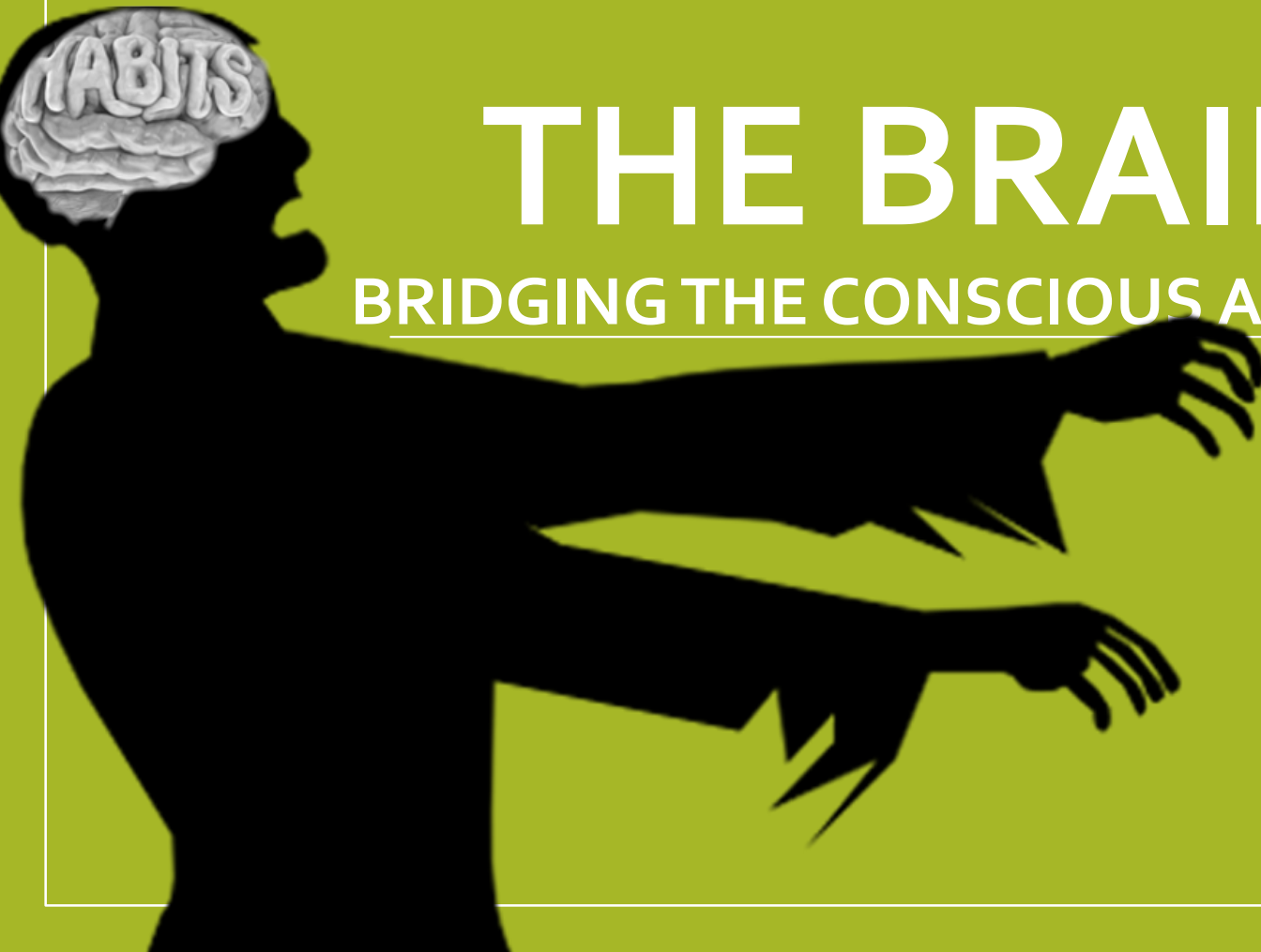


Part 2

THE BRAIN HABIT

BRIDGING THE CONSCIOUS AND UNCONSCIOUS MIND

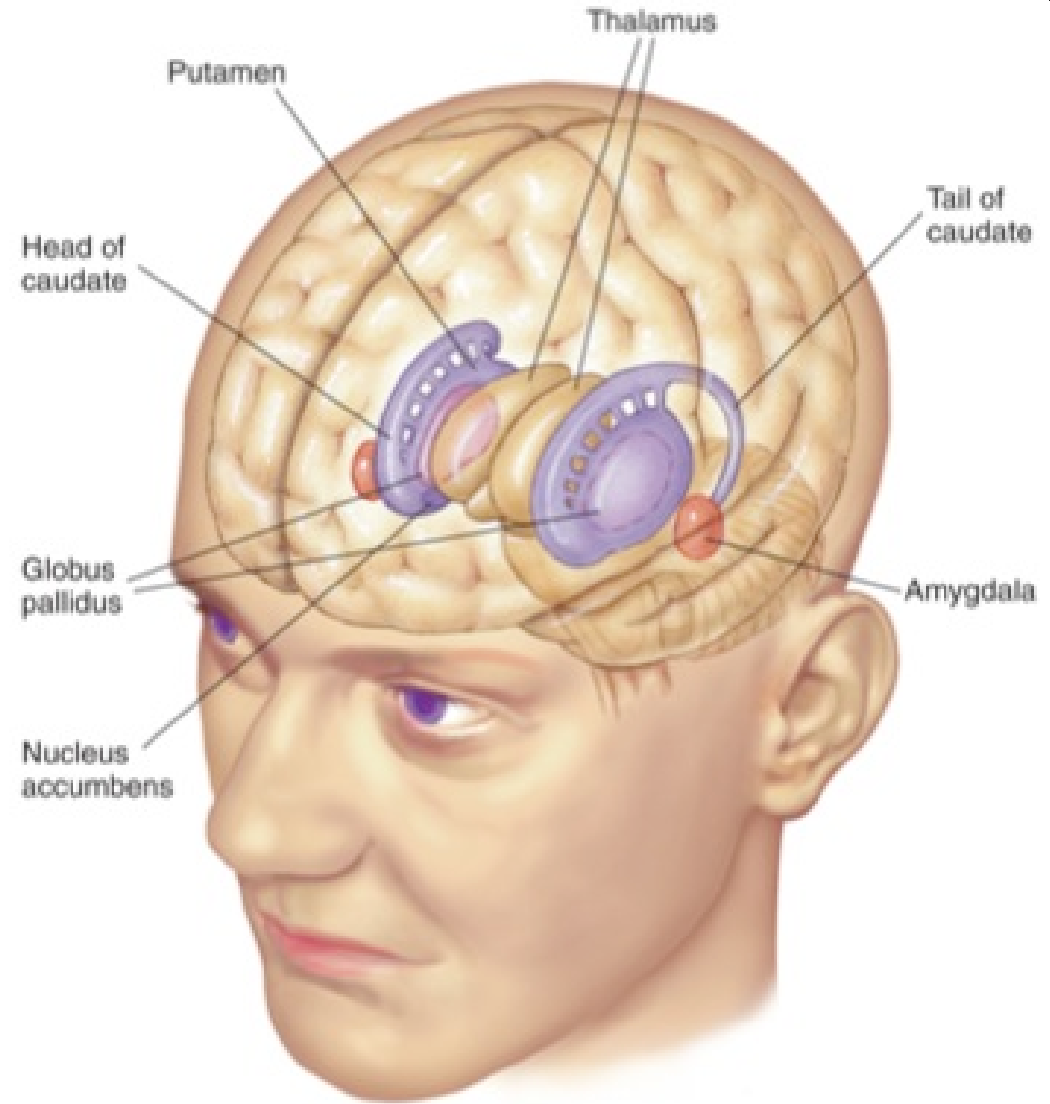
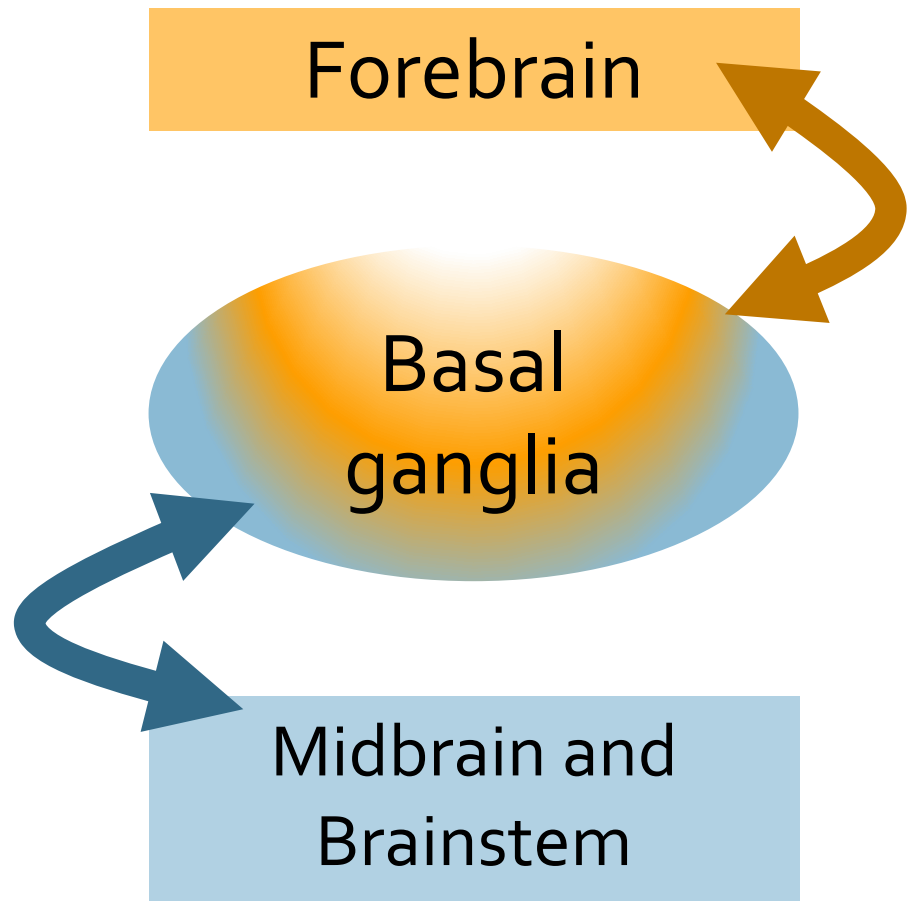


Mary ET Boyle, Ph. D.

