

Highlights from Pesticides Lecture

- ✓ Prior to World War II pesticides were _____, while post-WW II they were _____.
- ✓ What is meant by the biomagnification of pesticides and what are its consequences?
- ✓ Differentiate between acute and chronic pesticide toxicity.
- ✓ Define the term LD_{50} .
- ✓ Provide two major consequences of the wide-spread use of Mirex (chlorinated hydrocarbon) to control fire ants.
- ✓ Characterize chlorinated hydrocarbon insecticides (DDT and its relatives).
- ✓ What is meant when it is said that the use of pesticides is contextual?
- ✓ Define the term naturally-occurring inorganic pesticide and provide an example.

Highlights from Biological Control Lecture

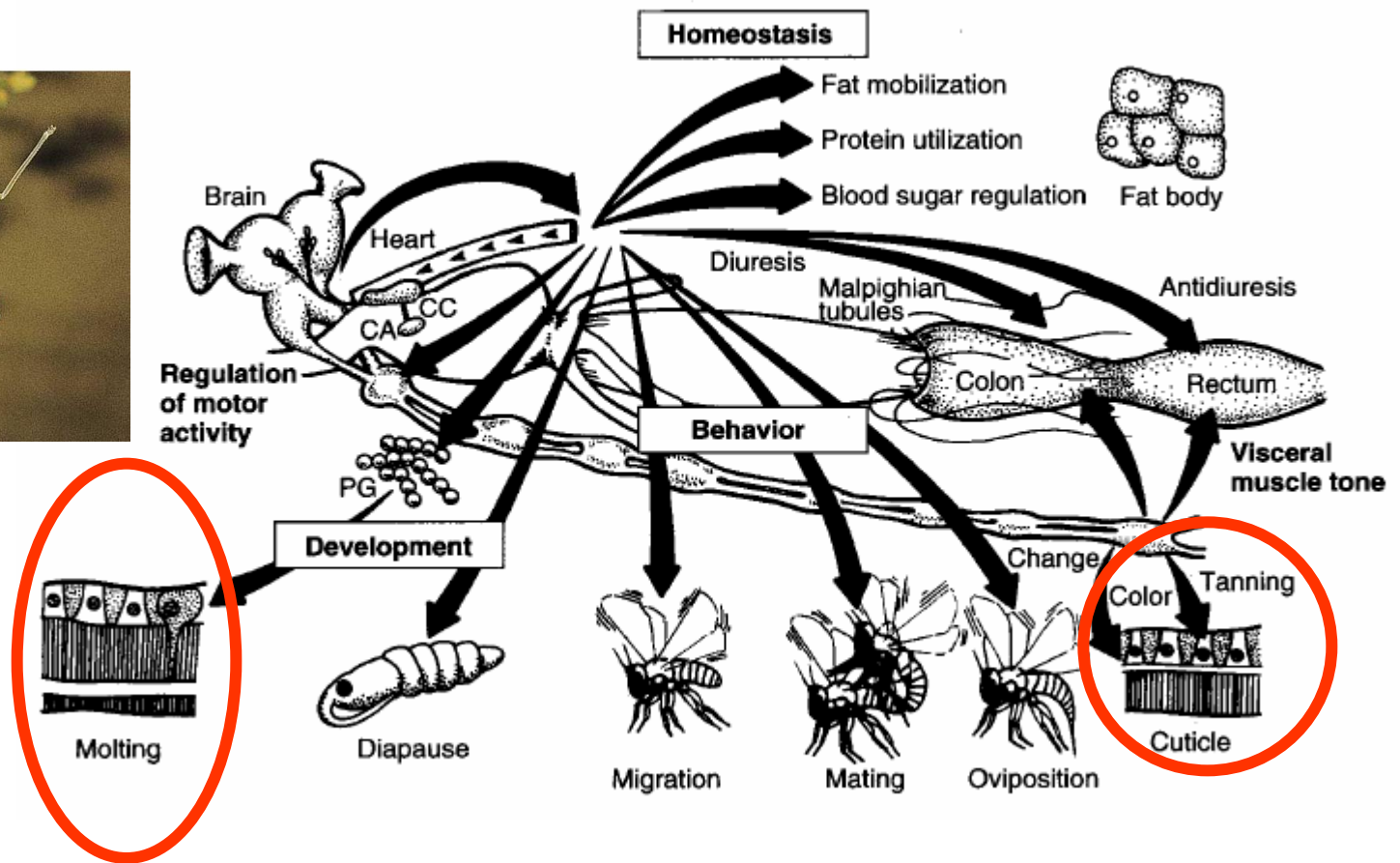
- ✓ Define the three types of biological control and provide an example of each.
- ✓ Contrast predators and parasitoids.
- ✓ What is a density-dependent mortality factor?
- ✓ How has the introduction of the soybean aphids affected pest management in the Midwest?
- ✓ Name one untoward effect related to controlling tamarisk trees on the Colorado River.
- ✓ What is meant by an inoculative release of a biological control agent?
- ✓ How do pesticide economic thresholds affect biological control programs?

