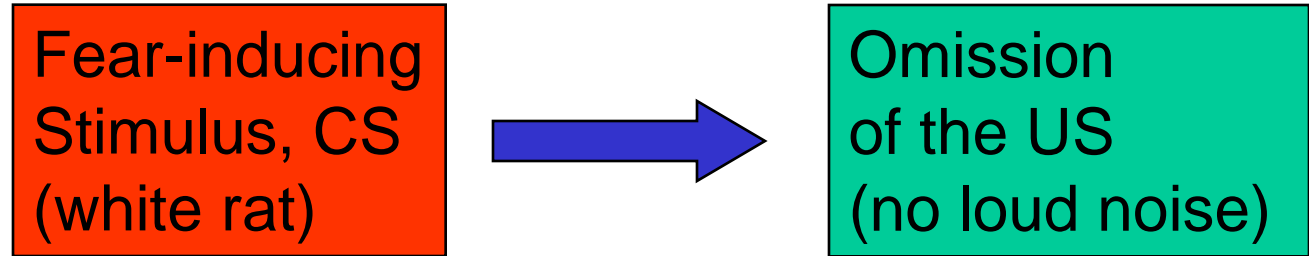


Properties of fear

- Unconditioned fears
 - Open spaces (agoraphobia)
 - Large/intense objects (startle reflex)
- Acquisition of fear
 - Conditioning (individual experience)
 - Social learning (vicarious experience)
- **Elimination**
 - **Extinction**
 - **Counterconditioning**
- Relapse
 - Spontaneous recovery
 - Reinstatement
 - Disinhibition
 - Renewal
- Reacquisition
 - Reexposure to the Pavlovian pairing
- Brain mechanisms

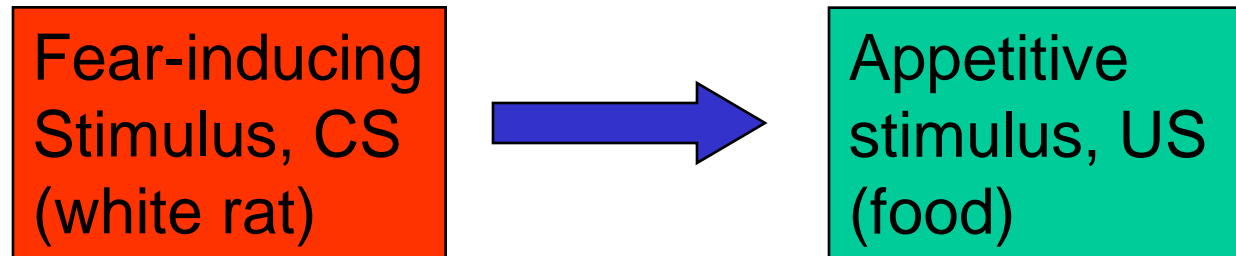
Elimination of fear

(1) Extinction of fear



(2) Counterconditioning of fear

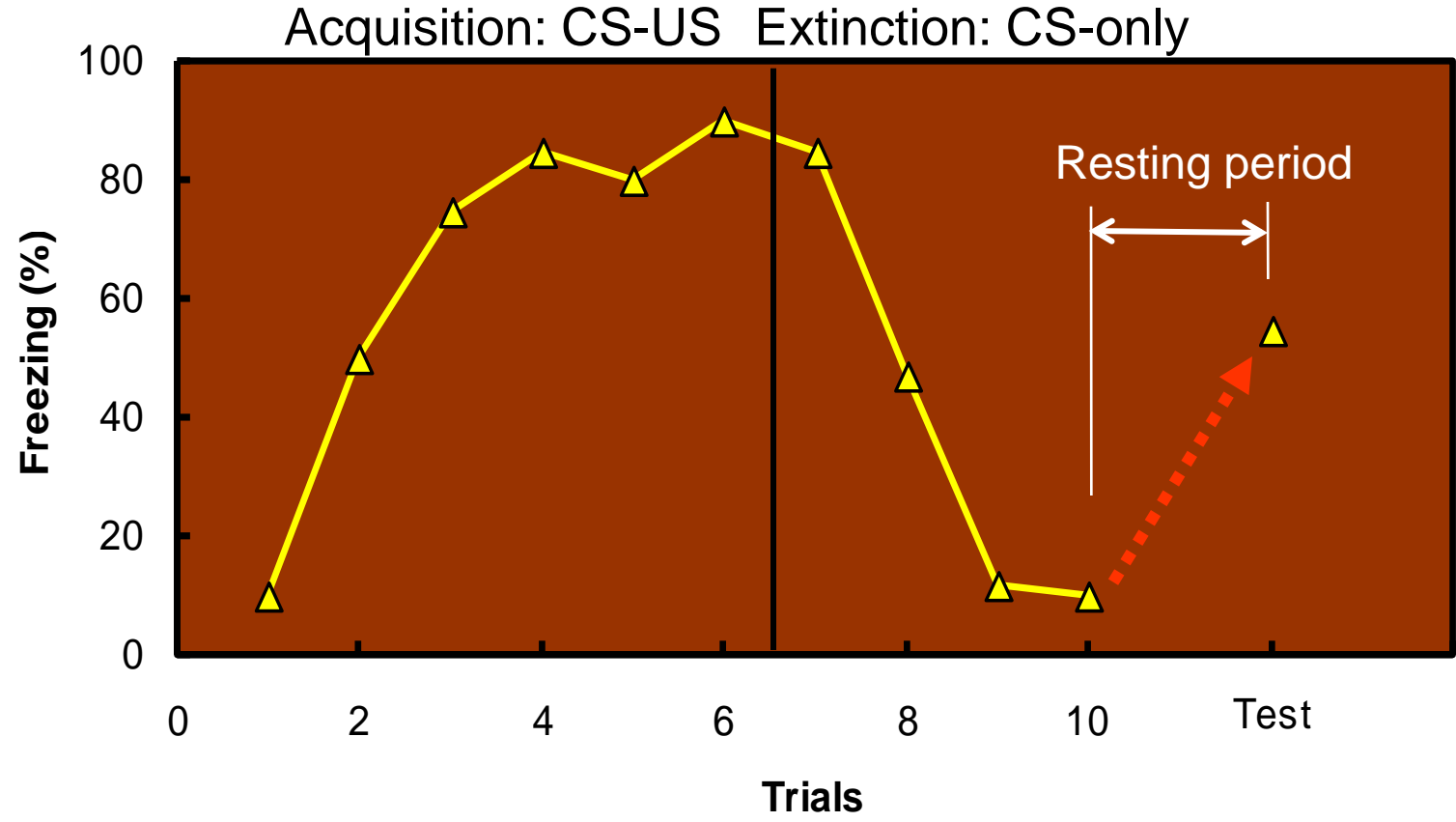
Counterconditioning changes the hedonic response to one stimulus when that stimulus is paired with another of opposite hedonic value.



