Part 6. Battle of the sexes

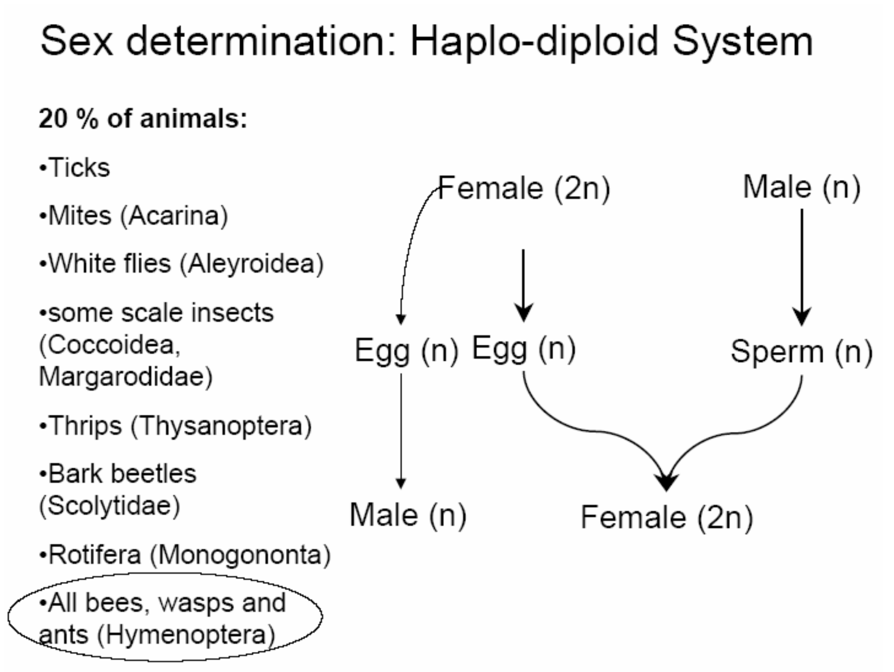
Why two sexes?
How sex is determined?

maleness sex chromosome is not absolute

X/Y, W/Z

Temperature, Wolbachia,

Probably an universal rule is to having two sexes



The major transitions in Evolution

Genes to Genomes
Unicellular to Multicellular organisms
Organisms to Societies

Within-group conflict: Selfish Reproduction

- Evolution of extreme cooperation potentially undermined by selfish reproduction among group members
- Non-cooperative 'free-rider parasites' can outcompete altruists
- Selfish reproductive workers exploit insect societies
- Mechanism moderating conflict: queen and worker policing

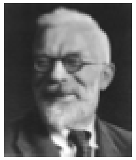
Natural selection

Species and environment
Interspecies selection
Intraspecies selection

Sexual selection
- Intersexual selection: sexual conflict
- Intrasexual selection: ex. sperm competition

Subcellular selection: organelle competition

8. Sex Ratio Allocation



A. Fisher Sex Ratio Theory
B. Deviations from 1:1 Sex Ratios
- Local Mate Competition
- Local Resource Enhancement
- Maternal Condition
- Sex Ratio Distorters

C. Hermaphroditism

D. Conflict over sex ratios

Parents that overproduce the minority sex will have offspring that enjoy greater than average reproductive success. Therefore any heritable variants that tend to cause overproduction of the minority sex will increase in frequency, as they do so the sex ratio imbalance will decrease.

• Parents invest “a certain amount of biological capital” in each offspring.

• The total reproductive value of all the M young in a population must equal the total reproductive value of all the F young“because each sex must supply half the ancestry of all future generations”. Therefore, the only evolutionarily stable sex allocation strategy is to invest equally in daughters and sons.

• If F (say) were receiving less total PI than M across the whole population, then daughters would be providing their parents with more fitness (grandchildren) per unit of PI than sons.

• This situation would select for re-allocation of PI toward the less-invested-in, higher-yield F … but only until F were receiving the same PI as M . The argument is symmetrical.

Thus, at equilibrium, there cannot be a less-invested-in sex.

