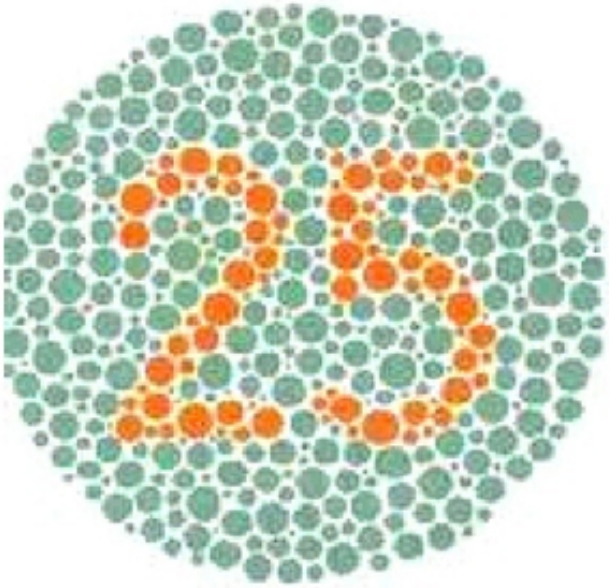
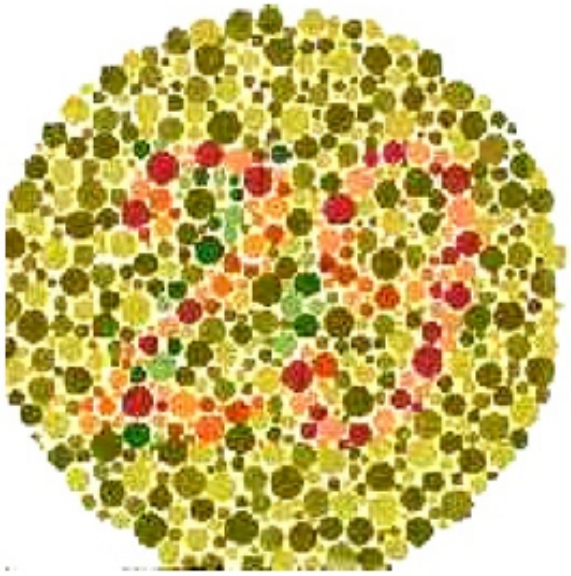
BIOL2107, Fall ’23

Lecture 14

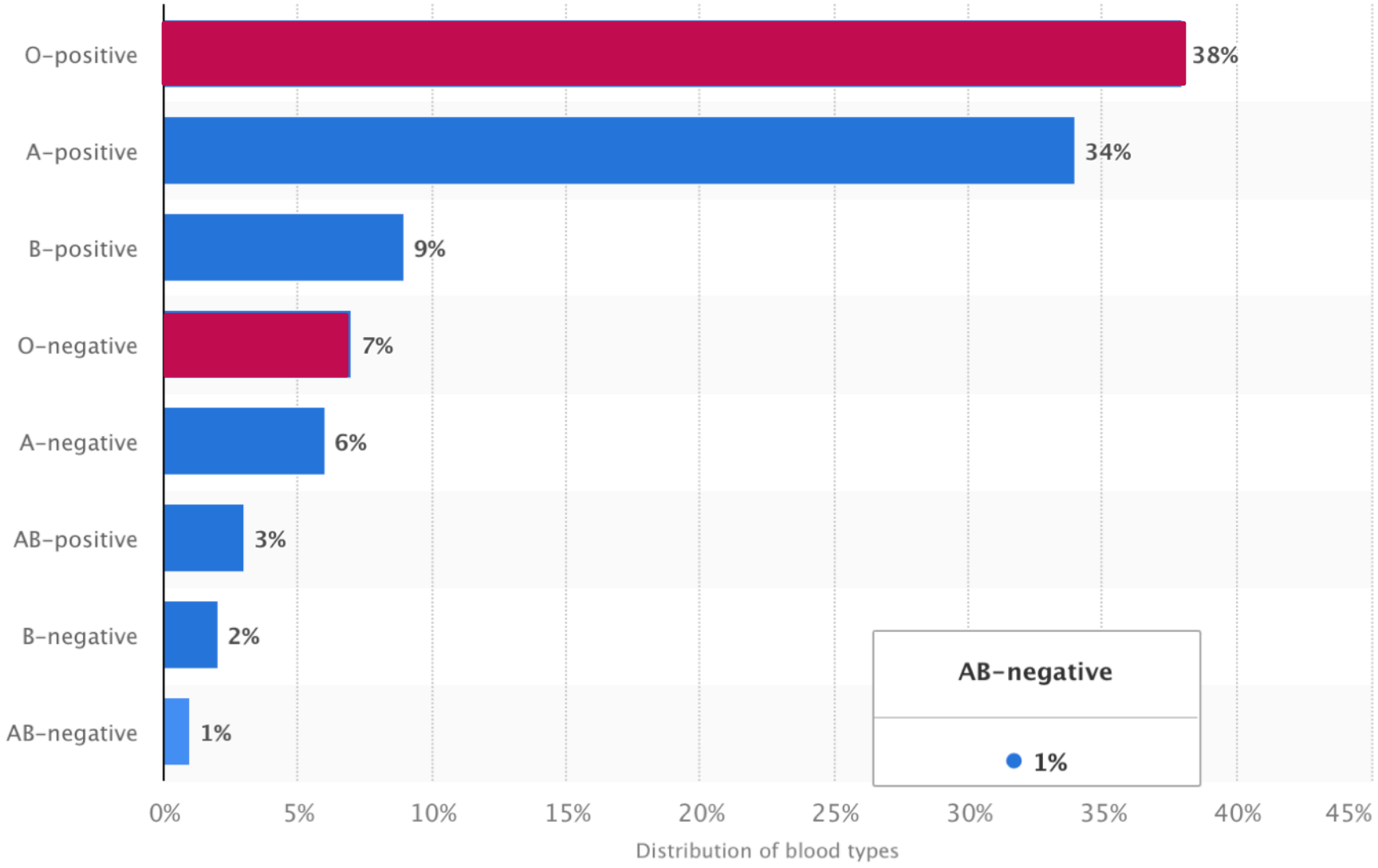




Extensions of Mendelian Genetics

Incomplete dominance:	**eg.** Four o' clocks, carnations. **BLENDING 1 : 2 : 1**
Codominance:	**eg.** M and N blood groups on chromosome 4 having specific antigens M and N **1 : 2 : 1** but where the heterozygotes (MN) gives rise to a distinct phenotype
Multiple alleles	ABO blood types, **A** and **B** are dominant to **O** , but **A** and **B codominant** to each other
Epistasis	essentially **"eliminates"** or **"masks"** phenotypic expression of other genes, **eg.** Labrador dogs fur colour**,** albinoism in mice
Lethal alleles	**eg.** Yellow colouration in mice fur. **2 : 1** . **Pleiotropy(?)**
Several genes / same character	Coat colour in mammals, **eg.** mice A (agouti), B (black/brown), C, (colour) D (intensity), S. (distribution) genes
Complementary gene activity	**eg.** Pea plants, purple colouration. **9 : 7**
Duplicate gene activity	**eg.** Shepherd's purse, Round over narrow fruits, where both A_1 - and A_2 - can cause heart shape **15 : 1**

AVERAGE DISTRIBUTION OF BLOOD TYPES IN US



Rhesus monkey

NOT

Reeses chocolate

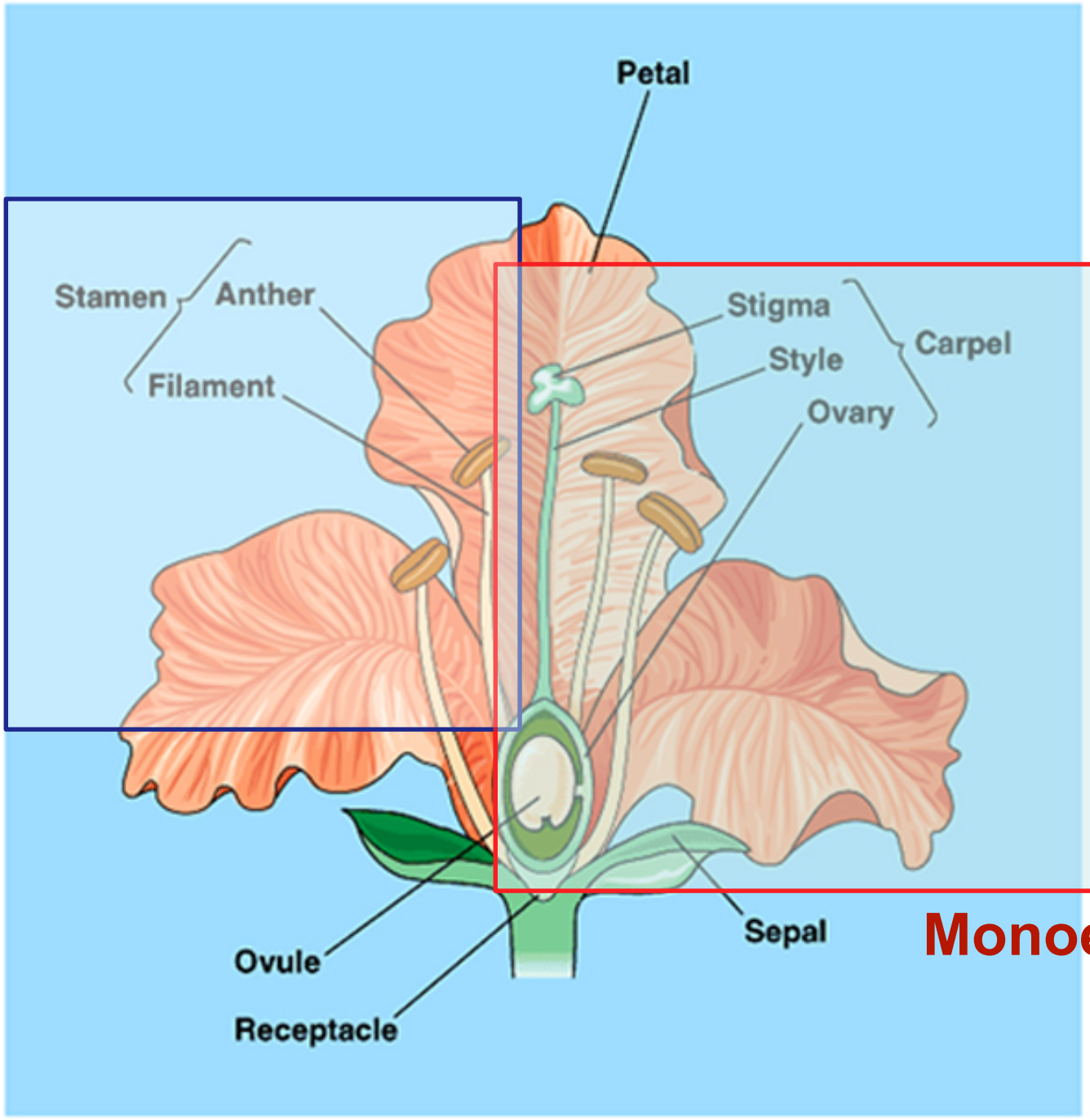
What about **Rhesus factors (Rh)**?… these are a whole new class of antigens and are quite distinct from the ABO factors, discovered when blood from "rhesus monkeys" was injected into guinea pigs (circa 1940's). There are over 50 different types of similar Rh factors in humans, but the most commonly known one is the **D antigen** (**Rho[D]**), which -if it is present- indicates that that person is **Rh-positive**; if the D antigen is absent, that person is **Rh-negative**.

85% of people are Rh-positive.

The rhesus (Rh) state only really begins to play a role during pregnancy
if the mother is Rh-negative, the father is Rh-positive **and** the baby is also Rh-positive, then the mother can be induced to produce the 'anti' D antibody…

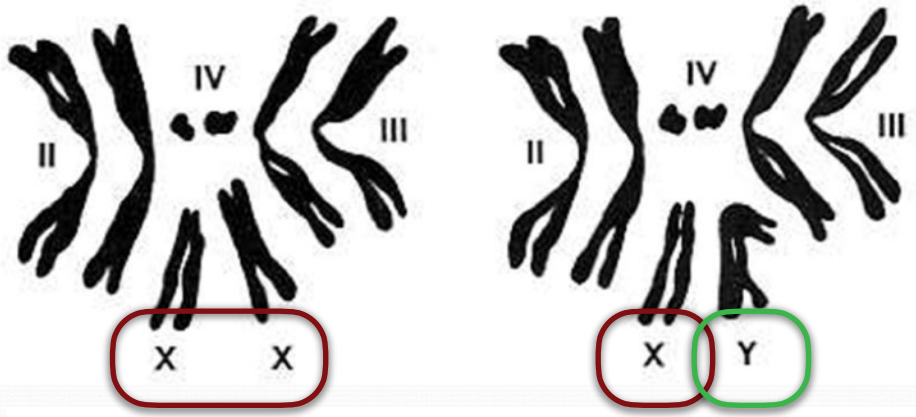
NO problems with the first pregnancy.

Major complications could occur, however, with a similar 2nd pregnancy as the Rh antigenic response has already been activated in the mother.


Petal
Stamen
Anther
Filament
Stigma
Style
Ovary
Carpel
Sepal
Ovule
Receptacle

Monoecious

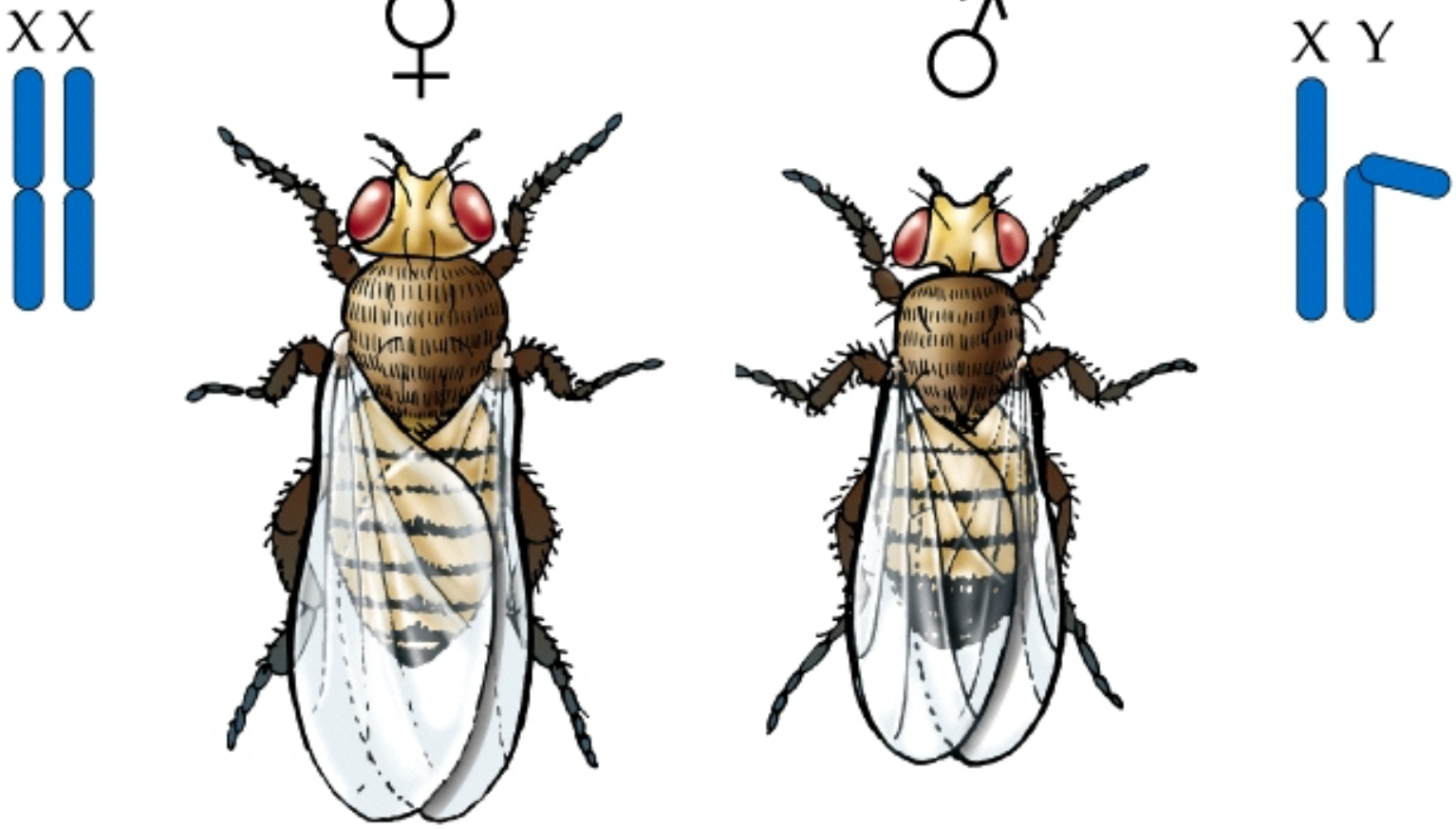
Dioecious



Drosphila melanogaster or "fruit fly"

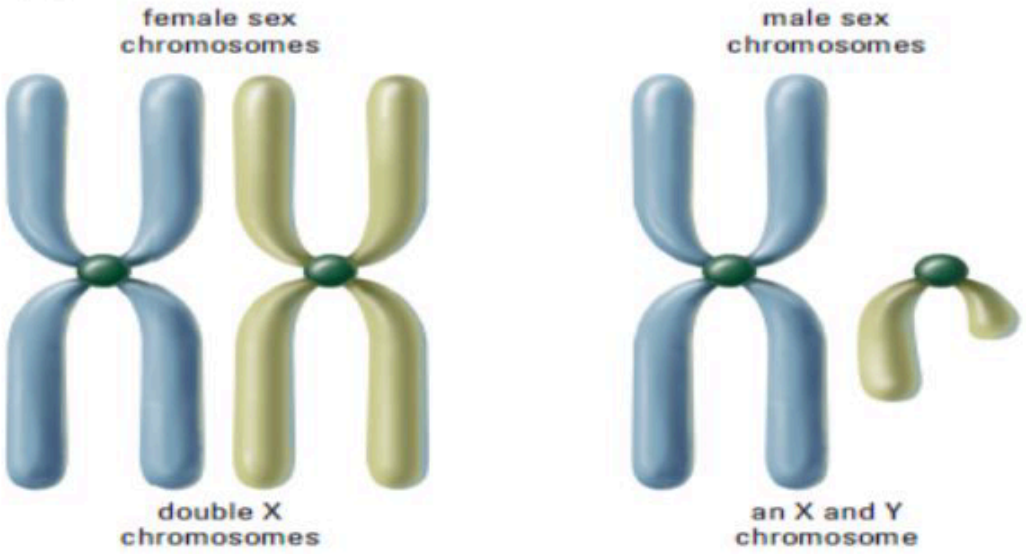
Dioecious

Homo sapiens or "humans"



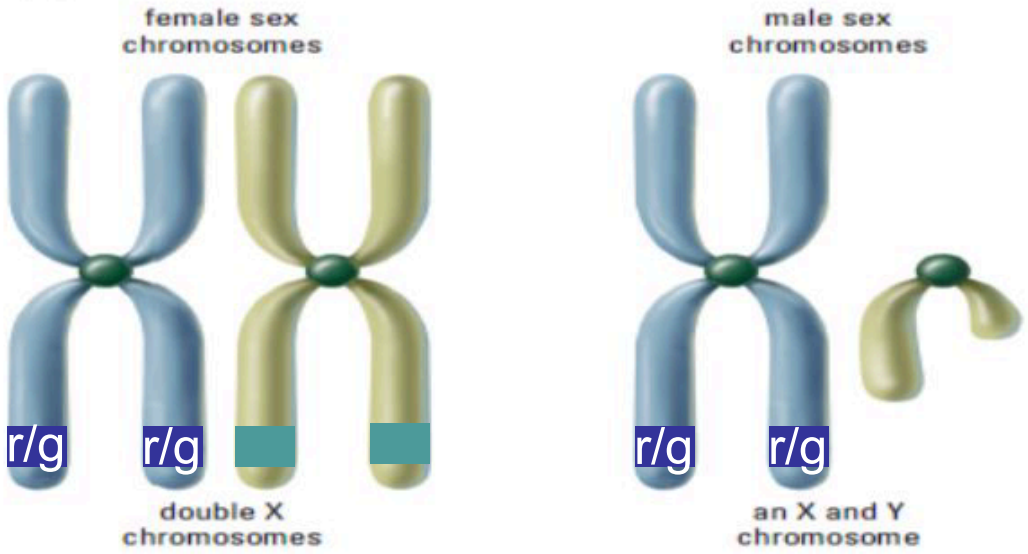
Dioecious

autosomes: the chromosomes not involved in sex determination



sex chromosomes: the pair of chromosomes that have a role in the sex of an individual

