The nuts and bolts of behavior- adaptations of sensory, CNS, and motor systems.

• I. Sensory systems influence behavior (sensory systems evolve to permit adaptive behaviors). Examples:

  – “seeing” in the dark
    • Bats, cetacean echolocation
    • Star mole somatosensory system
    • Honeybee vibrotactile communication
    • Electroreception

  – Navigating long distances
    • Magnetoreception
    • Polarized light detection
II. CNS adaptations to handle & facilitate interpretation of special sensory input; filtering to enhance detection of biologically relevant signals.
  - Bat auditory cortex
  - Somatosensory cortex non-linear mapping
  - ELLL
  - Feature detectors (& toad retina), IRMs
  - Ability to learn/memorize (from last lecture set)

III. Motor mechanisms to more efficiently produce common behaviors.
  - CPGs
    - Cricket calling
  - Command neurons
    - Cricket calling, escape behavior
    - Mauther cell escape behavior
Sensory “enhancements” can result from improvements of existing systems or the evolution of entirely new modalities or submodalities.

Some examples:

- Retinal enhancements for acuity, low-light vision
- Thermoreception in pit vipers
- Cochlear enhancements for ultrasound
- Jumping spider image forming eyes
Sensory systems influence behavior (or sensory systems evolve to permit adaptive behaviors)

... and those sensory systems would be:

Visual
Auditory
Somatosensory
Olfactory
Gustatory
Volmerolfactory
Nociception
Vestibular
Lateral line
Magnetoreception
Electroreception
(to name a few!)
Some sensory adaptations that enable behavior are enhancements to common sensory systems.

Example 1: the visual system of a hawk is similar to that of most vertebrates, but it is enhanced for better acuity at the expense of being able to see in low light conditions.
The fovea has highest density of photoreceptor cells, and therefore is the part of the retina with the greatest acuity.

In a mixed rod/cone retina, the fovea is populated by the smaller cones.
Rods, that can respond to lower light levels compared with cones, are found everywhere except the fovea.

Adaptations for low-light/nocturnal activity: more rods (no cones), tapetum
Adaptations for mixed diurnal/crepuscular activity: mixed retina as above
Adaptations for highest acuity: cone-only retina, extreme foveola (retinal pit)
Raptors have the highest acuity of any animal.
Example 2: Enhancements for nocturnal vision (at the expense of acuity)

The tapetum is a reflective layer behind the photoreceptor cell layer in the retina.

Photons that travel through the retina and are not intercepted by a retinal molecule in a rod or cone bounce off of the tapetum and reflect back for a second chance!

Since the reflection angle is not perfect, some scattering of photons occurs and this blurs the image slightly.
Animals with a tapetum (for example most non-primate mammals, some reptiles like Caiman) exhibit “eye shine” when a flash photograph is taken. Also readily visible with a flashlight.
Example 3: The enhanced auditory system of bats.

Bats have extended the basilar membrane in the cochlea so that higher-frequency sounds can be detected, and can be neurally processed with an extraordinary degree of precision.

This is an example of building an enhanced version of an existing sensory system. It is enhanced to perform a specific function very well!

To see how, first let’s look at the basic structure of the mammalian inner ear...
The basic anatomy of a mammalian cochlea
Low frequency sounds cause a traveling wave along the basilar membrane that peaks at the far end (apex). High frequency sounds cause a similar travelling wave but it peaks at the near end, and dies off quickly.

Human high frequency hearing extends to 20,000 Hz.

Rodents may extend to 60,000 Hz.

Bats, depending on the species, hear frequencies up to 120,000 Hz.
Bats have a number of special auditory adaptations, starting with the outer ear that captures sound energy... all the better to hear you with...
The inner ear is enhanced to extend the "normal" frequency range of a similar-sized mammal well into the ultrasonic range.
In some cases a sensory system is enhanced such that a new “sub-modality” evolves.

Example: The visual system of jumping spiders includes single-image-forming eyes analogous to vertebrate eyes.