The asymmetry of sex: size & number

Large immobile egg: a small number of large gametes
Small motile sperm: a large number of small gametes (which are disposable)
Competition between the needs of the offspring and the parents
Minimizing the cost to the parents, while maximizing the benefit to offspring

Sperm behaves like a parasite
It may be beneficial when fertilization is external, but why it occurs in internal

<u>Uniparental inheritance</u>

Algae & fungi: isogamous
They have mating types (*Schizophyllum commune* has more than 28000 types)

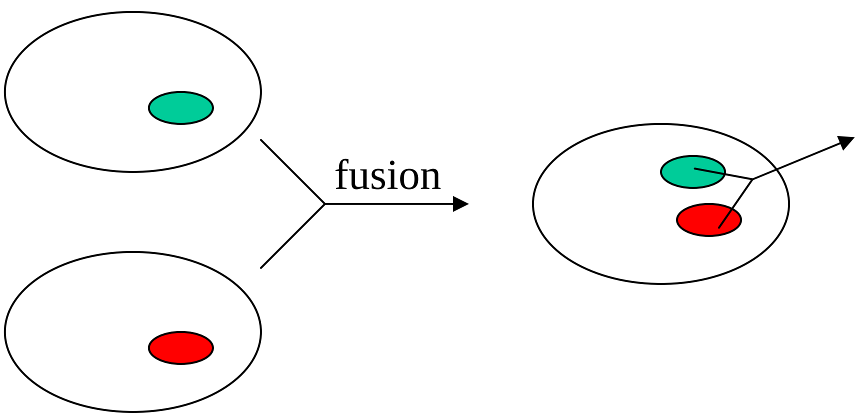
Incompatible cytoplasm
Vulva: produces isogamates
after cell fusion, organelles attack each other to eliminate the other
Chlamydomonas: selective silencing
organelle DNAs are digested

➡ Two cells fuse, but only one of them passes on the organelle (or its DNA)

Incompatibility: how they discriminate the other?

S. commun: no cytoplasmic fusion
Flowering plants (angiosperms): hermaphrodites
- developed mating types in order to maintain outbreeding
- mitochondria sterilize the male sex organ (cytoplasmic sterility): male gametes decrease
- it will upset sex ratio of the population (only female & hermaphrodites)
- nuclear genes counteract the selfish mitochondrial action

Selfish competition theory
Conflict between different cytoplasmic genomes: Leda Cosmides & John Tooby, 1981
- two sexes developed because it the most effective means of preventing conflict between selfish cytoplasmic genomes



competition between the two
their interests differ from the host’s

<u>Selfish mitochondria contributed to the asymmetry of gametes</u>

Human egg: 100000 mitochondria
Human sperm: 100 mitochondria
Many strategies to completely remove male mitochondria
    human, fly, *Vulva*, *Chlamydomonas*, etc
    meaning of independent evolution?

Then why heteroplasmy is found?
    fungi, angiosperms (20% are biparental inheritance), bat