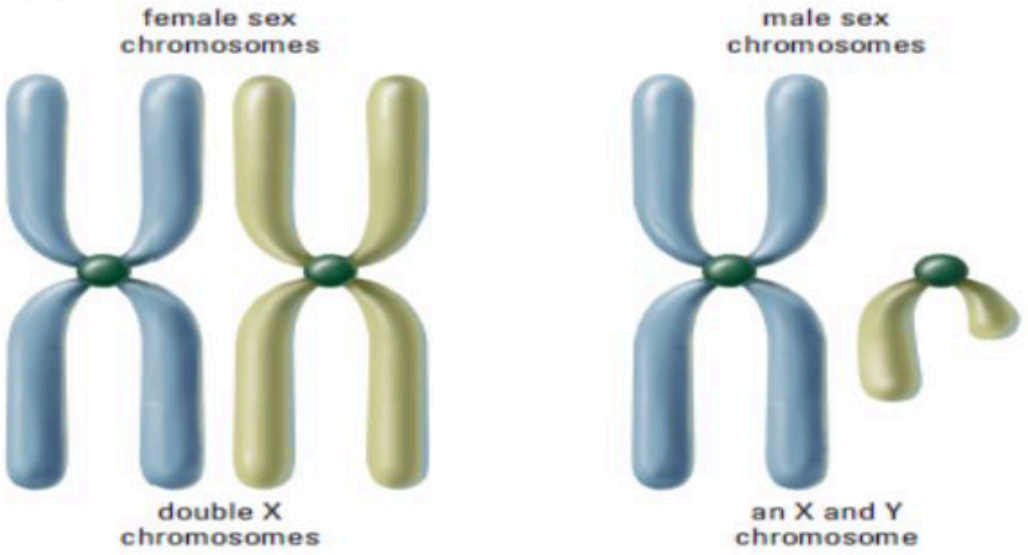
Red Green Colour blindness

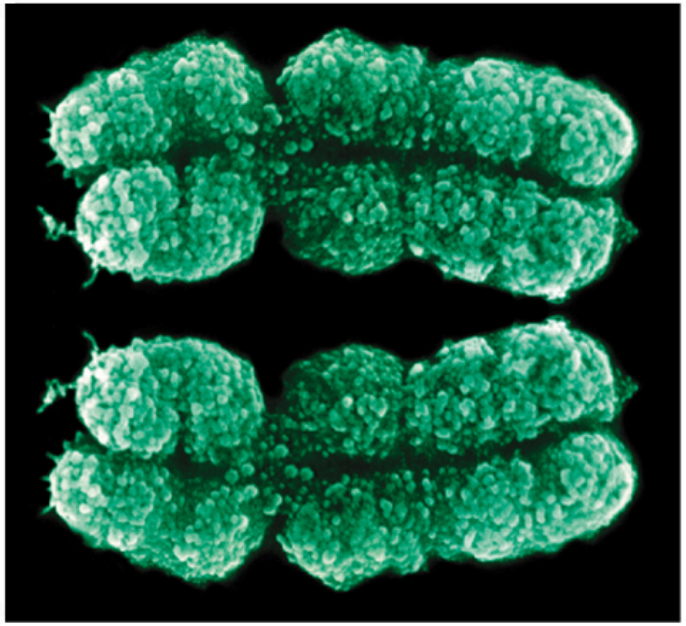


sex chromosomes: the pair of chromosomes that have a role in the sex of an individual

All genes that are present on the X-chromsome, demonstrate a genetic phenomenon called…

X-linkage

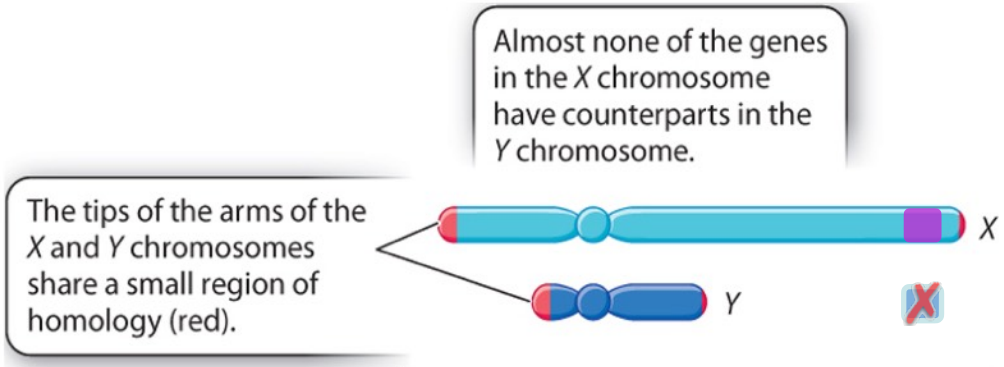


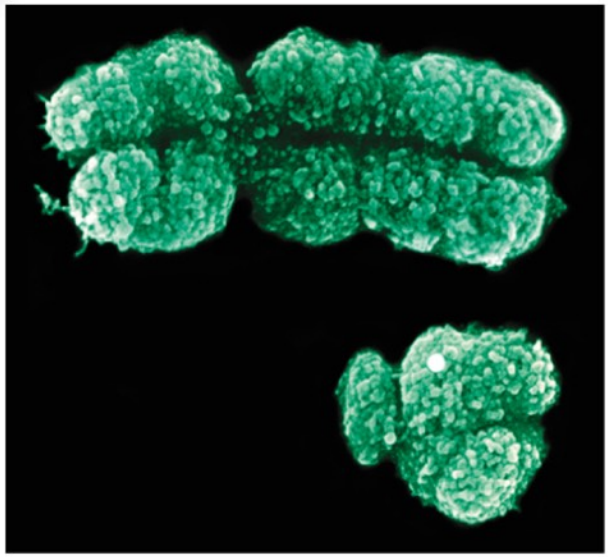


So while a females can carry an X- linked trait, if it is recessive- the other X chromosome would probably not, and it's expression would DOMINATE giving a WT phenotype.

Hence Females can often be carriers of an X- linked trait, but rarely demonstrate the phenotype.

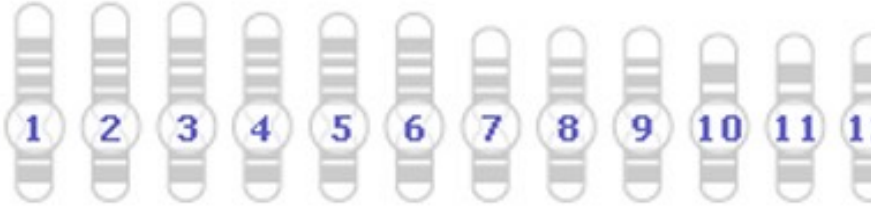
Giving rise to the following inheritable signs for **X- Linkage**





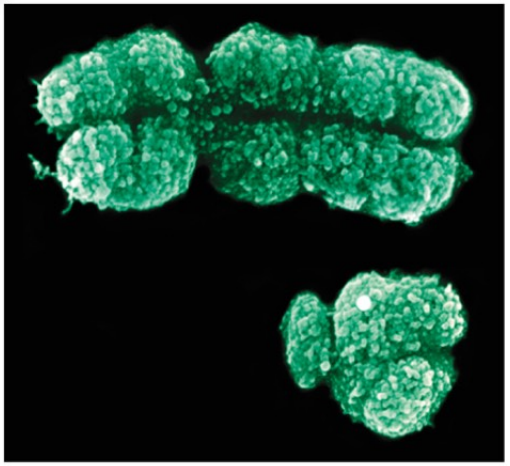
For Males it's a different story, if the X chromosome carries the trait… there is NO compensating X chromosome to help hide the trait, and if it is present it WILL ALWAYS SHOW THROUGH

human

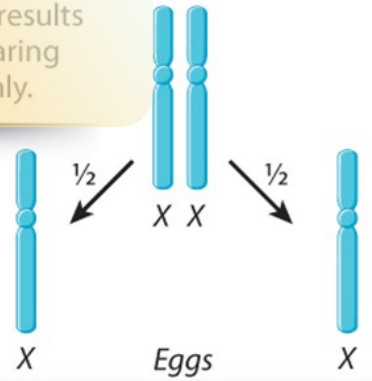


Almost none of the genes in the *X* chromosome have counterparts in the *Y* chromosome.

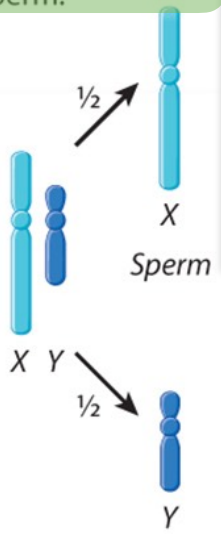
The tips of the arms of the *X* and *Y* chromosomes share a small region of homology (red).

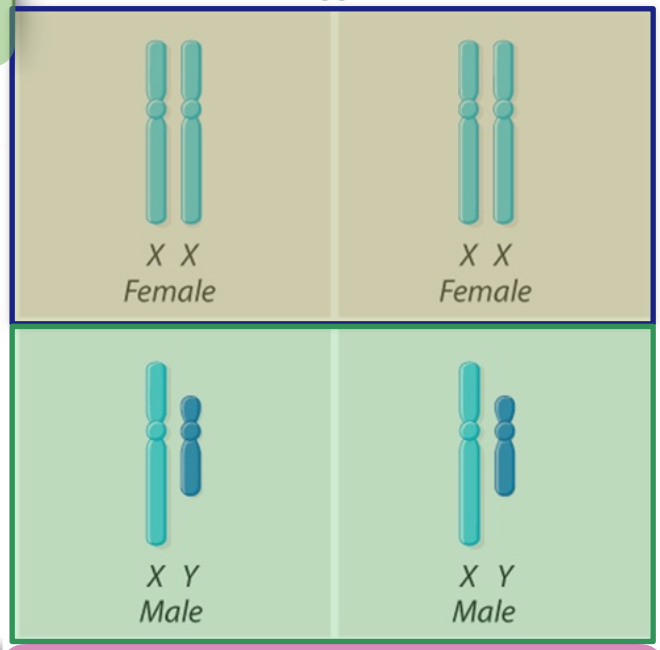


Meiosis in a female results in *X*-bearing eggs only.



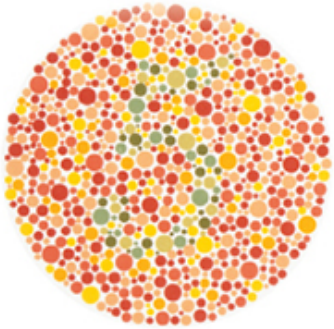
Meiosis in a male results in a 1:1 ratio of *X*-bearing and *Y*-bearing sperm.





Random fertilization results in an expected ratio of ½ *XX* (female) and ½ *XY* (male) progeny.

People with red–green color blindness cannot see the number 5 in this figure.

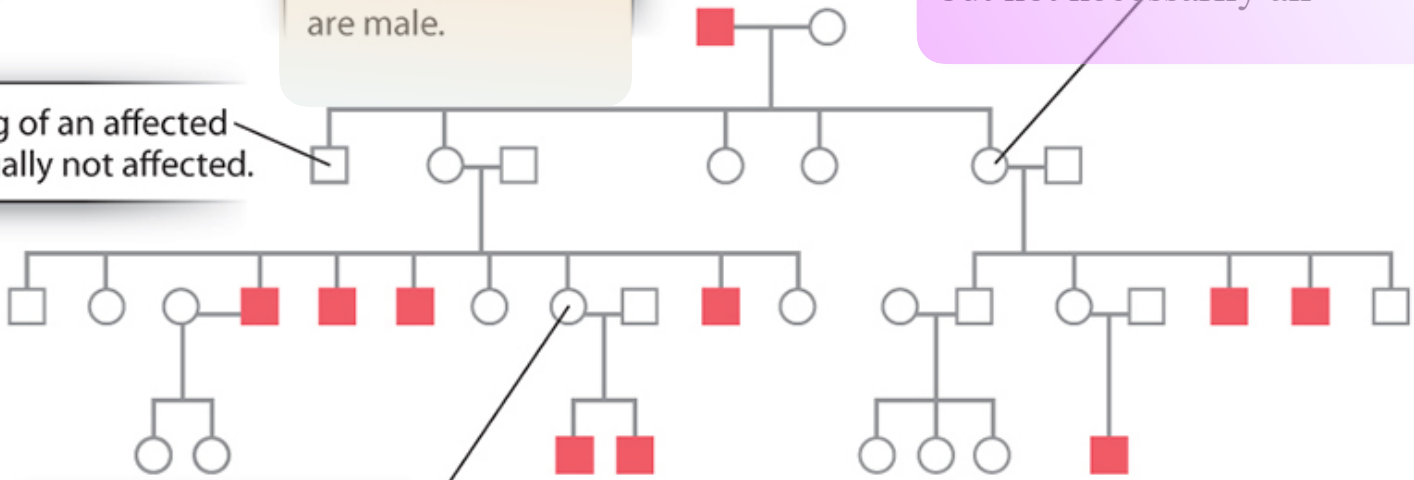


Unaffected male
Unaffected female
Affected male

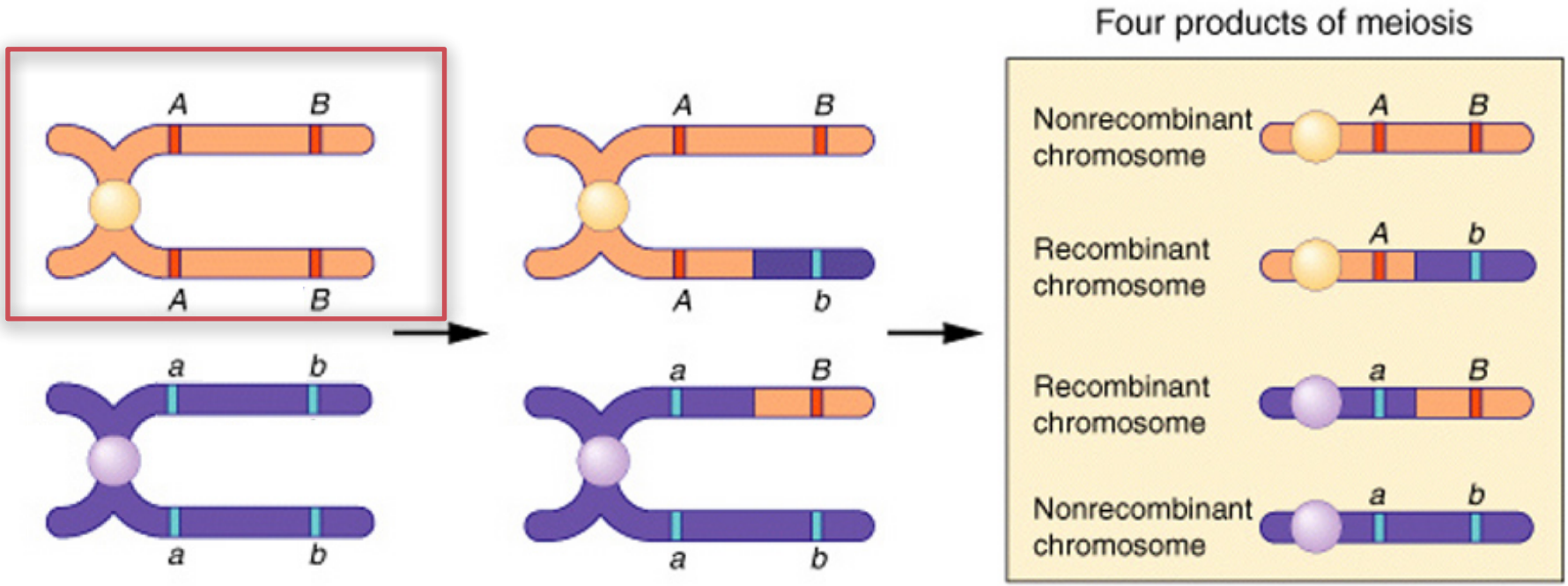
For a rare *X*-linked recessive trait, most affected individuals are male.

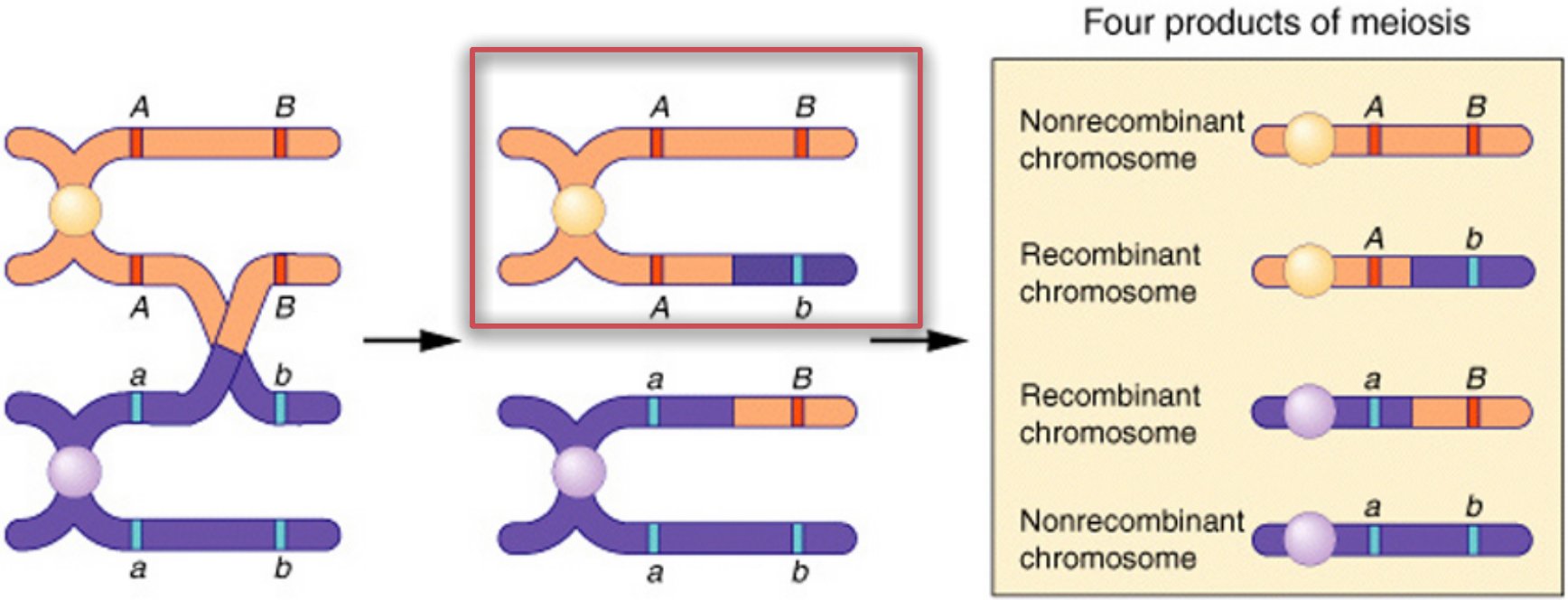
The daughters of affected males can have affected sons.
but not necessarily all

The offspring of an affected male are usually not affected.



The sisters of an affected male can have affected sons.