The single-image-forming eyes have evolved in addition to the common arthropod compound eye (which the spider also possesses).
The common arthropod eye has multiple facets, each one is a complete photoreceptor. This provides a granular (but rapidly updating) image.
Several of the jumping spider eyes are analogous to those of vertebrates.
Diagram of the eye configuration in a jumping spider
Show video 1
From Dr. Natalie Vasey, PSU Anthropology:

Dear Colleagues,

I would like to let you know about the Primatology Field Methods courses I will lead over Spring Break 2010 and in May 2010 after semester courses end. Both will take place at the Lemur Conservation Foundation in western Florida. Please let your students and associates know about this great opportunity via the link below and the attached flyer.

http://www.lemurreserve.org/vasey.html

(The course flyer is posted on the animal behavior web site)
Primatology Field Methods
Spring 2010
Includes a week-long session at the Lemur Conservation Foundation’s Myakka City Lemur Reserve in Florida led by Professor Natalie Vasey of Portland State University
Session dates:
March 20th-28th – this session corresponds to Spring Break for quarter system campuses
May 17th-25th
Course fee:
$1675 -- includes field school tuition, lodging, meals, & ground transportation in Florida
Enrollment:
Learn more about the course and download an application at the website (http://www.lemurreserve.org/vasey.html). Information on how to achieve academic credit at the undergraduate or graduate level is also available online. Enrollment is capped at ten participants per session. Apply early!
Application due dates:
March session: December 20th, 2009
May session: February 15th, 2010
Sometimes an entirely **new sensory modality** evolves. (not just a submodality)

Example 1 of a special modality: The infrared detector system of pit vipers.

The pit organs provide a binocular I.R. image of the world in front of the snake.

The pit organ system is innervated by the trigeminal nerve, not the optic nerve, and projects to its own brain nuclei. It eventually is mapped into the optic tectum in register with the visual field.
The pit acts like a pin-hole camera to focus light onto a thermo-sensitive membrane.
Example 2 of a special (maybe) modality: The vomeronasal system

In addition to olfaction and gustation, most vertebrates have this special chemosensory system. Depending on the taxon, vomeronasal olfaction is used for mating behavior and mate tracking, prey tracking, and finding home.

The vomeronasal system includes a special sense organ, Jacobon’s organ, and special nerve branches to unique brain nuclei.

Jacobson’s organ in a snake receives chemicals captured on the tongue during a tongue flick.
Point for discussion…..

If vomerolfaction is a primitive, rather than a derived, condition for vertebrates is it fair to call it a “special” sense?

In fact, the sense is found in humans, but the influence or effect is small. Compared with other mammals, humans have lost a good deal of their chemosensory ability and instead become more reliant on vision and hearing. Thus a poor VNO is almost certainly a secondarily reduced condition. Any sense we do not have, or do not do very well, is considered “special purpose”.
Sensory systems evolve not just to be better at some task (e.g., more sensitive, handle a wider range of frequencies) - - they evolve to **enhance certain** signal parameters of interest to the organism and **reject less-relevant** parameters.

The enhancement of some signal properties and minimization of others is one kind of **filtering**.

**Examples:**

1. Ambient light level *vs* contrast enhancement in the vertebrate retina
2. Bug detectors in frog eyes
3. Contrast enhancement in vertebrate retinas
4. Gating in electric fish
Mach band “illusion” due to sensory filtering in the retina. The retina pre-processes visual information before it goes to the brain. In this case the filter artificially enhances contrast.

In some cases, the filtering can be extreme. The frog’s retina is an example…
All information in

Retina

Photoreceptors and information processing neurons in five layers

Only information of biological relevance out
Record: frog optic nerve

Electrode

More on this later

More object past frog: what gives best neural response?

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<thead>
<tr>
<th>Shading</th>
<th>Object</th>
<th>Background</th>
<th>Object Size</th>
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The frog’s retina filters the visual world and gives the frog’s brain only relevant information.

In this case, the only relevant information is the presence or absence of a bug.

Bug-detector neurons that respond only to small, dark moving objects on a light background are an extreme example of sensory filtering.
CNS mechanisms evolve to be very good at processing particular kinds of sensory data streams.

Example: pulse-echo delay mapping in the bat brain.

Bats use an “active” acoustic sense when echolocating objects.

It is active because the bat emits the sound (chirp) it then detects as an echo bouncing off a surface.

The bat’s central nervous system must process the time delay between the time the chirp is made and the time it returns to the bat’s ears.