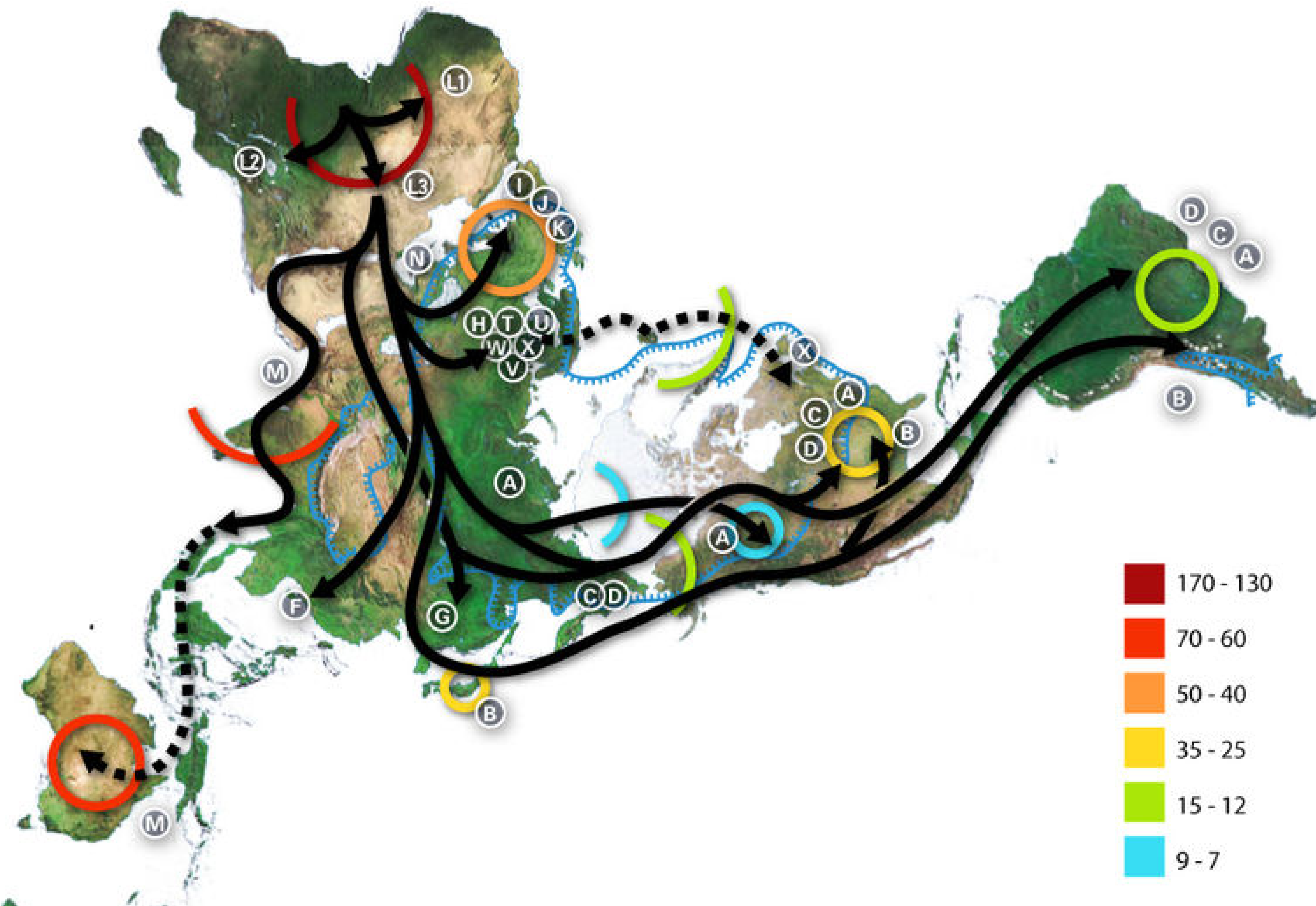
14. Human pre-history about sexes

Molecular Eve: Rebecca Cann, Mark Stoneeking, Allan Wilson, 1987
    mito DNA analysis of 147 living people from 5 continents

<u>Two theories about modern human origin</u>

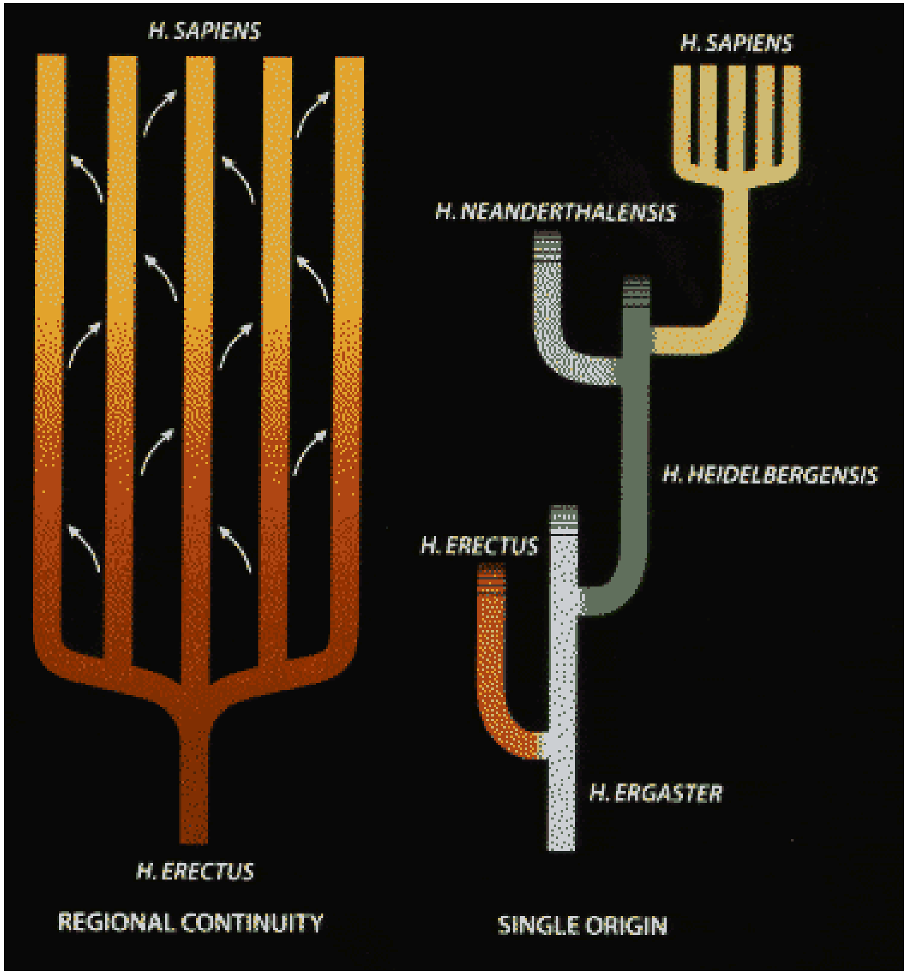
Out of Africa (single origin hypothesis, replacement hypothesis)
Multi-regional hypothesis: interbreed with the earlier descendents
The disappearance of Cro-Magnons: result of genocide or interbreed