A
B
A
B
a
b
a
b
A
B
A
b
a
B
a
b
Four products of meiosis
Nonrecombinant chromosome
A
B
Recombinant chromosome
A
b
Recombinant chromosome
a
B
Nonrecombinant chromosome
a
b


A
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A
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A
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B
a
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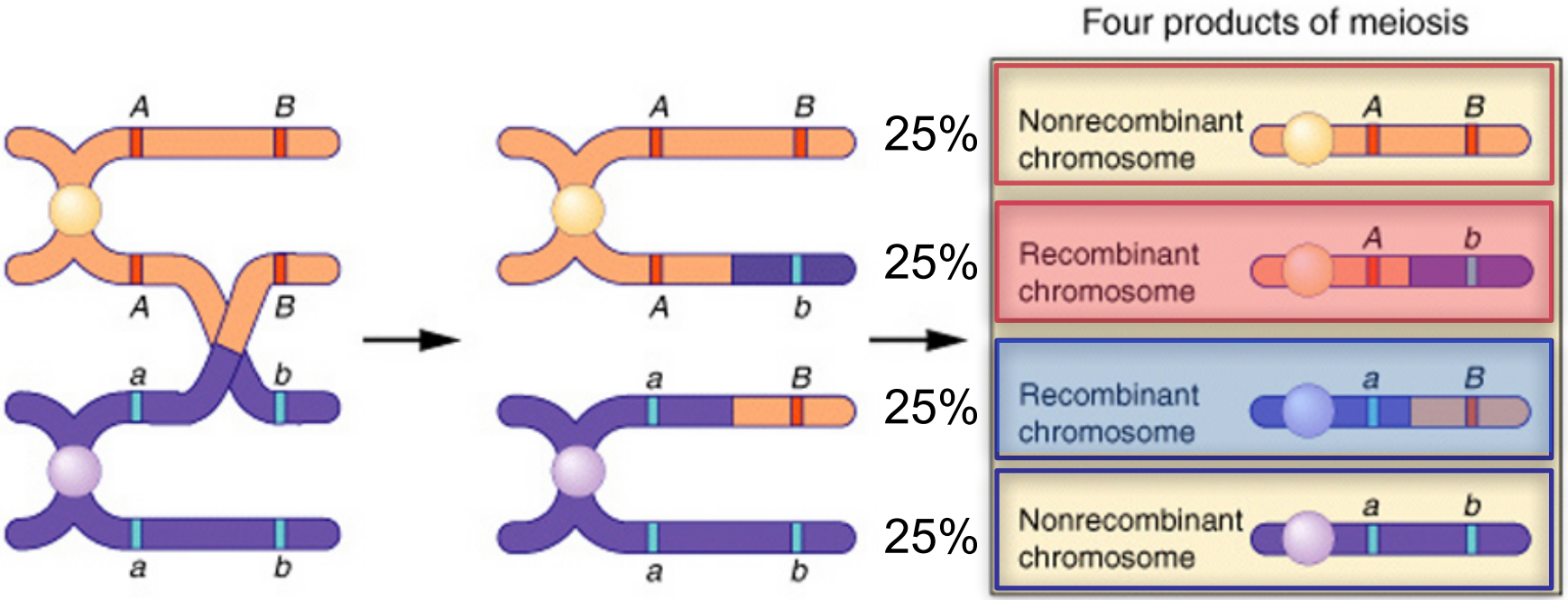
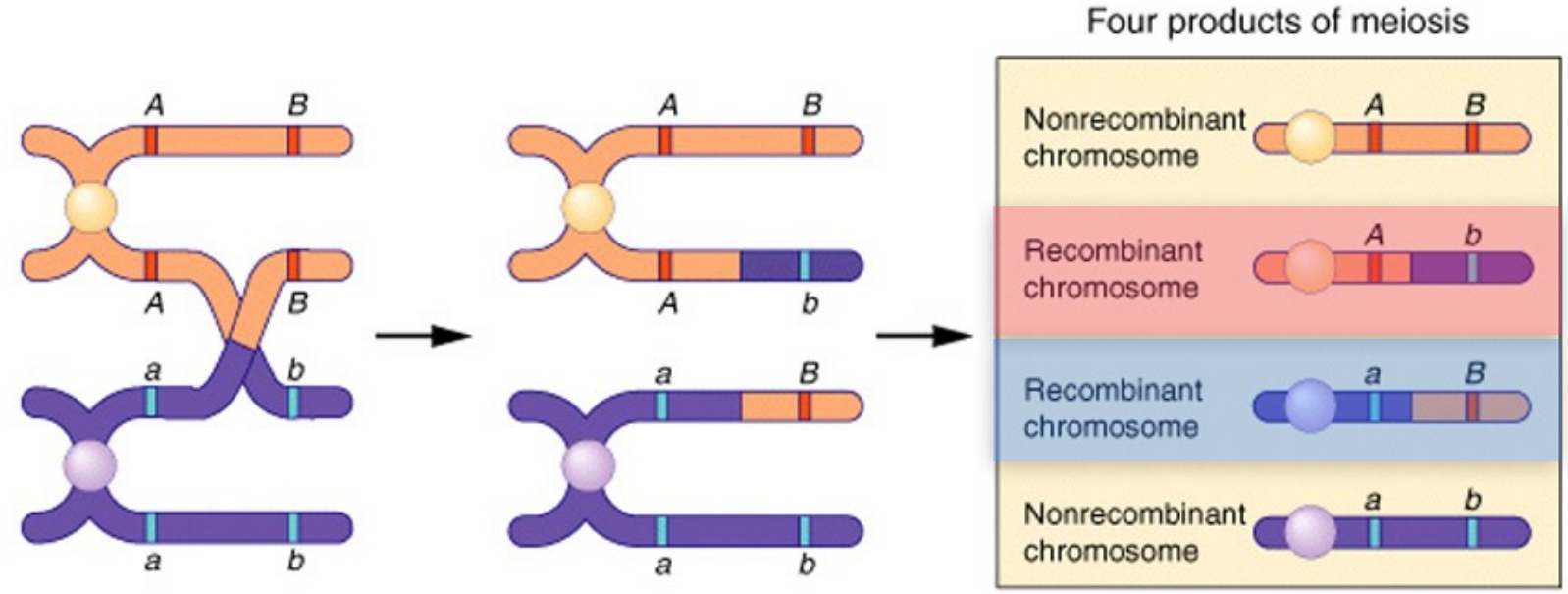
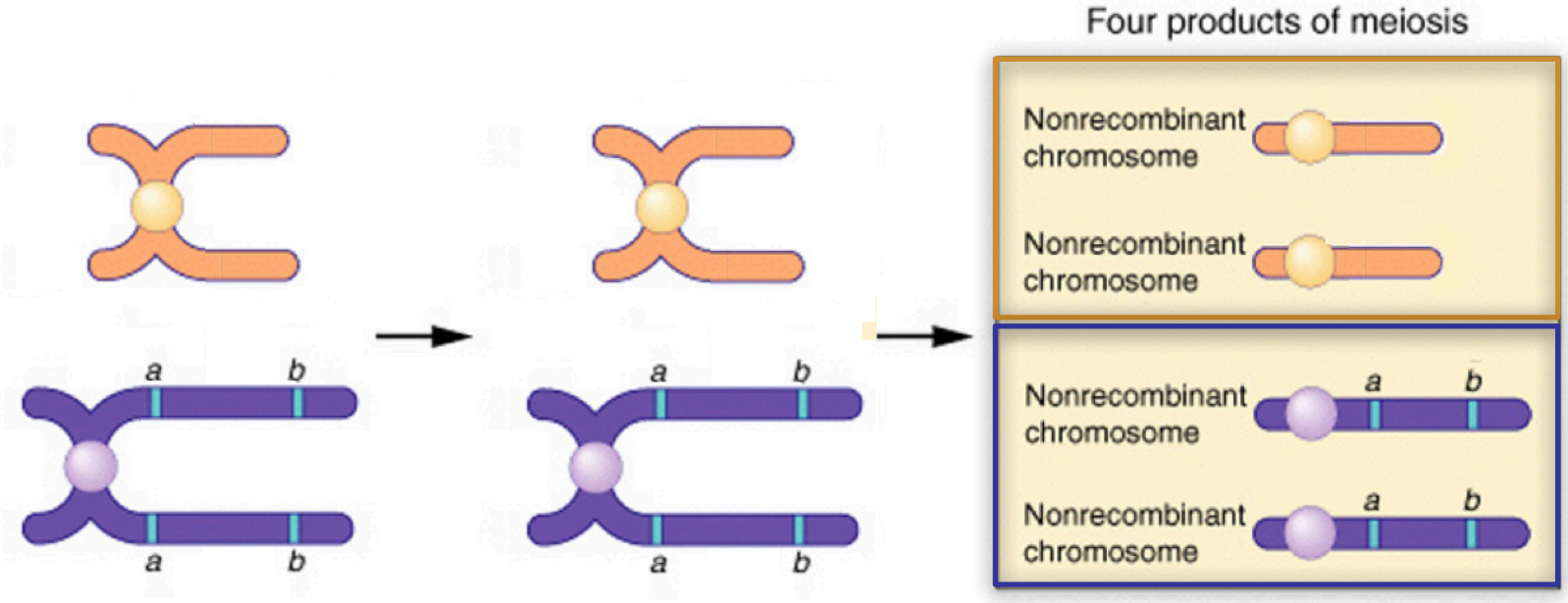
Four products of meiosis
Nonrecombinant chromosome
A
B
Recombinant chromosome
A
b
Recombinant chromosome
a
B
Nonrecombinant chromosome
a
b

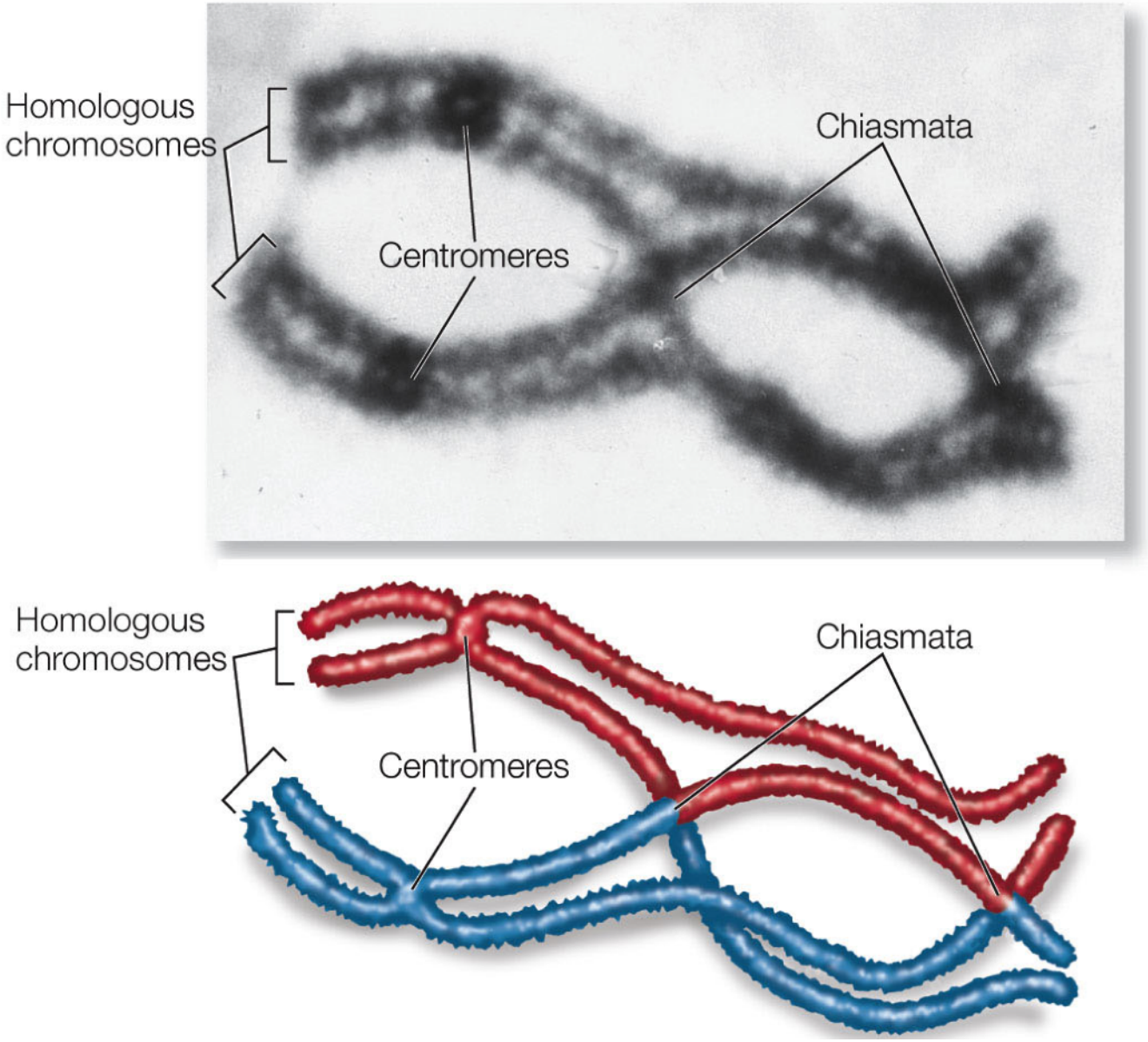


Full agreement with Mendel's 2nd law

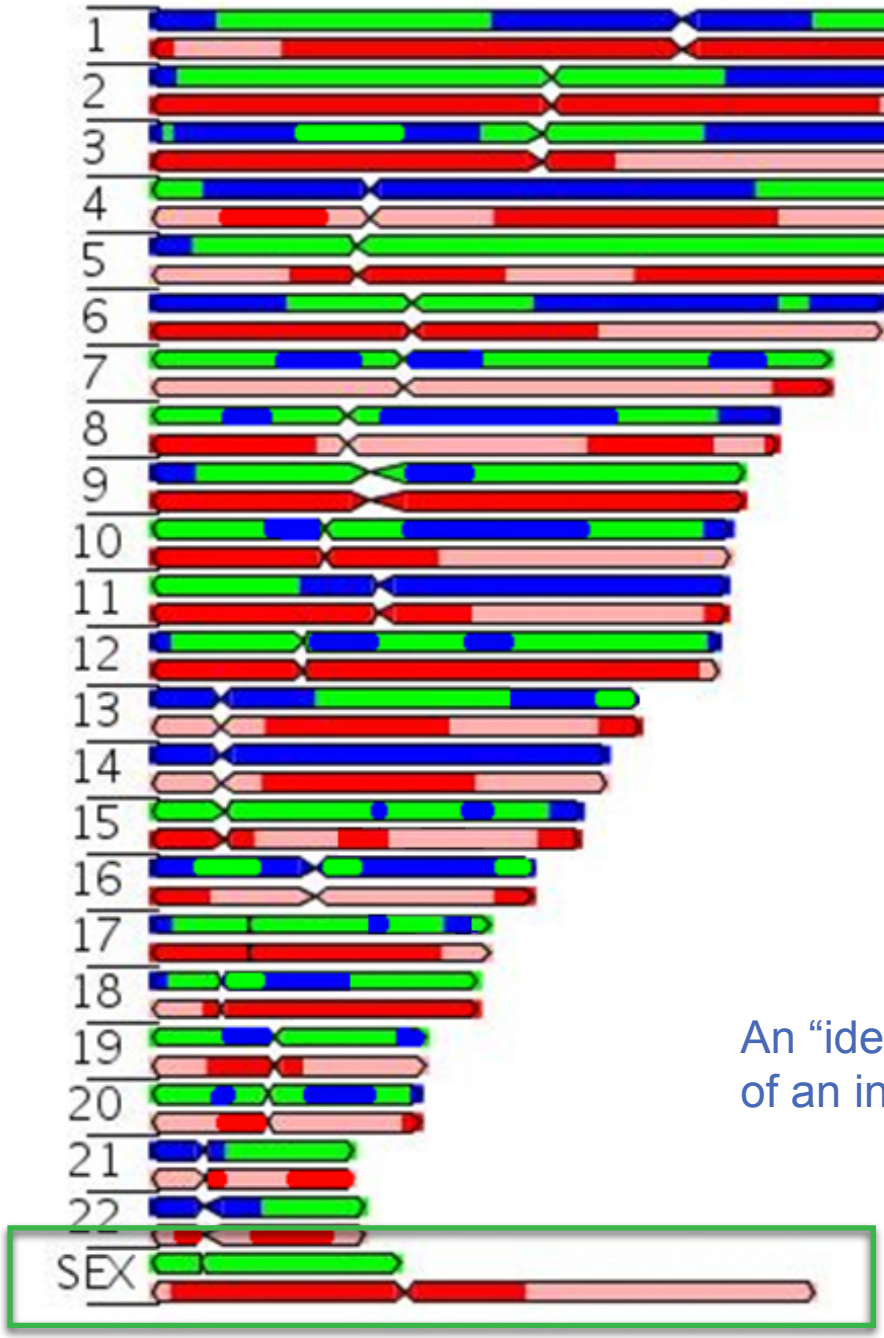




NO RECOMBINANTS with X and Y chromosomes



Chiasmata formation requires homology between the two homologous chromosomes



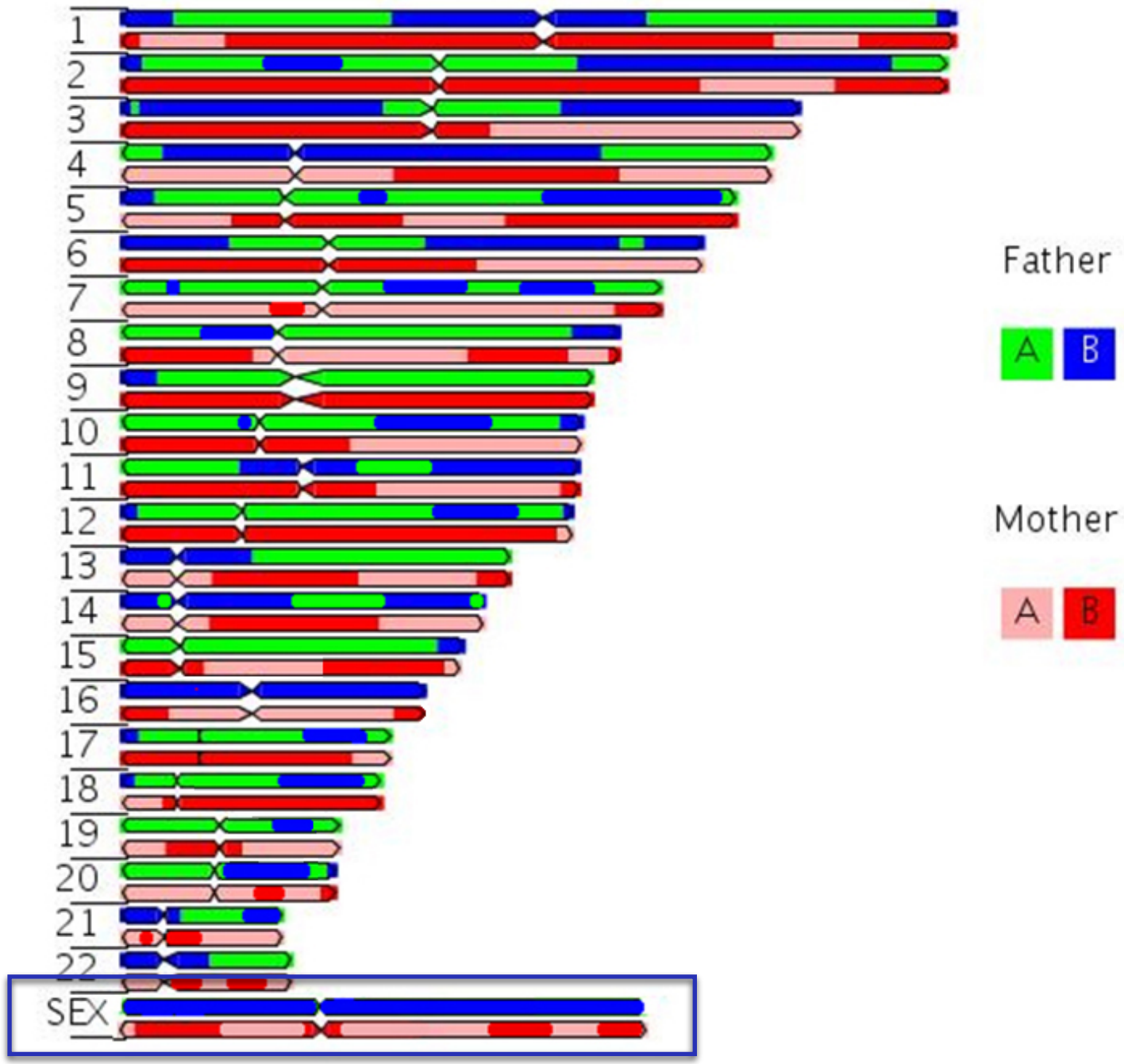
Father

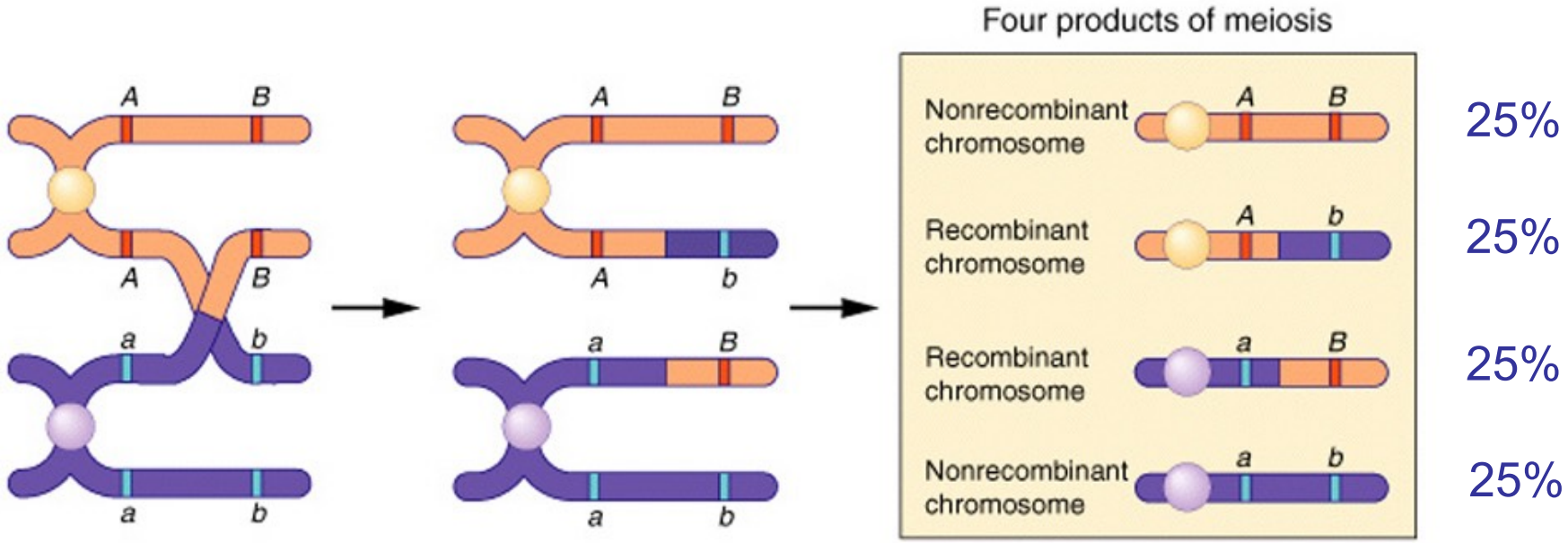
A B

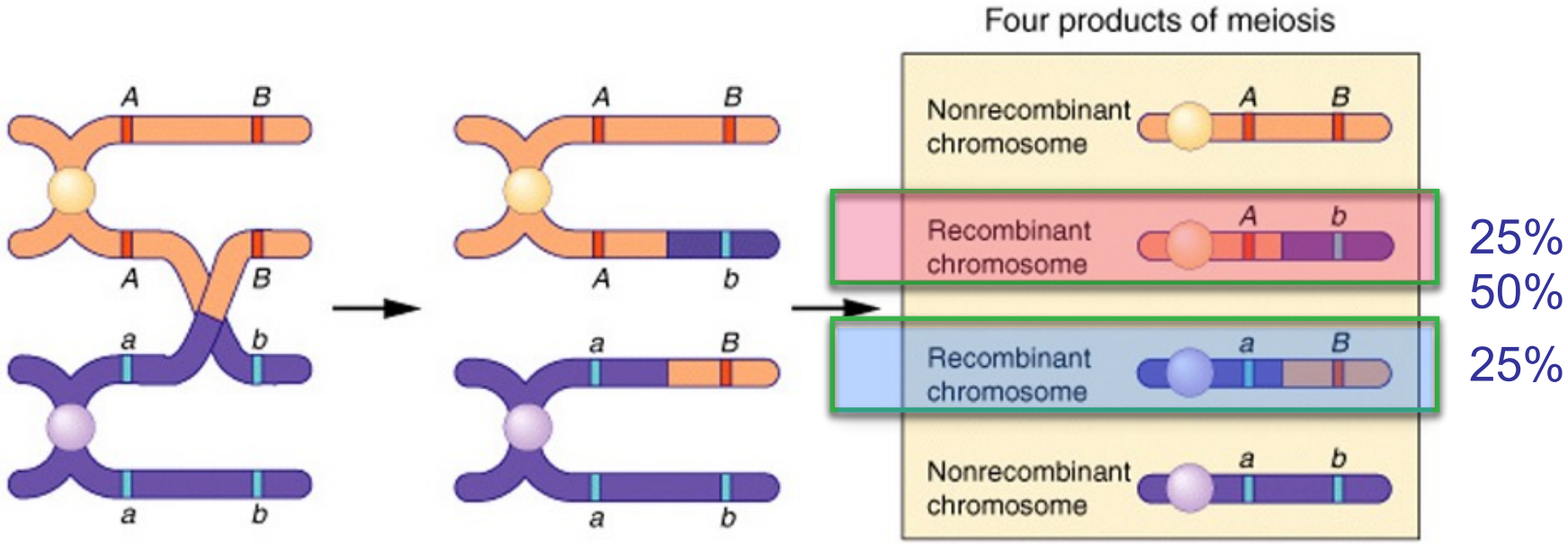
Mother

A B

An “ideogram” shows recombinatory origins of an individual’s chromosomes


1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18
19
20
21
22
SEX
Father
A
B
Mother
A
B


