Our life cycle consists of a haploid phase and a diploid phase

Adults make sex cells (gametes), which contain one half of the donor’s genetic information; these gametes contribute genetic information to the next generation.

We reproduce sexually.

Humans are sexually dimorphic, just like most other animals.

Presence of a Y chromosome will determine genetic maleness or femaleness (i.e., an XXY individual will become male)

Once ovaries develop, one X chromosome in females degenerates into a Barr body
Gender determination depends on the Y Chromosome

Early embryonic tissues make bipotential structures—they are indistinguishable as male or female.

There are two pairs of ducts: Wolffian Ducts and Müllerian Ducts (internal bipotential genitalia).

The presence of the SRY gene on the Y chromosome will determine which set of bipotential ducts develop into genitalia, and which degenerate.

Gender determination depends on the Y Chromosome

The SRY gene produces a SRY protein (= testis-determining factor) that acts as a transcription factor for other genes, such as SOX9, WT1, and SF1.

As testes develop, three hormones are secreted:

- anti-Müllerian hormone (secreted by Sertoli cells)
- Dihydrotestosterone (DHT) (Leydig cells)
- Testosterone (Leydig cells)
Gender determination depends on the Y Chromosome

The Y chromosome consists of only 27 genes on the Y-specific region (the X chromosome has ~1500 genes). Only one of those genes, the SRY, accounts for the largest phenotypic differences between males and females. Other genes have nothing to do with maleness; one codes for tooth enamel, and another for a ribosomal protein.
Gender determination depends on the Y Chromosome

The Y chromosome is particularly vulnerable to mutation because it does not recombine with any other chromosomes during crossing over (the X chromosome can cross over with another X in XX individuals).

Many of these mutations result in the loss of genes within a population.

Conservative estimates suggest that the remaining genes on the Y chromosome will completely disappear in the next 10 million years or so.

What of a maleless world?

Will humans become extinct?

Will we evolve some sort of parthenogenesis, in which female eggs will develop without sperm?

This Amazonian scenario cannot happen in mammals—we are tied to sexual reproduction, but other sex determination genes may be involved that we do not know about yet. As the Y chromosome continues to degenerate, these other genes might be discovered.
Things to remember about gametogenesis

In males, spermatogenesis proper (meiosis) does not begin until puberty, under the direction of gonadotropins (FSH, LH) and testosterone.

In females, oogenesis (meiosis) begins before birth and is halted before birth, leaving primary oocytes “frozen” in the ovaries until puberty.

Beginning at puberty, once a month, one primary oocyte goes through its second meiotic division, and will finish meiosis only if fertilized by a sperm.
Semen contains many components

The bulbourethral glands, seminal vesicles, and prostate contribute about 99% of the fluid volume of semen

In addition to helping the sperm succeed in the vagina, secretions of the male accessory glands play a role in the male’s own immunity by flushing the urethra with lysozyme and antibodies

Sperm must be capacitated prior to fertilization

By reorganizing molecules in the sperm, capacitation allows them to swim quickly up the fallopian tubes to an egg.

Eggs can only be fertilized for a few hours after ovulation, and fertilization is aided by chemicals produced by the egg (positive chemotaxis) (but sperm remain viable for 4-6 days in the female reproductive tract)
The egg presents several barriers to fertilization

Two membranes, the outer corona radiata, and the inner zona pellucida must be crossed by a sperm to fertilize an egg.

The acrosome of the sperm releases enzymes to dissolve these layers (the acrosomal reaction).

Once a sperm fertilizes an egg, polyspermy is prevented by the cortical reaction, in which the egg's cortical granules create a new, impenetrable layer around the egg.

Developmental patterns

Germ layer formation begins with the first cell division in the newly fertilized zygote — on its way to becoming a multicellular embryo.

Through cell movements, blastula becomes a hollow sphere.
Next stage is an invagination of the hollow blastula, called gastrulation. This process makes a diploblastic embryo as it forms the first two germ layers: Ectoderm (on the outside) and Endoderm (on the inside).

In most (but not all) animals, the diploblastic embryo adds a third primary germ layer called mesoderm, becoming triploblastic. Mesoderm forms between ectoderm and endoderm, and arises by a process called Enterocoely. Enterocoely (mesoderm arises from endoderm) occurs in Deuterostomes.

The three primary germ layers go on to form all the other cell types and tissues in the animal. This is what happens in triploblastic animals:

- **Ectoderm**: Skin and nervous system
- **Endoderm**: Gut and associated organs and structures
- **Mesoderm**: Muscles, gonads, internal skeletons
Development requires increasing **differentiation**, from totipotent stem cells (can become any cell type) to full specialized cells with fixed **fates**.

Example: differentiation of muscle

<table>
<thead>
<tr>
<th>Time</th>
<th>Early mesoderm: muscle, bone, gonads</th>
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<tbody>
<tr>
<td></td>
<td>2gote: source of all cells</td>
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**Developmental patterns**

- **Acceolomates**: no body cavity; the space between ectoderm and endoderm is filled with mesoderm (mesenchyme)
- **Pseudocoelomates**: body cavity (pseudocoel) derived from the embryonic blastocoel; partially lined with mesoderm (the gut has no mesoderm)
- **Eucoelomates**: body cavity – the coelom – derived from embryonic mesoderm (completely lined with mesoderm)