Chapter 48  Animal Reproduction
Many animals can switch between asexual and sexual reproduction.

When sexual reproduction occurs, fertilization may be external or internal.

Egg development may take place inside or outside the mother’s body, depending on the species.
Two examples of asexual reproduction in animals. The advantage is speed!

**Budding in hydra**

**Fission in flatworms**
Asexual repro faster... but over long term less diversity in population

->

extreme individuals not so well adapted

some fitness parameter (say temperature tolerance)

++

individuals

Not a problem... unless weather patterns shift
**Cnemidophorous sp.**

So... all individuals in the population are haploid females!

Parthenogenesis → evolve secondarily an asexual mating scheme from a sexual mating pattern.

sexual → [haploid eggs -> diploid embryo]

Eggs produced via mitosis.

partheno. → haploid eggs → trick into development without fertilization

haploid adult

haploid embryo
In other animals, parthenogenesis makes diploid offspring (e.g., the crustacean *Daphnia*). *Daphnia* produce diploid eggs asexually.

Sexual reproduction is more common in crowded populations than in sparse populations.

And these guys can switch!!

*Probably*... crowding → stress → conditions are not favorable → asexual repro not the best strategy... better build some diversity into the population for uncertain future conditions.
Sexual reproduction is closely tied to the endocrine system.

There are three epochs in the life of a vertebrate where hormones are critical relative to reproduction.

1. Embryonic differentiation of sex organs
2. Transitioning from larva to adult
3. Control of gamete production
Embryo

Differentiation of sex organs according to the genetic sex of the embryo

Hormone influence #1

Hormone influence #2

birth

Hormone influence #3

growth

larva

adult

(biological definition)
Hormone influence #1

- Haploid (1N) egg
- Haploid (1N) sperm
- Zygote (2N)
- Many mitotic divisions
- Diploid single cell
- Embryo

A small # of cells are triggered to become future "germ cells". Diploid now, but will become haploid eggs or sperm.
Maternal hormone gets into fetal circulation

At about 7 wks for humans

Bruce

Δ = Chorionic gonadotropin

Embryonic gonad. Many cell types, incl. Leydig cells
Only XY Leydig cells make the hormone receptor

Note “T” is the abbreviation for testosterone

cholesterol

Testosterone (T)

cholesterol

Testosterone

XX Leydig cell

XY Leydig cell
DUAL
♀/♂
repro. syst.
@ 7 wks

♀

♀ ovaries
Fallopian tube
Uterus

♀ no testosterone
♀

♂

♂ testes
epididymis
vas deferens
prostate
semen vesicle

♂ + testosterone
♂
No testosterone → male developing structures do not get the necessary hormone signal and fail to continue development.

Female developing structures do not need the hormone...

So why don’t genetic XY embryos make both sets of structures?

Leydig cells when stimulated with CG also make Mullerian Inhibiting Substance (M.I.S.) that blocks development of Mullerian duct system.
Genetic XY embryo (♂)
castrate at equivalent of 7 wks
→ ♂ genitalia at birth

Genetic XX embryo (♀)
give T at 7 wks (equiv)
→ ♂ genitalia at birth
Note that testosterone effect is critical, but only at a particular time in early development. At birth both $\delta$ and $\varphi$ have zero $T$. 
End of hormone influence #1
(early development)
Hormone influence #2

Making an adult

Adolescent brain

Hypothalamus

GnRH
(Gonadotropin-releasing hormone)

LH
(Luteinizing hormone)

FSH
(Follicle-stimulating hormone)

Pituitary gland

Secretes both

Gonads

Testosterone

or

estradiol

Target tissues

(brasets in females, larynx in males, etc.)

Same in males & females
LH + FSH

males → testosterone

Growth, development of penis, prostate, etc.

Brain neurons develop testosterone receptors

Secondary sex characteristics (pubic hair, etc)

Sperm production

Egg production

Aggression

Territoriality

nesting

Brain neurons develop estrogen receptors

females ← estrogens

Growth, development of uterus, etc.