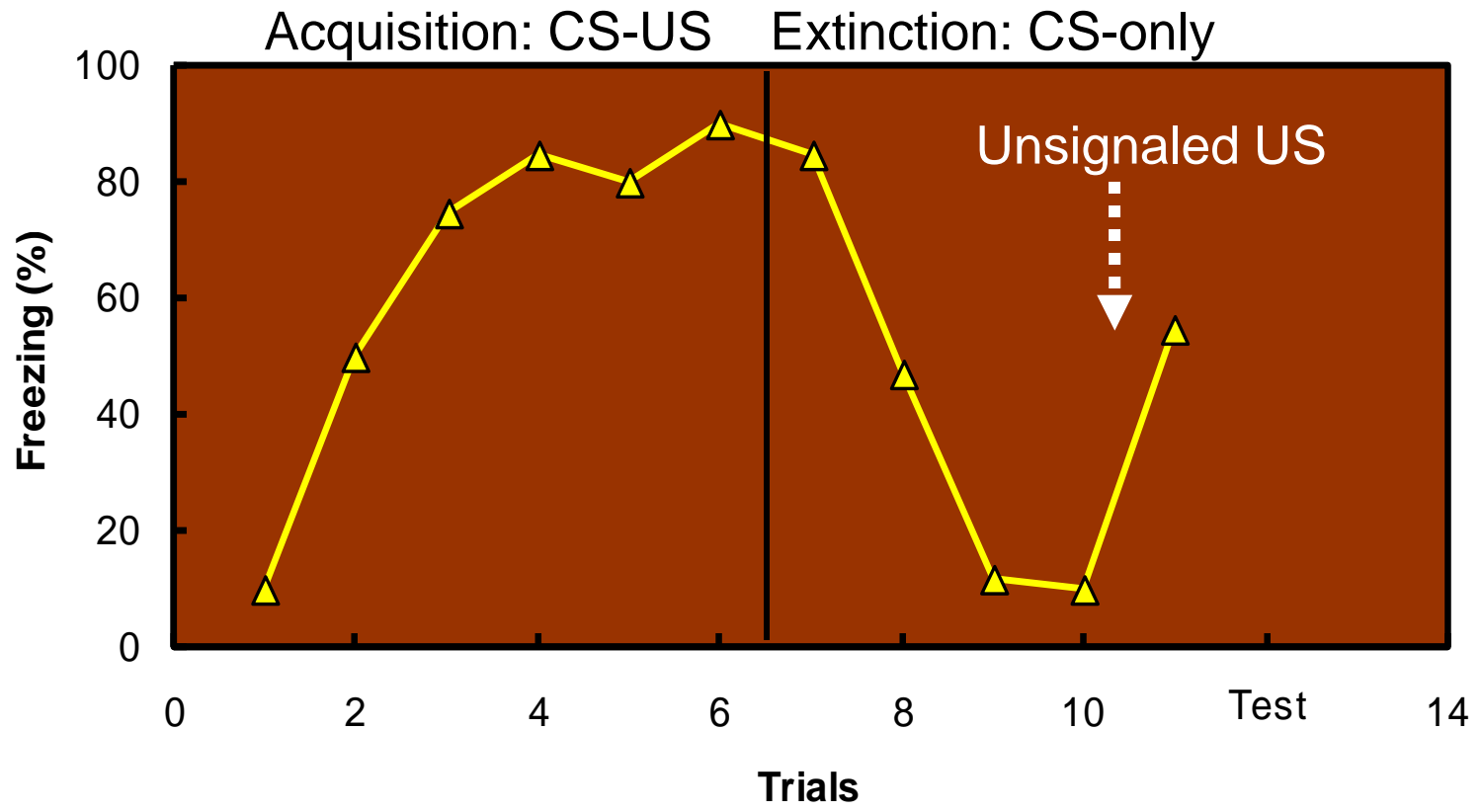
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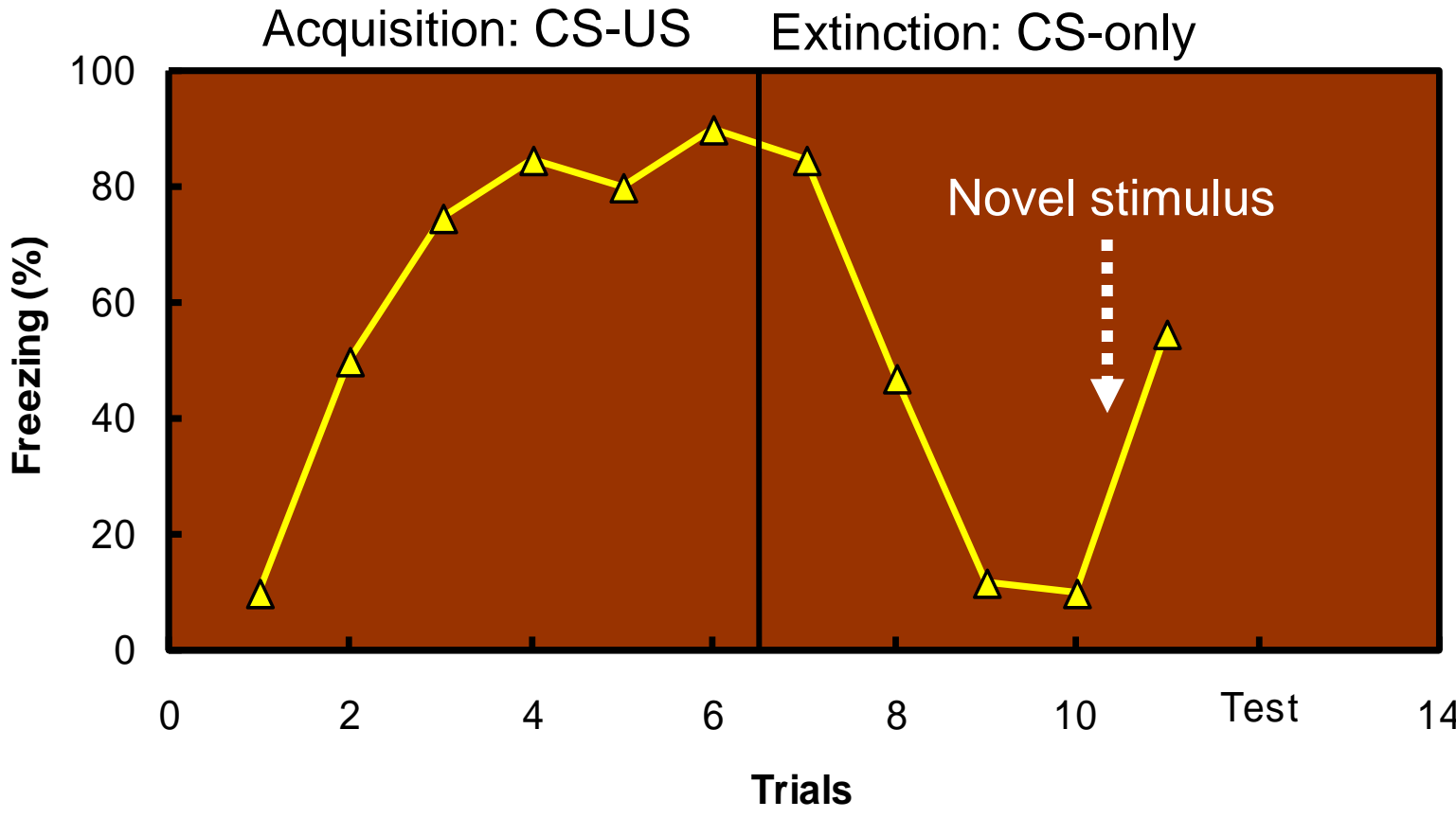
Relapse: Spontaneous recovery of fear