A
B
A
B
a
b
a
b
A
B
A
b
a
B
a
b
Four products of meiosis
Nonrecombinant chromosome
A
B
25%
Recombinant chromosome
A
b
25%
Recombinant chromosome
a
B
25%
Nonrecombinant chromosome
a
b
25%


A
B
A
B
a
b
a
b
A
B
A
b
a
B
a
b
Four products of meiosis
Nonrecombinant chromosome
A
B
Recombinant chromosome
A
b
Recombinant chromosome
a
B
Nonrecombinant chromosome
a
b
25%
50%
25%

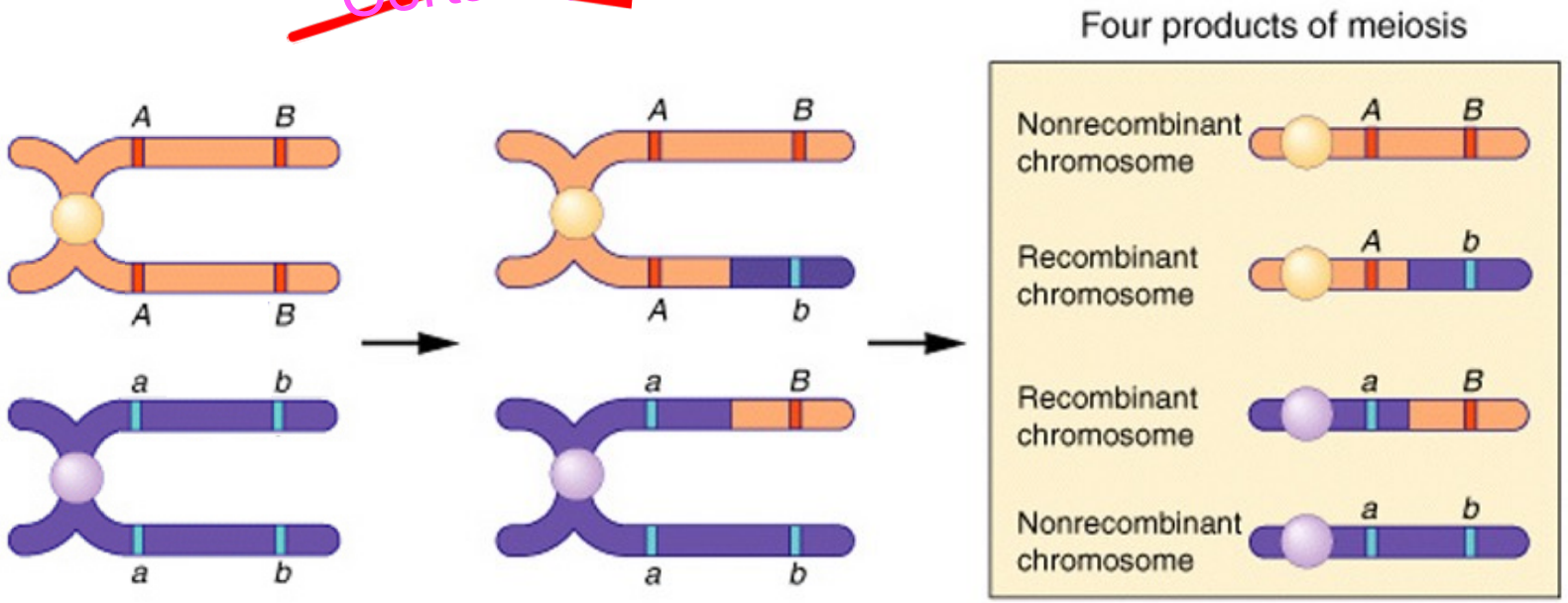
Mendel's Laws

Chiasma / Chiasmata

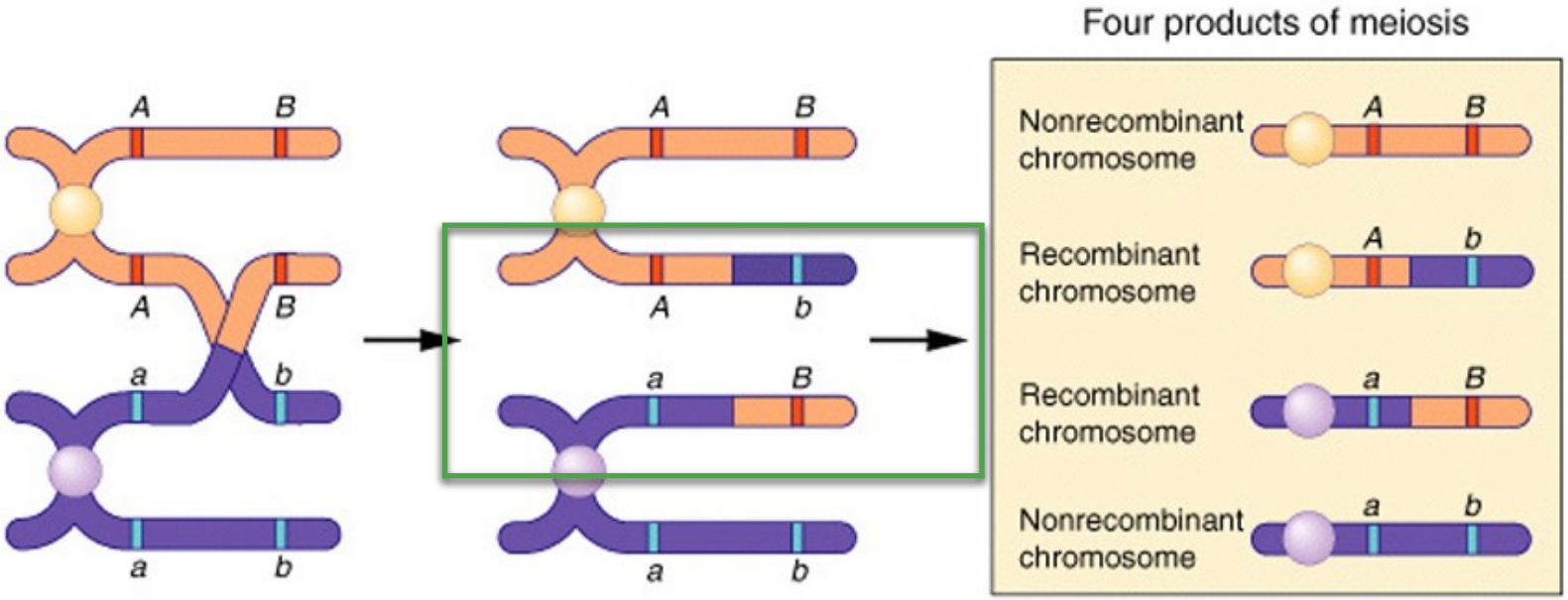
Certainty…

Probability = 1

~~Certainty…~~



Probability < 1


Four products of meiosis
A
B
A
B
a
b
a
b
A
B
A
b
a
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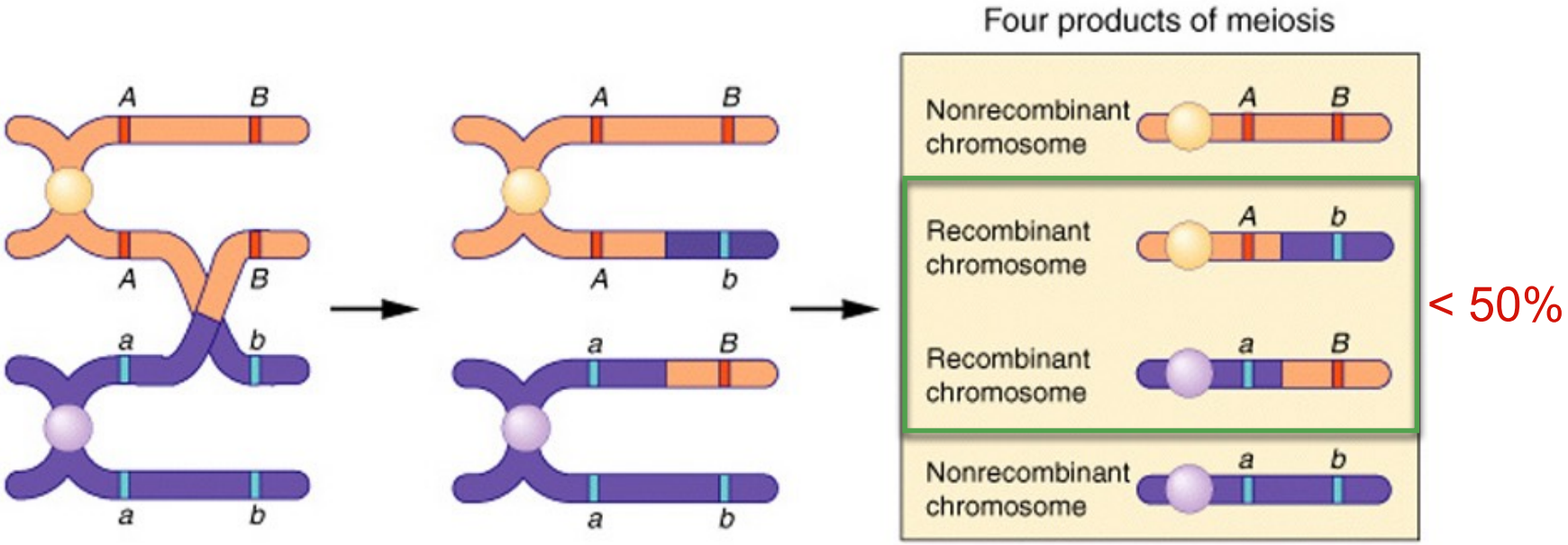
Nonrecombinant chromosome
A
B
Recombinant chromosome
A
b
Recombinant chromosome
a
B
Nonrecombinant chromosome
a
b


A
B
A
B
a
b
a
b
A
B
A
b
a
B
a
b
Four products of meiosis
Nonrecombinant chromosome
A
B
Recombinant chromosome
A
b
Recombinant chromosome
a
B
Nonrecombinant chromosome
a
b
< 50%

Thomas Hunt Morgan

From Wikipedia, the free encyclopedia

For other people named Thomas Morgan, see Thomas Morgan (disambiguation).

Thomas Hunt Morgan (September 25, 1866 – December 4, 1945)[2] was an American evolutionary biologist, geneticist, embryologist, and science author who won the Nobel Prize in Physiology or Medicine in 1933 for discoveries elucidating the role that the chromosome plays in heredity.[3]

Morgan received his Ph.D. from Johns Hopkins University in zoology in 1890 and researched embryology during his tenure at Bryn Mawr. Following the rediscovery of Mendelian inheritance in 1900, Morgan began to study the genetic characteristics of the fruit fly *Drosophila melanogaster*. In his famous Fly Room at Columbia University, Morgan demonstrated that genes are carried on chromosomes and are the mechanical basis of heredity. These discoveries formed the basis of the modern science of genetics.

During his distinguished career, Morgan wrote 22 books and 370 scientific papers.[2] As a result of his work, *Drosophila* became a major model organism in contemporary genetics. The Division of Biology which he established at the California Institute of Technology has produced seven Nobel Prize winners.

Contents [hide]

Early life and education [edit]

Morgan was born in Lexington, Kentucky, to Charlton Hunt Morgan and Ellen Key Howard Morgan.[3][4] Part of a line of Southern plantation and slave owners on his father's side, Morgan was a nephew of Confederate General John Hunt Morgan; his great-grandfather John Wesley Hunt had been the first millionaire west of the Allegheny Mountains. Through his mother, he was the great-grandson of Francis Scott Key, the author of the "Star Spangled Banner", and John Eager Howard, governor and senator from Maryland.[4] Following the Civil War, the family fell on hard times with the temporary loss of civil and some property rights for those who aided the Confederacy. His father had difficulty finding work in politics and spent much of his time coordinating veterans reunions.

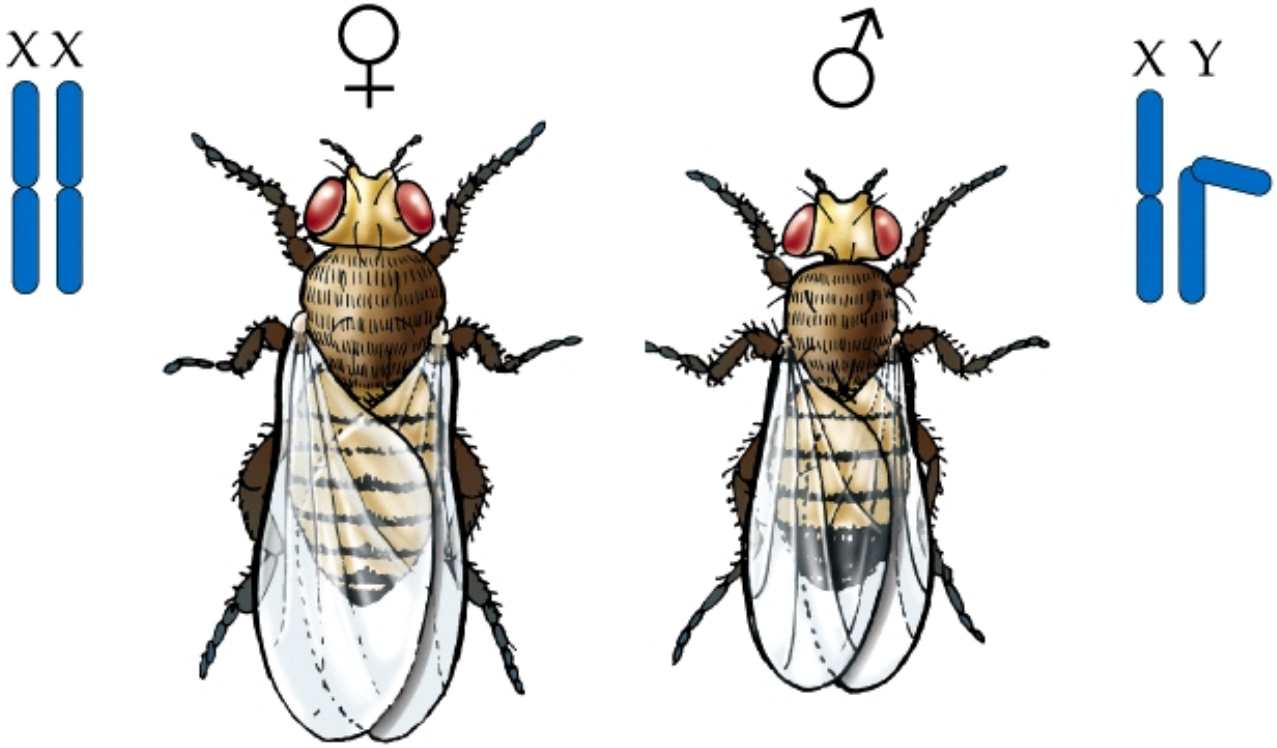
Beginning at age 16 in the Preparatory Department, Morgan attended the State College of Kentucky (now the University of Kentucky). He focused on science; he particularly enjoyed natural history, and worked with the U.S. Geological Survey in his summers. He graduated as valedictorian in 1886 with a Bachelor of Science degree.[5] Following a summer at the Marine Biology School in Annisquam, Massachusetts, Morgan began graduate studies in zoology at the recently founded Johns Hopkins University, the first research-oriented American university. After two years of experimental work with morphologist William Keith Brooks and writing several publications, Morgan was eligible to receive a master of science from the State College of Kentucky in 1888. The college required two years of study at another institution and an examination by the college faculty.[*citation needed*] The college offered Morgan a full professorship; however, he chose to stay at Johns Hopkins and was awarded a relatively large fellowship to help him fund his studies.[*citation needed*]

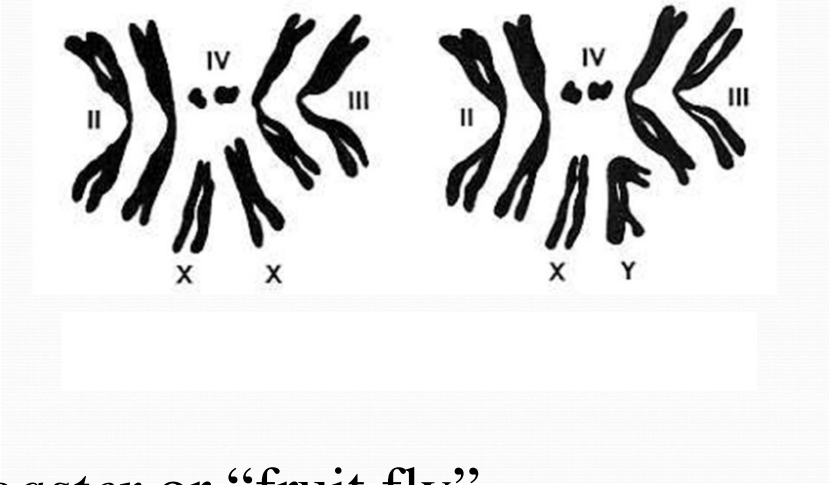
Under Brooks, Morgan completed his thesis work on the embryology of sea spiders—collected during the summers of 1889 and 1890 at the Marine Biological Laboratory in Woods Hole, Massachusetts—to determine their phylogenetic relationship with other arthropods. He concluded that with respect to embryology, they were more closely related to spiders than crustaceans. Based on the publication of this work, Morgan was awarded his Ph.D. from Johns Hopkins in 1890, and was also awarded the Bruce Fellowship in Research. He used the fellowship to travel to Jamaica, the Bahamas and to Europe to conduct further research.[6]

