Consider this:

- Other species clearly manipulate offspring sex ratio - it is not "normal/typical."
- Physogastric mites, birds, whales, snakes...
  - but it does occur in a wide variety of species
  - AND
- Other species manipulate peri-natal environments
  - (birds manipulate maternal deposition of testosterone into yolk)
  
  **Start**

- Precocial birds: acoustic cues speed up hatching of last couple of eggs
- Altricial birds: last 2 eggs hatch 1 or 2 days late
  - but the last-hatched young are able to "catch up" by fledging
  - (being 1 or 2 days behind could be detrimental)

- Last laid eggs \( \rightarrow \) contain more T
  - Last hatched young are more aggressive
  - Begging for food \( \rightarrow \) helps them "catch up"

Regardless of offspring sex, mother deposits extra T into last laid eggs:

- Presence of egg T influences nestling behavior shortly after hatching

Any longer term consequences?

Remember:
- If it is a \( \Phi \) offspring, any negative consequences of early T
  - \( 2\Phi \Phi + 2\Phi\Phi \)
- If it is a \( \delta \) offspring, any positive consequences?
  - \( 2\delta\delta + 2\delta\delta \)
From a parent's point of view
Could there be selection for producing 7 embryos in the last 2 eggs rather than just 5/4 at random?
(How could parents skew offspring sex ratios?)

Now, back to the proximate influence of hormones
Organizational effects
Activational effects

Lots of good examples Ch 13 411-419

Read through the examples and note
1) imp env cue
2) specific hormone activated by env cue
3) behavioral effect/consequence/outcome

Consider activational effects of testosterone
- Some are obvious, some less so
- Some beneficial, some negative (trade offs)

High T is usually associated with reproduction, territoriality, aggression
(These behaviors can be induced by ↑ T in ♀♀, too)

Dunnocks - a polygynandrous species

Remember our terms for mating systems
Social monogamy - 1♂ 1♀ function as a pair to raise young  
(may or may not show genetic monogamy)

Polygyny - 1♂ mates w/ multiple ♀♀
Polyandry - 1♀ mates w/ multiple ♂♂

Polygynandry - looks like a chaotic free for all  
- everybody seems to have multiple mates

But, there are distinct strategies w/ the ♀♀:

In a given breeding season

low ♀♀ - tend to form solitary monogamous pairs
- receive good ♂ parental care assistance

high ♀♀ - exist w/ polygynandrous groups  
- ♂♂ try to mate w/ multiple ♀♀  
- ♀♀ try to keep extra ♂♂ away

♀ success depends on ♂ parental assistance, and
♂♂ are more likely to help if they are only ♀♀

In these groups, high ♀♀ are able to garner better/more ♀♂ parental care

Imp note: High T levels are imp. to stimulate/modeled all sorts of seasonal repro behaviors, but it is also imp. for T levels to drop  
- low T levels / gonadal regression
The pattern of testosterone secretion in free-living populations of Song Sparrows.
Plasma levels peak in April and May as breeding got underway and then were maintained at a lower "breeding baseline" during the rest of the breeding season. As prebasic molt ensued, plasma levels of testosterone were basal and remained so throughout autumn and winter.

**TESTOSTERONE**

Biological actions of the steroid hormone testosterone. The morphological, physiological and behavioral actions of testosterone that are essential for male reproductive function are given on the right hand and lower sides of the figure. The "costs" of prolonged high levels of testosterone are given on the hand side in italics. The patterns of plasma testosterone levels may be a function of secretion patterns to maintain male reproductive function, and "costs" of testosterone that require that plasma levels be low. From Wingfield et al. (2000).
Why? Why turn T/gonads on/off?
What are the costs of high T?
How is T production turned off/turned on?

1) What are the costs?

\[ ^{\uparrow}T \rightarrow^{\uparrow}T \text{met and energy consumption (even when at rest)} \]
\[-^{\downarrow}T \text{fat storage,} \quad ^{\uparrow}T \text{muscle mass (energy)} \]
\[-^{\uparrow}T \text{aggression,} \quad ^{\downarrow}T \text{parental care behavior} \]
\[-\text{unreproductive effects} \]
\[-^{\downarrow}T \text{corticosteroids (stress hormones),} \quad ^{\downarrow}T \text{immune function} \]
\[-^{\uparrow}T \text{elicits} \rightarrow^{\uparrow}T \text{elicits more aggression} \]

For example, \( ^{\uparrow}T \) barn swallows have red/rusty plumage
\[-^{\uparrow}T \text{higher T} \rightarrow^{\uparrow}T \text{plumage color intensity} \]
\[-^{\uparrow}T \text{more attractive to potential mates} \]
\[-^{\uparrow}T \text{aggression from other dominant} \quad ^{\uparrow}T \text{males} \]
\[-^{\uparrow}T \text{tends to be} \rightarrow^{\uparrow}T \text{elicits extra aggression} \]