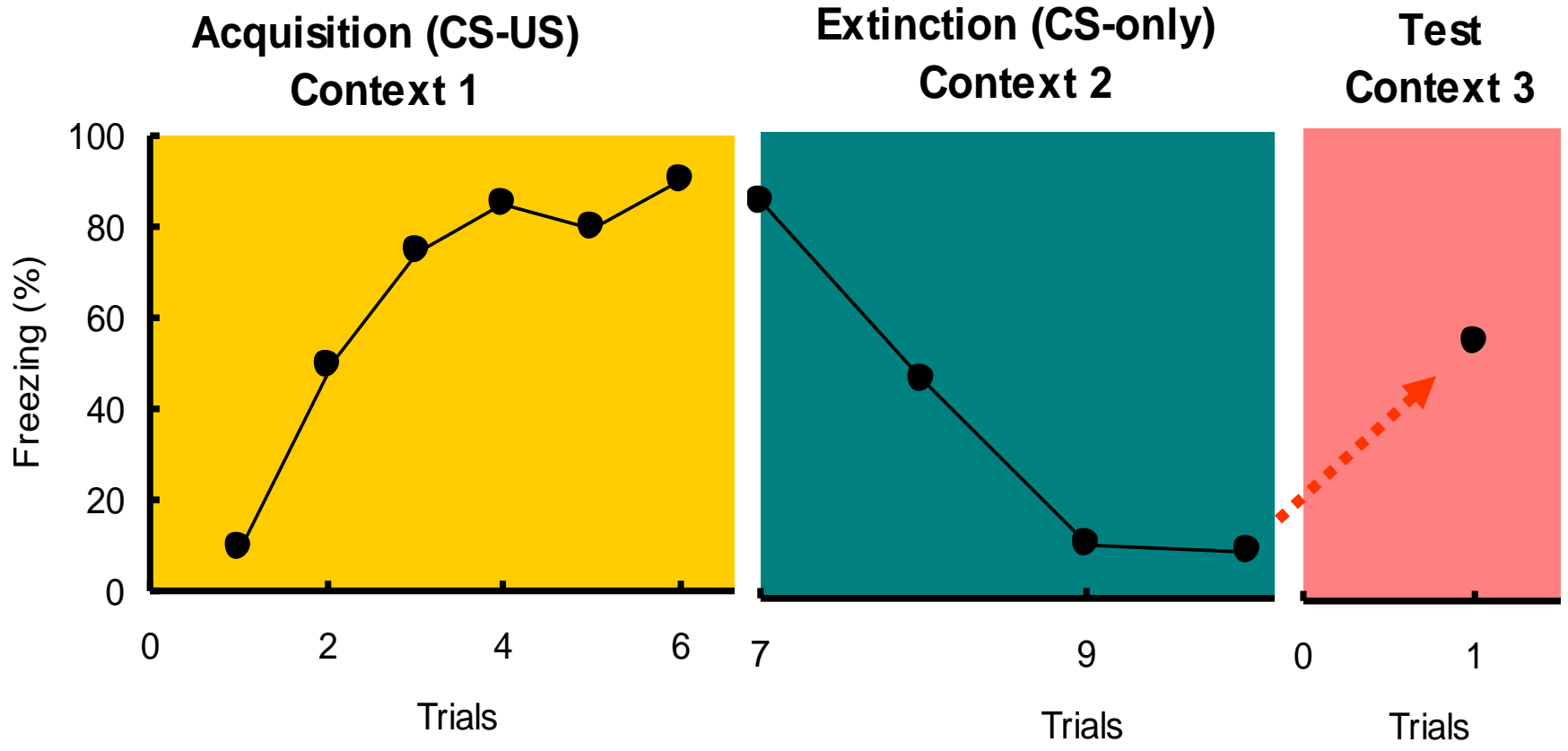
Relapse: Reinstatement of fear



Relapse: Disinhibition of fear

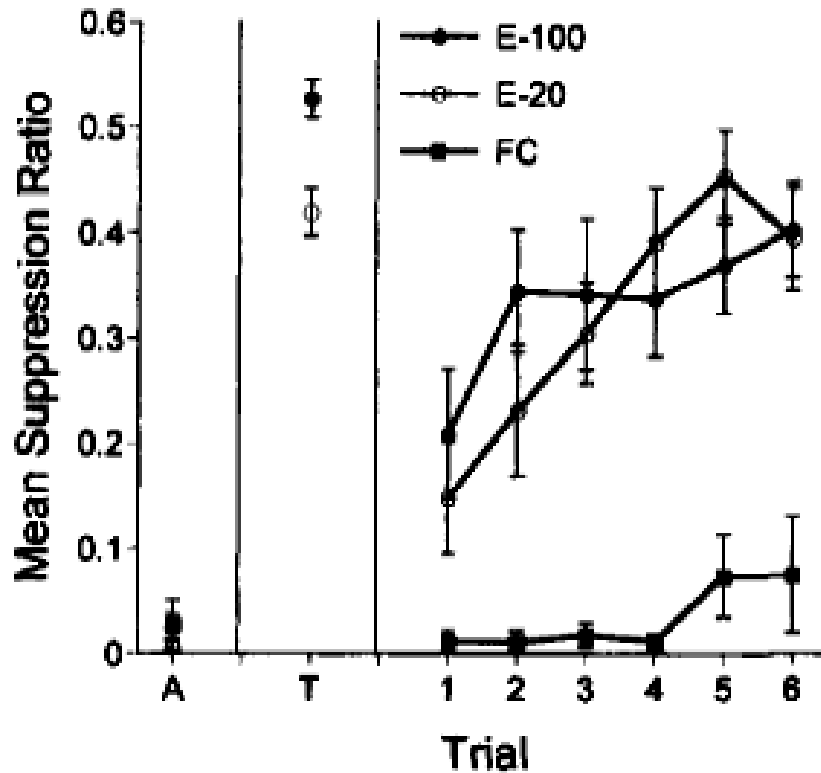