Lecture 19. Endocrine system II

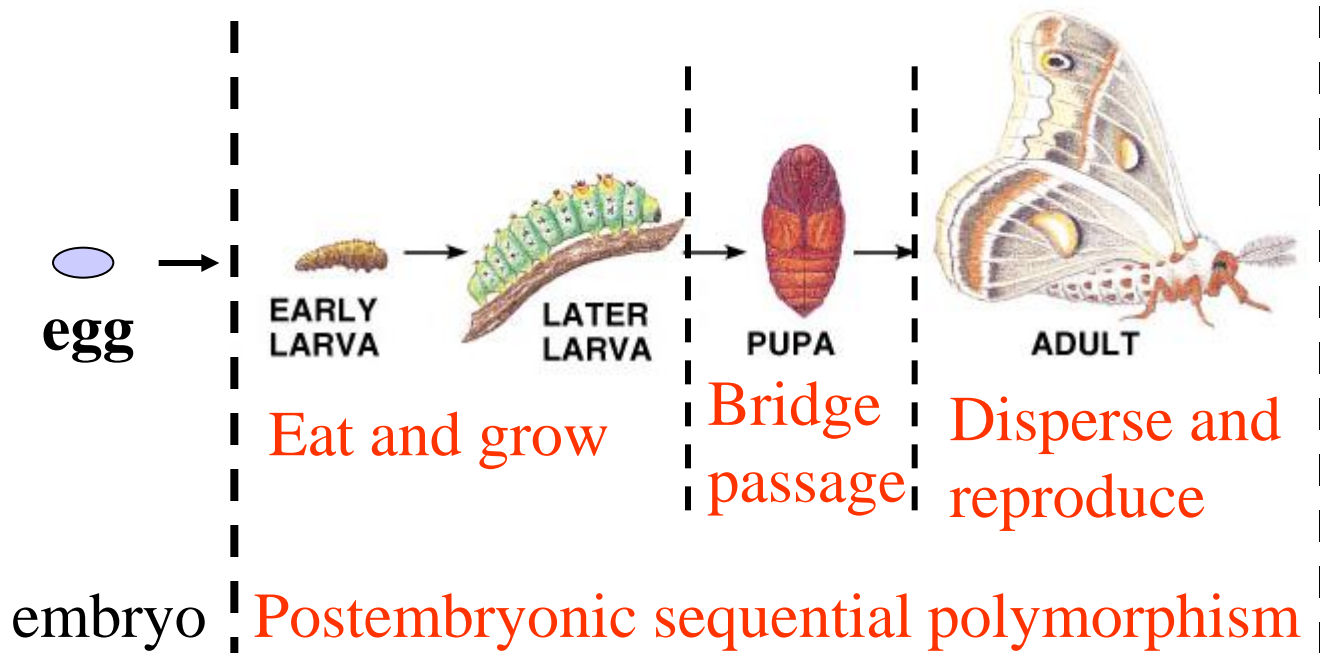
Physiological functions of hormones

- Anatomy
- Hormones: 14
- **Functions**

Functions of insect hormones: diversity

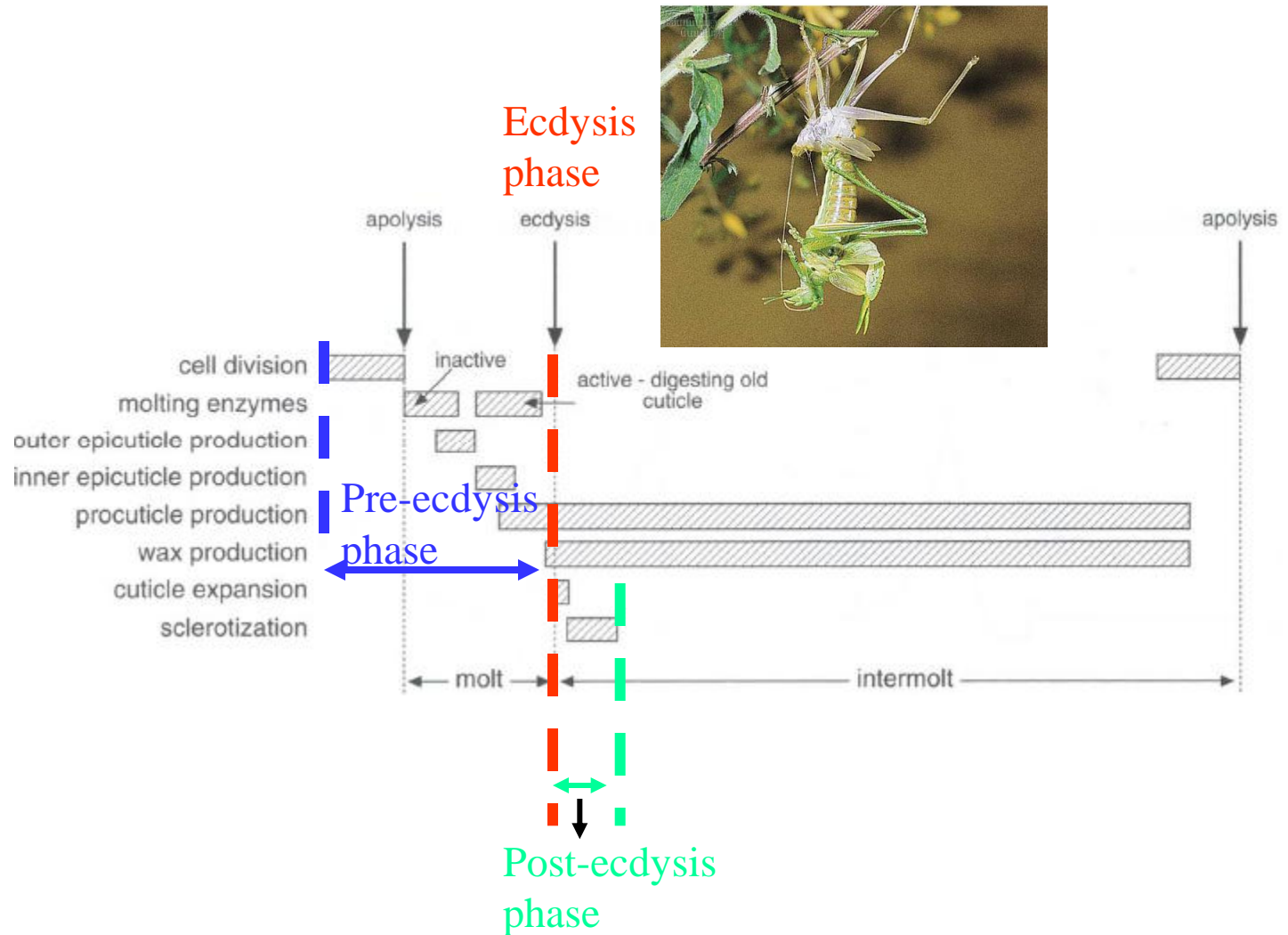


Hormonal functions: Molting as a paradigm



- Nature of molting, growth or metamorphosis?
- When and how to molt?

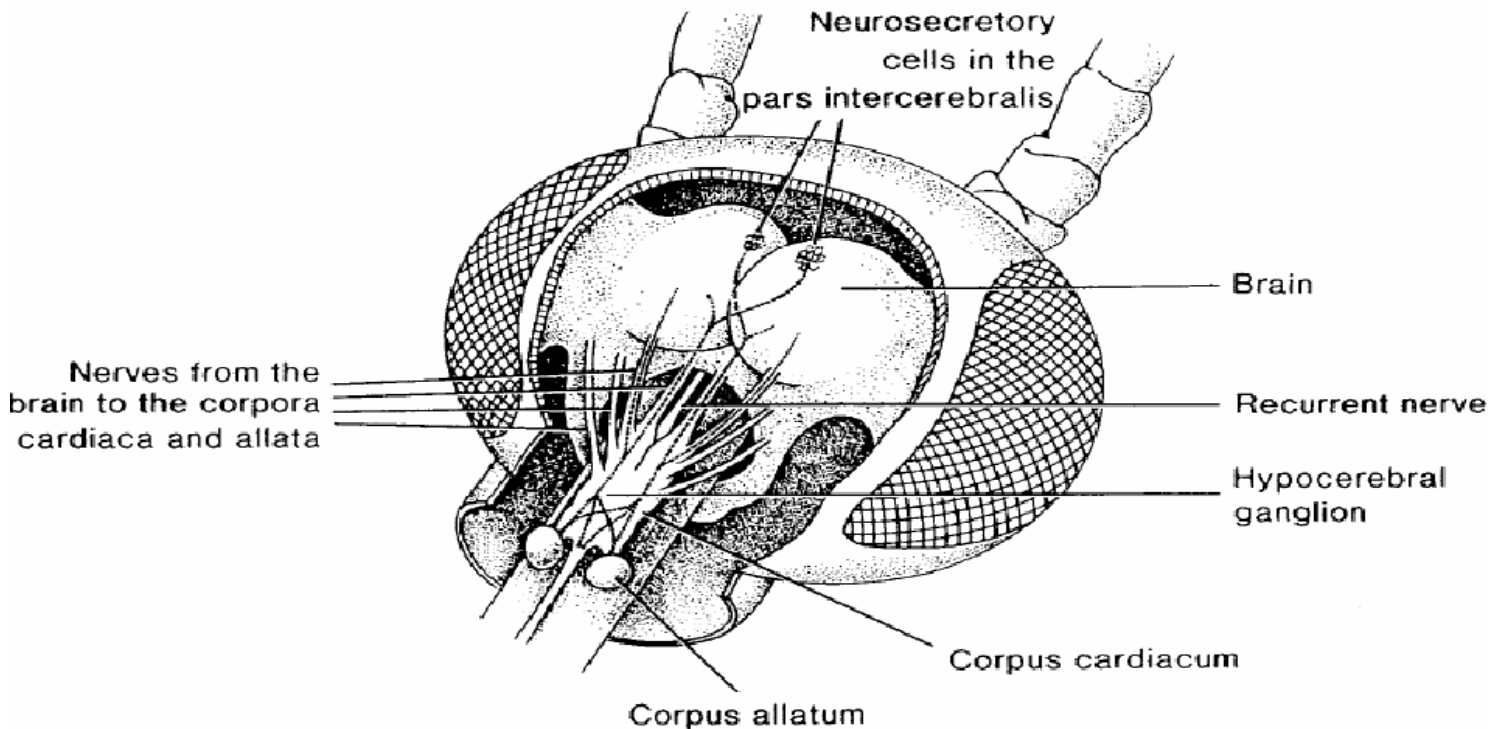
The molting process



Overview

- About 90 years' study (1917-2000): 7 hormones are involved in regulating molting / metamorphosis
- 3 in Pre-ecdysis preparatory phase: the initiation and determination of new cuticle formation and old cuticle digestion, regulated by PTTH, MH (Ecdysteroids), and JH
- 3 in Ecdysis phase: Ecdysis, i.e. shedding old cuticle, regulated by EH, ETH, and CCAP
- 1 in Post-ecdysis phase: Tanning, i.e. hardening (sclerotization) and darkening of new cuticle, initiated by bursicon

Pre-ecdysis phase : Kopec's brain removal experiments (1917)



The gypsy moth larval pupation

Three surgical treatments : remove larval **brain**, **SG (Subesophageal ganglion)**, or **surgical control** on day 7 and 10.

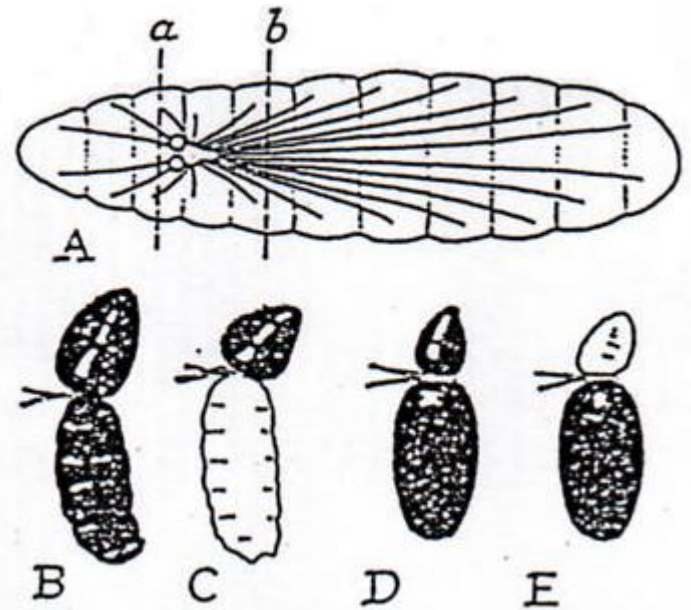
Pre-ecdysis phase: Brain is required

Surgical treatments	Pupation rate	
Day 7, Brain removal	5/25	20%
Day 10, Brain removal	12/15	80%
Day 7, SG removal	12/13	92.3%
Day 10, SG removal	7/8	87.5%
Day 7, brain removal control	13/16	81.3%
Day 10, brain removal control	9/10	90%

Conclusion: brain is an organ of internal secretion;
There is a “critical period (CP)” for brain activity

Pre-ecdysis phase: Brain is required (Fraenkel, 1934, *Nature*)

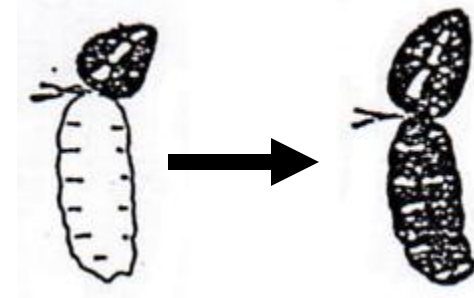
- Blowfly (*Calliphora erythrocephala*) pupation.
- Four ligature treatments:
 - B--ligature at position **b** and **after CP**, both parts pupated
 - C--ligature at position **b** and **before CP**, only the anterior part containing brain pupated
 - D--ligature at position **a** and **after CP**, both parts pupated
 - E--ligature at **a** and **before CP**, only the posterior containing brain pupated



**Conclusion: brain is an organ of internal secretion;
There is a “critical period (CP)” for brain activity**

Pre-ecdysis phase: brain secretion is a hormone (Fraenkel, 1934, *Nature*)

- **Experiments:** injection of hemolymph from a pupating larva into the posterior part of a larvae ligated at the position **b before CP**.



- **Result:** back end pupated
- **Conclusion:** brain is an organ of internal secretion in fly too; “critical period” exists for brain activity; signal is definitely **blood-borne, i.e, true hormone**.