Department of Cognitive Science

UCSD

Linking thought and movement **simultaneously!**



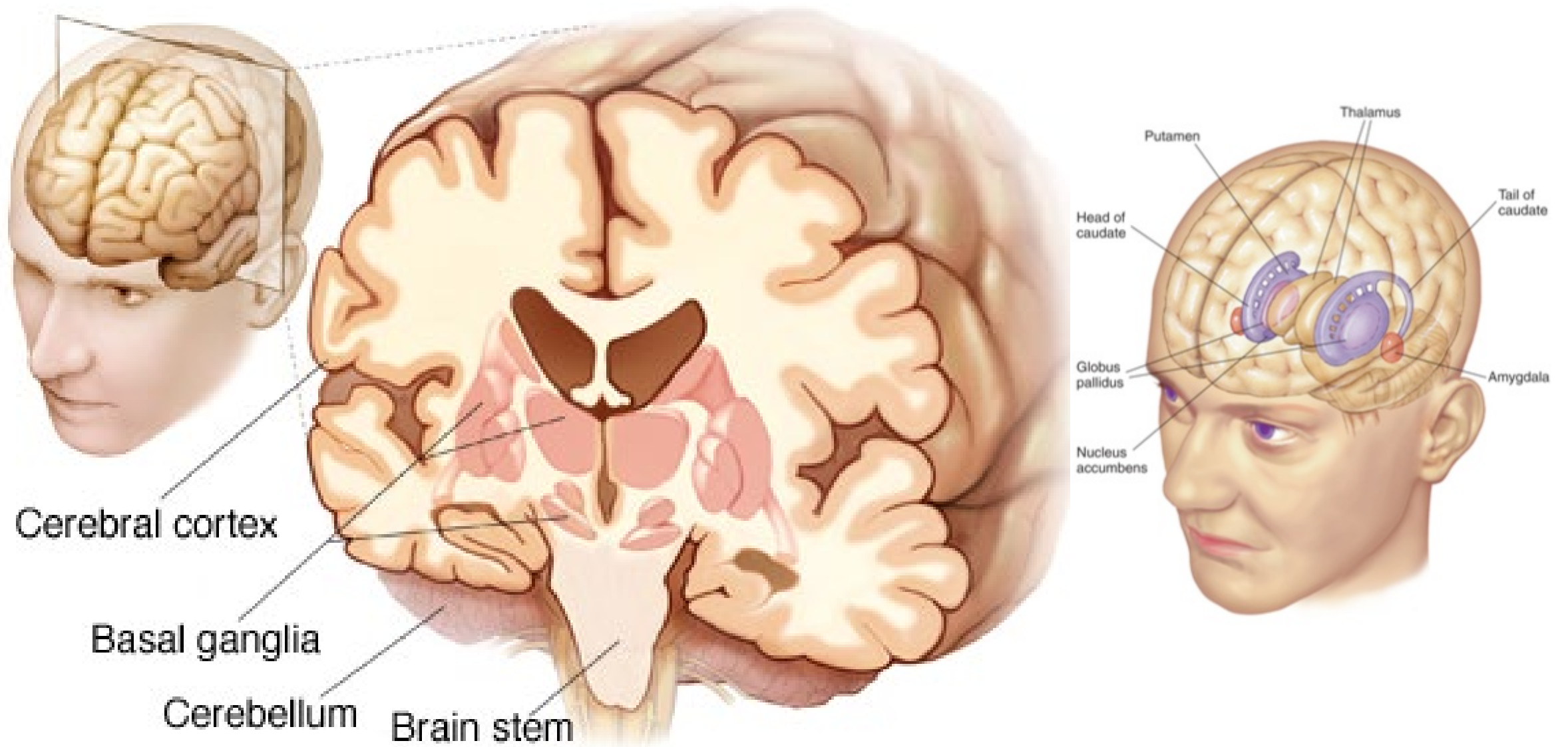