Thomas Hunt Morgan
ForMemRS

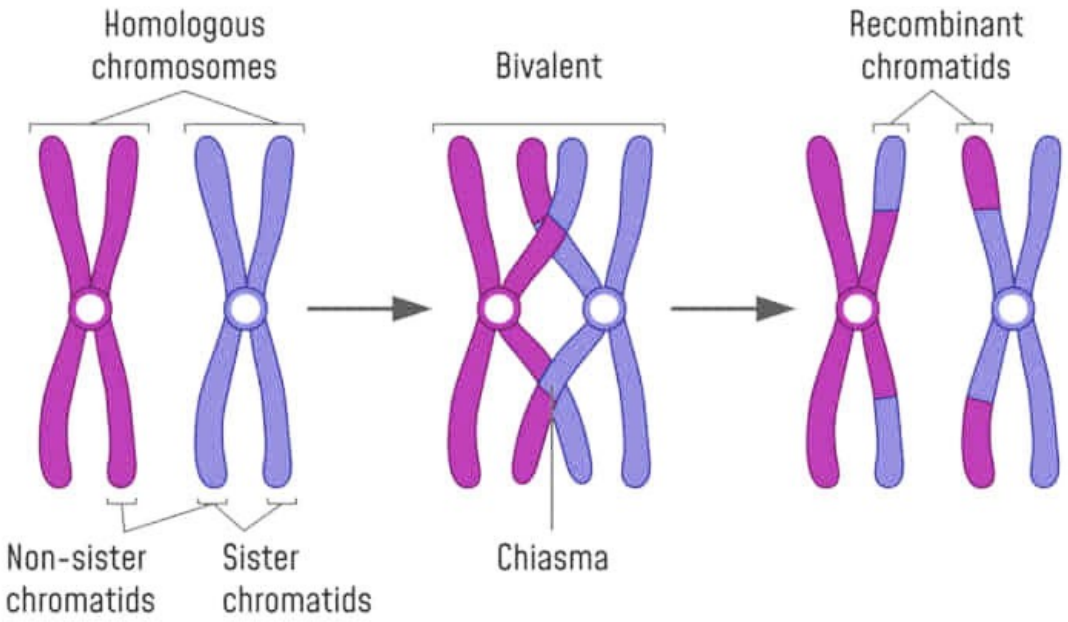
Johns Hopkins yearbook of 1891

Born	September 25, 1866 Lexington, Kentucky
Died	December 4, 1945 (aged 79) Pasadena, California
Nationality	United States
Alma mater	University of Kentucky (B.S.) Johns Hopkins University (Ph.D.)
Known for	Establishing *Drosophila melanogaster* as a major model organism in genetics Linked genes
Awards	Member of the National Academy of Sciences (1909)[1] Foreign Member of the Royal Society (1919)[2] Nobel Prize in Physiology or Medicine (1933) Copley Medal (1939)
Scientific career	
Fields	Genetics Embryology





Drosphila melanogaster or “fruit fly”

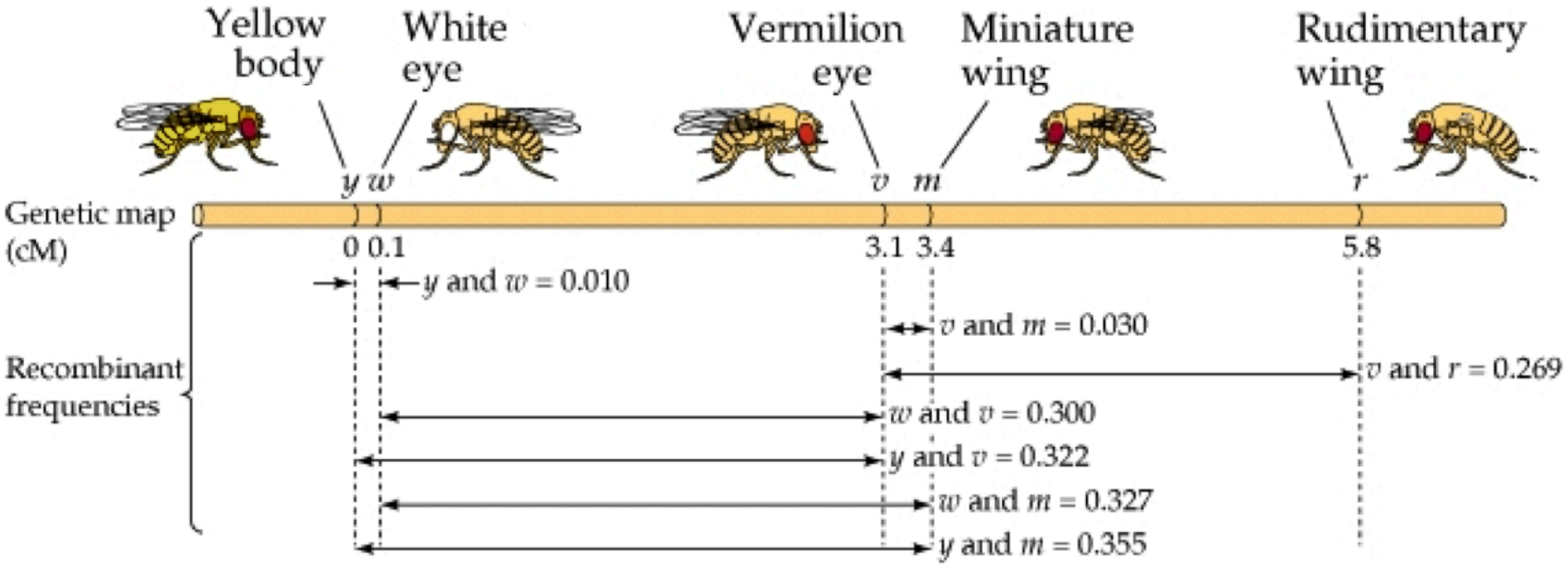


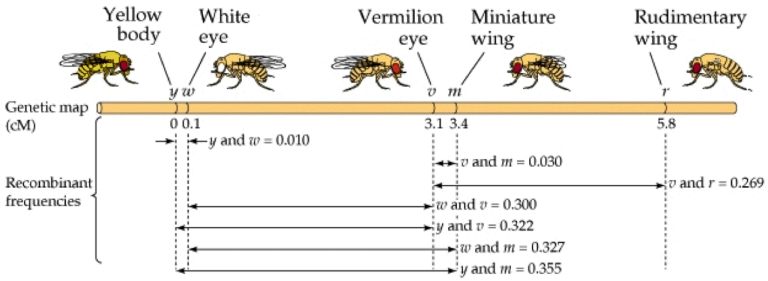
Morgan went further. He proposed that the frequency of cross-over events (occurring between two gene pairs) was a **function of the genetic distance** between the two loci.... ONLY if the gene pairs were relatively quite close to each other on the chromosome.

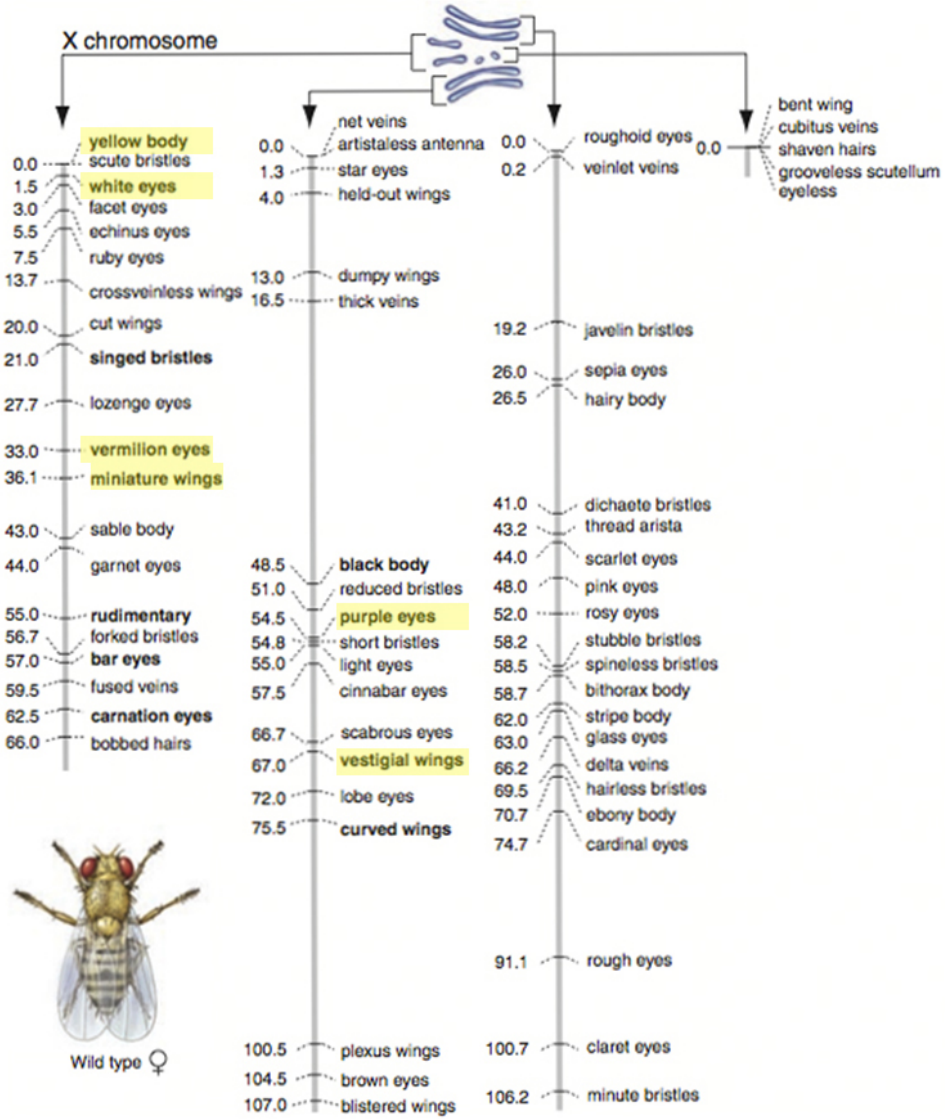
He correlated the **frequency of crossing over** between genes that are located very close to each other on the same chromosome with their actual distance apart…

He thus defined the unit of genetic distance as being:

one **crossover event/100 products of meiosis** = one map unit or 1 **centiMorgan (cM)**.


Yellow body
White eye
Vermilion eye
Miniature wing
Rudimentary wing
y w
v m
r
Genetic map (cM)
0 0.1
3.1 3.4
5.8
Recombinant frequencies
y and w = 0.010
v and m = 0.030
v and r = 0.269
w and v = 0.300
y and v = 0.322
w and m = 0.327
y and m = 0.355


Yellow body
White eye
Vermilion eye
Miniature wing
Rudimentary wing
y w
v m
r
Genetic map (cM)
0 0.1
3.1 3.4
5.8
Recombinant frequencies
y and w = 0.010
v and m = 0.030
v and r = 0.269
w and v = 0.300
y and v = 0.322
w and m = 0.327
y and m = 0.355


X chromosome
0.0 yellow body
0.0 scute bristles
1.5 white eyes
3.0 facet eyes
5.5 echinus eyes
7.5 ruby eyes
13.7 crossveinless wings
20.0 cut wings
21.0 singed bristles
27.7 lozenge eyes
33.0 vermilion eyes
36.1 miniature wings
43.0 sable body
44.0 garnet eyes
55.0 rudimentary
56.7 forked bristles
57.0 bar eyes
59.5 fused veins
62.5 carnation eyes
66.0 bobbed hairs
0.0 net veins
1.3 artistaless antenna
4.0 star eyes
13.0 held-out wings
16.5 dumpy wings
48.5 thick veins
51.0 black body
54.5 reduced bristles
54.8 purple eyes
55.0 short bristles
57.5 light eyes
66.7 cinnabar eyes
67.0 scabrous eyes
72.0 vestigial wings
75.5 lobe eyes
100.5 curved wings
104.5 plexus wings
107.0 brown eyes
blistered wings
0.0 roughoid eyes
0.2 veinlet veins
19.2 javelin bristles
26.0 sepia eyes
26.5 hairy body
41.0 dichaete bristles
43.2 thread arista
44.0 scarlet eyes
48.0 pink eyes
52.0 rosy eyes
58.2 stubble bristles
58.5 spineless bristles
58.7 bithorax body
62.0 stripe body
63.0 glass eyes
66.2 delta veins
69.5 hairless bristles
70.7 ebony body
74.7 cardinal eyes
91.1 rough eyes
100.7 claret eyes
106.2 minute bristles
0.0 bent wing
cubitus veins
shaven hairs
grooveless scutellum
eyeless
Wild type ♀

So, now we have analyzed at least three "variations" from the "predictable" Mendelian-type of inheritance,

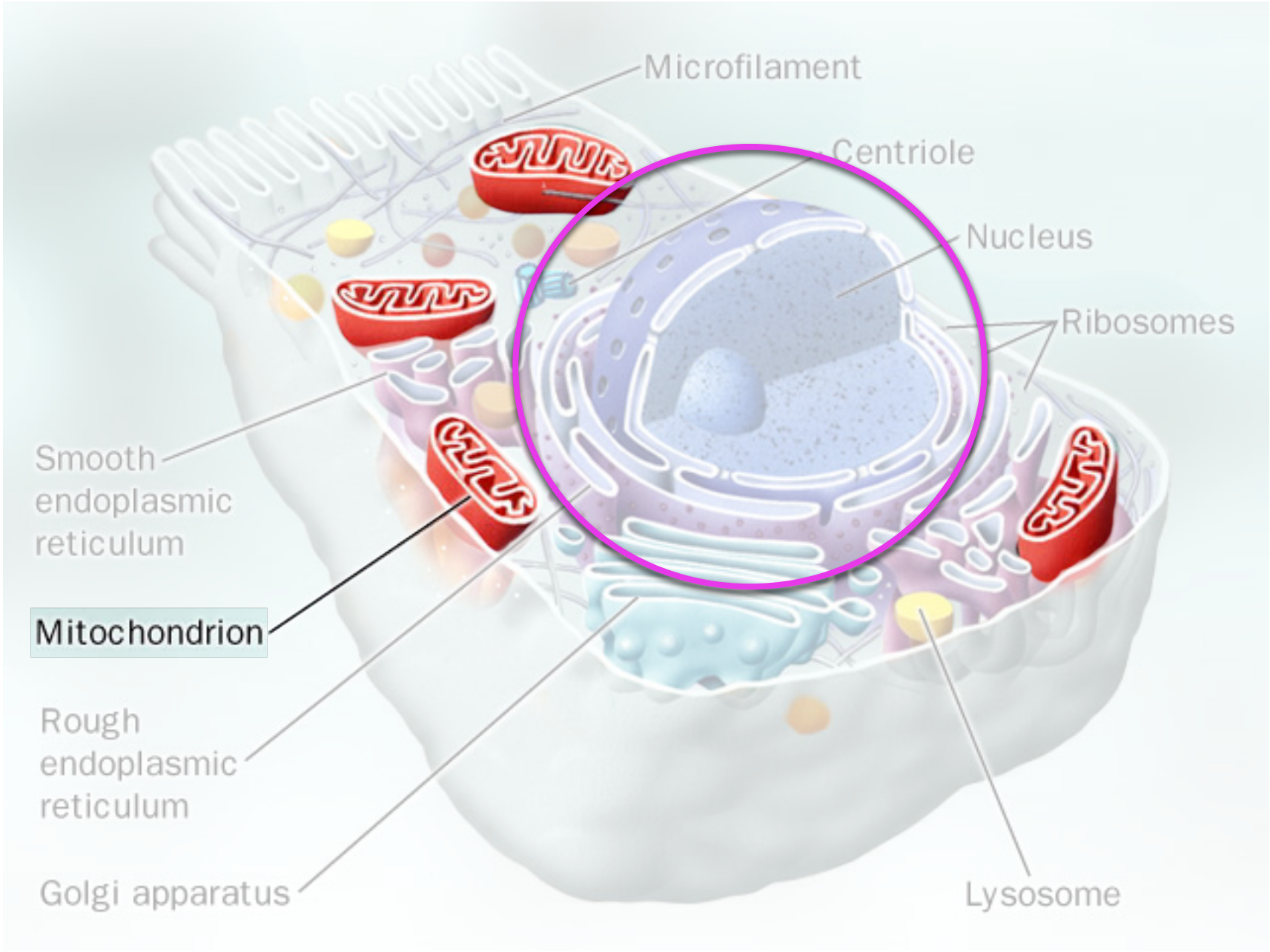
(a) variations that arise as a consequence of "**extensions**" to Mendelian genetics, where the function of the genes in question may interact to give different F_2 phenotypes.

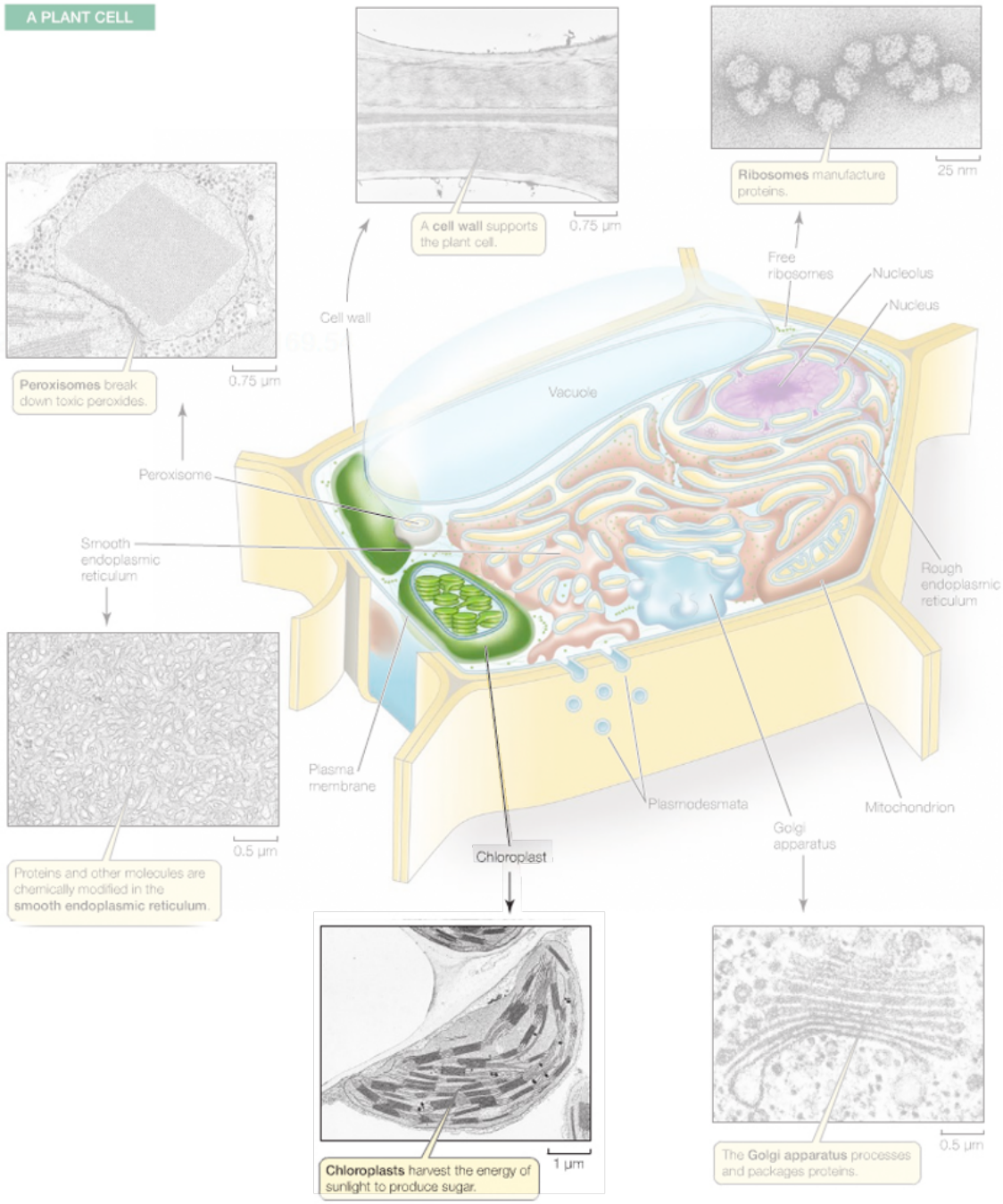
(b) variations that arise because of **X-linkage** -defying Mendel's 2nd law

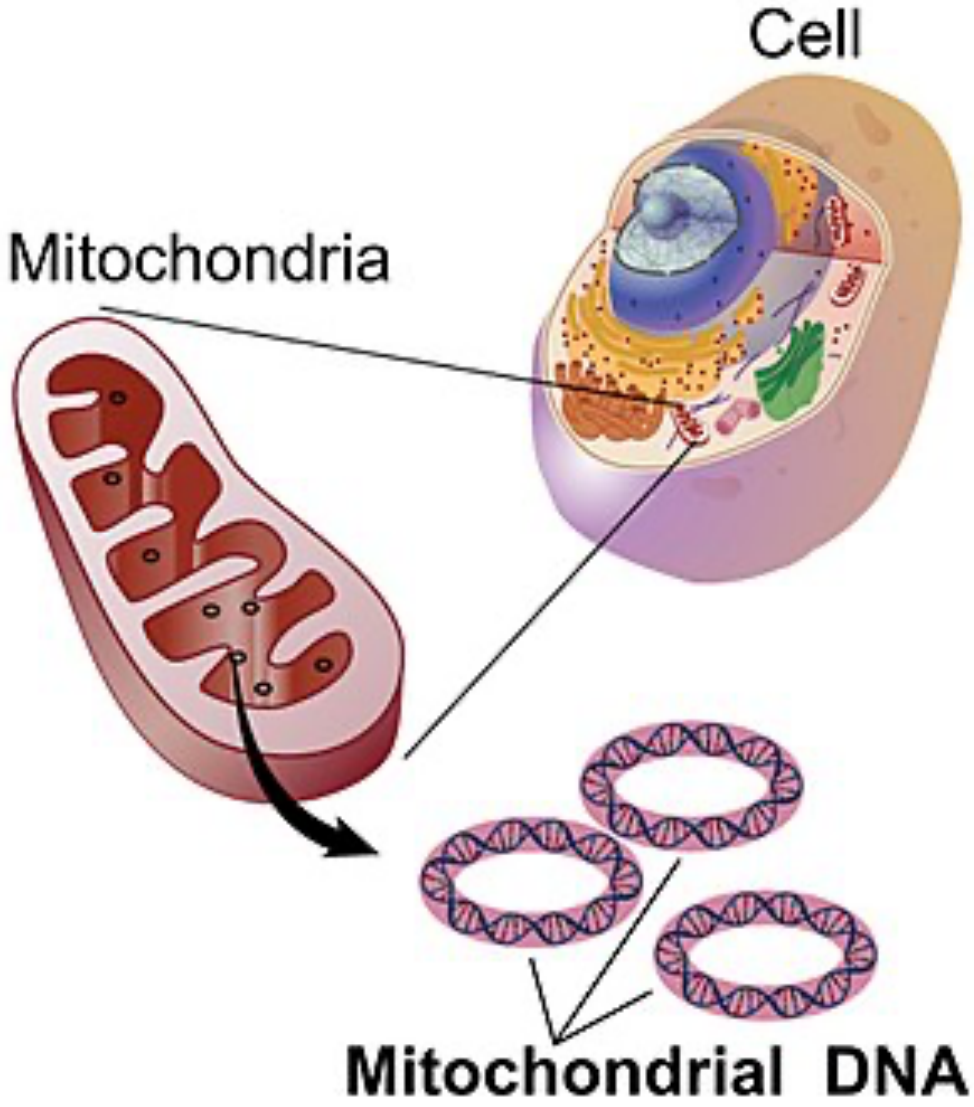
(c) variations that arise because "**chromosomal linkage**" on the autosomes (again defying Mendel's 2nd law, but not "totally").