Relapse: Renewal of fear

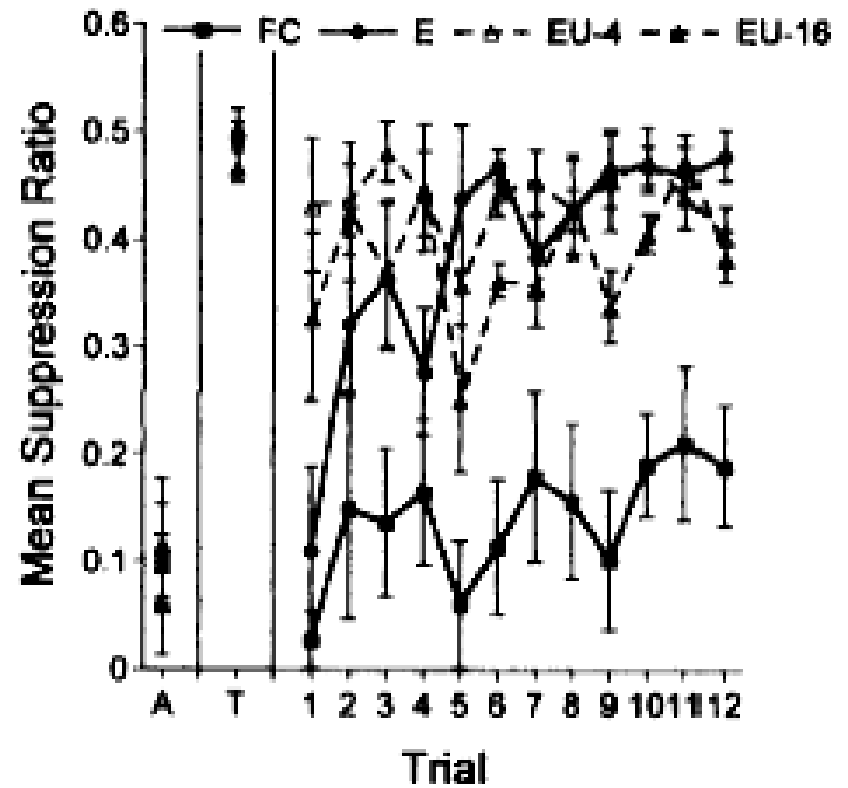


Reducing the renewal of fear

(1) Extensive extinction does not work well.