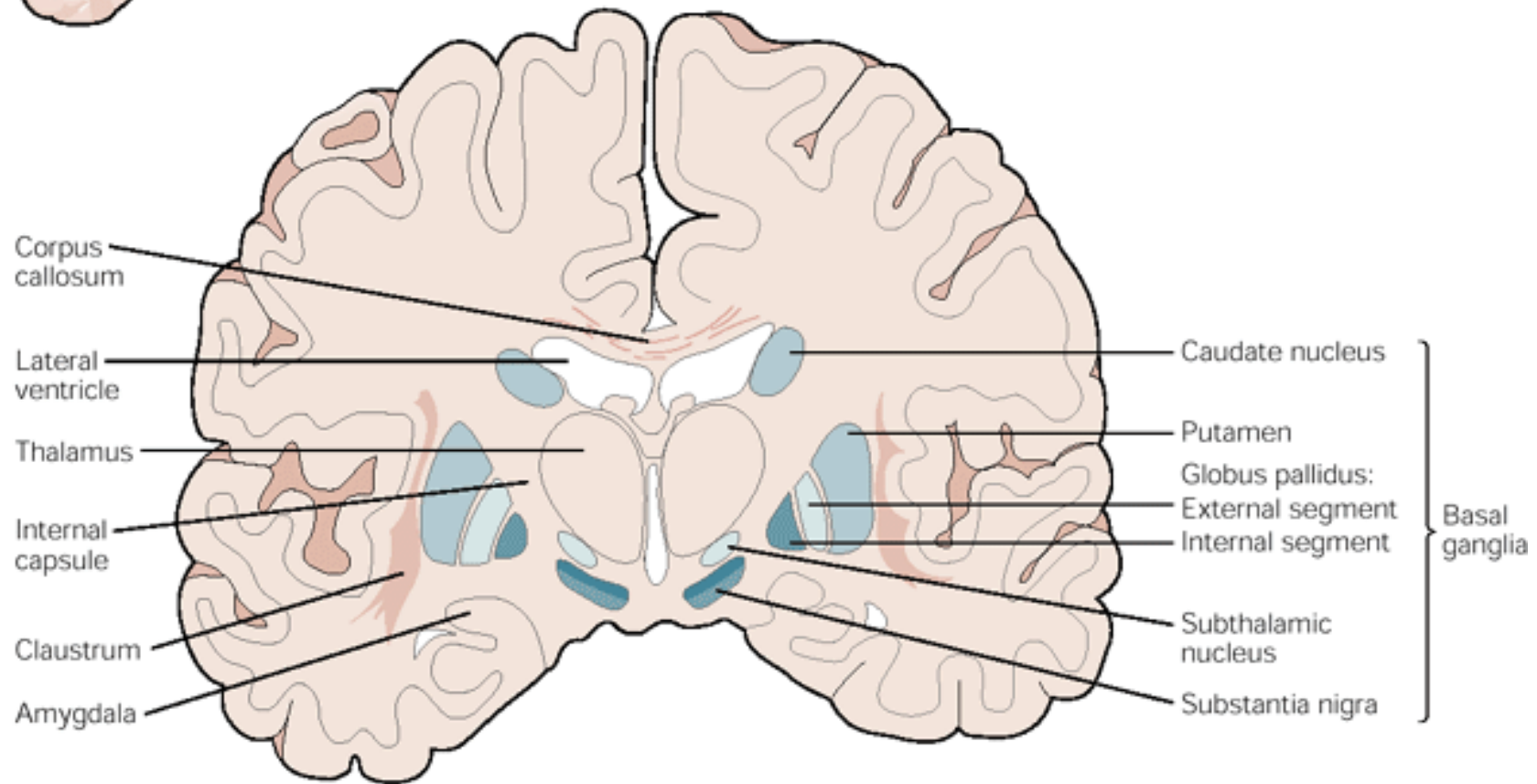
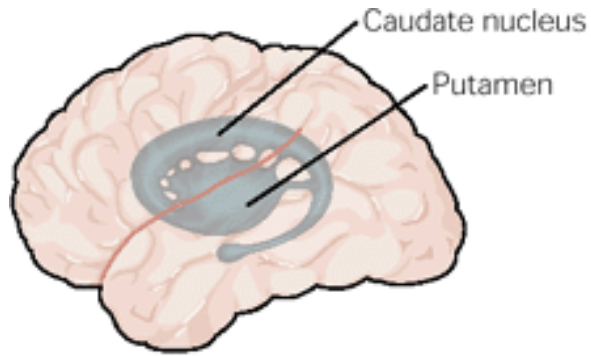
