g$ and $G$.

Rarely, recombination between loci gives rise to the $gA$ combination, yielding anthers and style at the same level.

**EVOLUTION 2e, Figure 9.19**
Inversions block recombination in heterozygotes
Super genes

‘coadapted combinations of several or many genes locked in inverted sections of chromosomes and therefore inherited as single units.’ (Dobzhansky, 1970).
A supergene determines highly divergent male reproductive morphs in the ruff
Clemens Küpper, Michael Stocks, Judith E Risse, Natalie dos Remedios, Lindsay L Farrell, Susan B McRae, Tawna C Morgan, Natalia Karlionova, Pavel Pinchuk, Yvonne Verkuil, Alexander S Kitaysky, John C Wingfield, Theunis Piersma, Kai Zeng, Jon Slate, Mark Blaxter, David B Lank & Terry Burke

Faeder-Independent inversion
4Mb

Structural genomic changes underlie alternative reproductive strategies in the ruff (*Philomachus pugnax*)
Sangeet Lamichaney, Guangyi Fan, Fredrik Widemo, Ulrika Gunnarsson, Doreen Schwochow Thalmann, Marc P Hoepner, Susanne Kerje, Ulla Gustafson, Chengcheng Shi, He Zhang, Wenbin Chen, Xinning Liang, LeiHuan Huang, Jiahao Wang, Enjing Liang, Qiong Wu, Simon Ming-Yuen Lee, Xun Xu, Jacob Höglund, Xin Liu & Leif Andersson

Independent genotype

Satellite genotype

Faeder genotype
The cost of sex.

Why risk breaking it up a winning genotype.

Finding and attracting a mate are costly and may be impossible, and mating is dangerous.

The two fold cost of sex

Sexual organisms only contribute $\frac{1}{2}$ of their genome to their offspring. While asexual organisms contribute their entire genome. This is sometimes called the cost of males.

Despite this sexual reproduction persists.
• Why have sex?
Vast majority of eukaryotic organisms reproduce sexually

Many species are not obligate sexuals and can reproduce clonally (i.e. asexually)
e.g. Vegetative growth in plants.
However, they will only do so for a few generations

Vertebrate asexual species can evolve

Unisexual Cnemidophorus

Unisexual *P. formosa* (left) sexually parasitizes the sexual *P. latipinna* (right)

Despite this sexual reproduction persists.
Asexual species emerge often in animals/plants but are generally short-lived as species.
Hypotheses for the evolutionary advantage and maintenance of sex

• Asexual species accumulate deleterious mutations
  • Hitchhiking of deleterious mutations
  • Due to Muller’s Ratchet

• Asexual species adapt slower
  – forced to fix advantageous mutations sequentially (Clonal interference)
  – Creation of novel haplotypes in asexuals is mutation limited. Hard to keep pace with rapidly evolving pathogens (Red queen hypothesis)
The Red Queen Hypothesis

Hosts have to constantly adapt to changing pathogen environment

Recombination generates novel combinations of alleles (i.e. haplotypes)

So that sexual species can more rapidly evolve to resist parasites.
Evidence for red queen hypothesis

- Increase in sex with higher parasite load.

*Potamopyrgus antipofarum*

*EVOLUTION 2e, Figure 15.7*
Clonal interference hypothesis

Selected alleles must fix sequentially in absence of sex

\[ s_A = 0.08 \]
\[ s_B = 0.06 \]
\[ s_{AB} = 0.14 \]
Clonal interference hypothesis

Selected alleles can fix simultaneously in presence of sex.

Without recombination

With recombination (r=0.001)
Evidence of Clonal Interference

Clonal interference also plays a key role in thinking about evolution of drug resistance in pathogens.
Muller’s Ratchet in asexuals

- Deleterious allele

Loss by drift of haplotype with no deleterious alleles

Loss of haplotype w. Only 1 deleterious allele

Hermann J. Muller

Time 1

Frequency of occurrence

Time 2

Number of deleterious mutations

Time 3
Muller’s Ratchet

Ratchet in asexuals: Progressive loss by drift of haplotype with lowest number deleterious alleles. Lowers fitness of population.

Sexual organisms avoid the effects of Muller’s ratchet.

Loss of haplotype with zero deleterious alleles

Recombination can reform that haplotype
Recurrent loss of sex is associated with accumulation of deleterious mutations in *Oenothera*

Jesse D. Hollister\(^1\)\(^,2\)\(^,\ast\), Stephan Greiner\(^3\), Wei Wang\(^1\), Jun Wang\(^4\), Yong Zhang\(^4\), Gane Ka-Shu Wong\(^4\)\(^,5\)\(^,\ast\), Stephen I. Wright\(^1\)\(^,6\), Marc T. J. Johnson\(^2\)\(^,6\)

Showy evening primrose
Marc Johnson

Proportion of genes with premature stop codons

D

\(^{\ast}\) Corresponding author.
A species having sex is not the same as a species having different sexes

- The fundamental difference between male and female function is Anisogamy (Gametes differ in size)
- Male sexual function:
  - Small mobile gametes
- Female sexual function:
  - Larger less mobile gametes
  - Maternal provisions

Male and female functions do not necessarily mean sexes are separate individuals. But separate sexes have arisen many times. Perhaps because of:
Selection for specialization or inbreeding avoidance.
Sex Determination: Why So Many Ways of Doing It?