Only echolocating mammals have neurons that compute chirp-echo delay, and these neurons are found in a special part of the brain.
In auditory cortex a systematic map of chirp-echo delay is made. There is quite a large amount of neuronal machinery devoted to this task.
Even in cases where a sensory system is enhanced (not a new modality) a great deal of extra CNS processing space can be devoted to that system.
So… CNS mechanisms exist to process specialized sensory information.

Not surprisingly, other CNS mechanisms have evolved on the motor side to more efficiently produce common behaviors, like fixed action patterns.

A good candidate for building a “boilerplate” neural mechanism is any repetitive behavior. Rhythmic activities are often produced by “central pattern generators” or CPGs.

Some examples:

- Walking
- Swimming
- Repetitive bouts of acoustic calling

Many CPGs are constructed of simple oscillators, which can be built from sets of reciprocally connected neurons.
In crickets, a command neuron in the brain controls or releases a ganglionic CPG that drives muscles involved in stridulation (how chirps are made).
This CPG oscillates at a fairly precise interval. Note the use of a frequency distribution to get at the variation in this behavior. This chirp would be considered highly stereotyped – the mean interval between chirps is 381 +/- 3.1 ms, or a variation of less than 1%.
Another CNS oscillator controls when the first command neuron is activated. That is, a hierarchical layering of control. The second oscillator operates on a diurnal cycle, slightly longer than 24 hours. When no sensory input is allowed to “trim up” the longer term oscillator (LL), it free-runs. Each trace below shows one 24-hour period. The vertical tics are bouts of calling.

**Teleogryllus: Endogenous Calling Rhythms**

![Teleogryllus: Endogenous Calling Rhythms](image)

from Lohr 1989

T.G. Forrest, [http://www.unca.edu/~tforrest](http://www.unca.edu/~tforrest)
A neural pacemaker sets the rate at which bursts of action potentials are produced by sonic motor neurons.
A classic vertebrate command neuron: The Mauthner Cell

The Mauthner cells (M-cells) control a simple CPG, a spinal network of just a few neurons that drive tail and body muscles during an emergency escape maneuver, the “S-start” or “C-start”.

Sensory input directly from many sensory systems activates the M-cell. The diagram in the next slide shows just auditory input from hair cells, though.
The Mauthner Cell pathway

Motor neuron

CPGs typically must be inhibited until they are needed, often the job of a command neuron.

This goes directly to the behavioral concept of the innate releasing mechanism.

Once “released”, that is, once the inhibition is removed, the CPG takes over and completes one or more cycles of oscillation.

So the IRM releases a complete bout of behavior, either a fixed or learned action *pattern*.

The releasing is effected by a command neuron.
A classic example of an innate releasing mechanism: gull chicks pecking parent’s beak to get food.

* Command neuron effectively disinhibits the CPG
The red spot on the yellow bill is detected by the visual system. A CNS releasing mechanism (AKA neural feature detector) qualifies the signal and releases a pattern generator to start pecking.
Note the subtle but important distinction between the “releaser” or releasing stimulus and the releasing mechanism.

The releaser is a key or sign stimulus, but the releasing mechanism is a neural process that receives input from recognition circuits and communicates with motor control circuits to activate (release) a behavior.
Releasing mechanisms can be very simple, requiring just one or two key stimulus parameters.

This means they are easily tricked!

Why do such vulnerable neural mechanisms persist?

- Processing a few simple key stimuli is faster, relative to CNS processing.
- Predators and parasites that do “code break” and deceive animals based on particular stimuli they are looking for will indeed drive an evolutionary response for the receiver to be more selective. If there is no code-breaking, there is no selective pressure to build a more elaborate recognition mechanism.
…and finally – nuts & bolts at a more fine-grained level:

What sort of signals do you see in the nervous system during singing (chirping), pecking, or sensing some external stimulus like a sound?

We need a little more basic neurobiology to understand Alcock’s figure on cricket chirping…
Basic neuron morphology and connectivity.
1. Sensory cells generate small graded electrical potentials. They are graded in that a larger stimulus produces a larger potential. The electrical potential is measured as trans-membrane voltage. Graded potentials last for nominally a millisecond and are a few millivolts in amplitude.