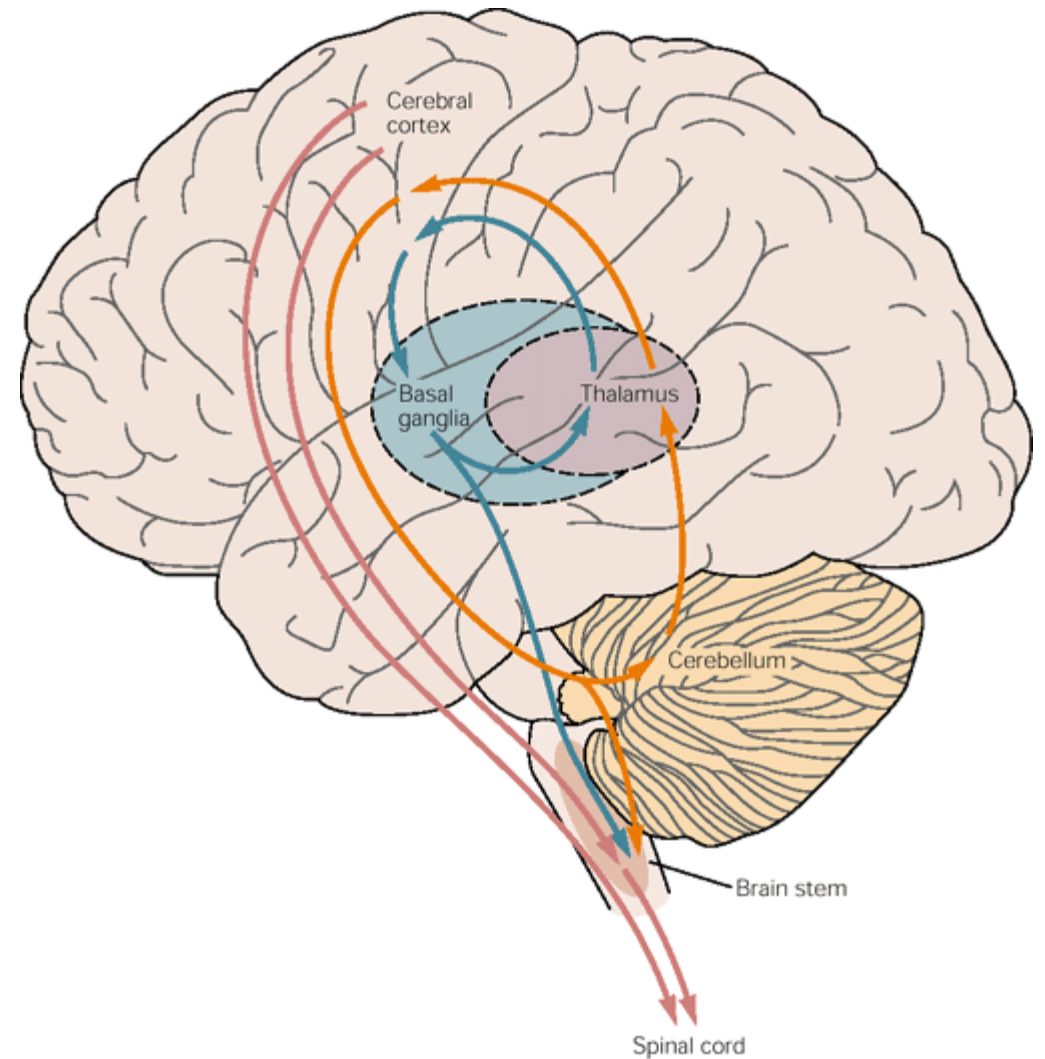
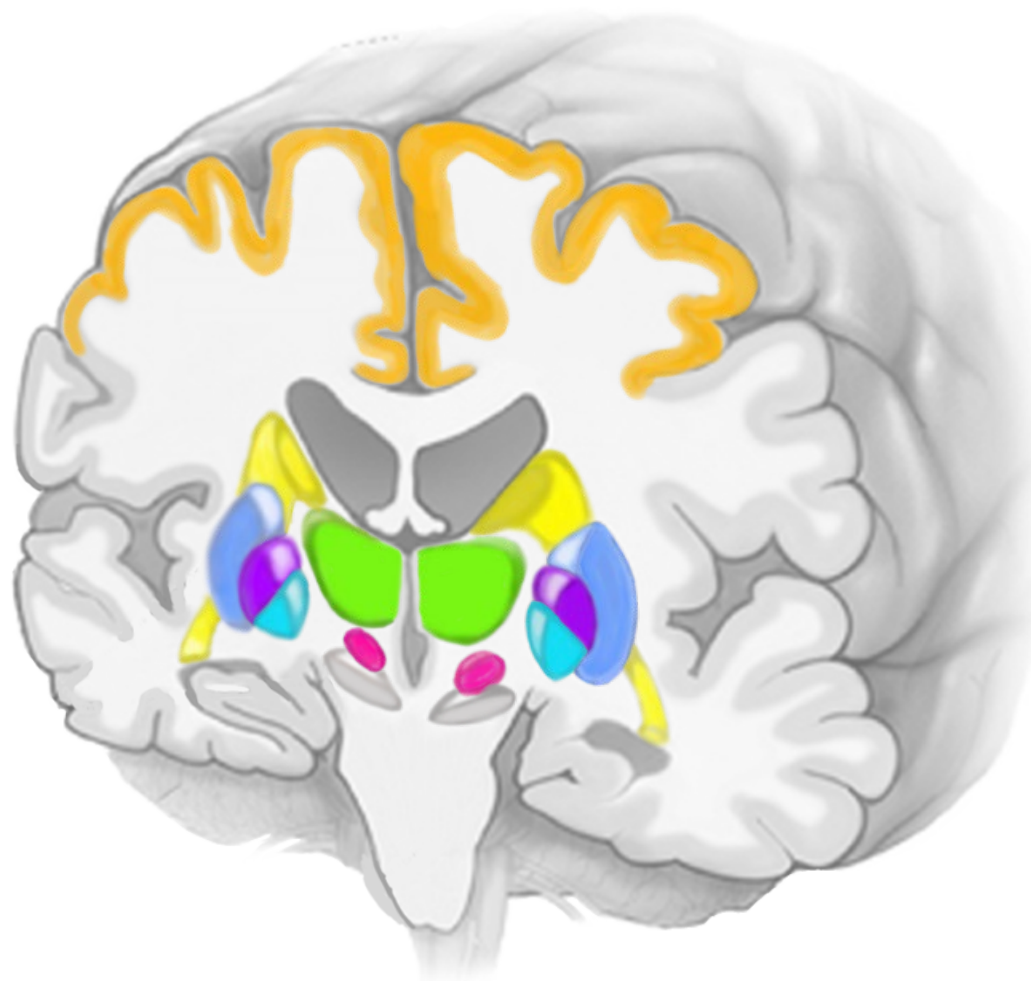
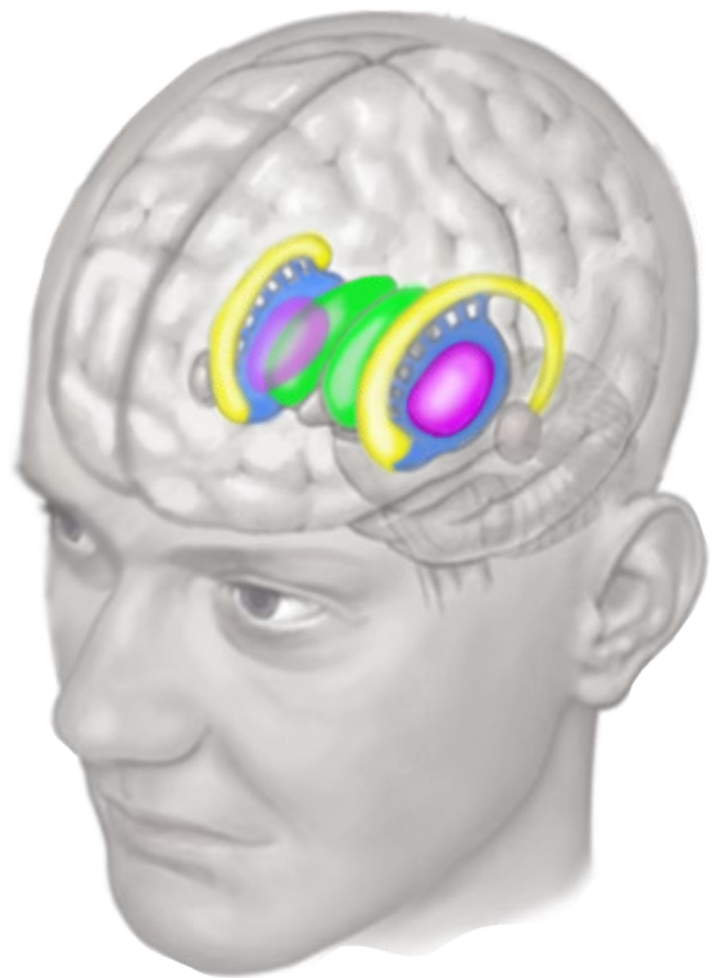