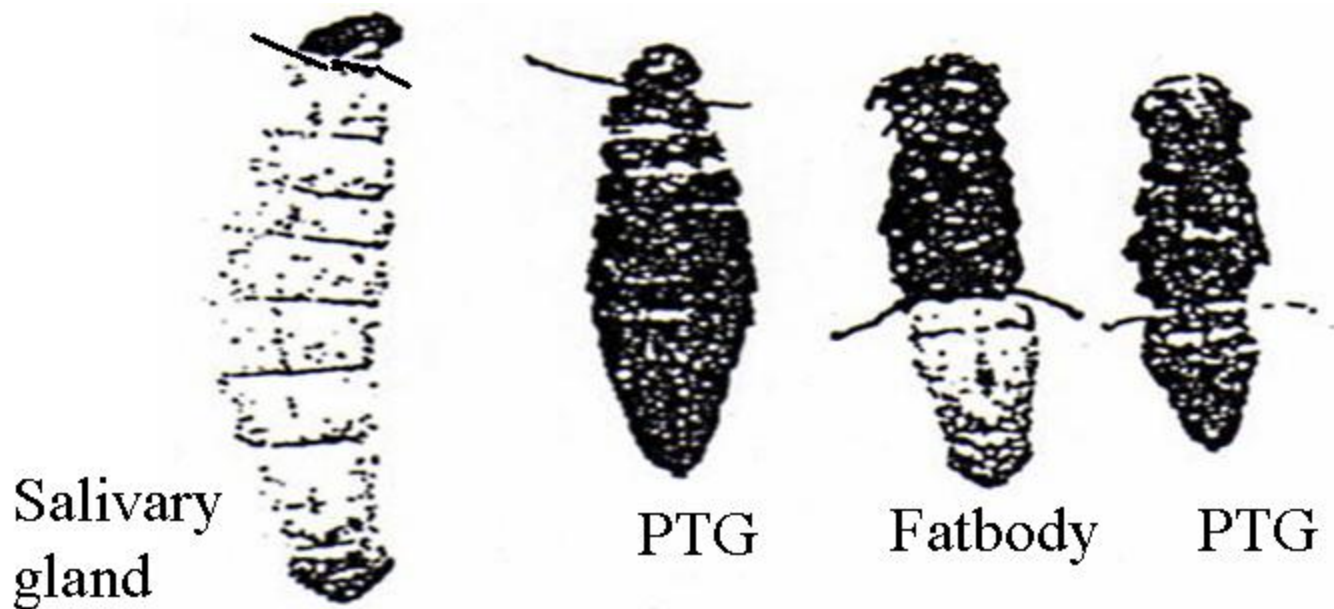
Pre-ecdysis phase: PTG is required (Fukuda, 1940)

- The silkworm (*Bombyx mori*) larval pupation
- Implant PTG (prothoracic gland), salivary gland, or fatbody from pupating larvae into the 9th abdominal segment of day 2 5th instar larvae
- ligature six days later



Soichi Fukuda

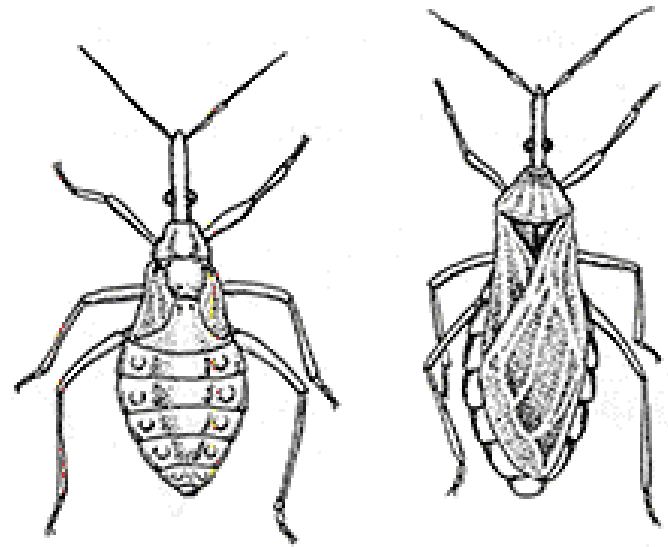
Pre-ecdysis phase: PTG is required (Fukuda, 1940)



Conclusion: silkworm need both the PTG and brain to molt.
This initiates a controversy: which is really important, the brain or the PTG?

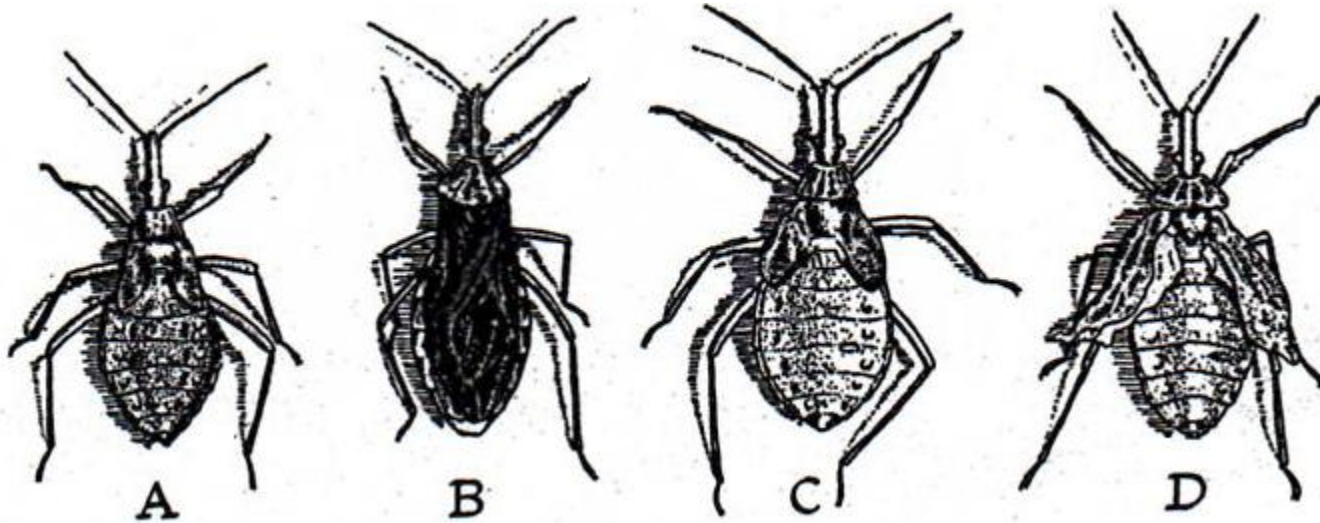
Pre-ecdysis phase: Wigglesworth and the “kissing bug”

Sir V.B. Wigglesworth



Rhodnius prolixus

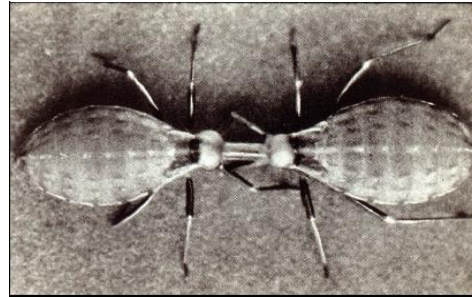
Pre-ecdysis phase: CA prevents metamorphosis



A=normal 5th instar nymph of *Rhodnius*, B=Normal Adult
C=6th instar produced by **implanting** a CA from 4th instar into the abdomen of 5th instars

D=similarly-produced 6th instar, with larval cuticle all over abdomen.
Wings & thorax are intermediate between larva and adult (**adultoid**)

Pre-ecdysis phase: CA sensitive time coincides with brain CP

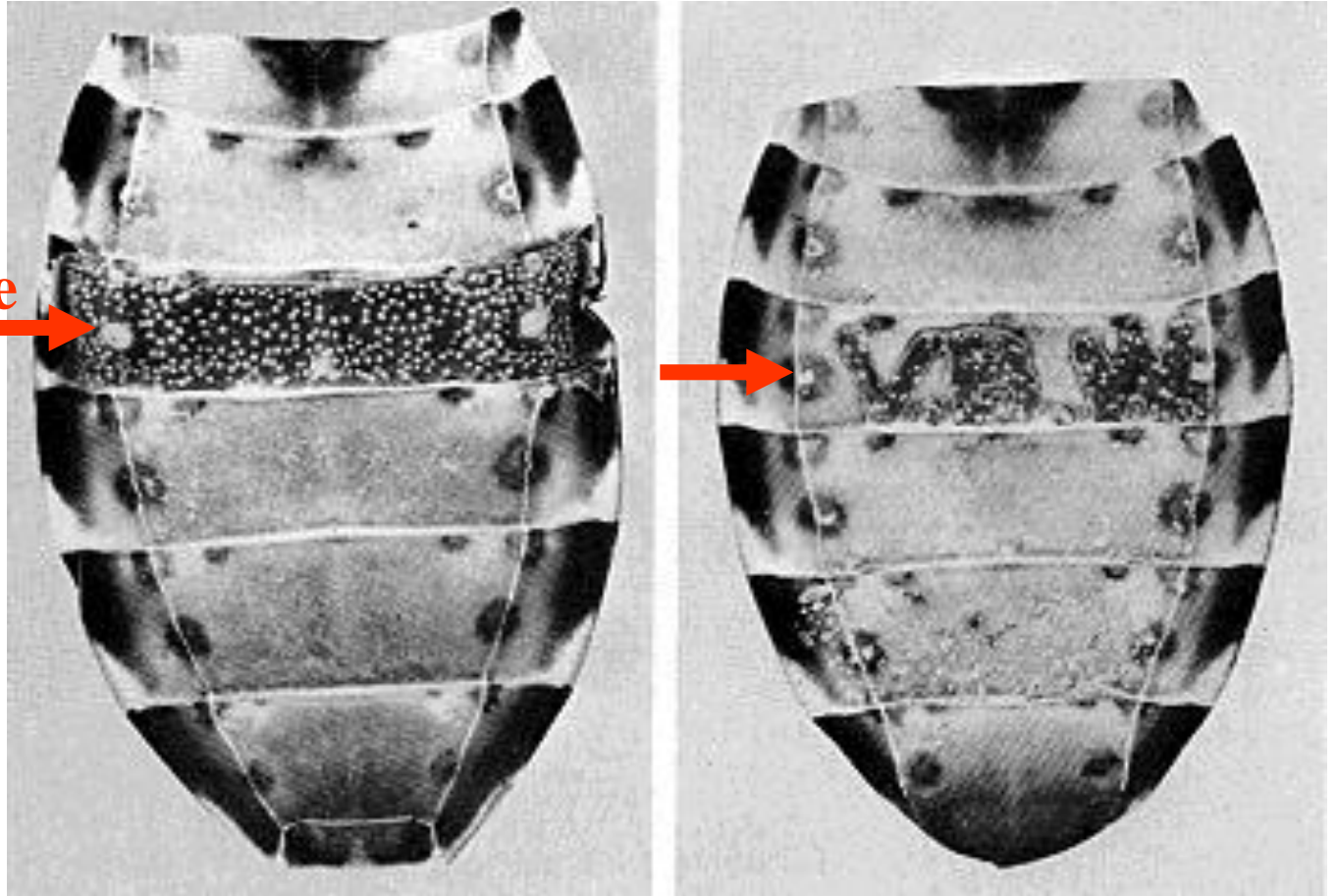


Decapitation & parabiosis

Insect A (before brain CP)	Insect B (after brain CP)	Results at next molt
4 th instar	5 th instar	A,B=adults
1 st instar	5 th instar	A=tiny adults B=adults
5 th instar	4 th instar	A, B=adultoids
5 th instar	1 st instar	A, B=larvae