There is a 3rd form of **non-Mendelian genetics…..**

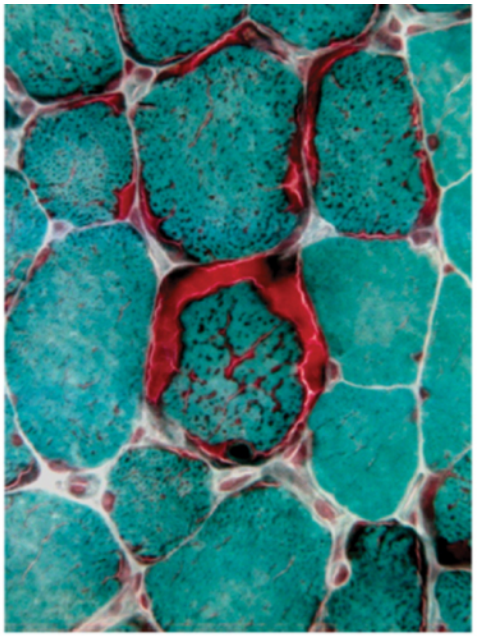
(d) Cytoplasmic / Maternal Inheritance


Microfilament
Centriole
Nucleus
Ribosomes
Smooth endoplasmic reticulum
Mitochondrion
Rough endoplasmic reticulum
Golgi apparatus
Lysosome

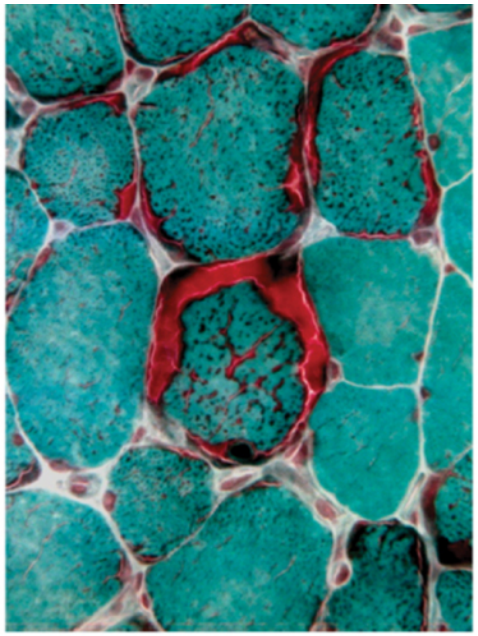

A PLANT CELL
A cell wall supports the plant cell.
0.75 µm
Ribosomes manufacture proteins.
25 nm
Peroxisomes break down toxic peroxides.
0.75 µm
Cell wall
Free ribosomes
Nucleolus
Nucleus
Vacuole
Peroxisome
Smooth endoplasmic reticulum
Rough endoplasmic reticulum
Plasma membrane
Plasmodesmata
Mitochondrion
Golgi apparatus
Chloroplast
0.5 µm
Proteins and other molecules are chemically modified in the smooth endoplasmic reticulum.
1 µm
Chloroplasts harvest the energy of sunlight to produce sugar.
0.5 µm
The Golgi apparatus processes and packages proteins.



….only 37 genes



Inherited mitochondrial diseases are often associated with muscle weakness reflecting deficient production of ATP. The red patches in the microscopic image result from clumps of defective mitochondria in muscle fibers observed in one form of epilepsy due to mutation in mitochondrial DNA.



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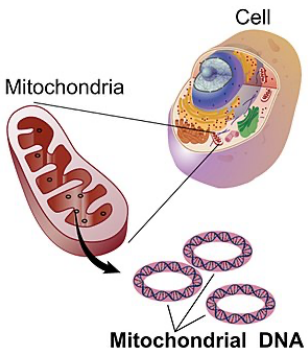
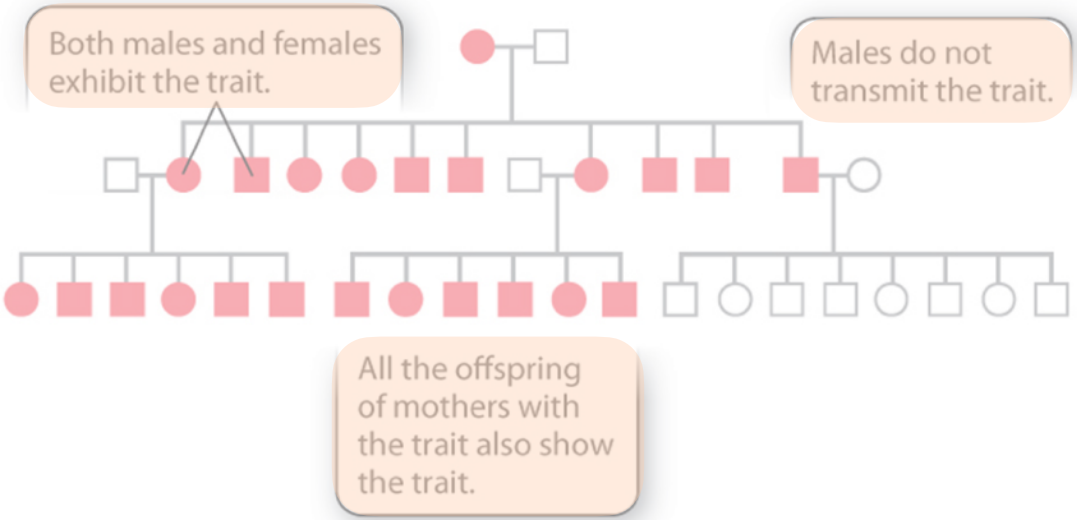




70,000 years ago
N
M
60,000 years ago
L3
80,000 - 100,000 years ago
L2
70,000 - 110,000 years ago
Mitochondrial Eve
152,00 -234,000 years ago
L0
110,000 - 190,000 years ago
L0k
L0d

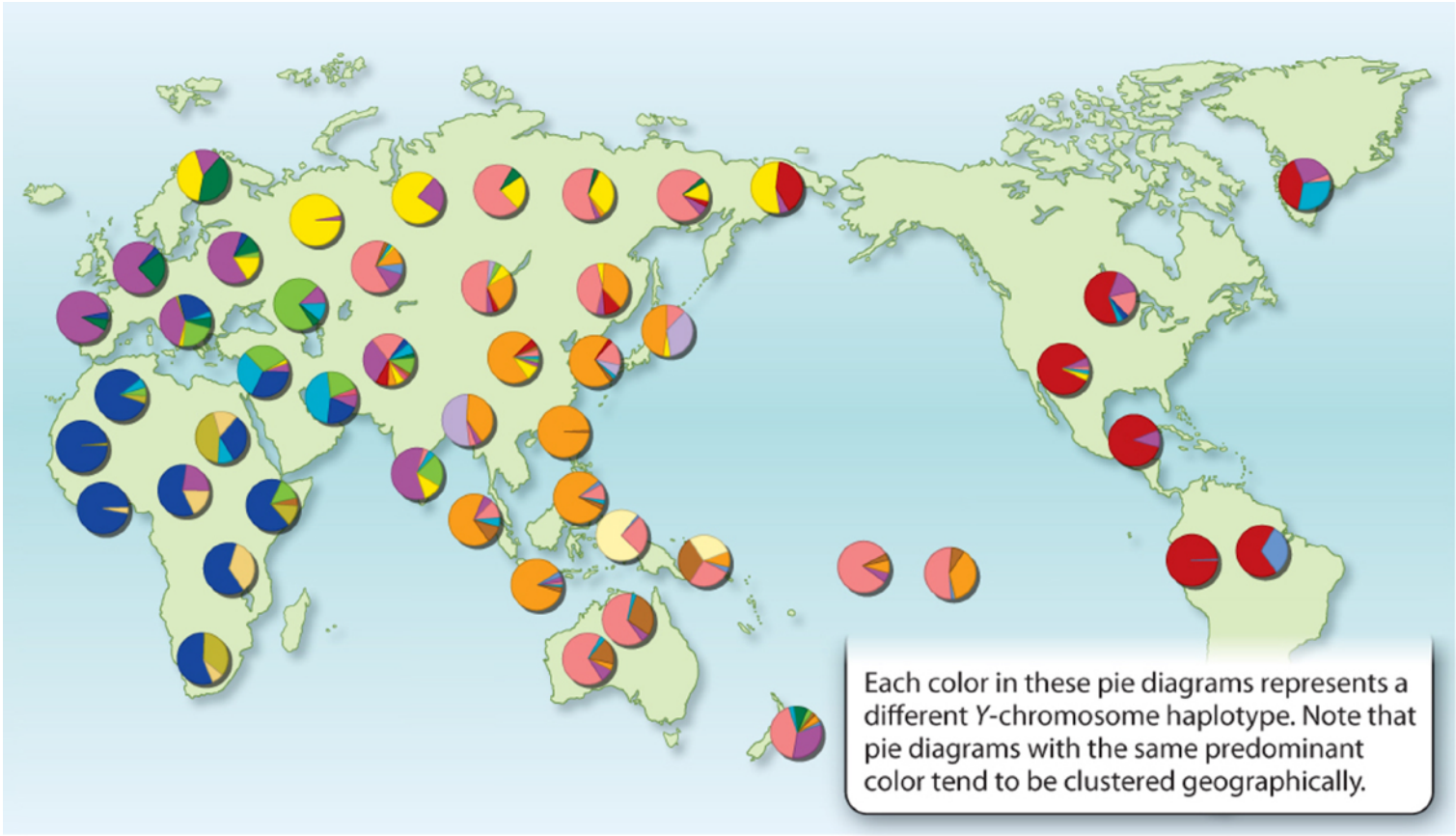
Is there a male equivalent to this purely female based inheritance?

Well Yeah, but it's not cytoplasmic...

it is sex-linked... on the **Y chromosome**

A **haplotype** is a set of DNA variations, or polymorphisms, that tend to be inherited together.

A **haplotype** can refer to a combination of alleles or to a set of single mutations or multiple nucleotide polymorphisms (SNPs)/mutations that are found on the same chromosome.



Bustamante and his team sequenced the **Y chromosomes of 69 males from around** the world and uncovered about 9,000 previously unknown DNA sequence variations.

NATURE | NEWS

Genetic Adam and Eve did not live too far apart in time

Studies re-date 'Y-chromosome Adam' and 'mitochondrial Eve'.

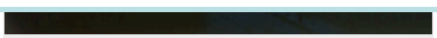
Ewen Callaway

06 August 2013

Bustamante and his team sequenced the **Y chromosomes of 69 males from around** the world and uncovered about 9,000 previously unknown DNA sequence variations.
They used these variations to create a more reliable molecular clock and found that Adam lived between **120,000 and 156,000 years ago**.

A comparable analysis of the same men's mtDNA sequences suggested that Eve lived between **99,000 and 148,000 years ago**[1].

(as is likely to have happened for long periods of human history), men have, on average, just one son. In this case, evolutionary theory predicts that for any given man there is a high probability that his paternal line will eventually come to an end. All of his male descendants will then have inherited Y chromosomes from other men. In fact, it is highly probable that at some point in the past, all men except one possessed Y chromosomes that by now are extinct. All men living now, then, would have a Y chromosome descended from that one man — identified as Y-chromosome Adam. (The biblical reference is a bit of a misnomer because this Adam was by no means the only man alive at his time.)



Hemera/Thinkstock

A Sardinian fisherman. Using DNA from men from the island, researchers have reconstructed a tree of paternal descent.

journalists around the world.

So, now we have analyzed two "variations" from the "predictable" Mendelian-type of inheritance,

(a) variations that arise as a consequence of "**extensions**" to Mendelian genetics, where the function of the genes in question may interact to give different F_2 phenotypes.

(b) variations that arise because of "**chromosomal linkage**" (thus defying Mendel's Second law).

(c) Cytoplasmic / Maternal Inheritance

There is actually a 4th form of **non-Mendelian genetics…..**

(d) ??

So, now we have analyzed at least three "variations" from the "predictable" Mendelian-type of inheritance,

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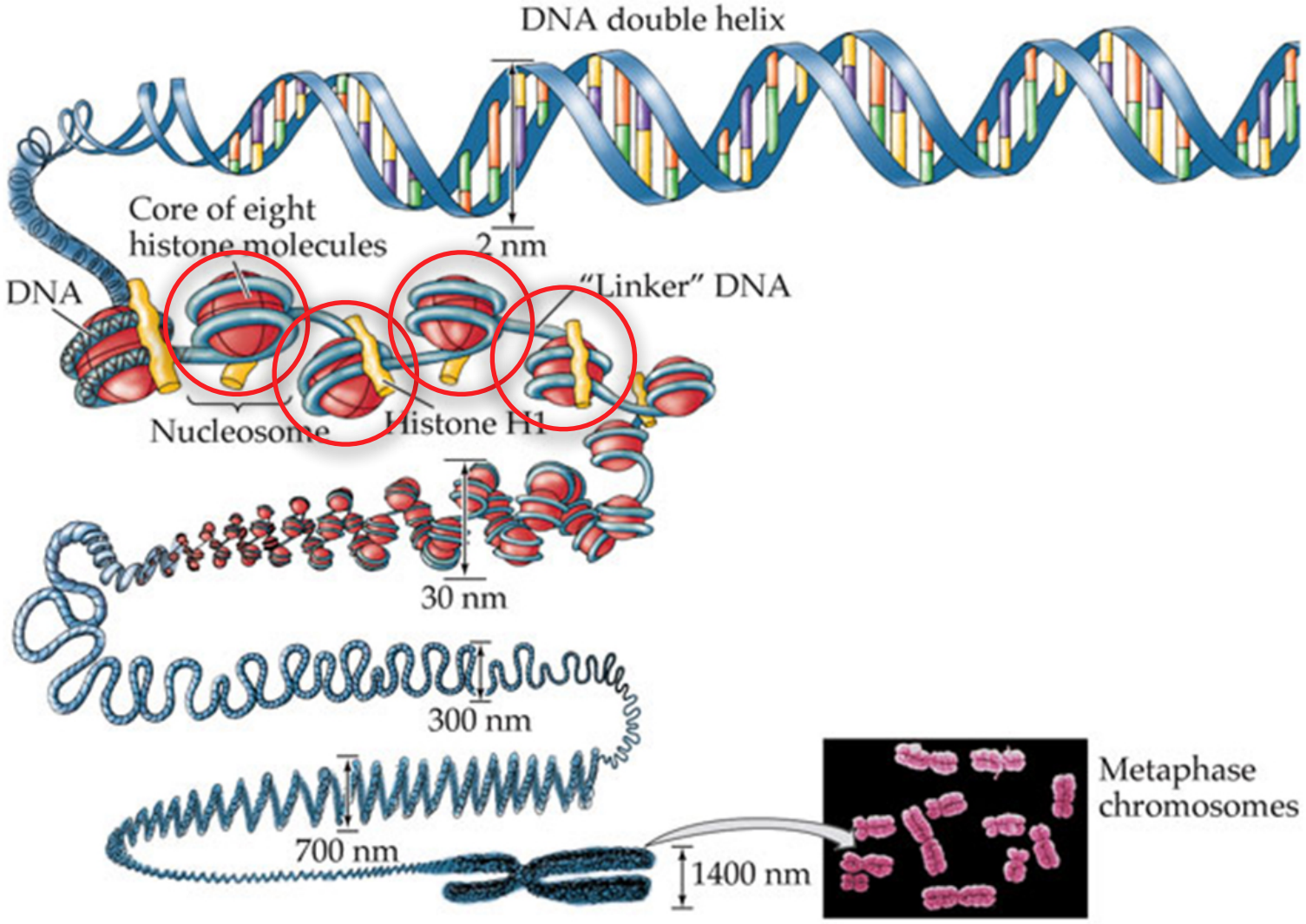
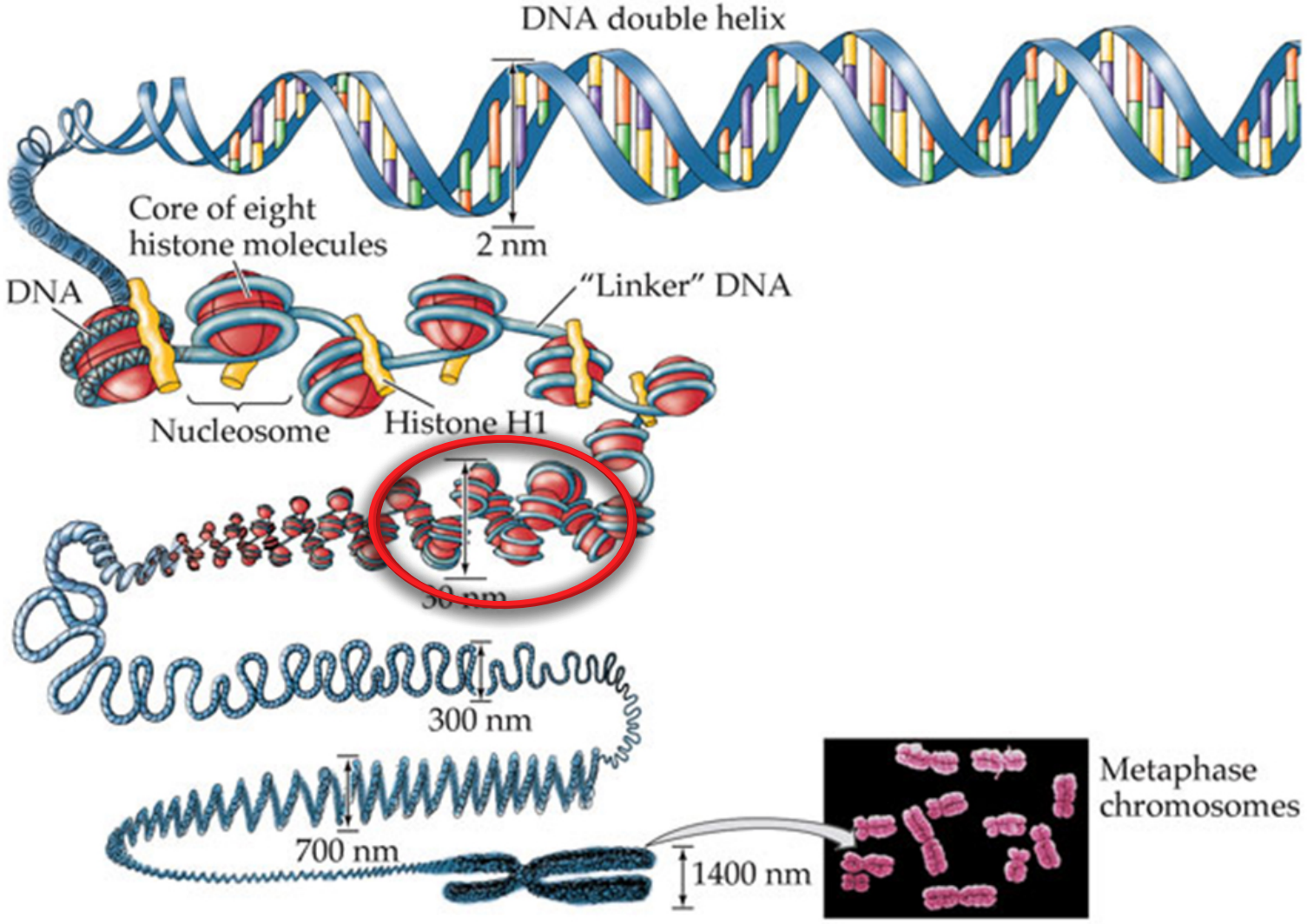
(d) **Cytoplasmic** / **Maternal** Inheritance

There is actually a 4th form of **non-Mendelian genetics…..**

(e) ??