2. Some sensory cells (those that are true neurons) use graded potentials to make action potentials (APs). Action potentials are larger (40 – 90 millivolts) and have the advantage that they do not diminish with distance along a neuron’s axon.

3. Sensory cells that are not true neurons only make graded potentials. Such sensory cells communicate with true neurons. The true neurons almost always do make action potentials.

4. Action potentials also last for about a millisecond. Since APs are the same size in a given neuron, the magnitude of the sensory input is encoded by the rate of action potentials.
Stimulus

Graded potentials

Action potentials

Short hand action potentials

1 AP/second

2 APs/second

4 APs/second

↑ Stimulus magnitude
inputs to neuron become graded potentials

APs generated here

APs propagate along axon membrane

1 to 100 meters/second! (depends on axon)
Glass micropipette. At end A, diameter is nominally 1.0 mm. Pipette is "pulled" under high temperature such that glass melts. At end B, diameter can be < 1.0 μm!
To electronics

EXTRA cellular recording

Insulated coating

Metal tip ~ 100µm diameter

"artifacts" of action potentials from many neurons picked up at once
What happens when we make an extracellular recording from a motor neuron or interneuron while bat ultrasound chirps are played to the cricket?

BTW, this is text figure 4.13 and is described as a neural network. A neural network is not shown in this figure!
Different functional classes of action potentials can be identified in neural recordings. Note that the B cell APs are not correlated in time with the acoustic chirps, but the A1 cell is time-locked to the chirps.

BTW, the figure caption (figure 4.17) describes the top trace as a sonagram. It is not. Rather it is an “oscillogram”, or amplitude vs time trace.
The point is that we can identify specific neurons in the moth that have evolved for the singular purpose of allowing a behavior – avoidance flight when ultrasonic chirps are detected.
So now we know about action potentials in neurons, pathways that connect sensory neurons to interneurons (for integration/computation) and then to motor neurons.

With this information we can see an entire functional neural “circuit diagram” to account for a behavior.

One such well studied system is the escape response of the sea slug (a mollusk) *Tritonia*. 
In this case the entire neuronal circuitry from sensory input to motor output is understood.

There are several examples from both invertebrates and vertebrates where this has been done, but not too many!
The DRI swim initiation neuron is a command neuron. It “kicks” the CPG.
4.21 Neural control of escape behavior in *Tritonia*

The central pattern generator involves reciprocal inhibitory interneurons that cause bouts of action potentials in motor neurons. The motor neurons control muscles on alternate sides of the slug.
Command neurons cont’d... segue to chapter 5, neuroendocrine control of behavior.

Cricket stops calling (wing motion) when hit with air puff:
The nervous system controls behavior… but the endocrine system has a huge influence over the nervous system!

The endocrine system is best suited to control longer-term changes in behavior, for example, daily and seasonal changes.
As seen in an earlier slide, a circadian rhythm

Circadian behaviors are often controlled by a neuro-endocrine mechanism. How does it work??
You might think it is easy to decide when it is time to do something. In fact, this is not so trivial.

Which environmental cues do you use?

What if they are in conflict?

For short-term changes, the nervous system has to integrate information from multiple sensory systems and make a best judgement.

For longer term changes, it is best to couple an intrinsic oscillator with sensory input.
Environmental Influences on Behavior

* short term: It's starting to rain
  \[\text{Sensory system discovers this!}\]
  \[\downarrow\]
  stop basking behavior
  start hunting for shelter behavior

* long term: Hmm, I think the days are getting shorter
  \[\rightarrow\]
  brain would have to memorize day lengths & compare older with current
  \[\downarrow\]
  stop basking behavior
  start flying south
Asking the brain to compare the length of the current day with that of the previous day is a hard task!!!

Instead, we’ll take advantage of a much older and more primitive mechanism for long-term timing, the construction of a chemical ramp...
Use stable compound.
Gene to produce it constantly transcribed.
Constant enzymatic production at rate X. So predictable accumulation rate in cell.
when threshold reached, trip a second mechanism

transcription regulator

turn on gene 4

gene 4 product degrades timer chemical

reset ramp
long term

So we can make long-term chemical oscillation — but how coupled to environmental signal?