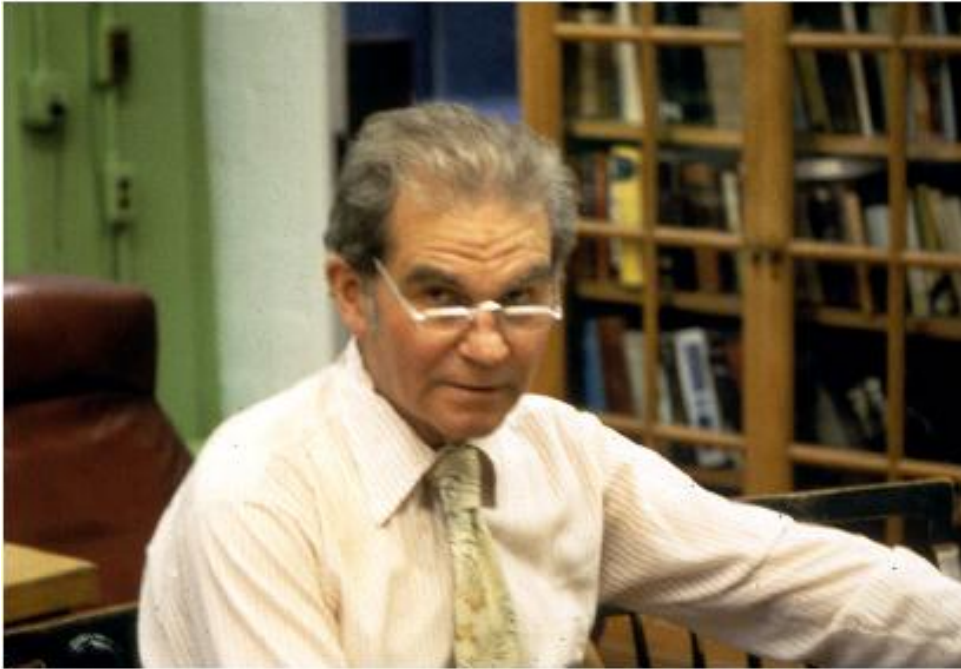
Pre-ecdysis phase: JH maintains larval characteristics

Apply JH here
At 5th instar



Pre-ecdysis phase: Williams's tissue implant experiments (1950)

Carroll Williams



Hyalophora cecropia
diapausing pupae requires
a period of low temperature
to molt



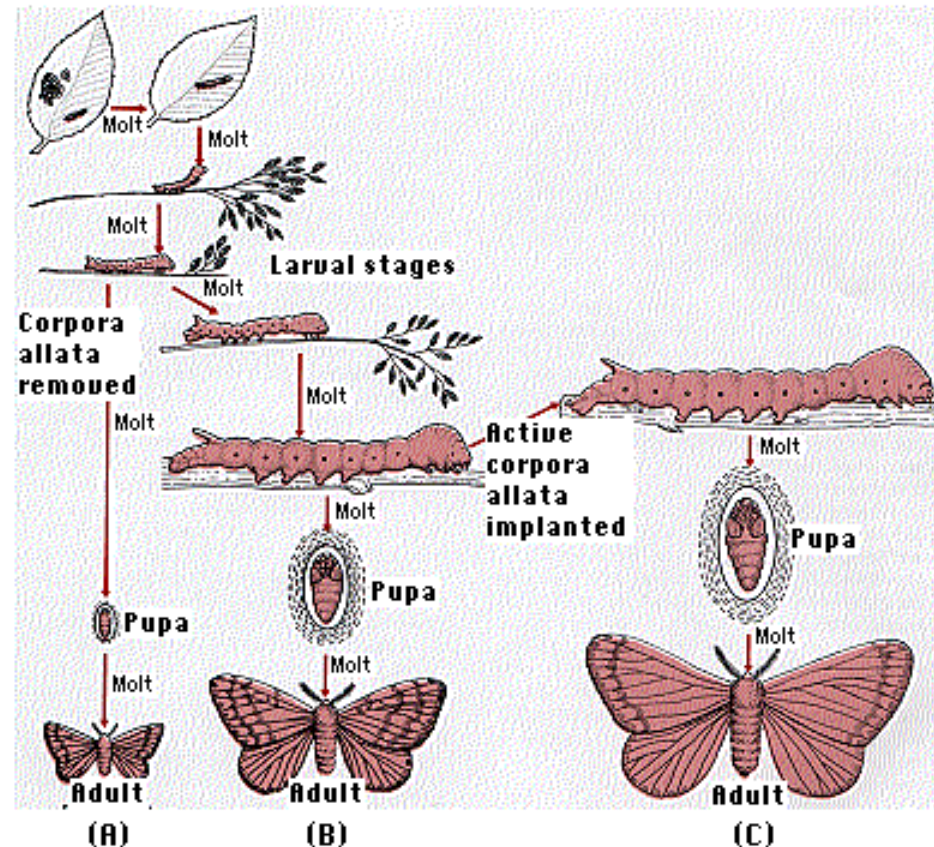
Pre-ecdysis phase: Williams's tissue implant results

Treatment	Result
Isolated pupal abdomen	Doesn't develop
Add a brain	Doesn't develop
Unchilled PTG	Doesn't develop
Chilled PTG	Doesn't develop
Chilled brain + PTG	Adult abdomen develops
Chilled brain + PTG + CA	2 nd pupal stage forms

Experiment: implants of tissues into isolated pupal abdomen.

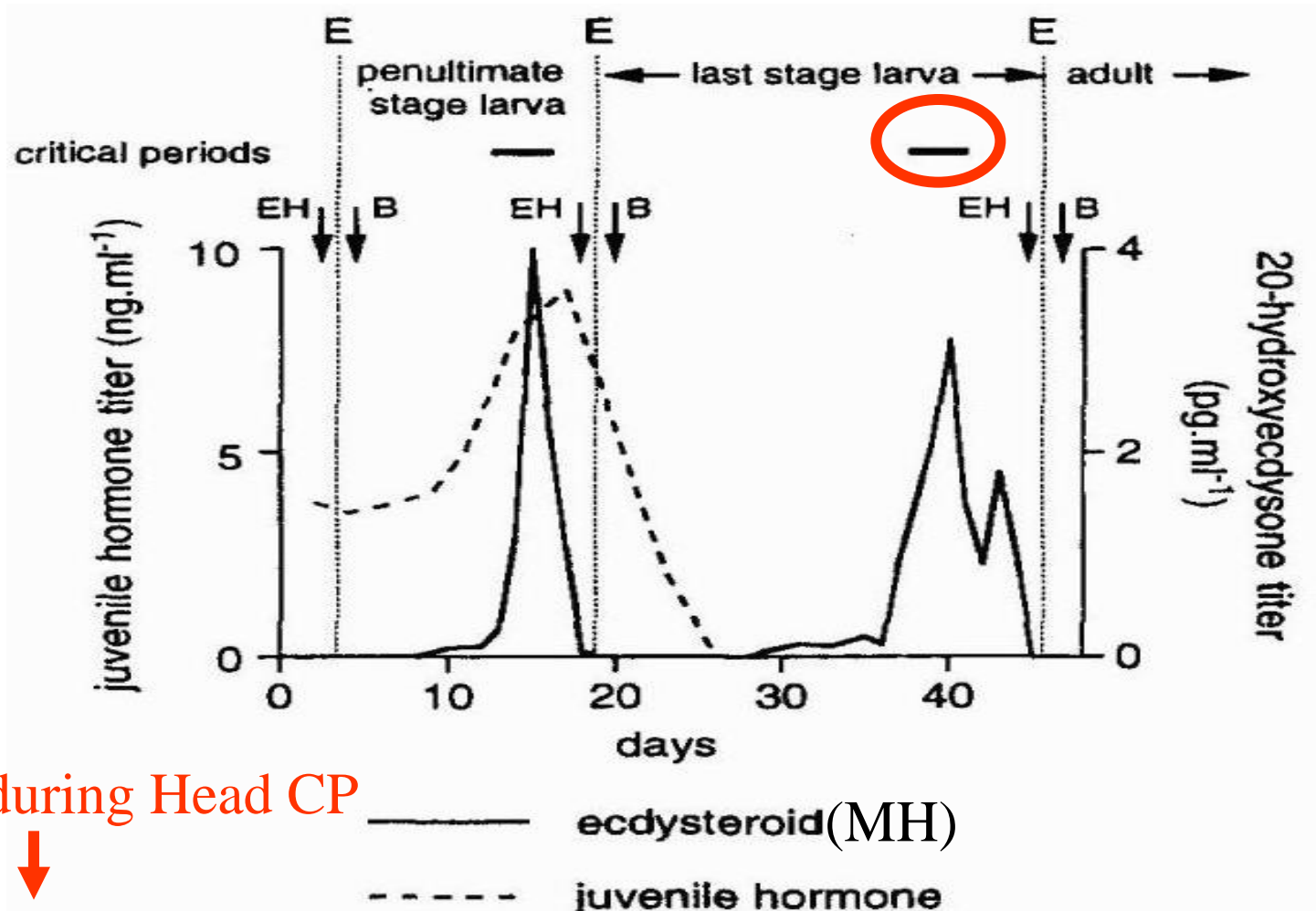
Conclusions: 1. brain is needed to “drive” the PTG
2. CA make a “peter pan” or “status quo” hormone