DNA double helix
2 nm
Core of eight
histone molecules
DNA
"Linker" DNA
Nucleosome
Histone H1
30 nm
300 nm
700 nm
1400 nm
Metaphase
chromosomes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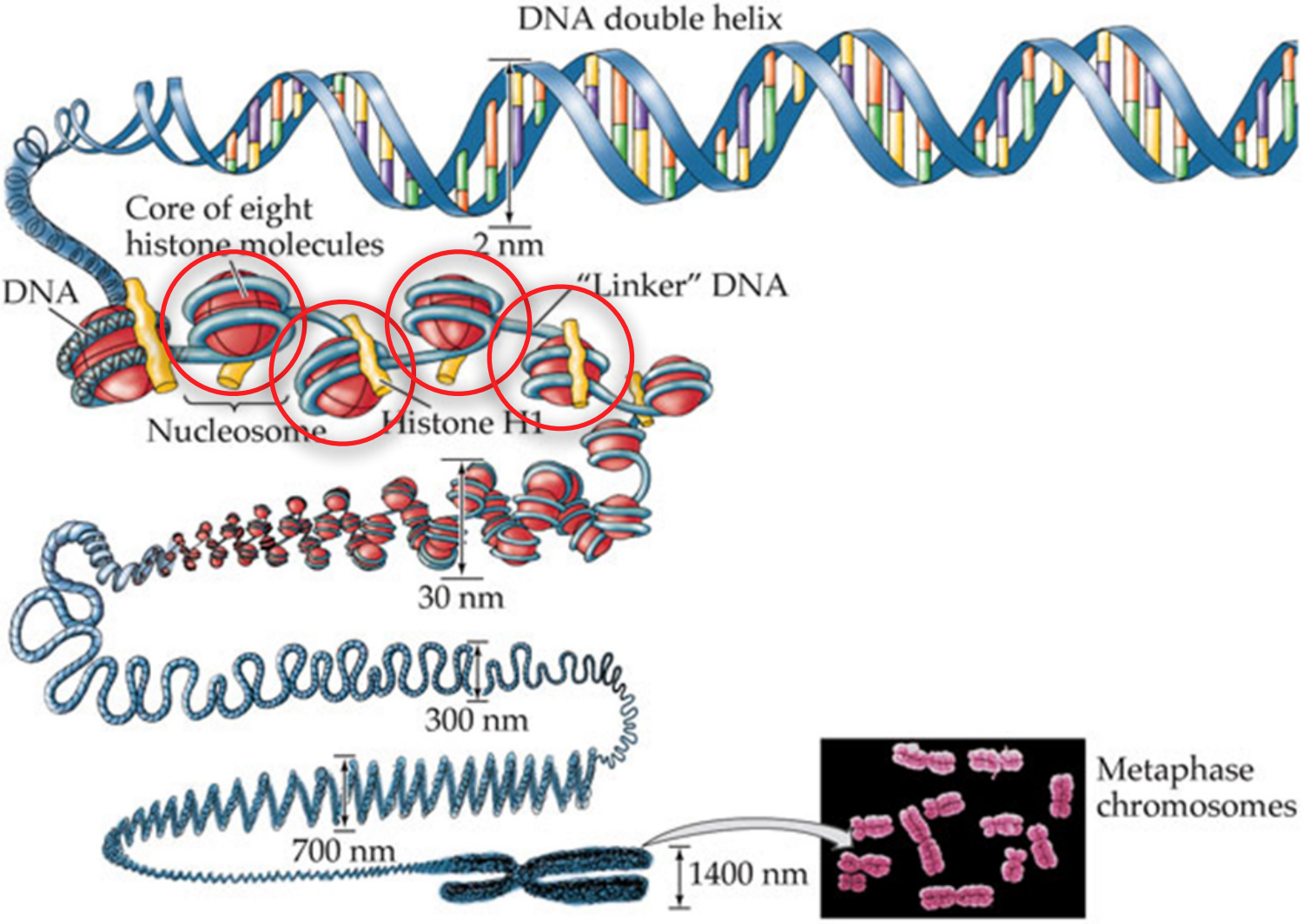


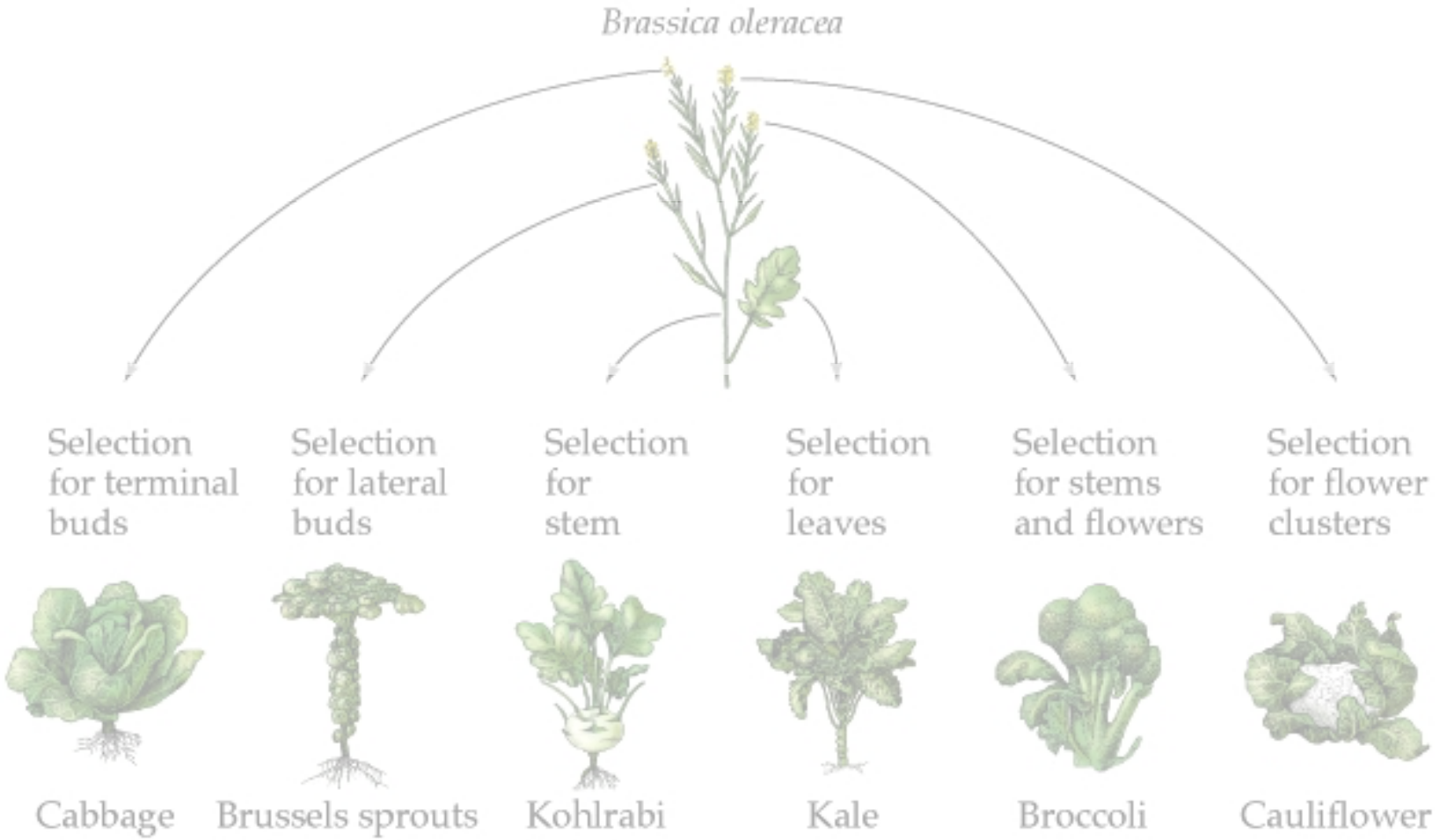
Epigenetics

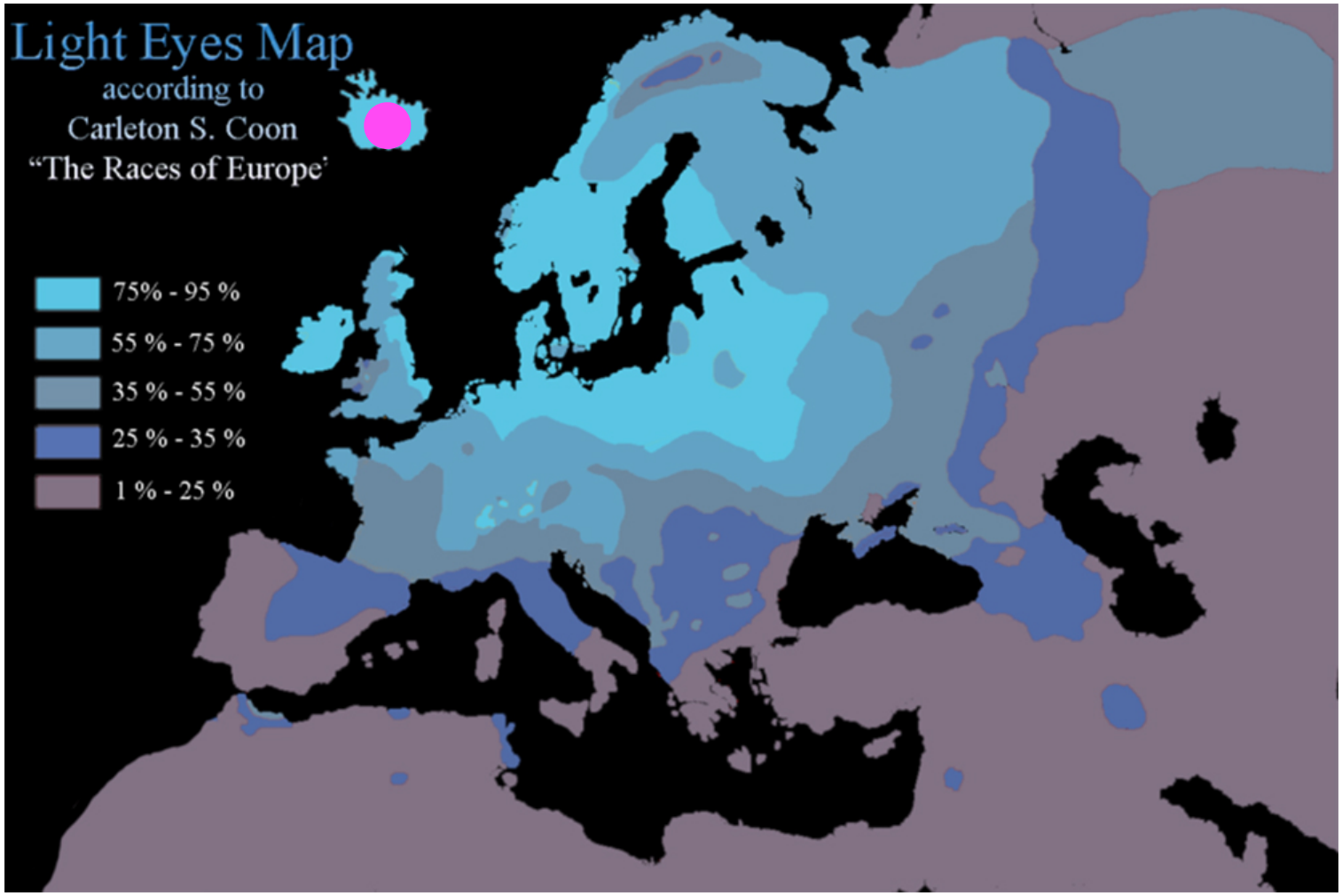

eight

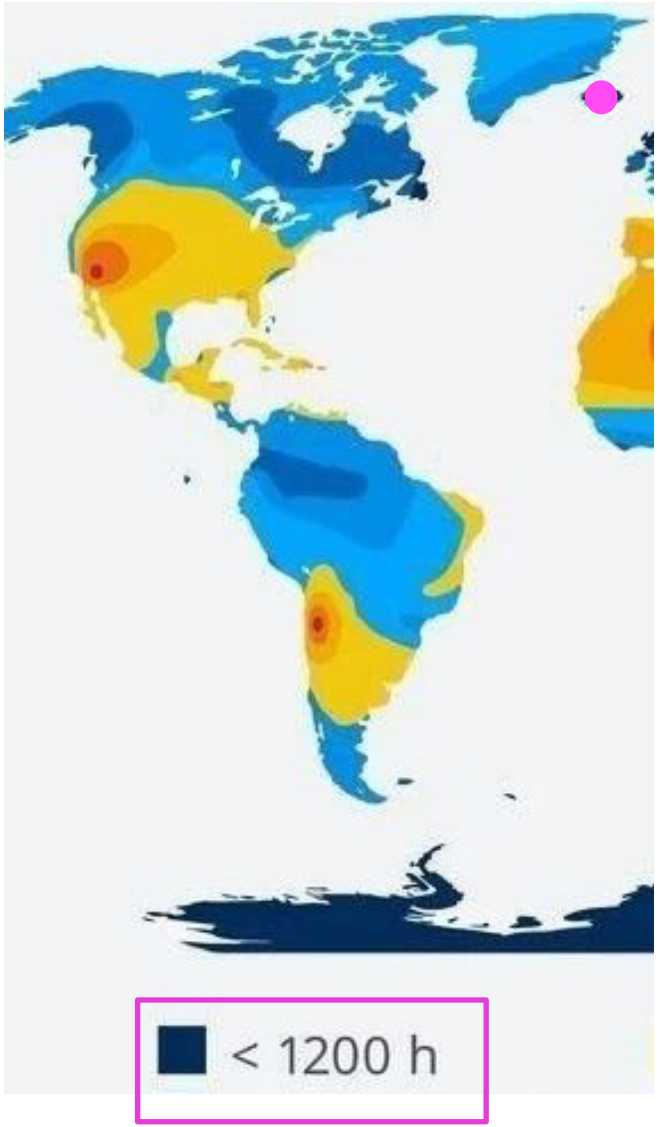
Genetic Variation within Populations

To recap (in light of the last few lectures):

For a population to evolve, its members must possess variation, which is the raw material on which "agents" or "forces" o evolution act (**genetic variation** within a **gene pool**).




Light Eyes Map
according to
Carleton S. Coon
"The Races of Europe"
75% - 95 %
55 % - 75 %
35 % - 55 %
25 % - 35 %
1 % - 25 %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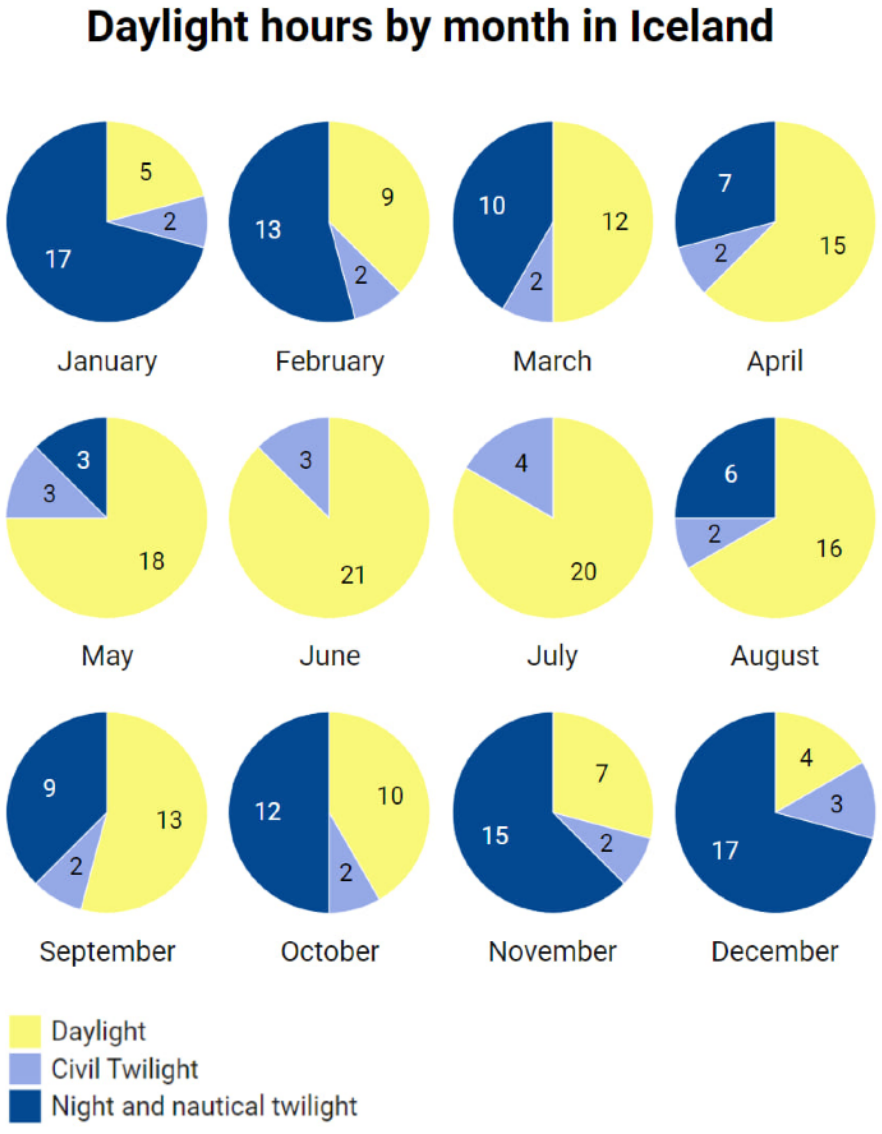

Daylight hours by month in Iceland
5
2
17
January
9
13
2
February
10
12
2
March
7
2
15
April
3
3
18
May
3
21
June
4
20
July
6
2
16
August
9
13
2
September
12
10
2
October
7
15
2
November
4
3
17
December
Daylight
Civil Twilight
Night and nautical twilight

Evolution: Natural Selection…

Genetic Toolkit movie (lecture 6)…

Over the course of the last 600 million years, "what is evolution really working on… it's the recipe, it's the genes"

Looking at evolution of populations through the eyes of a geneticist, you can think of **Natural Selection** in terms of **phenotypes** and **genotypes**.

Beneficial phenotypes -with some type of advantage will be selected over others… But, how are these genes "assessed"? -through the survivors passing on their particular form of genes… their "alleles" on to the next generation.

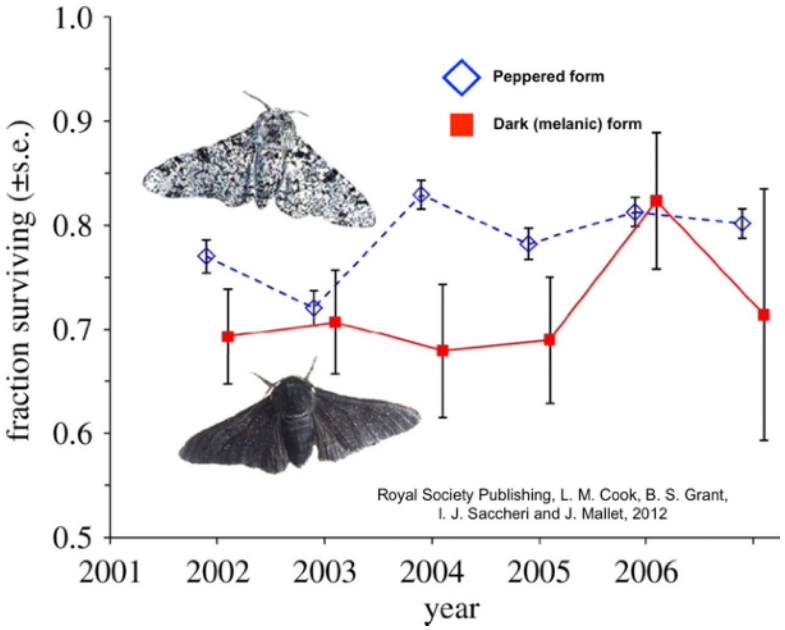
Over time, the gene pool of a given population will have more copies of those alleles that code for beneficial phenotypes, and less copies of alleles for harmful traits. The central thesis of this argument is that ***-through selection of phenotypes, natural selection actually changes the*** *allele frequencies* ***in a population's gene pool.***

What is **Natural Selection** working on? What is it selecting for?

For survival and reproduction.

In the same way that through “artificial selection”, crop breeders, farmers select the crops / animals with the most desirable traits…

In the experiment below; by eating the “easily viewed” moths the birds effectively change the phenotype of the moth population (the frequency of the two alleles) over time, i.e the allele frequencies will shift to match this selective regimen.



experimental data from studies
by Michael Majerus (1954-2009).

Such analyses demonstrated ~9% drop in highly pigmented moths (on average).. over just a 6 year time course.

Hardy Weinberg Principle:

"the frequency of alleles and genotypes in a population will remain constant over time -in the absence of other evolutionary influences".

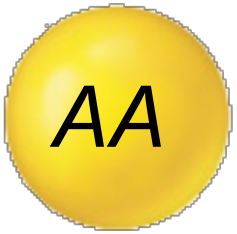
In essence, the Hardy–Weinberg equilibrium describes the

Perfect "Mendelian Population", without ANY Evolutionary variation.