So, T production comes with trade-off
2. organisms make strategic adjustments

But first - b/f searching for strategies is there
\[ \text{exist evidence that T causes these costs,} \]
\[ \text{not just a bunch of obs/correlations?} \]

Consider
House Sparrows - 3 discrete phases to the breeding cycle

1. Territory establishment - high aggression, high T
2. Mate attraction
3. Parental care - low aggression, low T

Assumption, T across season contributes to fitness

Experiment treatments → 3 groups of males

1. T implants - maintain high T
2. Flutamide implants - low T
3. Empty implants - normal T

Results:
- High T males - 2.6 yng/nest
- Low T males - 3.8 yng/nest
- Normal T males - 4.2 yng/nest

Plus lower survival for high T males during years

But here's another issue
- Northern pops have nice discrete, short breeding season
  (incur relatively few costs for high T)

But consider pops at equator → much longer breeding season
(At equator, HOSP breeding lasts for 8 months)

Not surprisingly
Continuous maintenance of territory
Multiple repeated breeding attempts
High levels of aggression/few parental care

\[ \text{How do \( S \)s survive the effects of high T for 8 months?} \]

- Not completely understood
- Basic obs: after early part of season (≈ 2 months), T levels drop, but

\[ \text{How are \( S \)' typical/high T' behaviors maintained/activated?} \]

\[ \text{Basic obs: \( S \)'s in these 707s have unusually} \]
\[ \quad \text{high levels of estrogen} \]
\[ \quad \text{AND} \]
\[ \quad \text{if estrogens are blocked with Fenrotide,}\]
\[ \quad \text{there is a decrease in \( S \)' typical behavior} \]

So, how does estrogen relate to \( S \) behavior in these 707s?
Does it activate \( S \) behavior do all the T costs?
If so, how?

Last issue - Aside from intrinsic feedback loops
and pineal activity, how else
are normal daily + seasonal activities maintained → Biological Clock/Rhythm

For Testosterone - important cues varied
- ↑ daylength / ↓ night → ↓ melatonin, ↑ T
- rainfall + food supply influences → ↑ T (phytochemicals)
- availability of mates → ↑ T
- \( \text{Chicken : case } \downarrow \ T \)
There is also the possibility that some of this cycling is "hard-wired."

- Consider extreme high latitude breeding
- Season is so short, migration is so essential
- Individuals need to start preparing for end of breeding season even when conditions are still good (long days, abundant food + mates)

Rather than utilizing env input for on/off cycling, these poor need to be hard-wired

**Biological Clocks** (**circannual + circadian rhythms**)

To a certain extent, we've talked about a variety of parts of the control process

- photoperiod, pineal gland, melatonin, FSH, LH, feedback

How does brain contribute to this?

So far, we've treated brain like "black box"

- inputs go in, responses come out

Can we be a bit more specific?

- nerve, endocrine brain, env. interaction

**Circadian Rhythm in Crickets** - good model system

"Simple" system b/c no pineal or analog of pineal
How do these organisms organize daily activities?

**Step 1** - Document that daily behavioral patterns exist

- Typically inactive during day, active searching for food at night
- Inactive during day, call for burrow at night

**Fig 13.5**

**Step 2** - What restricts/constrains/establishes behavioral pattern?

H₁: Hard-wired, 24 hour clock (env. independent)

Behavior starts up X hours after it stopped

→ X free-running - occurs w/o env. input

Not unreasonable given crickets are "simple" lack pineal

H₂: Env. dependence

- Neural mechanisms for calling & searching
  are activated/initiated as light levels drop

Both good ideas, but how can you test/evaluate H₁ vs H₂?

One manipulation: constant light

Under this condition, crickets call for about 4 hours, then go quiet - start up in ~20 hours

Good evidence for hard-wired, free-running clock (4 hours on/20 off; repeat)
Other manipulations
12L, 12D \(\text{crickets adjust the gap length}\)
14L, 10D \(\text{+ photoperiod so they}\)
16L, 8D \(\text{start singing at dusk}\)
\(\text{(not completely hard-wired to a 20 hour gap)}\)

Other manipulations
14L, 14D \(\text{Can adjust \(+\) non-24 hour days}\)
12L, 16D \(\text{(still start singing at dusk)}\)
12L, 18D

So, built-in/hard-wired free running clock, but it
can be over-ridden by env. cues

Fig 13.7
light/dark info is fed into optic regions/center
of brain to coordinate cycling
(\text{severed optic nerves, no input \(\rightarrow\) no adjustability})
\(\text{just free running}\)

"Clock" or "Pacemaker" must be in optic lobe

Given this basic cricket info, is there a vertebrate analog?