Map of human races migration, according to the mitochondrial DNA.



Number represent thousand years before present time. The blue line represents area covered in ice or tundra during the last great ice age.

General European: H, V Southern European: J, K Northern European: T, U, X Near Eastern: J, N African: L, L1, L2, L3, L3* Asian: A, B, C, D, E, F, G (note: M is composed of C, D, E, and G) Native American: A, B, C, D, and sometimes X


H. SAPIENS
H. ERECTUS
REGIONAL CONTINUITY
H. SAPIENS
H. NEANDERTHALENSIS
H. HEIDELBERGENSIS
H. ERECTUS
H. ERGASTER
SINGLE ORIGIN

<u>**Difference between nuclear and mitochondrial DNAs**</u>

1. The mutation rate of mitochondria is nearly 20 times faster than nDNA
   Mitochondrial mutation rate: 2~4% per a million yrs
2. Maternal inheritance only by asexual reproduction
3. Mitochondrial mutation rate is constant

<u>Mitochondrial recombination</u>

Inconsistency from the exclusive maternal inheritance
mtDNA recombination is known in
    Yeast & Rat (1996)

Any evidence of mtDNA recombination in human?
1999, two suggestions were published but not clear
2002, a human patient who inherited some paternal mtDNA from
    heteroplasmy in muscles cells: 90% paternal & 10% maternal
2004, 0.7% of the patient’s mtDNA recombine
    it does not mean inheritance to the next generation
    so far no evidence supporting recombined mtDNA inheritance

Calibrating the clock

Heteroplasmy

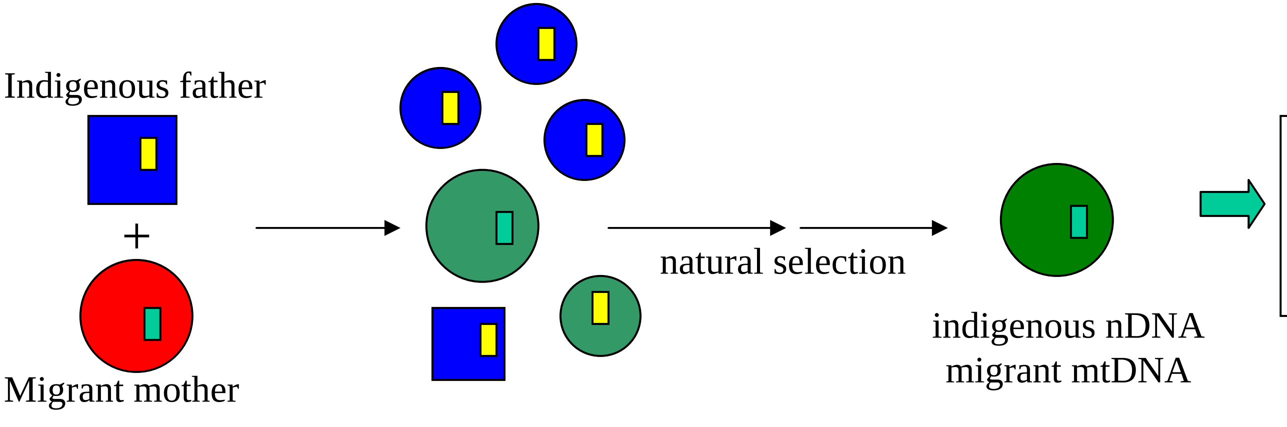
Usually results from mutation not from paternal mtDNA transfer