Pre-ecdysis phase: CA produces a juvenile hormone



Bombyx mori

Pre-ecdysis phase: hormone titers in hemimetabolous insects



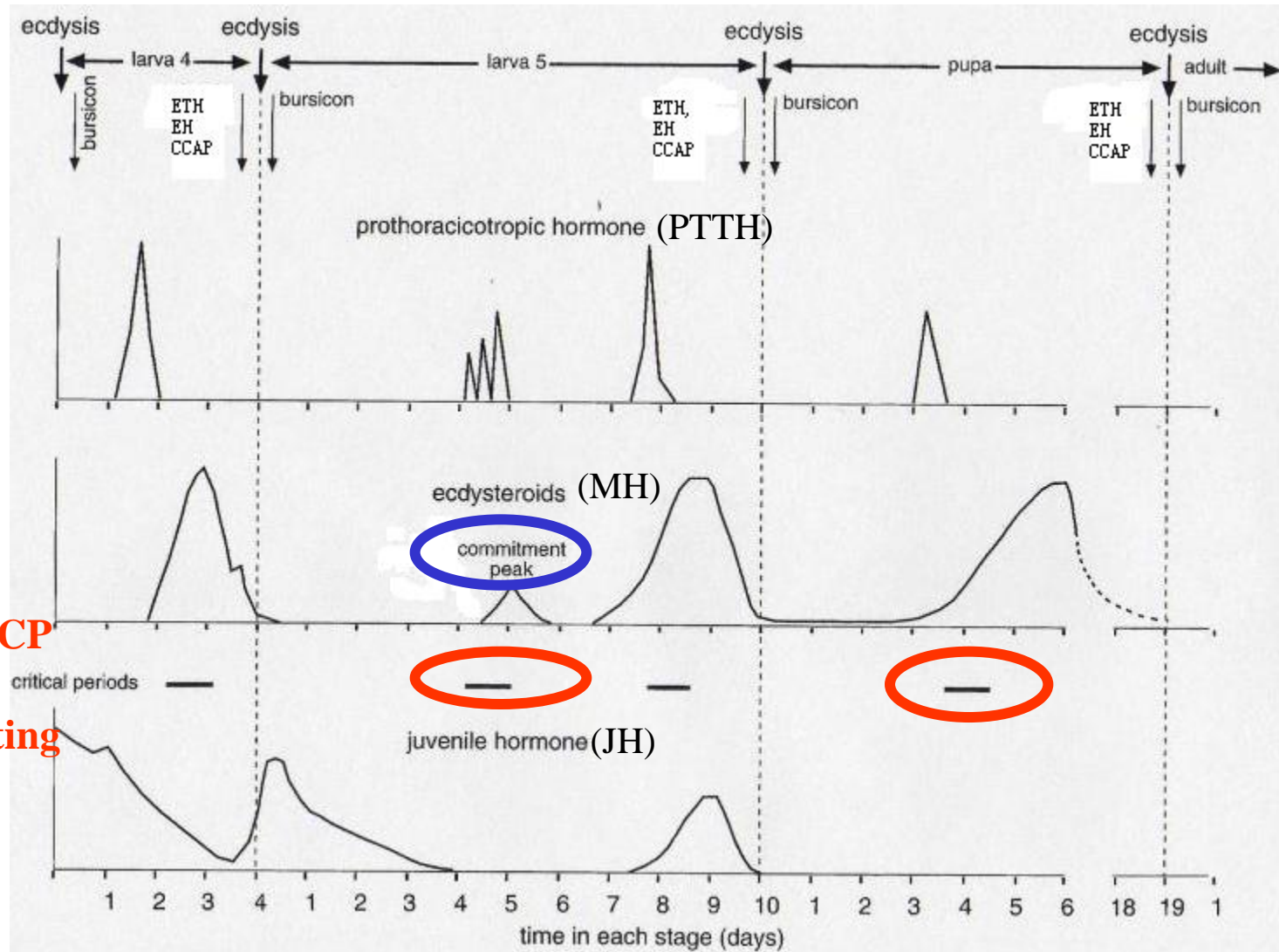
No JH during Head CP



Metamorphosis molting

— ecdysteroid(MH)
 - - - juvenile hormone

Pre-ecdysis phase: hormone titers in holometabolous insects



No JH during Head CP



Metamorphosis molting

Pre-ecdysis phase: Developmental patterns of PTH production

- PTH stimulates the PTGs to secrete ecdysone (MH)
- Once ecdysone secretion begins, PTH secretion stops and the molting cycle continues without further need for PTH
- PTH titers have been determined directly for very few insects
- Most studies on the control of molting determine the time at which the molt becomes independent of the brain
- The point in time at which half the animals are able to complete a normal molt in the absence of their brain = **head critical period**

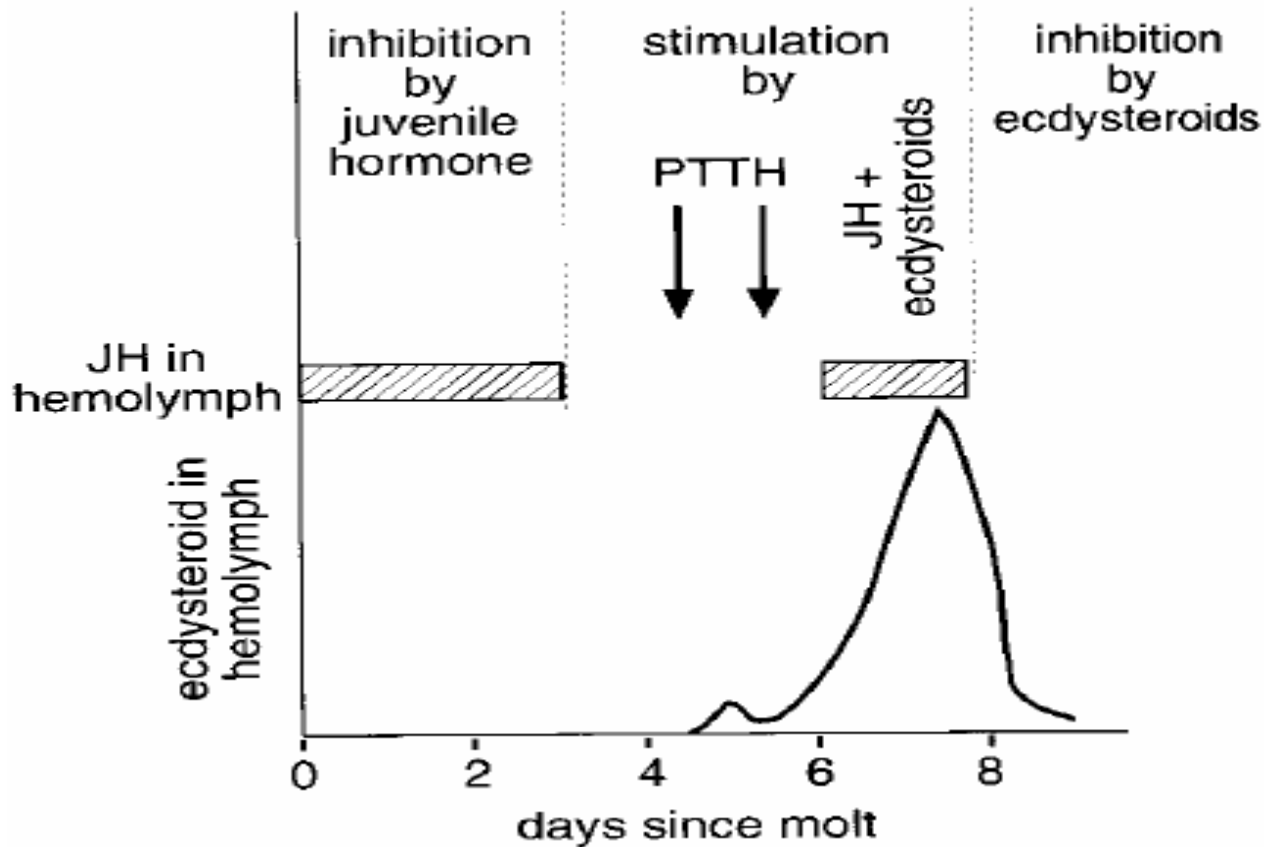
Pre-ecdysis phase: regulation of PTH and molt cycles

- Onset of a molt cycle is an indeterminate event
- Molt cycles are tightly linked to growth and nutrition
- Factors shown to regulate molt cycles:
 - stretch of the abdomen
 - attainment of a critical size
 - temperature
 - humidity
 - photoperiod
 - contact with specific substrates
 - injury
 - pheromones
- All of these factors directly or indirectly regulate the secretion of PTH
- Every molting cycles begins with the secretion of PTH by the brain

Pre-ecdysis phase: Developmental changes in hemolymph ecdysteroids

- Titer RISE and then FALL before each molt
- Titrers are always high during the second half of the final larval instar
- Titrers are high after the “critical period” for molting.
- A small increase (called **commitment peak**) in ecdysteroids often precedes the big rise in the last larval instar
- High levels of ecdysteroids are associated with adult development

Pre-ecdysis phase: Control of ecdysteroid titer in hemolymph



Pre-ecdysis phase: Developmental patterns of JH production

- Present during almost all of larval life
- Present in relatively high levels at each larval-larval molt
- Present at moderate levels at the larval-pupal molt
- Typically not present at the pupal-adult molt
- JH is present whenever ecdysteroids are present, except at the end of the last larval instar and during adult development