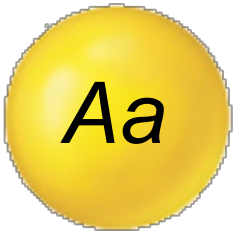
The resulting **HW equilibrium** relates "Genotypes**"** to measurable "**Allele Frequencies**".

and gives us some appreciation as to how such "Mendelian populations" will/ will not change over time

Genotypes



Homozygous DOMINANT



Heterozygous

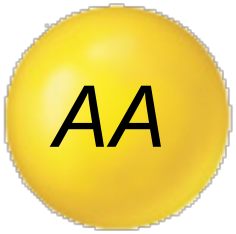
Homozygous recessive

Allele Frequency:

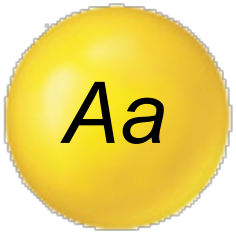
= frequency of **“A”** and the frequency of **“a”** in the above population

Genotype Frequency -in a population is the number of individuals with a given Genotype

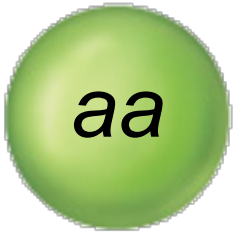
Let **Genotype frequency** **of "A" = "p"** and **of "a" = "q"**



p^2



$2pq$

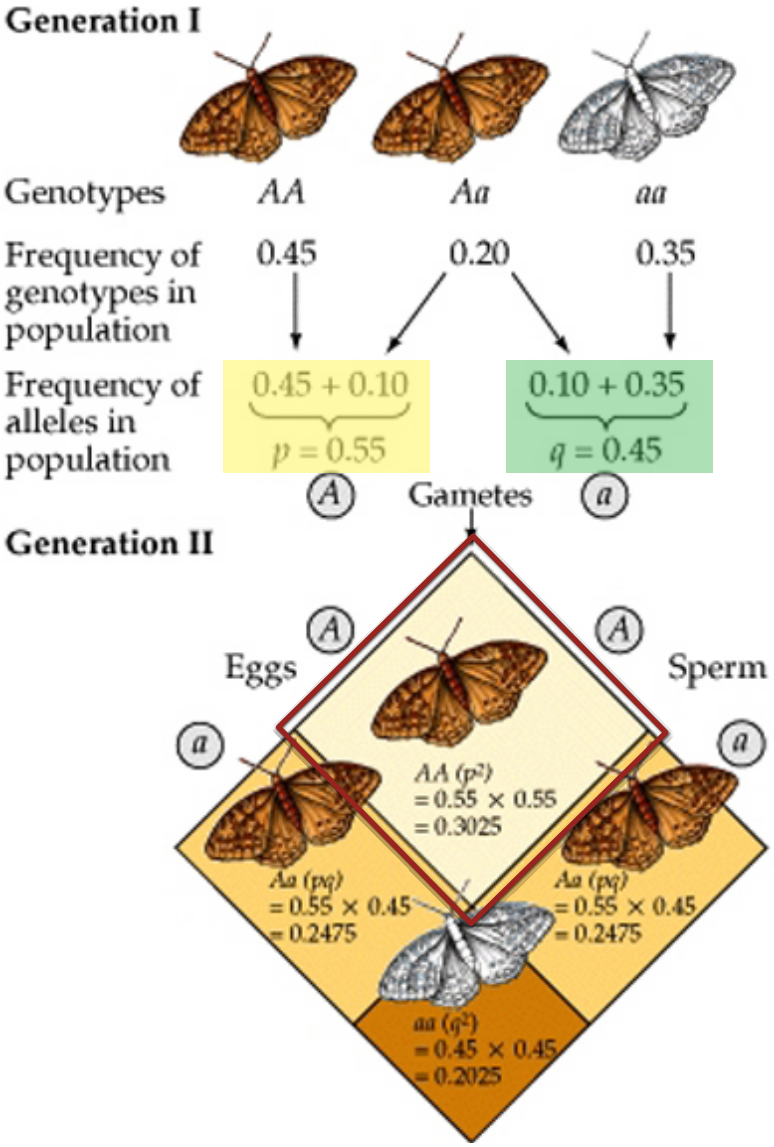


q^2

at equilibrium… **Genotype frequency = 1**

$$p^2 + 2pq + q^2 = 1$$

Hardy–Weinberg equation


Generation I
Genotypes
AA
Aa
aa
Frequency of genotypes in population
0.45
0.20
0.35
Frequency of alleles in population
0.45 + 0.10
p = 0.55
0.10 + 0.35
q = 0.45
A
Gametes
a
Generation II
A
A
Eggs
Sperm
a
a
AA (p²)
= 0.55 × 0.55
= 0.3025
Aa (pq)
= 0.55 × 0.45
= 0.2475
Aa (pq)
= 0.55 × 0.45
= 0.2475
aa (q²)
= 0.45 × 0.45
= 0.2025

Genotype Frequency -in a population is the number of individuals with a given Genotype

Let **Genotype frequency** **of "A" = "p"** and **of "a" = "q"**

AA

p^2

Aa

$2pq$

aa

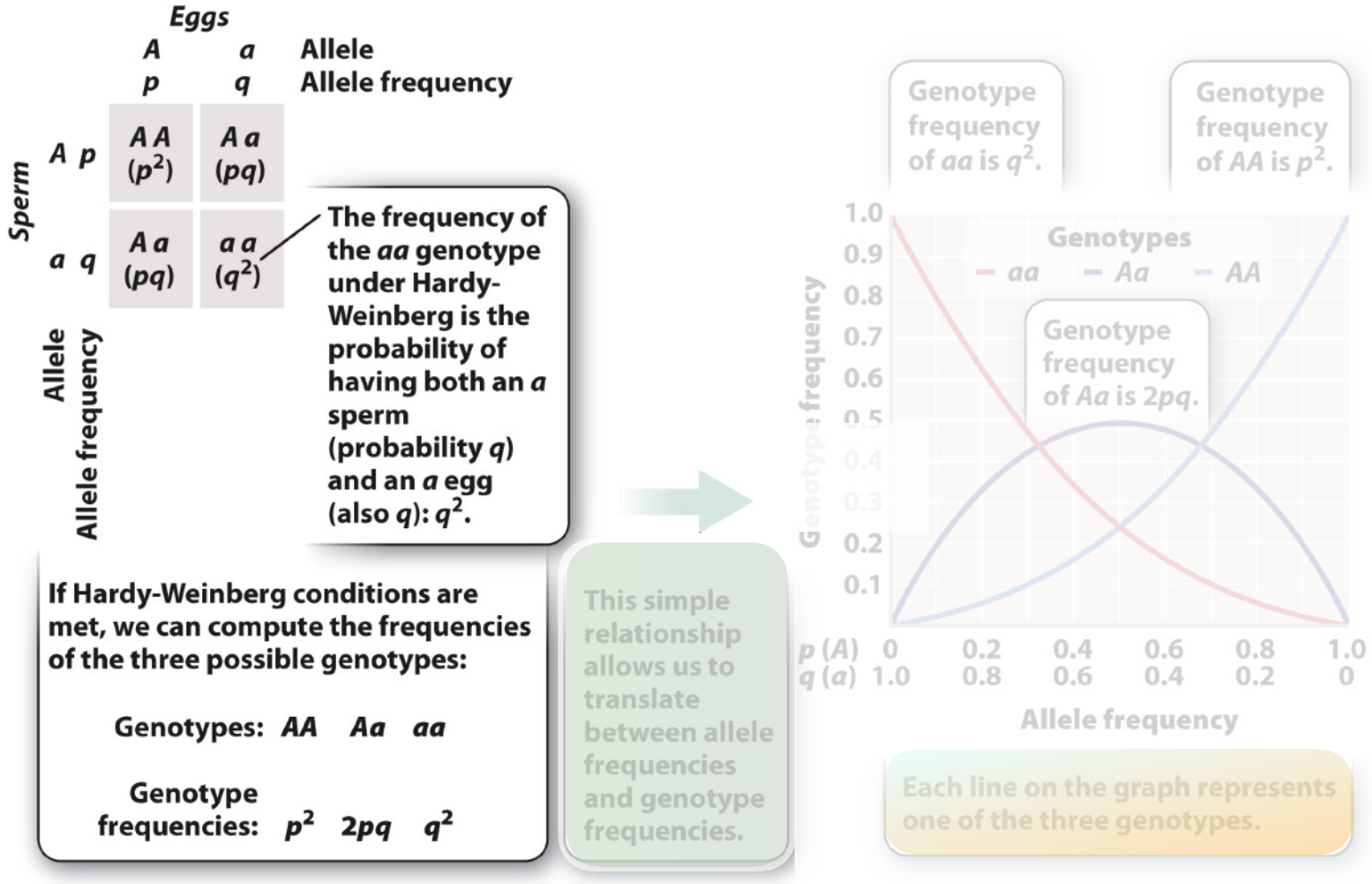
q^2

at equilibrium… **Genotype frequency = 1**

$$p^2 + 2pq + q^2 = 1$$

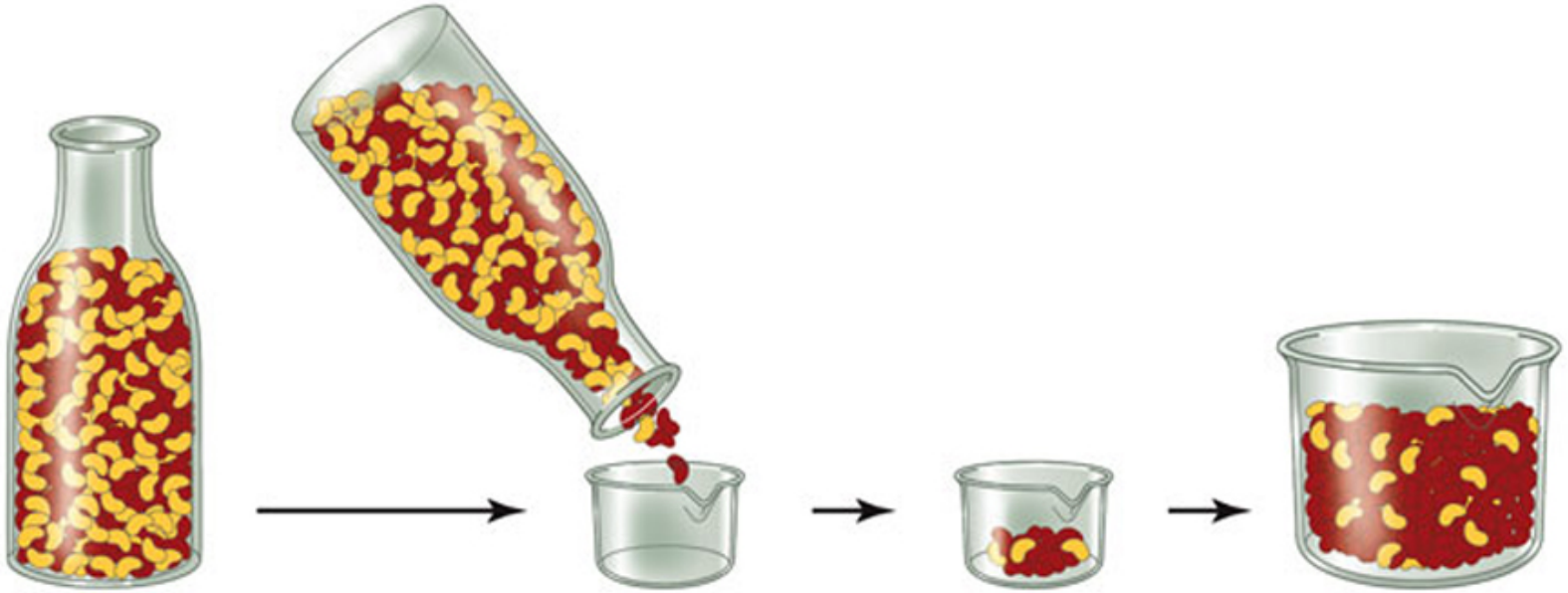
Hardy–Weinberg equation

Hardy–Weinberg equilibrium

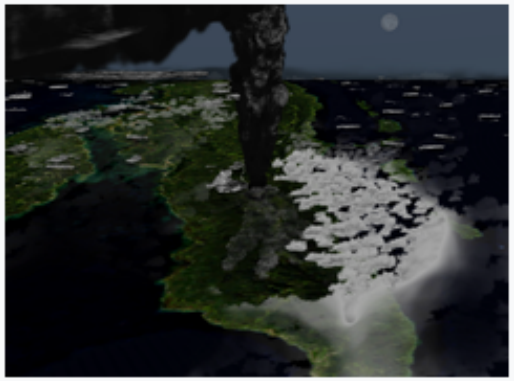


Major Changes in the **HW equilibrium** often signal dramatic changes in population stability…

It can also indicate recovery of a population from dramatic events… such as a **bottle neck effect**.



Youngest Toba eruption



Artist's impression of the eruption from about 42 kilometres (26 mi) above Northern Sumatra

Volcano	Toba Caldera Complex
Date	75,000 ± 900 years BP
Type	Ultra-Plinian
Location	Sumatra, Indonesia 2.6845°N 98.8756°E
VEI	8
Impact	Second-most recent supervolcanic eruption; impact disputed

Lake Toba is the resulting crater lake

The **most recent** Toba eruption was a supervolcanic eruption that occurred around 75,000 years ago at the site of present-day Lake Toba in Sumatra, Indonesia. It is one of the Earth's largest known explosive eruptions.

The **Toba catastrophe theory** holds that this event caused a global volcanic winter of six to ten years and possibly a 1,000-year-long cooling episode.

In 1993, science journalist Ann Gibbons posited that a population bottleneck occurred in human evolution about 70,000 years ago, and she suggested that this was caused by the eruption.

Geologist Michael R. Rampino of New York University and volcanologist Stephen Self of the University of Hawai‘i at Mānoa support her suggestion. In 1998, the bottleneck theory was further developed by anthropologist Stanley H. Ambrose of the University of Illinois at Urbana–Champaign. Both the link and global winter theories are controversial.[1]

The **Hardy–Weinberg equation** can also be used as the “ultimate” evolutionary “null hypothesis”…

Hardy–Weinberg equilibrium

When a population is at "equilibrium" **there can be no differences in the survival and reproductive success of individuals.** i.e there is NO selective elimination of *a* alleles (**NO SELECTION**), meaning that the frequency of *a* will gradually decline (and the frequency of *A* correspondingly increase) over the generations. As we discuss below, we call this differential success of alleles.

Populations must not be added to or subtracted from by migration. (**NO GENE FLOW).** Consider a second population adjacent to the one we used in the preceding example in which all the alleles are *A* and all individuals have the genotype *AA.* Then there is a sudden influx of individuals from the first population into the second. The frequency of *A* in the second population changes in proportion to the number of immigrants.

The population must be sufficiently large to prevent sampling errors. Population size affects the Hardy–Weinberg equilibrium such that it technically holds true only for "infinitely" large populations. A change in the frequency of an allele due to the random effects of limited population size is called So, effectively **NO GENETIC DRIFT**.

There can be no mutation. If *A* alleles mutate into *a* alleles (or other alleles, if the gene has multiple alleles), and vice versa, then again we see changes in the allele frequencies over the generations. In general, because mutation is so rare, it has a very small effect on changing allele frequencies on the timescales studied by population geneticists.

Individuals must mate at random. For the Hardy–Weinberg equilibrium to hold, mate choice must be made without regard to genotype, *AA, Aa,* or *aa* individuals should choose and be chosen at random. **non-random mating**

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Hardy–Weinberg equilibrium

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Populations must not be added to or subtracted from by migration. (**NO GENE FLOW).** Consider a second population adjacent to the one we used in the preceding example in which all the alleles are *A* and all individuals have the genotype *AA.* Then there is a sudden influx of individuals from the first population into the second. The frequency of *A* in the second population changes in proportion to the number of immigrants.

The population must be sufficiently large to prevent sampling errors. Population size affects the Hardy–Weinberg equilibrium such that it technically holds true only for "infinitely" large populations. A change in the frequency of an allele due to the random effects of limited population size is called So, effectively **NO GENETIC DRIFT**.

There can be no mutation. If *A* alleles mutate into *a* alleles (or other alleles, if the gene has multiple alleles), and vice versa, then again we see changes in the allele frequencies over the generations. In general, because mutation is so rare, it has a very small effect on changing allele frequencies on the timescales studied by population geneticists.

Individuals must mate at random. For the Hardy–Weinberg equilibrium to hold, mate choice must be made without regard to genotype, *AA, Aa,* or *aa* individuals should choose and be chosen at random. **non-random mating**

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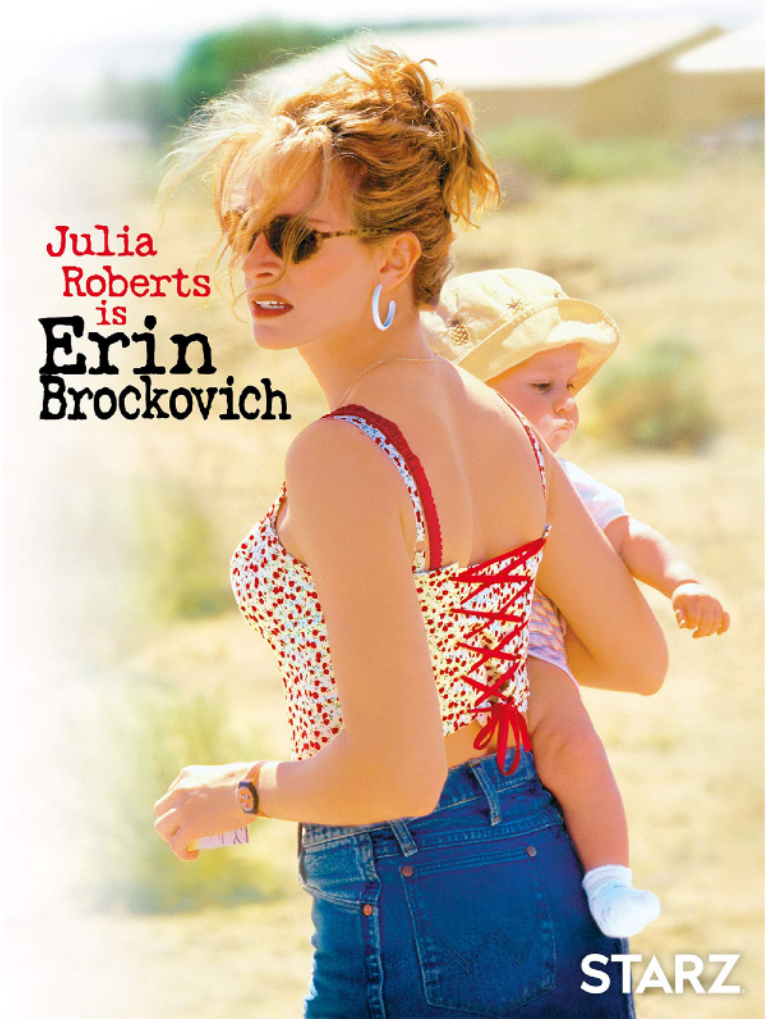
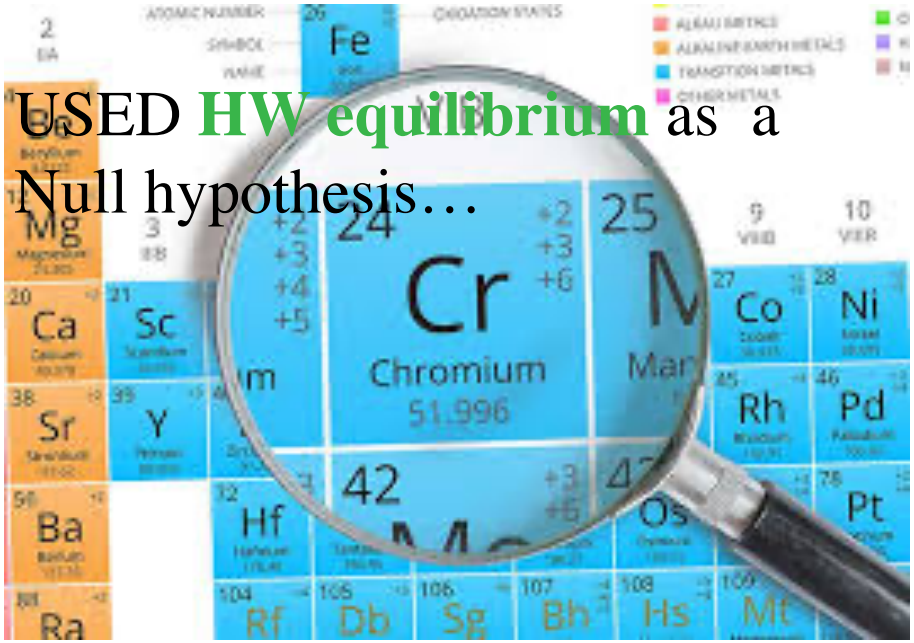


USED **HW equilibrium** as a Null hypothesis…



Erin Brokovich Julia Roberts

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increased Cancer in Hinkley -caused by INCREASE in mutation rate ?