How general is it? found to be 10~20%

1. Heteroplasmy is far more common than we had imagined
    - consequences for the selfish mitochondrial model of sexes
2. Mutation rate is far higher than expected
    - one mutation every 40~60 generations (800~1200 yrs)
    - according to the rate, molecular Eve dates back to about 6000 yrs ago
    - why such a big discrepancy? (see next slide)

1969, Mungo fossil: anatomically human dated to about 60000 yrs old

2001, mtDNA analysis showed no homology



Can be applied to Neanderthales, who has different mtDNA from modern human?

mtDNA & surname

1869, Francis Galton, Hereditary Genius
Domesday Book of 1086
1912, Australia

Reproductive success is extremely unevenly distributed in population
Most lines fall extinct; and just the same applies to mtDNA (by Jim Cummins)

Is it by neutral drift or natural selection (such as shown in Mungo fossil)?
The discrepancy between

high mutation rate & slow rate of divergence (evolution)

Natural selection abolishes the fastest evolving line
Survivors must have smaller evolutionary variations

Mitochondrial selection

2004, Douglas Wallace:
mitochondrial lineage (haplogroups: the daughters of Eve)
mitochondrial genes might be susceptible to natural selection (more than nuclear DNA)

Purifying selection

If predicted by the theory of neutral drift: the geographical distribution of mitochondrial genes in human populations was not random

But particular genes thrived in certain places (a telltale sign of selection at work)

Of all the abundant lines of mtDNA in Africa, only a handful of lineage left
The same were true in the rest of the world

Some mitochondrial genes are adapted to particular climates, and do better there, whereas others are penalized if they leave home.

mtDNA haplotypes & climate

Tight coupling & Loose coupling (linked to uncoupling)

Africans: tight coupling of ETC, more prone to heart disease and diabetes

less heat & more energy

Inuit: loose coupling of ETC, vulnerable to male infertility

more heat & less energy

sperm motility: vulnerable to energy failures

Asthenozoospermia

more common in people of haplotype T (widespread in northern Sweden)

than in haplotype J (widespread in southern Europe)

but no report in Inuits

The cold-adapted mtDNA uncoupling mutations

- Less ATP per calorie consumed
- Energy insufficiency & more prone to clinical problems
- However, burn calories more rapidly to generate both ATP and heat
- More oxidized mitochondria
- Less ROS production
- Cellular protection & less prone to degenerative and aging
- Increased life span

15. Why there are two sexes?

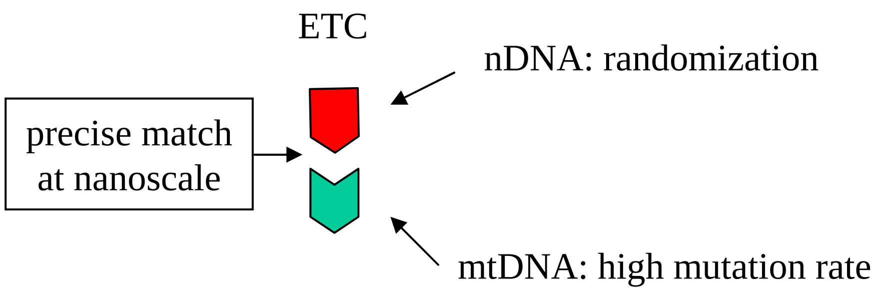
Mitochondrial recombination & heteroplasmy
High mtDNA mutation rate selected against by natural selection

Mitochondrial function is determined by
interactions of proteins encoded by mtDNAs and nDNAs

The problem of Mix-and-match
The requirement for a close match

Two sexes are needed because the dual genome system demands a close match

The dual-control hypothesis



Pretest with a single set of mtDNA genes with a single set of nDNA genes:
selection might occur in unfertilized eggs (the mitochondrial bottleneck)

What if mtDNA comes from two parents?