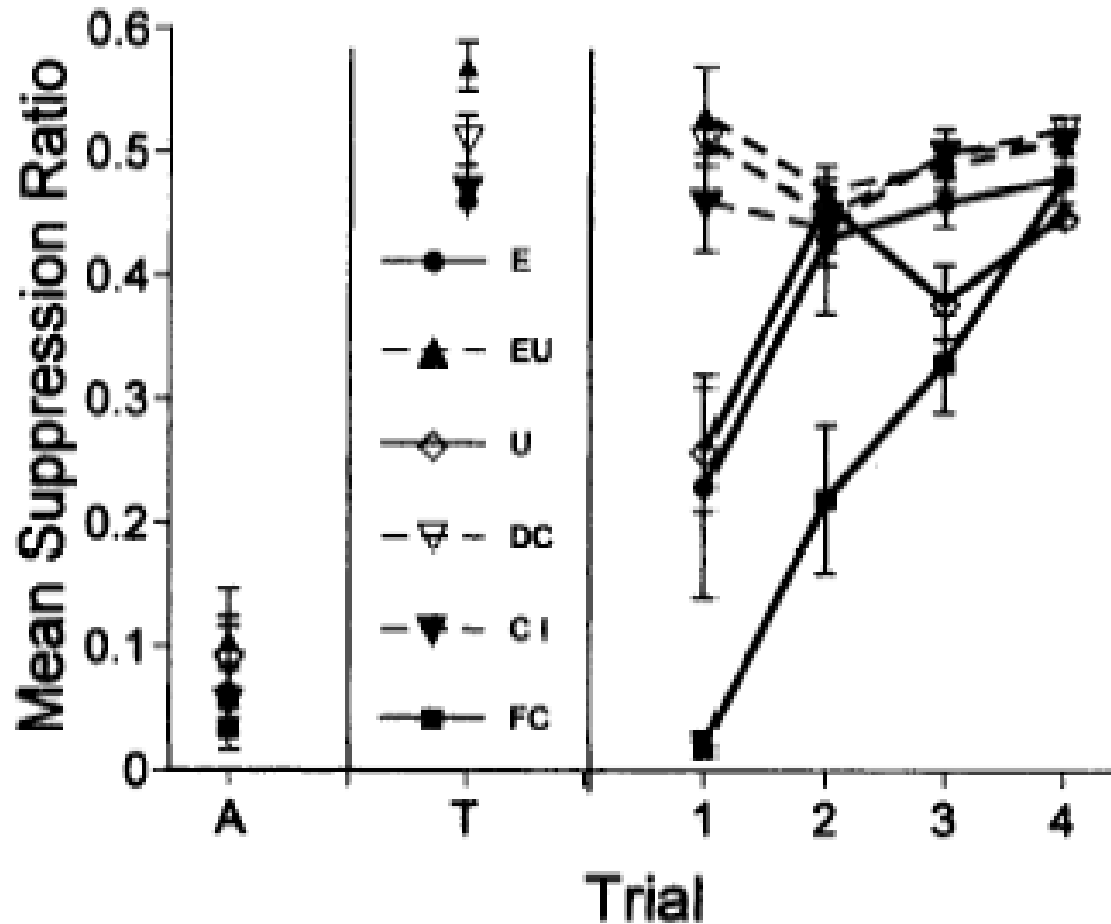
(2) Explicitly unpaired training eliminates renewal.



E (extinction): A-
FC (forgetting control): No treatment

EU (explicitly unpaired): A- / +

(3) Inhibitory treatments are maximally effective.