Image from Mayo Clinic



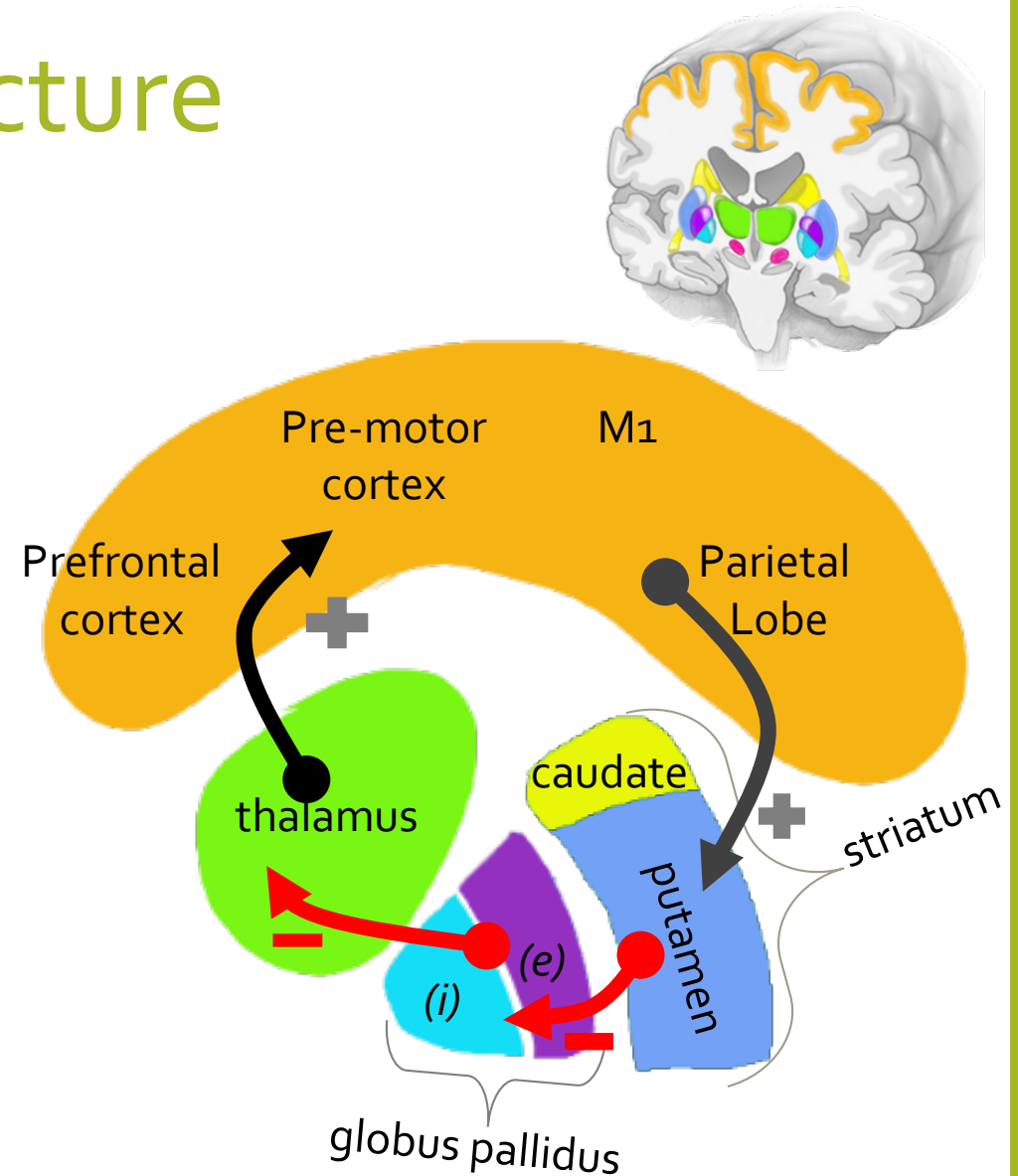
1. Have a major role in normal voluntary movement.
2. BG does not have direct input or output connections with the spinal cord.
3. BG nuclei receive primary input from the cerebral cortex and send output to the brain stem and, via the thalamus, back to the prefrontal, premotor, and motor cortices.
4. The motor functions of the basal ganglia are therefore mediated, in large part, by motor areas of the frontal cortex.