Let's say light is the signal.
As long as it is light out, a second chemical is expressed $\rightarrow$ binds with the cell making the timer chemical (or binds to another regulatory element) $\rightarrow$ speed up / slow down (slightly) T.C. rate.
Entrained rhythm: environmental signal (daylight) synchronized or "phase locks" chemical secretion.
In vertebrates, a major brain center controlling circadian activity is the suprachiasmatic nucleus (SCN):
OK... one more time here is our circadian pattern!!!

The PER gene mechanism runs independently of external influences but can be entrained to light/dark cycles...
1. **per gene**

2. **tau gene**

3. **tim gene**

At peak per production, more binding to tim & cry → less per breakdown by tau → more block → faster cycle reset.

More tau → less block → longer cycle.

**tau protein (CKI)** breaks down per protein.

Per protein temporarily blocks per gene (negative feedback).
Flies use almost the same mechanism as mammals.
SCN neuron levels of PER, etc, oscillate on a diurnal basis, but how does this influence other cells/tissues?

SCN neurons also make prokineticin 2 (PK2). The PK2 gene is clock-controlled and is probably the output molecule of the SCN. There are PK2 receptors expressed in all target tissues of the SCN.
SCN is entrained by light input via the retina, and via the parietal eye in some vertebrates.

SCN is also regulated in a hormonal feedback loop with melatonin:

Melatonin produced in the dark and turns down activity of SCN neurons. The mechanism for this is complex, and just one of several regulators.
Figure 5 | Signalling within a suprachiasmatic nucleus synapse and neuron. The suprachiasmatic nucleus (SCN) circadian clock is affected by light (photic cues) through the retino-hypothalamic tract (RHT) leading to use of glutamate (Glu) as a neurotransmitter. Glutamate receptor triggering induces various intracellular responses, leading ultimately to gene expression and phase shifts. Non-photic cues involve a variety of other neurotransmitter and signalling pathways (three of which are shown here). The photic and non-photic pathways can cross-regulate each other pre- and postsynaptically, this scheme is therefore an oversimplification.

- AC, adenylate cyclase; CRE, cAMP response element; ER, endoplasmic reticulum; ERK, extracellular signal-regulated kinase; GC, guanylyl cyclase; H, histone; ieg, immediate early genes (for example, c-fos); IGL, nerve terminus from the intergeniculate leaflet (thalamus); NMDA, N-methyl-D-aspartate; NOS, nitric oxide synthase; NPY, neuropeptide Y; PKA, cAMP-dependent protein kinase; PKC, protein kinase C; PKG, cGMP-dependent protein kinase.
There are also peripheral circadian oscillators in many tissues, at least circadian gene expression in liver, heart, kidney and pancreas.

These oscillators can be phase-shifted by feeding:

Lab experiments in which the time that experimental animals are fed is moved 12 hours. This changes the peripheral oscillators, but does not influence the SCN/CNS oscillator.

On the other hand, the peripheral oscillators are entrained by the SCN if there is no over-riding influence, like the change in food schedule.
What is the benefit of entrained short term oscillators?

Why don’t organisms just run at full speed all the time?

1. Need rest periods to maintain & restore tissue and organ function? ✗

2. Stay out of trouble when your sensory/motor systems are not optimal for finding prey or predators? ✓
Finally, note that not all animals have circadian oscillators.

If you live underground, not much point in attending to photoperiod!
longer behavioral cycles
- monthly / lunar
- circannual
  Seasonal adjustment
spring / summer
  food abundance
  favorable weather
  longer day length → longer
  more leaf cover
  foraging periods
  → smaller likelihood of predation
- synchronize with tides
- operate when have moonlight ... or when no moonlight to aid predators
Example of tide synchronization:

Make sure the tide won’t be so high again until after your eggs have developed – so lay at peak high tide of the month.

Grunion deposit eggs in beach sand during early stages of the ebb of higher high tides on the three or four days following maximum spring tidal range.

Flood tides erode sand and free grunion eggs during higher high tide as maximum spring tidal range is approached.