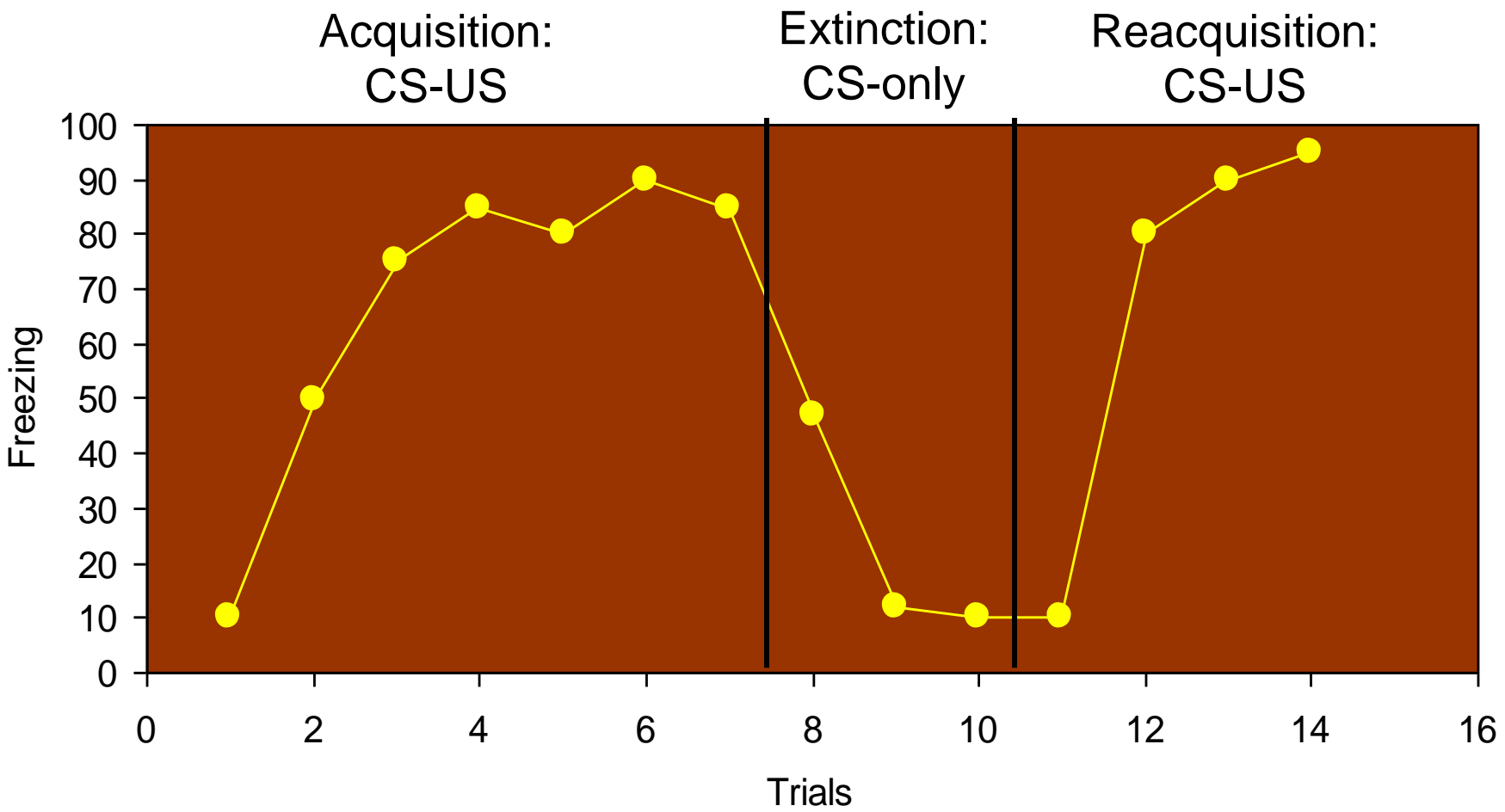
DC (differential conditioning): A+ / B-

CI (conditioned inhibition): A+ / AB-

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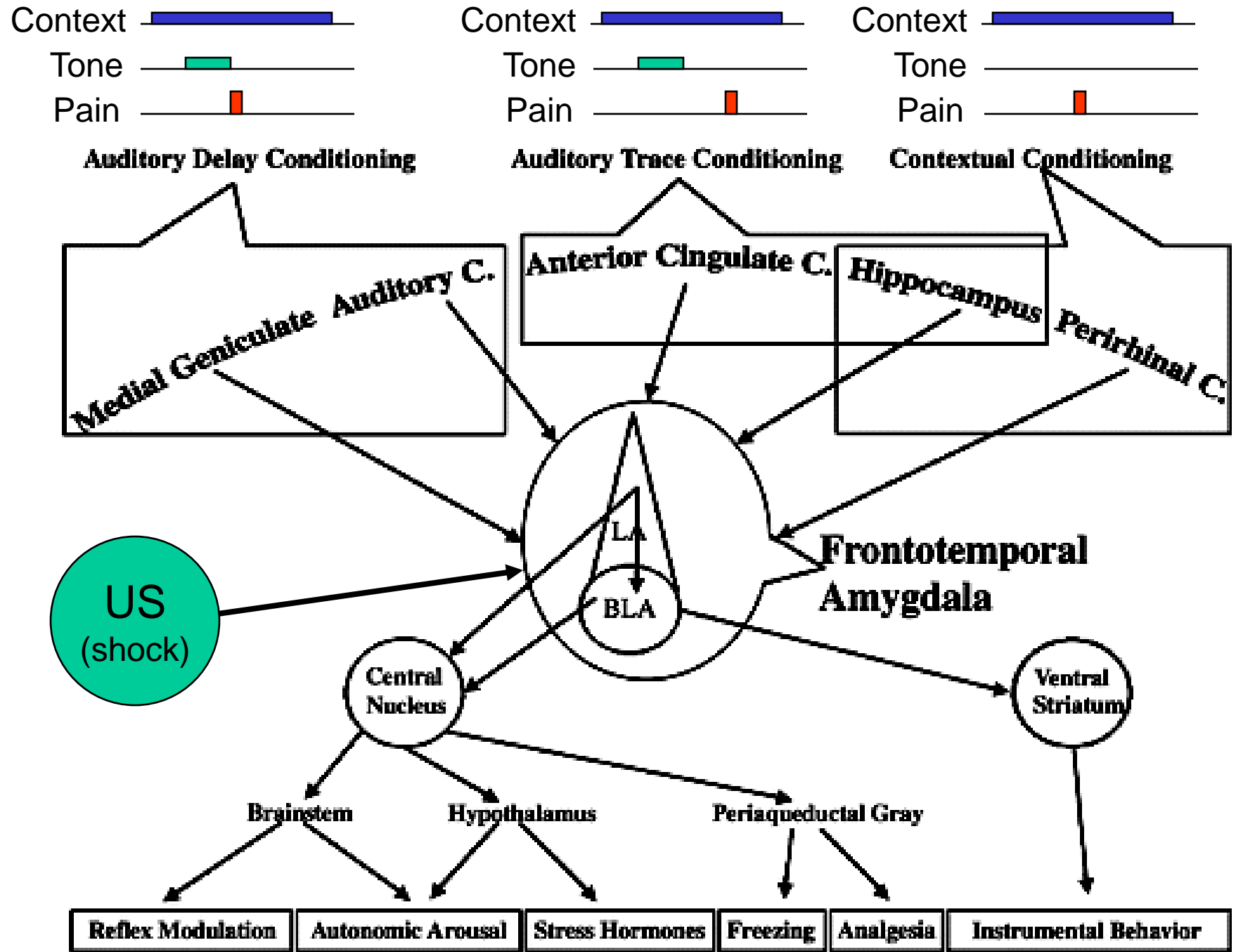
Reacquisition of fear