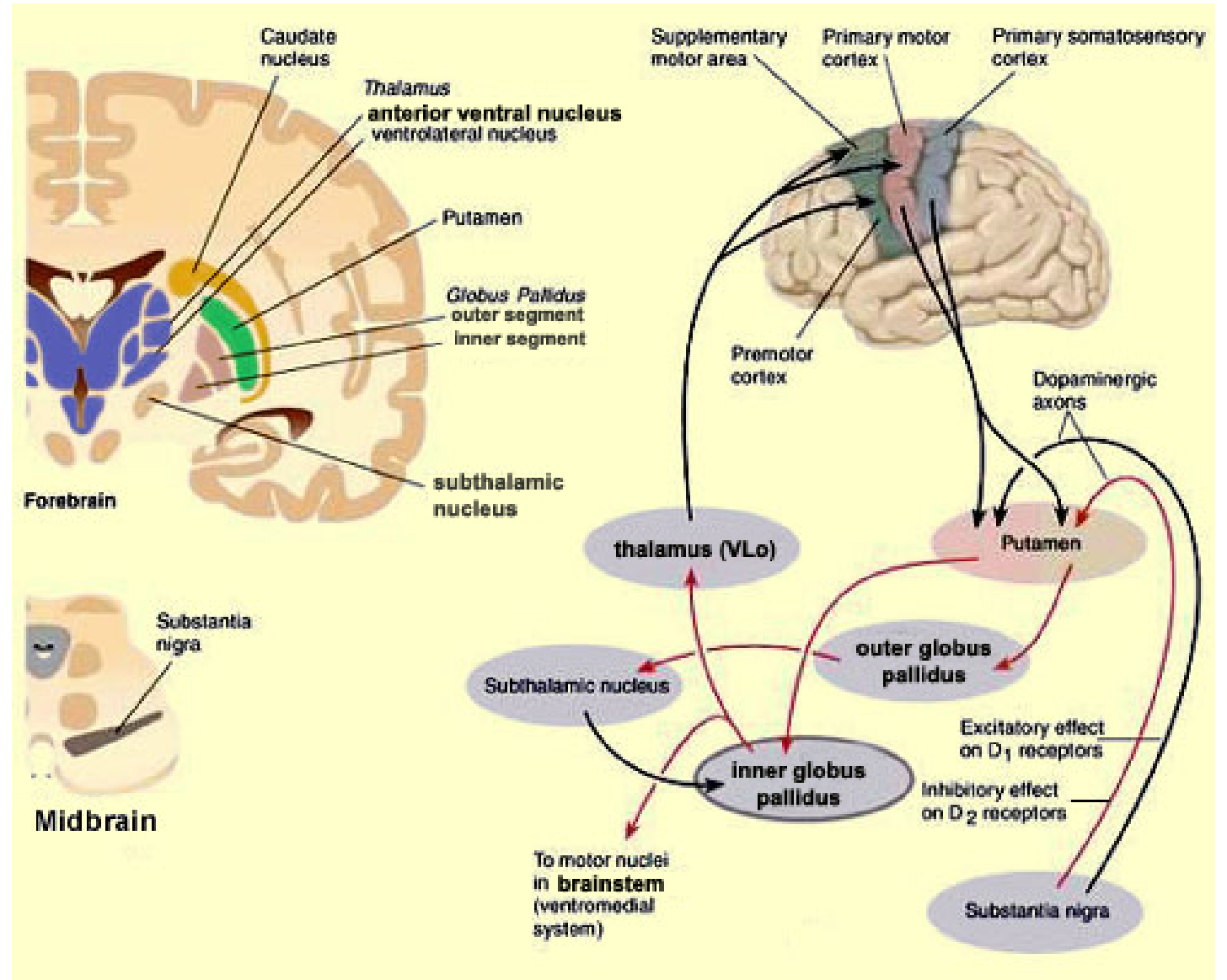
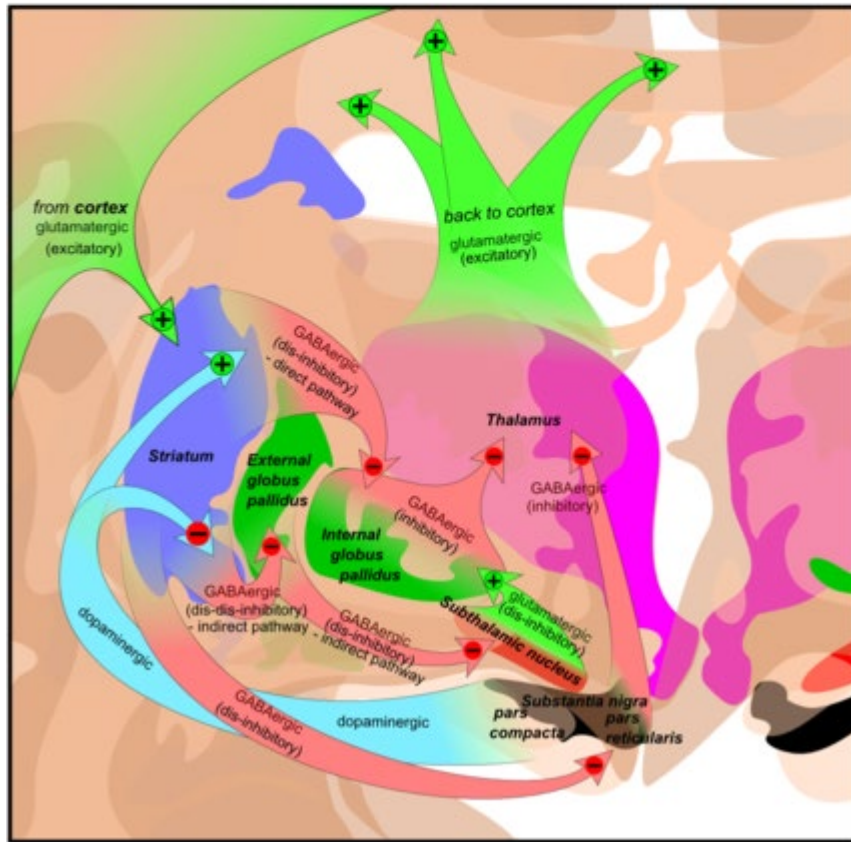
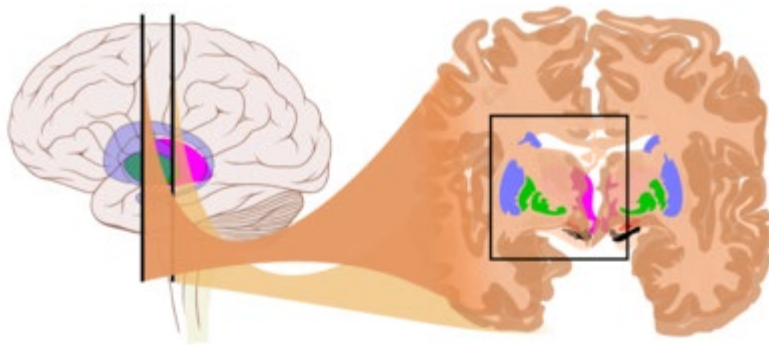