Pre-ecdysis phase: Control of JH titer in hemolymph

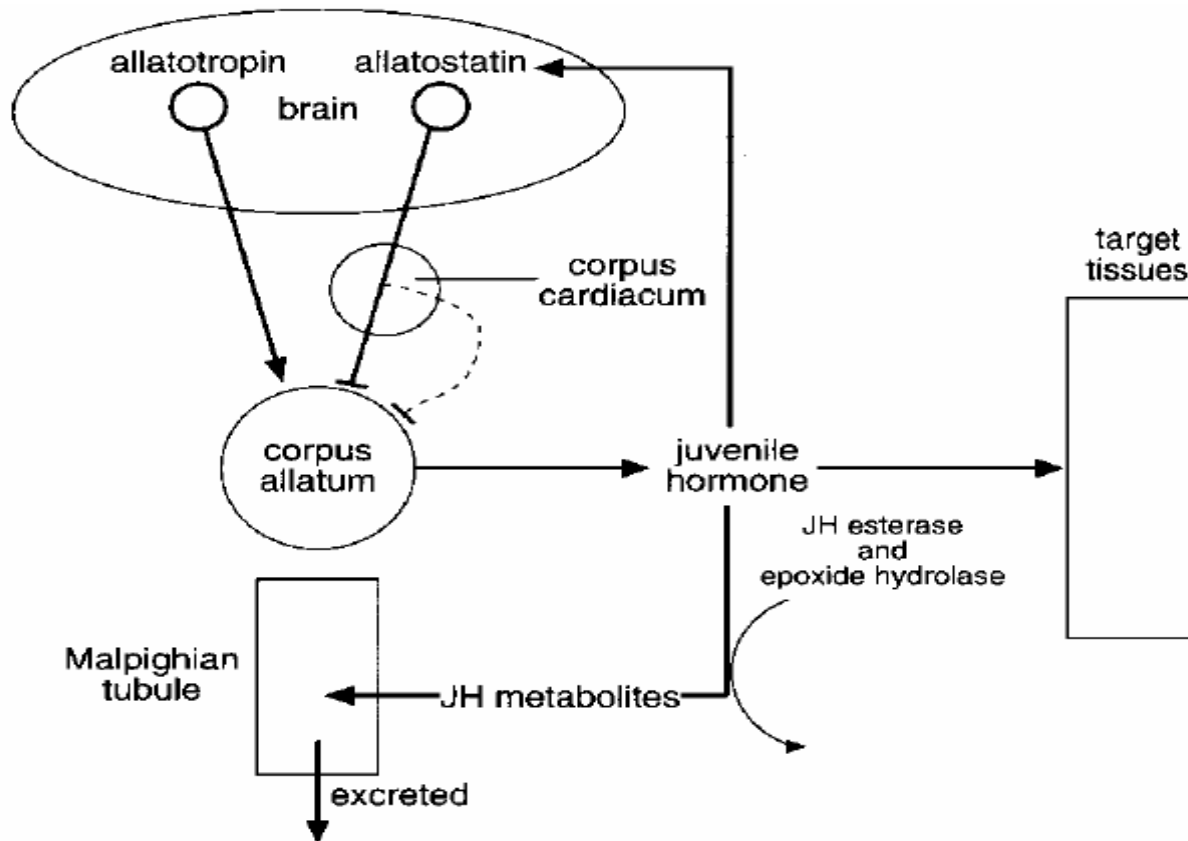


Fig. 21.11. Juvenile hormone. Regulation of hemolymph titer involves the balance between synthesis in the corpora allata and degradation and excretion by the Malpighian tubules.

Pre-ecdysis phase: induction chromosomal puffing by ecdysteroid



Fig. 1 The pipe as a Lamarckian characteristic. The "Chef" without his pipe was quite unimaginable



Pre-ecdysis phase: ecdysone activates gene transcription

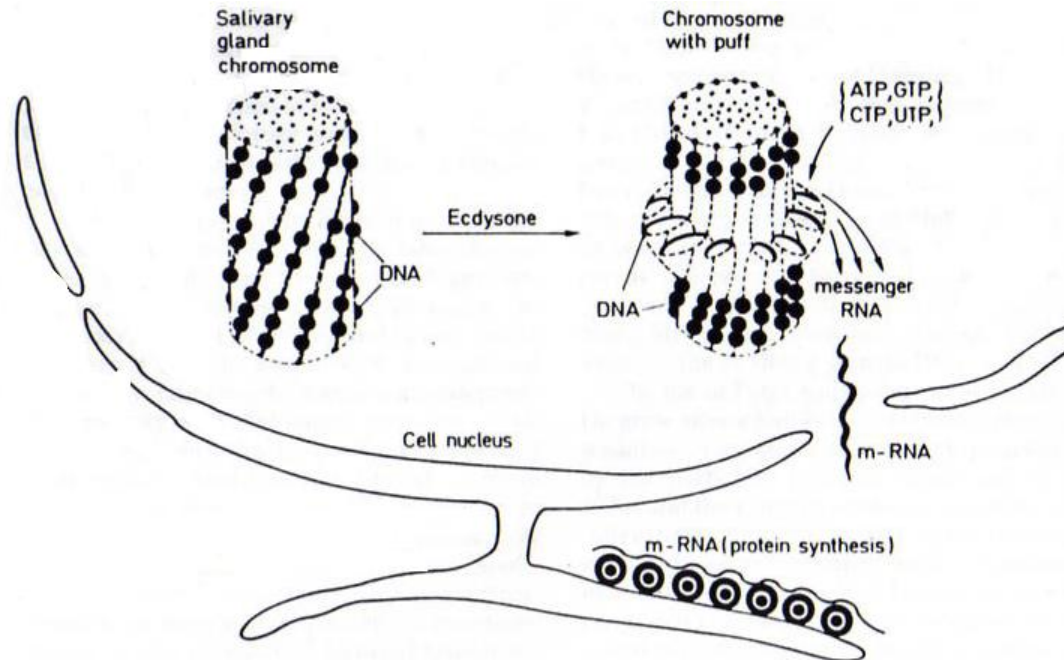


Fig. 4 The "blueprint". Karlson's hormone-gene activation theory, which, complemented by the receptor concept, served as the basis of experimentation for almost three decades in laboratories all over the world

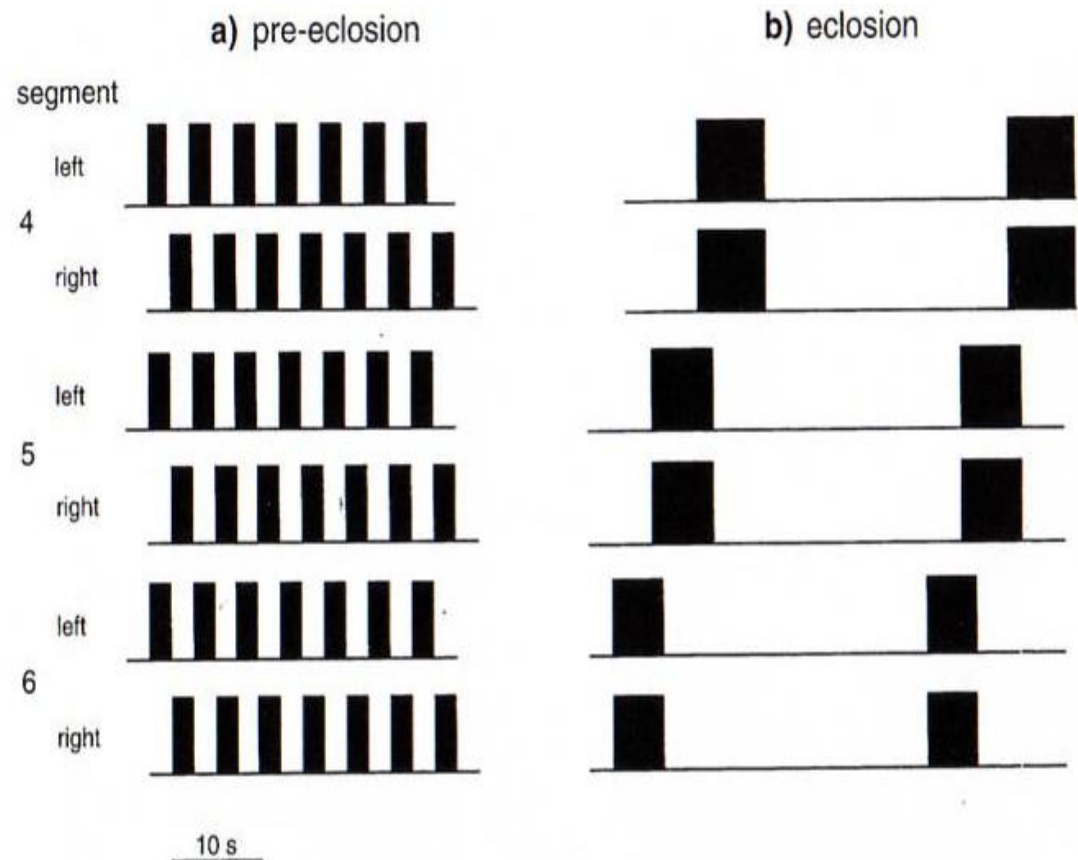
JH modulates ecdysone-induced activation of gene transcription

Ecdysis phase

- **Ecdysis**: shedding old cuticle
- **Two stereotyped ecdysis behaviors**
 - Pro-eclosion behaviors
 - Eclosion behaviors
- **Regulated by three hormones**
 - EH
 - CCAP
 - **ETH**