Maximum spring tidal range
From California Fish & Game website:

Grunion spawn only on these higher tides, and after the tide has started to recede. Since waves tend to erode sand from the beach as the tide rises and deposit sand as the tide falls, it is obvious that if grunion spawn on a rising tide the succeeding waves would wash the eggs out. This danger is eliminated since spawning usually is confined to the falling tide. In addition, grunion nearly always spawn on a descending series of tides when succeeding tides are lower than tides of the previous night. The eggs would be washed out prematurely by succeeding tides if spawned during the ascending tidal series. The eggs mature and are ready to hatch in about 10 days or about the time of the next series of high tides. Thus, spawning must take place soon after the highest tide in a series if the eggs are to have adequate time to develop before the next series of high tides. Looking at the tidal cycle, it becomes apparent that there are only 3 to 4 nights following the highest tide that spawning conditions are right, and it is on these nights that grunion spawn.
Everybody has the same idea!!!
… but how do you find the shore?

… or for that matter, how do you find the ocean if you are already on the shore?

Some interesting answers from turtle navigation.
Loggerhead sea turtles

Diagram from http://www.unc.edu/depts/oceanweb/turtles/
Hatchlings

- Turtles use light contrast to find the ocean; typically the ocean is brighter than the hills/coast behind. Although this picture shows otherwise, turtles usually hatch at night.

- Eggs moved from the east to the west coast of Costa Rica: Turtles were still able to go in the direction of the water, implying that turtles do not inherit a specific direction (Carr and Ogren, 1960).

Adult ♀ making ▶️ west
Finding Open Ocean

The hatchlings use wave direction to navigate out into pelagic waters.

Facing waves:
- up → back
- forward < down

With waves:
- up → forward
- back < down

http://www.unc.edu/depts/oceanweb/turtles/
Open ocean migration

• Deep open water - cannot use waves as a navigation tool.

• First orientate towards light, then use magnetic fields to keep going in that direction.

• Reversing the magnetic field after the initial orientation causes the turtles to turn around.

• Latitude recognized by detecting differences in magnetic field inclination and intensity.

(Lohmann and Lohmann, 1994)
Turtle references


• Lohmann Lab Website http://www.unc.edu/depts/oceanweb/turtles/

• Caribbean Conservation Corporation and Sea Turtle Survival League http://www.cccturtle.org/

Photo courtesy Blair Witherington
For circannual oscillator → how to synchronize?

Ans: A seasonal cue

- Temperate zones
  - Day length
  - Temperature
  - Food availability

- Tropical zones
  - Rainy season
  - e.g. breeding in weakly electric fish
Eignemannia virescens

This weakly electric fish uses its electrosensory system to detect water conductivity. When the rainy season starts, conductivity drops and this is the cue for breeding!
How are seasonal cues processed?

... some sensory cue

→ nervous system

→ neuro-endocrine system

Δ hormone level → good long-term signal system

→ feedback to brain neurons

Δ behavior

Δ social interactions
BUT → → If we seasonal cues, why need oscillator??

Many behaviors need to be ready to go as soon as conditions are favorable.

So... start a developmental cycle ahead of environmental cue in anticipation of sensory input.

Sensory input → accelerate on-going process... faster readiness.
still... not all behaviors that change circannually are driven by internal oscillators.

→ How to test??

1. Control [environmental variable] + see if behavior still cyclical

   - photoperiod
   - temperature
   - food availability
(2) Measure physiological variable assoc. with behavior

  \[ \text{lion's share of circannual behaviors have to do with} \]
  \[ a) \text{migration : food/nesting} \]
  \[ b) \text{reproduction directly} \]

measure hormone levels assoc. with reproduction - FSH
  - LH
  - estradiol
  - progesterone
If you provide lots of food at all times, and keep temperature constant – you still get change in a parameter associated with reproductive behavior ...implies that day length cues an oscillator.
Golden-mantled ground squirrels maintained in constant darkness, temperature, food availability. Shows that there is an intrinsic **long-term** oscillator.
If there is an internal circannual oscillator, how do environmental cues synchronize it??

Nice experiment with White Crowned Sparrows (see text pg 157-158) shows that the oscillator controls sensitivity to light level.

When daytime coincides with a period of light sensitivity, reproductive development is triggered.

The oscillator is reset every day at dawn. Light sensitivity occurs 16 – 20 hours later. Will it be light out 16-20 hours after dawn?
White Crowned Sparrow circannual synchronization

(see figure 5.17)

only long days trip mechanism

Summer daylight
Winter daylight
Light sens.

oscillator reset at dawn

light level req'd for activation
What the oscillator controls...