Egg production

Brain neurons develop estrogen receptors
<table>
<thead>
<tr>
<th>Stage</th>
<th>Boys</th>
<th>Girls</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (Prepubertal)</td>
<td>No sexual development</td>
<td>No sexual development</td>
</tr>
<tr>
<td>2</td>
<td>Testes enlarge</td>
<td>Breast budding</td>
</tr>
<tr>
<td></td>
<td>Body odor</td>
<td>First pubic hair</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Body odor</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Height spurt</td>
</tr>
<tr>
<td>3</td>
<td>Penis enlarges</td>
<td>Breasts enlarge</td>
</tr>
<tr>
<td></td>
<td>First pubic hair</td>
<td>Pubic hair darkens, becomes curlier</td>
</tr>
<tr>
<td></td>
<td>Ejaculation (wet dreams)</td>
<td>Vaginal discharge</td>
</tr>
<tr>
<td>4</td>
<td>Continued enlargement of testes and penis</td>
<td>Onset of menstruation</td>
</tr>
<tr>
<td></td>
<td>Penis and scrotum deepen in color</td>
<td>Nipple is distinct from surrounding areola</td>
</tr>
<tr>
<td></td>
<td>Pubic hair curlier and coarser</td>
<td>Pelvis begins to widen</td>
</tr>
<tr>
<td></td>
<td>Height spurt</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Male breast development (temporary)</td>
<td></td>
</tr>
<tr>
<td>5 (Fully mature adult)</td>
<td>Pubic hair extends to inner thighs</td>
<td>Pubic hair extends to inner thighs</td>
</tr>
<tr>
<td></td>
<td>Increases in height slow, then stop</td>
<td>Increases in height slow, then stop</td>
</tr>
<tr>
<td></td>
<td>Increased muscle mass</td>
<td>Deposition of fat in hips, buttocks, thighs, and breasts</td>
</tr>
</tbody>
</table>
End of hormone influence #2
Larval → adult transition
Hormone influence #3 – sperm & egg production.

1) Gametogeneis – general
2) Male gametogenesis
3) Sperm delivery system
4) Female gametogenesis
gametogenesis

**Spermatogenesis**

1. Spermatogonium
   - (May divide by mitosis to form more spermatogonia)

2. Mitosis

3. Primary spermatocyte

4. Meiosis I

5. Secondary spermatocyte

6. Meiosis II

7. Spermatids

8. Mature sperm cells

**Oogenesis**

1. Oogonium

2. Mitosis

3. Primary oocyte

4. Meiosis I

5. Secondary oocyte + polar body

6. Meiosis II

7. Ootid + polar body

8. Mature egg cell (ovum)
Sperm – inexpensive, made continuously, on-demand use.

Eggs – expensive, made intermittently and/or at slow regular intervals, and according to resource availability.
Who makes eggs, who makes sperm?
Hermaphrodites

--simultaneous: earthworms, snails.
Each individual has both testes & ovaries. Two individuals required for mating. Each fertilizes the other.

--sequential: some inverts, some fishes
Protogynous (female first, then male)
Protandrous (male first, then female)
Sperm production

Sertoli cells completely surround developing spermatocytes.
Differentiation of sperm cells -- this slide and next.

Sperm is foreign. Tight junctions between Sertoli cells prevent immune attack.
Cool drawing of sertoli cells at work
It takes 70-80 days to make sperm (humans), but production is continuous at about 200,000,000 per day.

Sperm maturation is faulty above 95 °F -- cremaster muscle raises/lowers testes to regulate temperature.
Swimming suppressed by inhibitory factors in epididymis & vas deferens

Epididymis - 6 m long... takes several days for transit
Vas deferens: sperm storage of 3 days - 1 month
Male Gamete Production & Delivery
An “intromittent” organ used by animals that have internal fertilization.
snakes & lizards

...have hemipenises – dunno which side you will end up on.
Human sperm delivery gizmo
Visual/Tactile stimulation

NO (nitric oxide gas) is a neurotransmitter released by motor neurons

- Relax smooth muscle on arterioles
- Increase blood flow into sinuses around cavernosus muscle

Contraction of vas deferens, prostate, seminal vesicle

- Fill urethra
- Pinch-off urethra
Positive feedback…

Urethra fills with semen

Activate pressure receptor neurons

Contraction of cavernosus muscle

Pressurize contents of urethra

Eventually urethra pressurization overcomes blood pressure block $\Rightarrow$ explosive release
<table>
<thead>
<tr>
<th>Source</th>
<th>Content</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Seminal vesicles</td>
<td>Fructose (a sugar)</td>
<td>Source of chemical energy for sperm movement</td>
</tr>
<tr>
<td></td>
<td>Prostaglandins</td>
<td>Stimulate smooth muscle contractions in uterus</td>
</tr>
<tr>
<td>Prostate gland</td>
<td>Antibiotic compound</td>
<td>Prevent urinary tract infections in males?</td>
</tr>
<tr>
<td></td>
<td>Citric acid</td>
<td>Nutrient used by sperm</td>
</tr>
<tr>
<td>Bulbourethral gland</td>
<td>Alkaline mucus</td>
<td>Lubricates tip of penis; neutralizes acids in urethra</td>
</tr>
</tbody>
</table>
Female Gamete Production & Delivery
Female gametogenesis

1. Formation of primary oocytes within follicles
2. Follicle growth
3. Maturation of follicle
4. Ovulation
5. Degeneration of corpus luteum

Secondary oocyte to oviduct

This is an ovary
Starting at puberty a single primary oocyte will develop each month.

Primary oocyte, in primary follicle, is diploid.