\text{Supra Chiasmatic Nucleus in hypothalamus (SCN)}

\begin{align*}
\text{blind the animal \(\rightarrow\) basic maintenance of 24 hour cycle} \\
\text{constant light \(\rightarrow\) basic maintenance of 24 hour cycle} \\
\text{variable photoperiods \(\rightarrow\) slight adjustments} \\
\text{damage to SCN \(\rightarrow\) no more pattern/irrhythmia} \\
\text{transplant fetal SCN \(\rightarrow\) restoration of normal cycling}
\end{align*}
For both verts + inverts, brain is "home" to clock/pacemaker (optic lobe, SCN)

Now, how does SCN maintain its 24 cycle? what is main output molecule/product of SCN?
how does it influence/communicate with rest of the body?

Brain is supercomplex, so it's like searching for a needle in a haystack w/ a little guidance

First - look for/document circadian activity in SCN (look for T1 in transcription-translate rate)

Fig 13.9 Rat SCN activity
2 impt genetic correlates - per (per = periodic) - tau

per gene codes for PER protein
- PER builds up + activates tau gene
- high [PER] causes decline in per gene activity

tau gene codes for enzyme to degrade PER
- as PER is degraded, ↓ PER activates per (per = periodic)

INNATE, HARD-wired 24h clock
- production, accumulating, degradation → takes about 24 hours
Fig 13.10 - same genes found in fruit flies + honey bees

- yng bees in hive - low perc activity / PER product (worker)
  - no circadian rhythm ble feeding
  - juveniles/larvae is constant

But, older individuals

- vigorous perc activity + PER product
- older ind. do have 24 hour of cycling behavior

This is a good example of a polyphenism

- occurrence of 2 (or more) inducible phenotypes

Different forms are result of gene activity / expression induced by env cues
(its not ble of different alleles)

So, honeybees switch from worker/rhythmic to adult/rhythmic
as optic lobe activity of per + tau start to oscillate

Go back to our 2 questions

1) How does SCN maintain its 24 h cycle?

2) What is main output molecule
   of SCN + influence/communicate
   with rest of the body?

Fig 13.8 - Does it contain via nerves?

Does it produce a chemical carried in circ. system?
Important product is **Prokineticin2/PK2** - circulatory system

**↑ PK2** stimulates sleep/suppresses wakefulness

Usual sequence/diurnal organism:

- **Dawn**
  - SCN $\rightarrow$ UPER $\rightarrow$ Tau $\rightarrow$ $\downarrow$ PK2 $\rightarrow$ wakefulness

- **Dusk**
  - Tau product causes $\downarrow$ PER $\rightarrow$ UPK2 $\rightarrow$ sleep

* These brain structures, genes, gene products establish the 24-hour free-running clock

* This clock can be shifted by environmental influence
  (Think of how you respond to jet lag)
  - Shift when your cycle starts

This is a nice proximate, mechanistic explanation of why an activity occurs in a particular way (immediate causation)

What controls behavior/How do animals do what they do?
- filters, tuning, signal transduction
- hormone production, feedback loops
- transcription, translation $\rightarrow$ genes for behavior

Remember
We don't always expect to find the direct genetic link for a behavior (per, tax, VNRT) but genes have a big causative effect/influence.

These genes have specific behavioral & fitness consequences, these genes (and their alleles) are targets of selection.

Another example - behavior, fitness, hormones, nerves, genes...

Ch. 11 Figs 11.24-11.25 Astatotia burtoni (egg-laying cichlid fish)

Populations consist of many breeding females and 2 forms of males:
- Few 0° males - dominant, aggressive, territorial → bright yellow
- Many 0° males - subordinate, non-fertile 0° males

Dominant yellow 0° males do not survive for long - high costs of elevated T ( Testosterone, T-cort, immune fit...)

So, when dominant 0° dies/disappears, he is quickly replaced by one of the subordinate 0° (it takes an individual about 5-6 days to switch from subordinate to bright yellow/fertile).

How does this occur? First - lack of a bright yellow 0° in population (bright yellow is inhibitory)
2) Preoptic lobe activity increases
   \[ \uparrow \text{transcription of erg-1 gene} \rightarrow \uparrow \text{ERG-1 protein} \]

3) \text{ERG-1 stimulus hypothalamus}

\[ \downarrow \text{yellow} \rightarrow \uparrow \text{ERG-1 and ERG} \rightarrow \uparrow \text{hypothalamic activity} \downarrow \text{CuRHH} \]
\[ \uparrow \text{Ant. Pit} \downarrow \uparrow \text{FSH} - \text{TSH} \]

Basic endocrine pathway is controlled by \[ \uparrow \downarrow \text{yellow, } \uparrow \downarrow \text{ERG-1} \]
\[ \uparrow \text{Testis, } \uparrow \text{testosterone} \]

\text{X Genes, Nerves, Hormones interact}
   \allow for appropriate behavior
   \text{at the appropriate time} \rightarrow \text{change from sub to dominant only at appropriate time}

Think of extir manipulation
    - If optic nerve is severed, then...
    - If optic lobe is disrupted, then...

\[ \text{Avoid costs of high T until there are benefits} \]

Separate (or maybe in addition to) from these immediate physiological/genetic controls/cause of behavior, there is also the issue of development of behavior \[ \rightarrow \text{Ch. 10 - 11} \]