- Environmental synchronization
- Change in hormone levels
  - Change in behavior: territoriality, aggression, mating, parental care
- Oscillator
Counting mechanisms

Artificial day length manipulation changes when mouse loses tendency for infanticide. It is the number of cycles post copulation, and not the “real” amount of time that has passed, that controls behavior?

→ Infanticide stops on day 22 for slow days and day 22 for fast days → counting

Day length manipulations make 22, 24, or 27 hour days.
Interpretation of previous slide:

Probably a stable chemical produced, one burst per day/night cycle. The rate of accumulation corresponds to the # d/n cycles, not on the absolute amount of time that has passed -- shown graphically on next slide.
The behavior match is with number of light/dark cycles since mating, not the “real” days since mating.

(B) Males committing infanticide (percent)

“Real” days since mating

1 16.3 20 24.8

“Fast-day” males

“Slow-day” males

Light–dark cycles since mating

1 18 22
What physiological mechanism does the counting mechanism operate, that controls infanticide?

RZ’s hypothetical model next two slides...

Extra credit alert:

Disprove model and/or find out what is really going on.
Neuron 1 excites neurons 2 and 3.

Neuron 3 is active (= infanticidal) if it binds Progesterone (P).

Neuron 2 inhibits neuron 3, but the strength of inhibition increases with binding of regulator A.

After n L-D cycles, N2 blocks activity in neuron 3.
Progesterone is produced in breeding season, and causes infanticide unless blocked by neuron 2, the activity of which is controlled by the level of regulator A.

Knockout mice lacking progesterone receptors are not infanticidal.
Progesterone receptor knockout mice (PRKO) do not show aggression toward their newborn offspring. (Why the w.t. does is not known!) So failure to detect hormone leads to change in behavior.

Alternatively, the number of receptors for a hormone could increase for each d/n cycle.

![Bar chart showing infanticide percent for C57BL/6 and PRKO](chart)

- **First litter**
- **Second litter**

<table>
<thead>
<tr>
<th>Infanticide (percent)</th>
<th>C57BL/6</th>
<th>PRKO</th>
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<tr>
<td>0</td>
<td>(0)</td>
<td>(0)</td>
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Progesterone receptor knockout mice (PRKO) do not show aggression toward their newborn offspring. (Why the w.t. does is not known!)

So failure to *detect* hormone leads to change in behavior.
In general, sex steroids (testosterone, estrogens) bind to brain neuron receptors and control a great deal of aggression and mating behaviors.

You must exercise caution, though, when interpreting whether a reproductive pattern is “associated” with hormone levels or not. See text pg 164, and next two slides.
According to Alcock, the anole reproductive behavior is “associated” with gonadal hormones while that of garter snake is not. Do you agree?
1. Association is not the same thing as causation. The snake hormone levels do seem to be correlated. Is this sufficient for “association”?

2. It is possible that the rise in hormone levels after mating prime the snake for the next round of mating, or for another hormone-controlled behavior associated with mating.

   Arguably, point 2 is not very likely, but still suggests caution in interpretation!
Indeed, testosterone levels are “associated” with birds that have one mating cycle in a season. For those that have two mating cycles --- testosterone is uncorrelated!!
Other cases for association are more clear...

Clearly there are species differences in the endocrine control of behavior, even in closely related species.

Again, it is best to show causation, not just correlation.
One confounding variable: testosterone is not the only hormone at work during the breeding season. Estrogen inhibitors block song production and territorial distancing.
Ideally, from an experimental perspective you would like to show that an endocrine regulator of behavior is both necessary and sufficient (as below). Since there can be complex interactions and multiple factors, often both criteria are hard to meet.

Guinea pig copulatory behavior
In many cases, brain neurons have steroid receptors throughout adult life, and binding of the appropriate hormone is necessary to maintain tonic activity in the neuron, or even to keep the neuron alive.

So hormones are not necessary just to trigger behaviors at the right season, etc, but can be necessary for the long-term maintenance of a behavior.

[Graph showing percentage of males courting receptive females over three years, with intact males in blue and castrated males in red.]

Garter snakes again