The hypothesis supports why heteroplasmy, mtDNA recombination, selections are possible

pureness of the population is not important

but how effectively the mt genes work against the nuclear background

The mitochondrial bottleneck

The fertilized egg contains 100000 mitochondria
During the first two weeks:

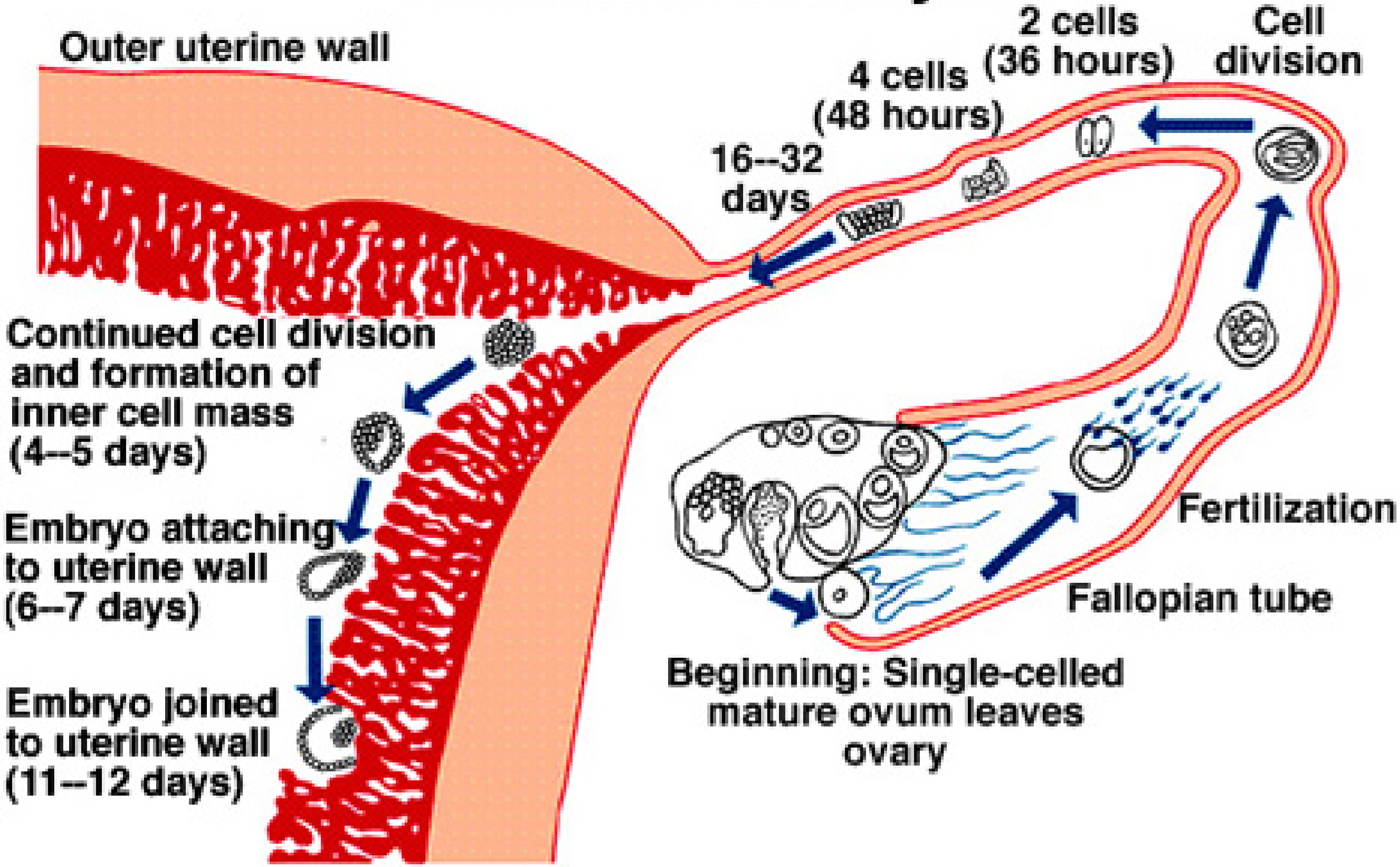
- active cell division, but mitochondria remain quiescent
- a couple of hundreds mitochondria per cell
- if their function is not sufficient to support development, the embryo die
    - probably early miscarriages
    - energy insufficiency may lead to chromosomal abnormalities

Female embryo:

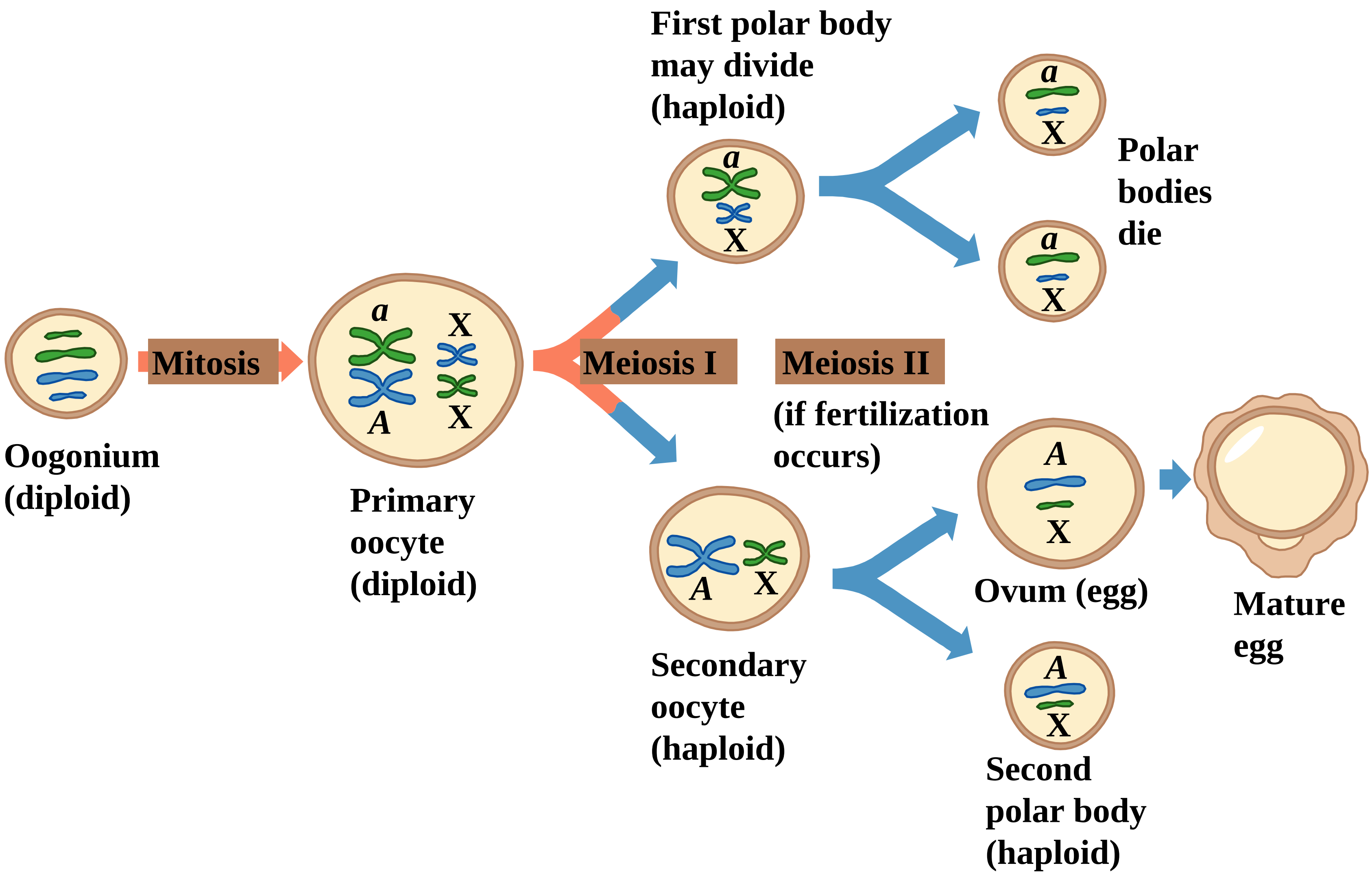
- 2~3 weeks of development: the earliest recognizable egg cells (the primordial oocytes)
- 10~200 mitochondria per the oocyte (start of the mitochondrial bottleneck)
- A surprising variety of mitochondrial sequences in different oocytes taken from the same ovary
- Probably a few mitochondria per a cell and a single copy of mtDNA per mitochondria
- Mitochondrial deficits are exposed and selection works
- Amplification and selection in number thereafter