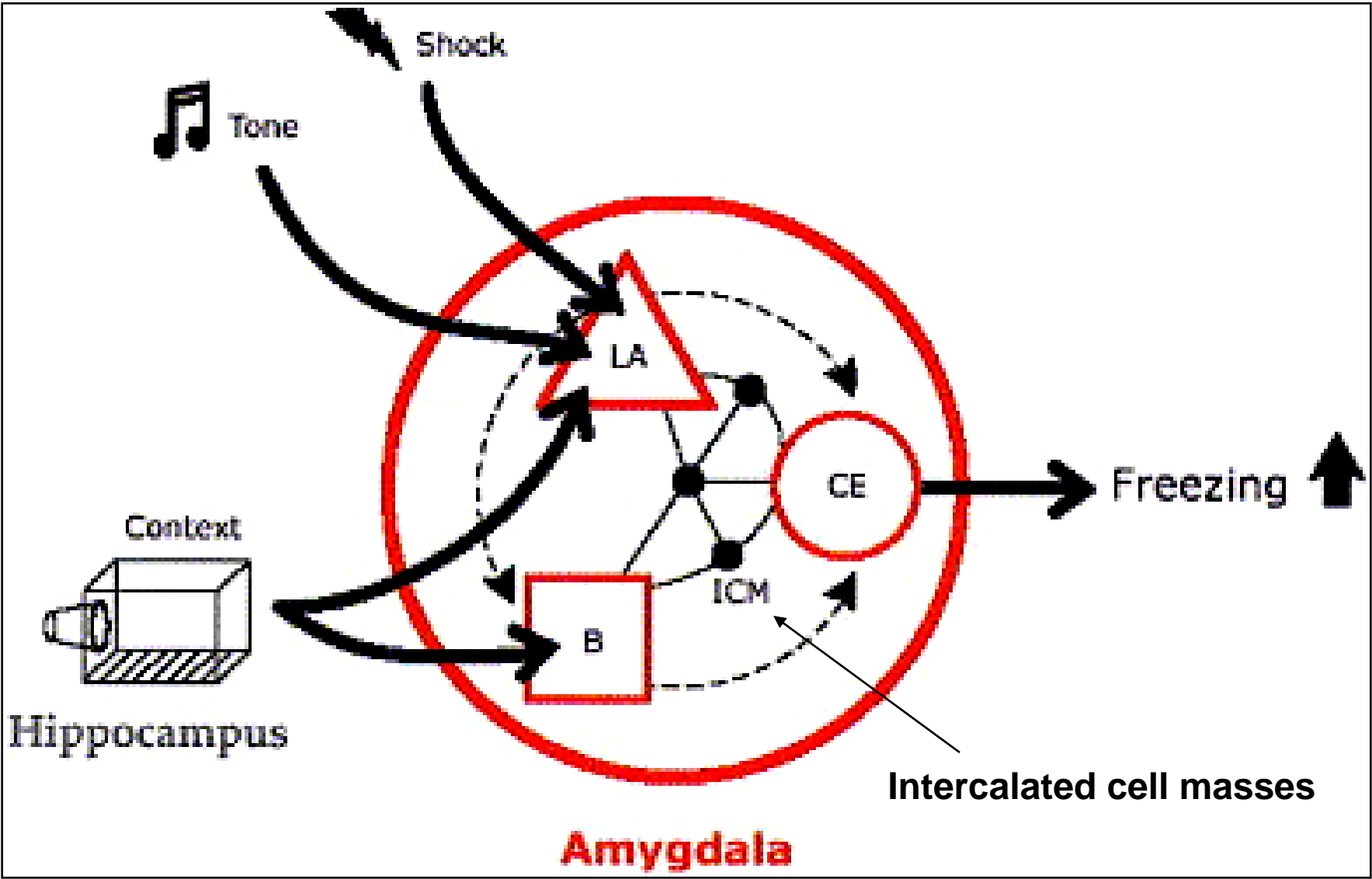
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Brain mechanisms of fear