Doris Bachsрог, Judith E. Mank, Catherine L. Peichel, Mark Kirkpatrick, Sarah P. Otto, Tia-Lynn Ashman, Matthew W. Hahn, Jun Kitano, Itay Mayrose, Ray Ming, Nicolas Perrin, Laura Ross, Nicole Valenzuela, Jana C. Vamosi, The Tree of Sex Consortium

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**Key**
- **Red**: XO, XY or complex XY
- **Blue**: ZO, ZW, or complex ZW
- **Yellow**: Homomorphic sex chromosomes or GSD
- **Green**: ESD
- **Gray**: Haplo-diploidy
- **Black**: Paternal genome elimination
- **Purple**: Hermaphroditism

**Animal-specific**
- **Monoecy**
- **Diocny**

**Plant-specific**
- **ESD**
- **Haplo-diploidy**
- **Paternal genome elimination**

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**Hermaphrodites**
- **Simultaneous hermaphrodite**
- **Sequential hermaphrodite**

**Environmental Sex Determination**
- **Temperature-dependent**
- **Social factors**

**Genotypic Sex Determination**
- **Male heterogamy**
- **Female heterogamy**
Evolution of sex chromosomes

In species with genetic sex determination the chromosomes containing the sex determining factors are often heteromorphic: One is much reduced in function and size. And does not recombine.

Human Y: 60 Mbp ~80 genes
Human X: 153 Mbp ~2000 genes

Heteromorphic sex chromosomes have evolved independently many times.
The evolution of sex chromosomes

Sex determining allele arises (e.g. dominant male determining allele)
Recombination between sexually antagonistic allele and male-determining allele
have lowers fitness.
Recombination between these loci suppressed by inversion

“Autosomal” region (Y)
Sexual Conflict Resolved by Invasion of a Novel Sex Determiner in Lake Malawi Cichlid Fishes

Reade B. Roberts, Jennifer R. Ser, Thomas D. Kocher*

“Autosomal” region (Y)
Sex determining allele arises (e.g. dominant male determining allele) 
Recombination between sexually antagonistic allele and male-determining allele have lowers fitness.
Recombination between these loci suppressed by inversion

Shutting off recombination now means that this section of Proto-Y no long recombines (note that Proto-X can recombine with itself in females)

This in turn leads to degeneration of Y sex chromosome genes due to: 
Muller’s Ratchet 
And the hitchhiking of deleterious alleles.
Accumulation of repeats and transposable elements.
Ancestral gene content in section of human Y chromosome

- Linear decay
- Exponential decay
- Exponential decay plus a constant baseline

Bellot et al 2015, Nature
The evolution of sex chromosomes has taken the same winding downhill path many times in the lineages to diverse taxa. Ranges. A close relative of *D. miranda* - common dark drosophila in the western states. The *D. pseudoobscura* autosome. The Drosophila miranda neo-Y adapters favored mutations -> "hitchhiking" deleterious mutations and genetic drift? neo-X2. will remain in males (no crossing over) they will Since the genes on this segment of the genome the old Y to produce a neo-Y (and a second X). About 1 MYA one of the autosomes fused with a larger neo-Y - chromosome translocation the pseudoautosomal regions the X-transposition regions - mini-neo-Y. Background selection? Muller's Ratchet? reduced levels of polymorphism. accumulation of "deleterious" mutations. increase rates of divergence, esp. nonsyn.

What is the population genetic process of Y do#not#experience#recombina##tion##? The autosome fused to the Y does not experience recombination

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A neo-Y chromosome region was formed in *Drosophila miranda* 1.25 Myrs ago and has rapidly decayed.

Majority of genes (open reading frames, ORFs) in neo-Y region have become non-functional due to:
- Muller’s Ratchet
- The hitchhiking of deleterious alleles.
- Accumulation of repeats and transposable elements.
The evolution of sex chromosomes

Convergent evolution and different ages of sex chromosomes

**Corresponding X**

**Non-recombining Y (gene poor)**

Asparagus

Cannabis also has old XY

- ~2 million years old
- ~10 million years old
- ~20 million years old
The evolution of sex chromosomes

Our own X and Y have different evolutionary strata corresponding to different ages that recombination ceased between X and Y (different inversions & transpositions).

Genes on Y in these different strata are in different states of decay.

Bergero, Charlesworth 2009