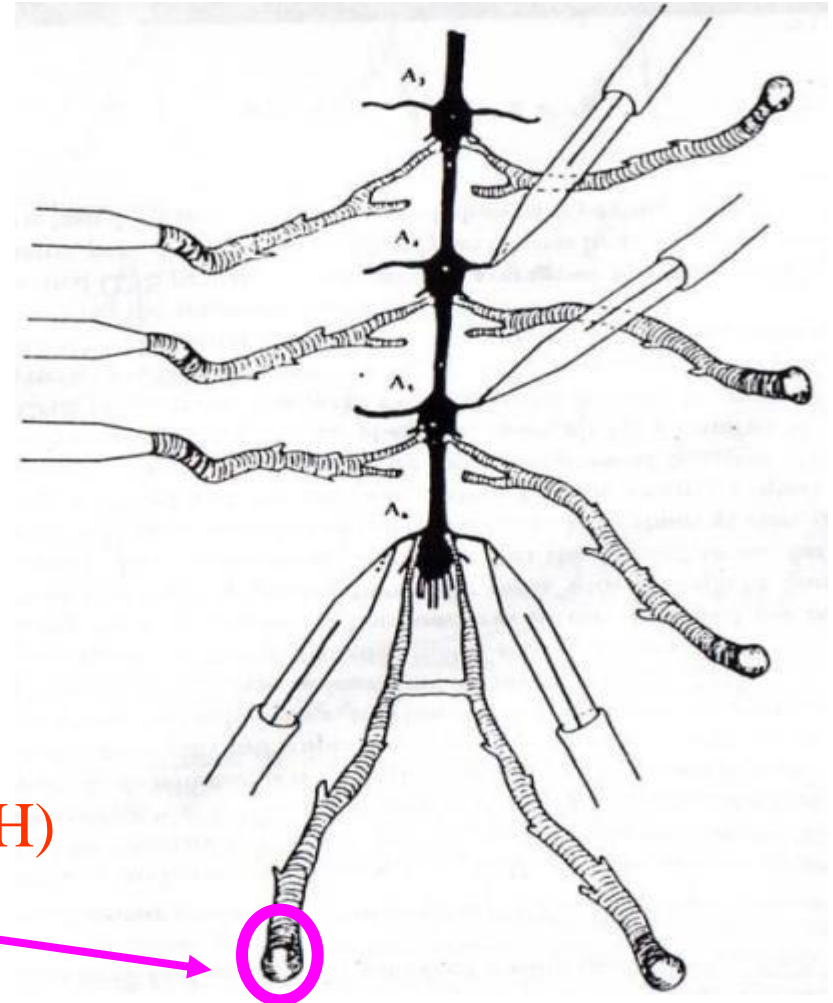
Ecdysis phase: two stereotyped ecdysis behaviors

- **Pro-eclosion behaviors:** a series of abdominal rotations
- **Eclosion behaviors:** waves of abdominal contractions



Ecdysis phase : EH can trigger the two motor program in vitro

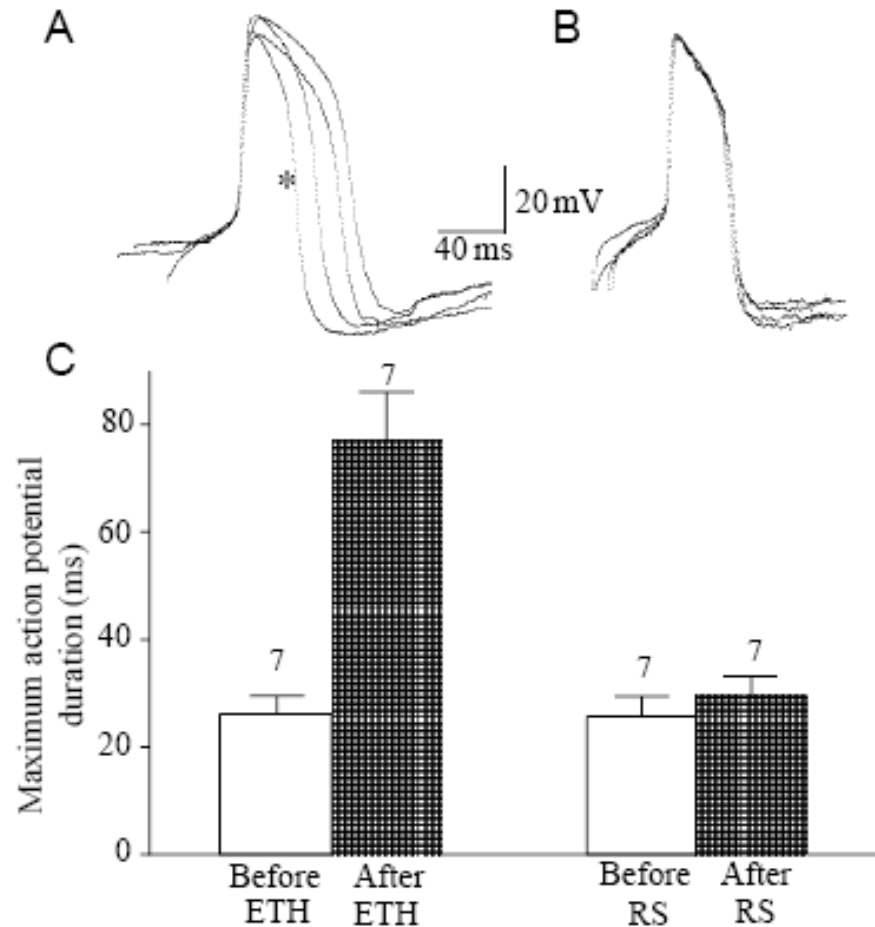
Ecdysis hormone (EH) from brain NSC can trigger both pro-eclosion and eclosion motor programs. But trachea must be there for pro-eclosion program (James Truman, 1980)



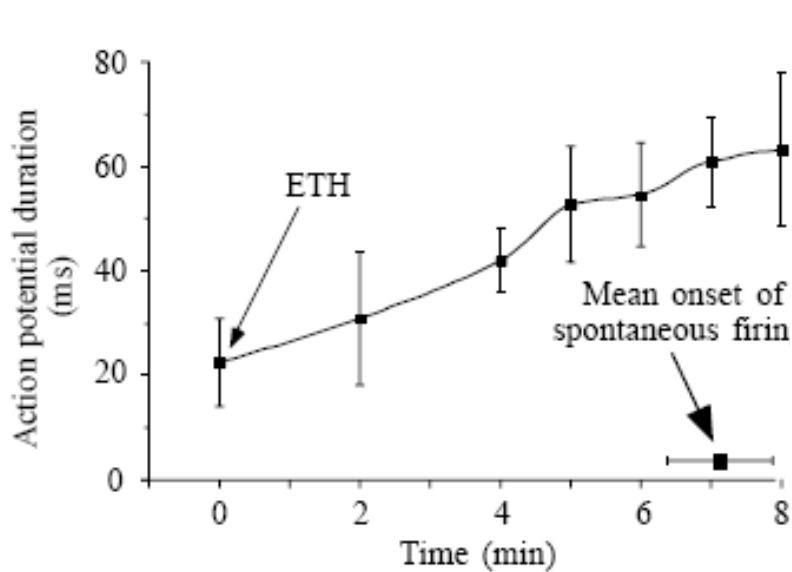
Why? Provide oxygen ?
Ecdysis-triggering hormone (ETH)
from epitracheal glands

Ecdysis phase: ETH cause broadening of AP in brain EH NSC

- ETH extend the duration of action potentials (AP) in EH neurosecretary cells (NSC) in brain
- Gammie & Truman, 1999