Role of the amygdala in fear acquisition



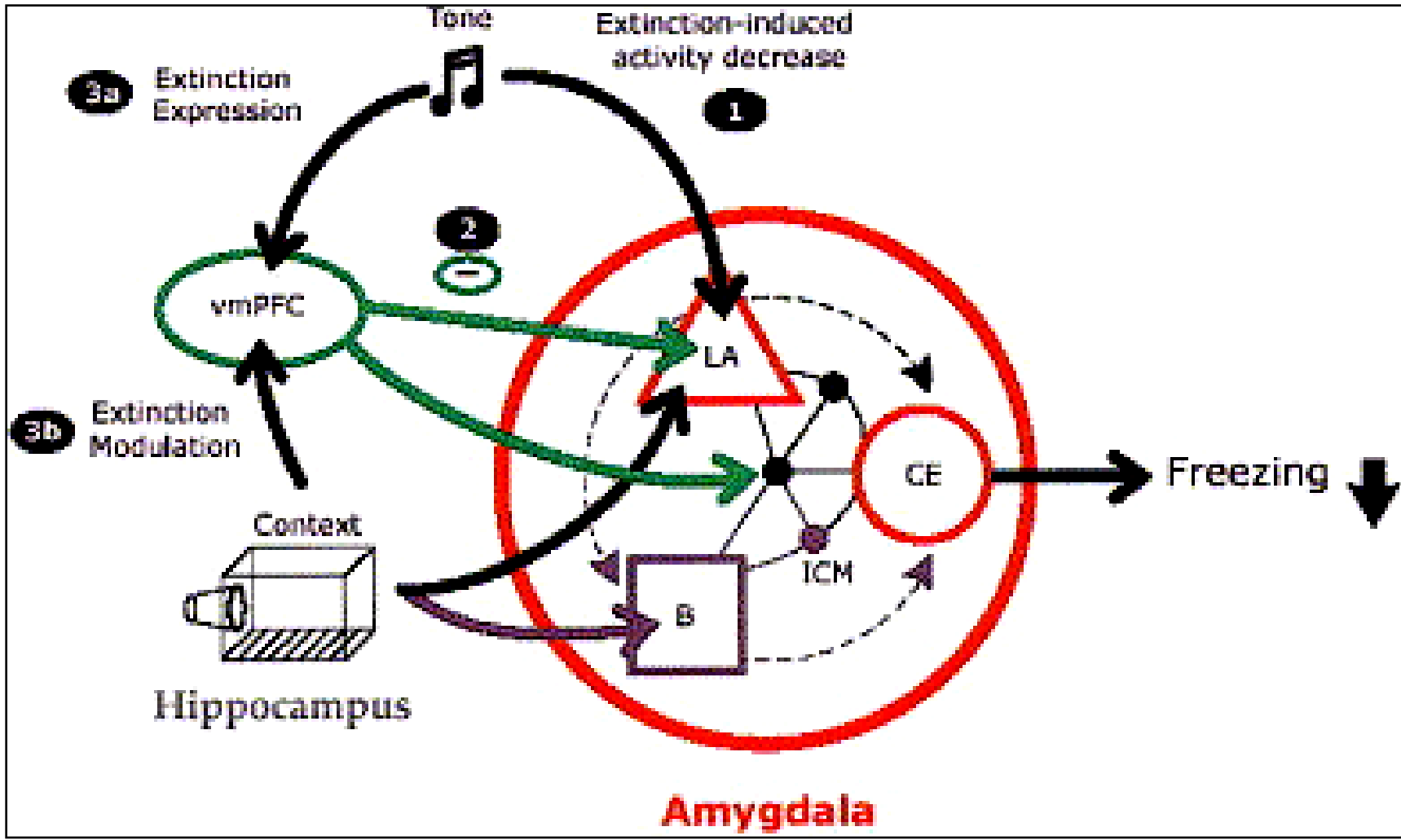
Fear conditioning circuitry

In auditory fear conditioning, animals learn to fear a tone. By pairing tone and shock, the tone acquires the capacity to elicit defensive reactions, such as freezing.

Tone and shock stimuli converge in the *lateral amygdala* (LA), resulting in associative plasticity in the tone→LA pathway. Subsequent presentations of the tone can now activate LA neurons.

The LA then communicates with the *central nucleus* (CE), which controls the expression of fear by way of connections to specific circuits that mediate freezing behavior. The LA connects with CE directly and by way of connections to other amygdala areas, including the *intercalated cell masses* (ICM), which gate the output, and the *basal nucleus* (B), which processes contextual information from the hippocampus.

Amygdala and prefrontal cortex during fear extinction

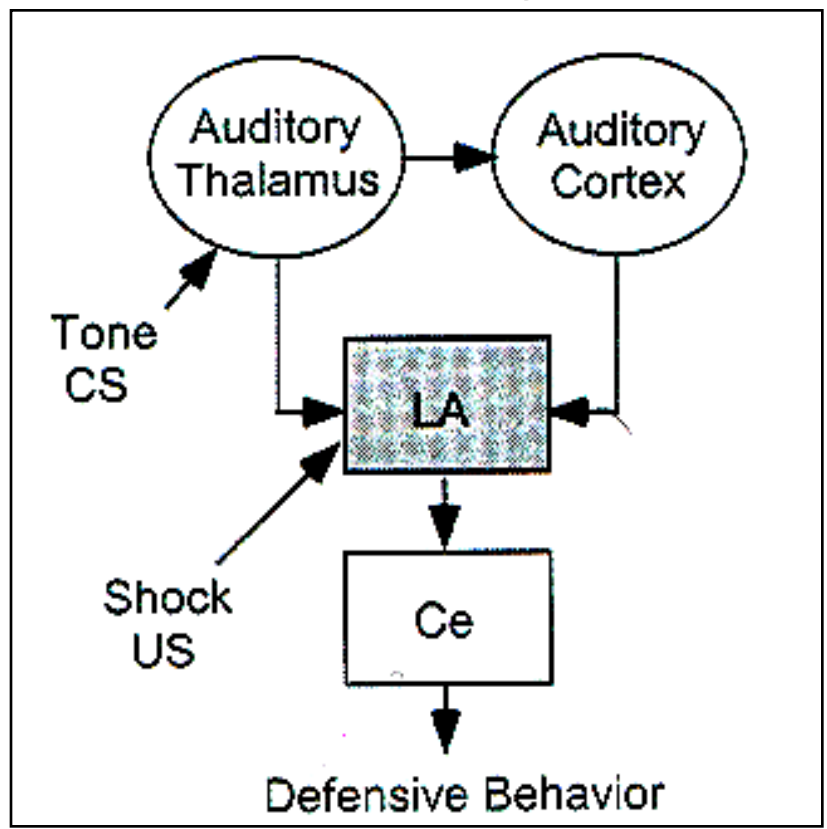


Fear extinction: neural model

- (1) In the early stage, after fear acquisition, T- trials cause some amygdala neurons to decrease their firing rate, through fear learning reversal and/or new inhibitory learning.
- (2) Next, at some point during consolidation of extinction, the inhibitory memory trace between the vmPFC and the lateral amygdala (LA) and/or intercalated cell masses (ICM) is established (mPFC-mediated amygdala inhibition; green arrows and minus sign).
- (3) After extinction learning, when the animal is required to retrieve the consolidated extinction memory, the vmPFC suppresses activity in the amygdala through inhibition of LA neurons and/or activation of the inhibitory ICM, resulting in a rapid decrease in freezing.
- (4) At the same time, hippocampus-based contextual memory modulates neural activity of the vmPFC and/or LA, but not through basal amygdala (B; grey), during extinction expression to regulate the animal's behavioral response (i.e., decrease freezing) if in the appropriate environment.

Cellular mechanisms of fear acquisition in the amygdala

Input convergence



Coincidence detector