- The last selection by male nDNA

Early Development of a Human Embryo

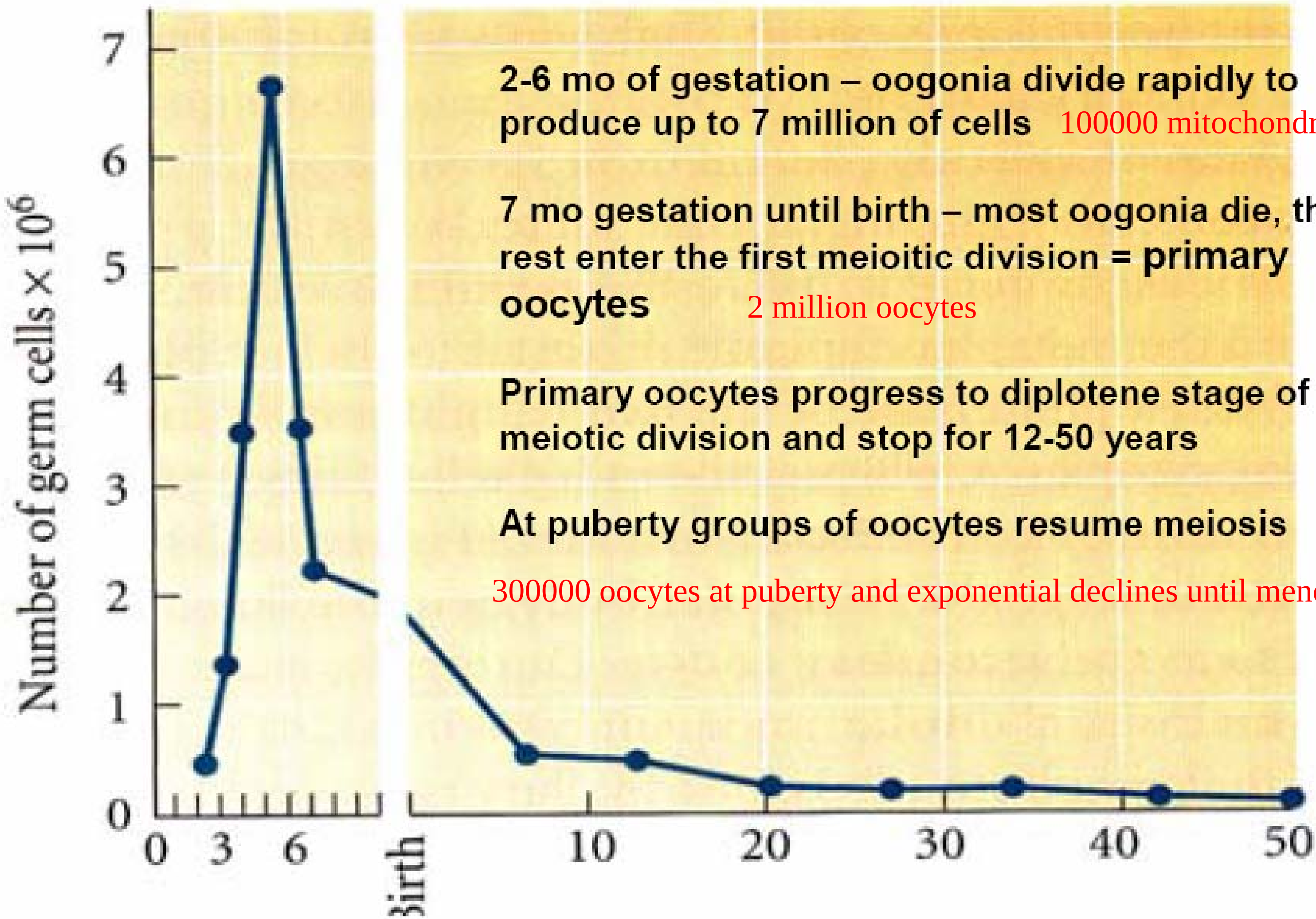


Oogenesis



First polar body may divide (haploid)
a
X
a
X
a
X
Polar bodies die
Mitosis
a
X
X
A
Meiosis I
Meiosis II
(if fertilization occurs)
Oogonium (diploid)
Primary oocyte (diploid)
A
X
Ovum (egg)
Mature egg
A
X
Secondary oocyte (haploid)
A
X
Second polar body (haploid)

Changes in the number of oogonia in human ovary over the life span



2-6 mo of gestation – oogonia divide rapidly to produce up to 7 million of cells 100000 mitochondria/cell

7 mo gestation until birth – most oogonia die, the rest enter the first meioitic division = primary oocytes 2 million oocytes

Primary oocytes progress to diplotene stage of 1st meiotic division and stop for 12-50 years

At puberty groups of oocytes resume meiosis

300000 oocytes at puberty and exponential declines until menopause