Meiosis I occurs to make haploid secondary oocyte. One polar body eliminated.

Secondary oocyte released from secondary follicle. Still arrested in meiosis.
How the polar body thing works

This is ovulated.

Fertilization occurs here.

This happens after fertilization.
Human females are born with about 400,000 diploid oogonia, all that will ever be made by mitosis.

Puberty-to-menopause is about 30 years x 12 oocytes per year \( \approx 400 \) oocytes that will develop.

Left ovary produces egg every other month

Right ovary produces egg every other month
Fallopian tube secretions dissolve mucous part of semen, wash out inhibitory proteins.

After 1-10 hours sperm become motile. Sperm can survive 1-2 days in female genital tract.
Meiosis completes at fertilization; polar body ejected. Occurs in Fallopian tube/oviduct.

Trip down oviduct takes 1 week (due to cilia beating), whether egg fertilized or not.

Ovulated oocyte only survives for 24 hours, so many oocytes in transit are duds.
Amazingly the recognition protein on sperm cells found in 2005 (Izumo1), and the matching protein on egg cells (Juno) found this year!

Fertilization fails in mouse egg cells lacking the protein.

A type of folate receptor (other folate receptors bind folic acid)
Tactile stimulation

- Vaso "congestion" → erection of clitoris & labia
- Vaginal lubrication
- Uterus elevates to form a depression at the back to receive sperm
At time of ovulation, cervical secretions become runny and more conducive to sperm swimming through. After ovulation, secretions are thick & pasty to block sperm. With successful implantation, secretions are so thick as to make plug completely blocking off uterus.
Time/day of ovulation…

…known to female in almost all vertebrate species.

…known to both males and females in many species.

Humans are rare exceptions in that neither males nor females are aware of ovulation.
Ovarian cycling

Feedback is negative (insufficient) at low levels, positive at high levels, negative at very high levels.
Peak in LH, FSH due to positive feedback

During luteal phase, CL secretes estrogens & progesterone

Estrogens stimulate growth of endometrium
CL has self-destruct mechanism activated - after 12 days CL-progesterone stops. This kills endometrium, allows the “insufficient” negative feedback condition (1) to resume.
Neg feedback has not yet kicked in.
B

Negative feedback $\rightarrow$ SLOW growth of follicle

Follicle estradiol $\rightarrow$ decrease sensitivity of AP to GnRH $\rightarrow$ reduce FSH, LH secretion by AP

Follicle growth slowed, but not stopped. So estradiol levels creep up.

Neg feedback phase: estradiol-FSH neg loop, LH minor player
Once a threshold level of estradiol reached

\[ \text{incr hypothalamus secretion GnRH} \]

positive feedback on hypothalamus via LH. Run away LH

ovulation

\[ \text{GnRH} \]

\[ \text{AP} \]

\[ \text{LH} \]

\[ \text{FSH} \]

\[ \text{estradiol} \]

\[ \text{follicle} \]

Pos feedback phase:
Estradiol-hypothalamus-LH/FSH loop dominates
Hypothalamus continues to secrete GnRH at low level. Progesterone feedback keeps GnRH low so new follicle does not start to develop.

After ovulation: ovum+CL

If no implantation

CL self-destruct

Progesterone levels drop

Remove inhibition on hypothalamus

GnRH levels increase, new follicle develops

LH loop dominates.

Adenohypophysis (anterior pituitary)
At ovulation...

1. Estradiol drops

2. Progesterone takes over

Corpus degen

Days

Progesterone

Negative feedback

LH

FSH
If no implantation:

- Ovum degenerates, corpus luteum degenerates.

- Progesterone levels drop
  - Remove inhibition on hypothalamus
  - GnRH levels increase, new follicle develops

- Corpus luteum self-destructs
  - Adenohypophysis (anterior pituitary)
How the egg gets out of the follicle

Estradiol increase just before ovulation

High estradiol induces granulosa cells of follicle to produce receptors for LH.

Binding of LH by granulosa cells…

1. decreases their adhesion.

2. switches on genes to make proteolytic enzymes to break down connective tissue of follicle.

Follicle falls apart
Not all ovarian cycles are the same...

copulation

Stimulate various brain regions

Induce hypothalamus to release large amounts of GnRH

(induced ovulation in cats, rabbits, minks)

(in some mammals repeated copulation required)
estrus vs menstrual cycles

**Nearly all mammals**

*Proestrus* – follicles grow

*Estrus* (heat, musth) – timed mating coincides with ovulation; female only receptive to male at this time

*Metestrus* – resorb endometrium

*Diestrus* – uterus diminishes in size

(cycle repeats)

**Old World Monkeys & humans**

*Menstruation* post-ovulation – discharge of endometrium
Female receptive to male at all times.
Fetus is very demanding re: oxygen!

At 30 mmHg, maternal Hb 60% saturated, fetal 85% saturated.