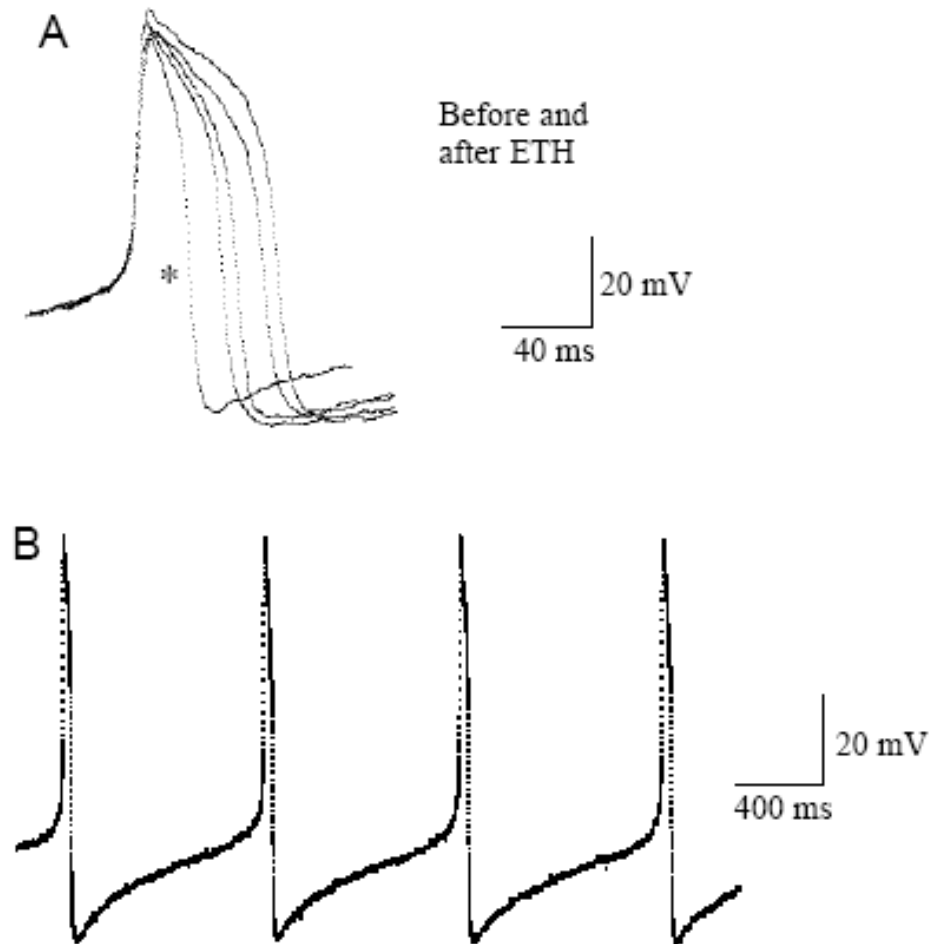


Ecdysis phase: ETH cause spontaneous AP in brain EH NSC



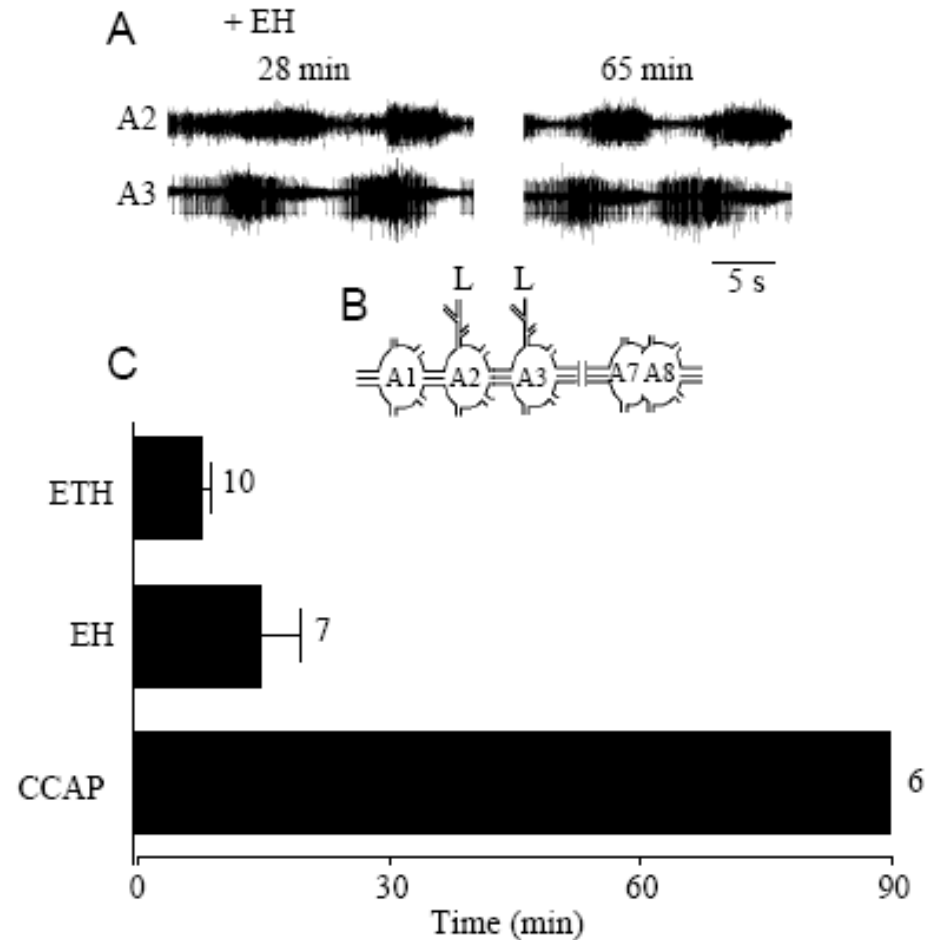
Amplitude also increased
from 67 ± 4 to 80 ± 5

ETH stimulate the release
of EH from brain NSC

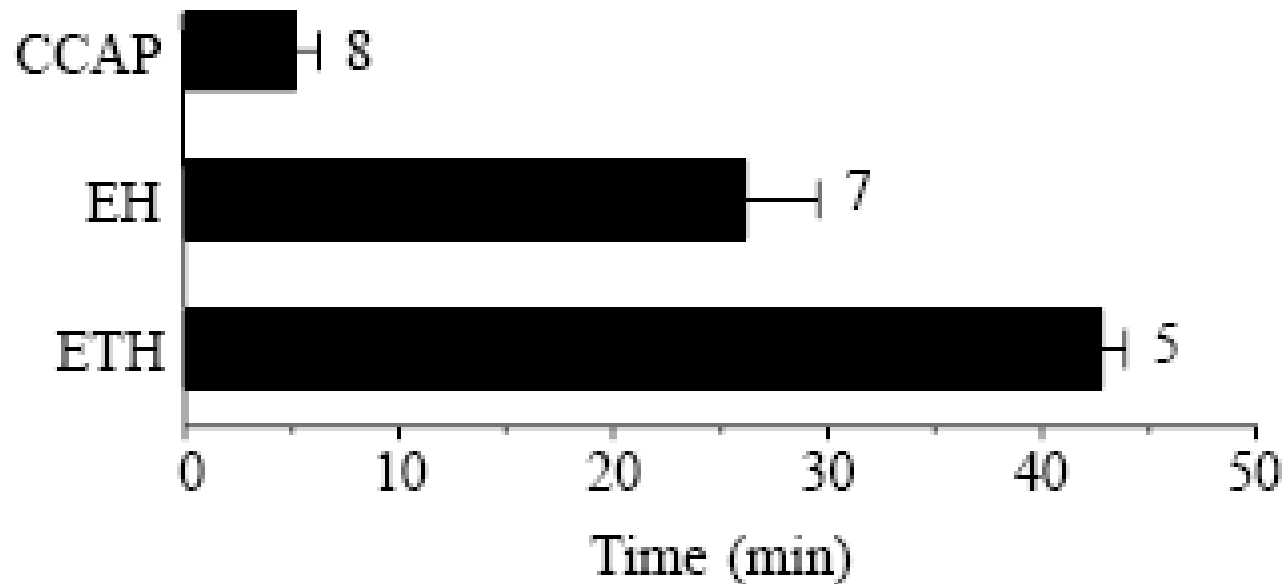


Ecdysis phase: The duration of the eclosion motor programs

- Add ETH, EH, and CCAP to abdomen ventral nerve cord preparations and record the duration of the eclosion motor program
- The longer the duration, the closer to the motor neuron
- Inference: ETH → EH → CCAP → Motor neurons in A2/A3



Ecdysis phase: time needed to switch on the ecdysis motor program



- ETH → EH → CCAP → motor neuron

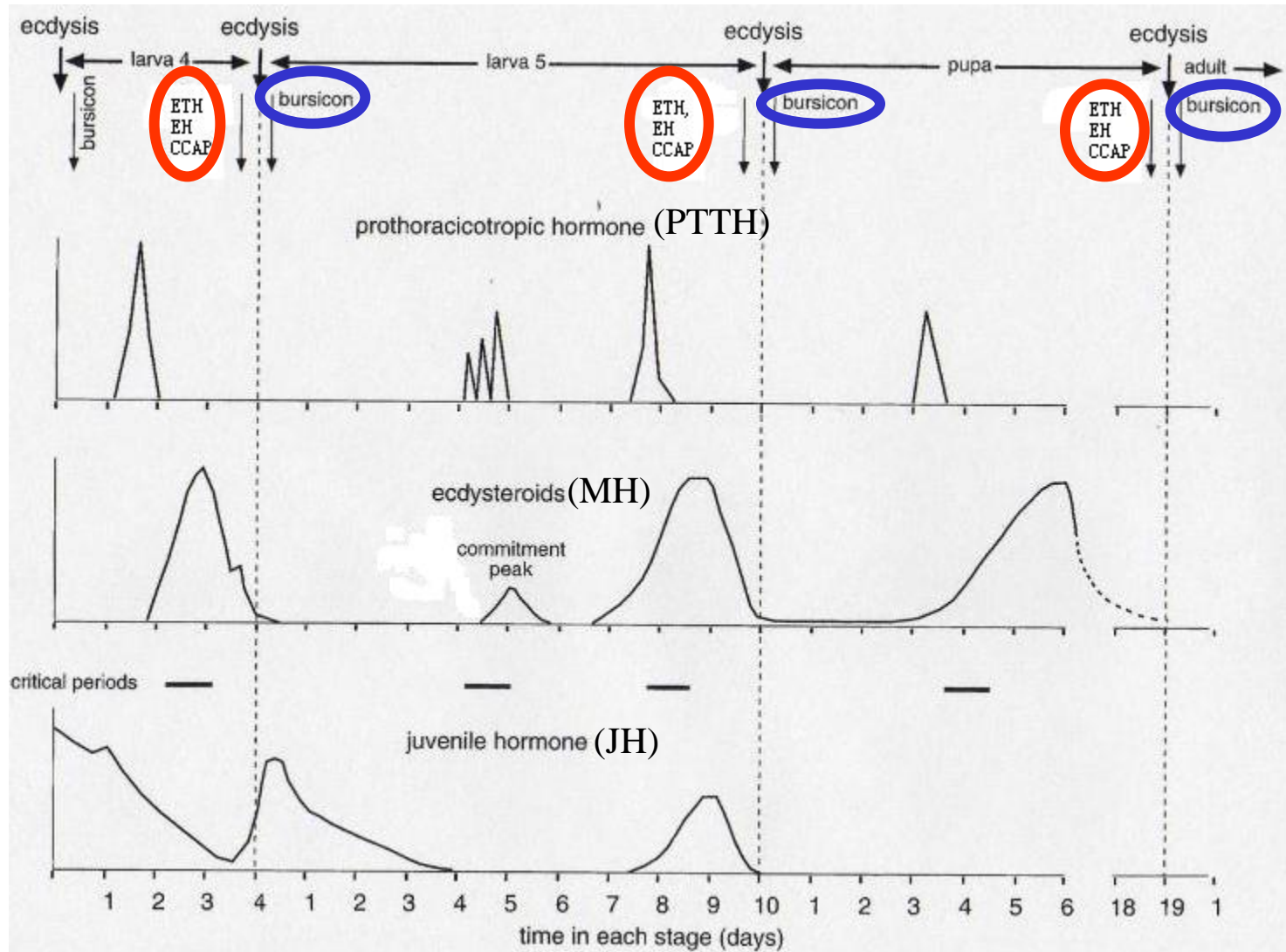
Ecdysis phase: summary

- ETH cause release of EH by NSC (neurosecretary cells) in the brain
- ETH switch on pre-eclosion behavior
- Release of EH cause release of CCAP (crustacean cardioactive peptide)
- CCAP switch on eclosion behavior, switch off pre-eclosion behavior

Post-ecdysis phase

- **Post-ecdysis phase:** hardening (sclerotization) and darkening of new cuticle, initiated by **bursicon**
- Identity of bursicon was determined by Luo et al. (2005)

Hormones involved in molting and metamorphosis



Summary

- Blood levels of the molting hormone (MH, i.e. ecdysone) peak before each molt (MH acts on the tissues of the body to regulate growth and differentiation)
- A peptide hormone produced in the brain, PTTH, regulates MH synthesis by the prothoracic gland. (regulation of PTTH release therefore regulates the timing of the molt)
- Whether or not metamorphic changes occur at the cellular level depends on whether JH is present when MH is present (the same tissues responds to both hormones)
- At least three peptide hormones interact to trigger molting behavior (ETH, EH, CCAP). Both release of these hormones and the ability of tissues to respond to these hormones is controlled by MH and JH.
- Another peptide hormone, bursicon is responsible for hardening (sclerotization) and darkening of the new cuticle.