Largest subcortical brain structure

“ The basal ganglia receive inputs from the neocortex and, by way of their output nuclei, the basal ganglia nuclei project massively to thalamic nuclei, which in turn project to the frontal cortex. This anatomy means the basal ganglia are in a prime position to influence the executive functions of the forebrain, such as planning for movement and even cognitive behaviors. ”





Operational definition of habit:

Initial stage

- Behaviors are **goal directed** → get food reward!
- Behaviors have **not** become **automatic**

Extended training

- Regularly performed behaviors on cue
- Cued response – even with lower or **no reward**

Context triggered behavior

- “Behaviors performed not in relation to a current or future goal but rather in relation to a (*successful*) previous goal.”

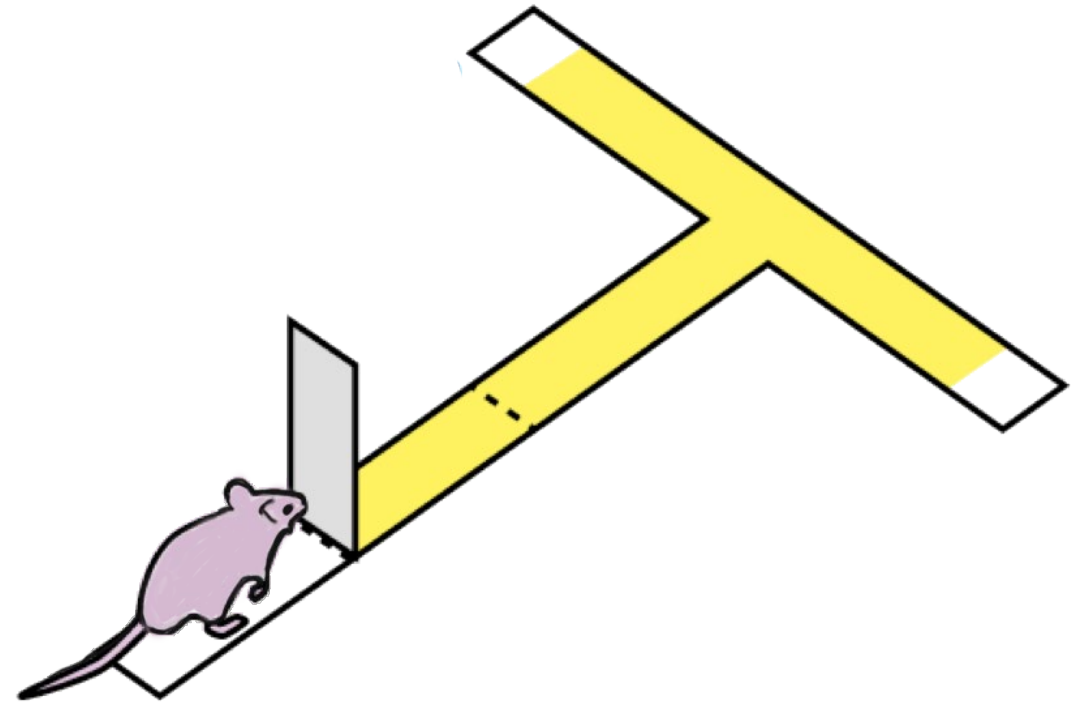
repetitive, sequential,
context-triggered
behaviors

forming a habit

Building Neural Representations of Habits

Mandar S. Jog,^{1*} Yasuo Kubota,^{2*} Christopher I. Connolly,³
Viveka Hillegaart,⁴ Ann M. Graybiel^{2†}

Memories for habits and skills (implicit or procedural memory) and memories for facts (explicit or episodic memory) are built up in different brain systems and are vulnerable to different neurodegenerative disorders in humans. So that the striatum-based mechanisms underlying habit formation could be studied, **chronic recordings from ensembles of striatal neurons were made with multiple electrodes as rats learned a T-maze procedural task.** Large and widely distributed **changes in the neuronal activity patterns occurred in the sensorimotor striatum during behavioral acquisition, culminating in task-related activity emphasizing the beginning and end of the automatized procedure.** The new ensemble patterns remained stable during weeks of subsequent performance of the same task. These results suggest that the encoding of action in the sensorimotor **striatum undergoes dynamic reorganization as habit learning proceeds.**

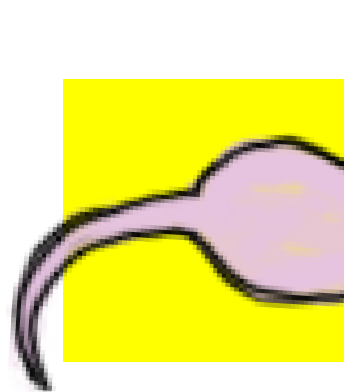
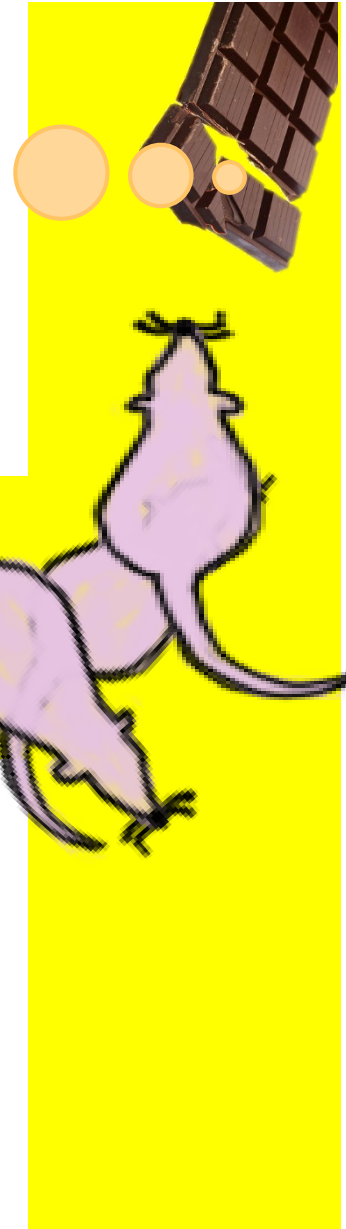


Animals with injured basal ganglia developed problems with tasks such as learning how to run through mazes.

“...no discernible pattern in their meanderings. It seemed as if each rat was taking a leisurely, unthinking stroll.”

What are the chances of finding chocolate here???

Yum!



Rat would wander

Sniffing corners

Scratching walls

Could not figure out how to find the chocolate at first.

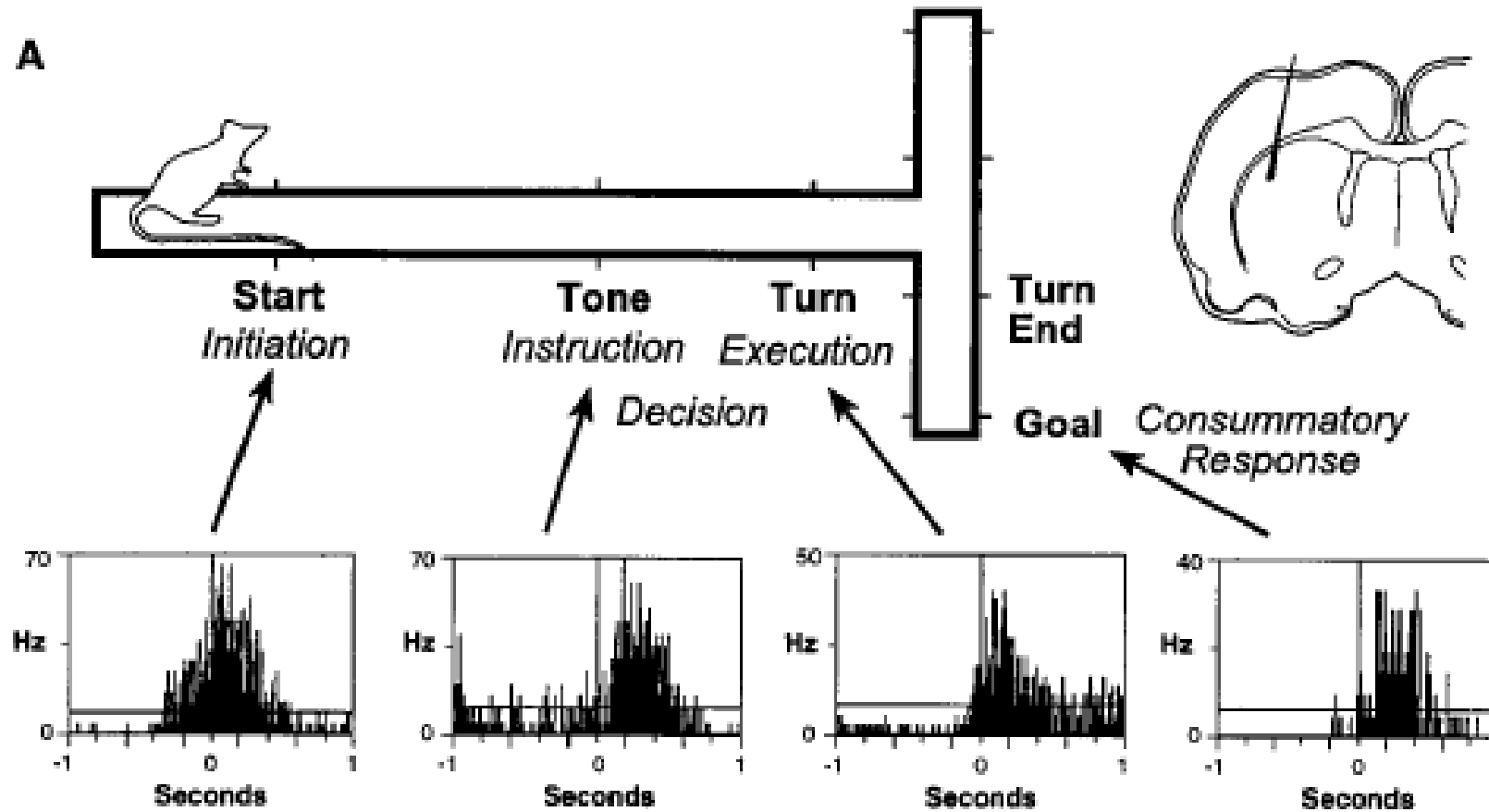
While the animal wandered through the maze – **the basal ganglia was firing furiously**. When the rat sniffed or scratched a wall the brain exploded with activity.

The experiment was repeated hundreds of times

- Rat's brain activity changed.
- Rat stopped sniffing corners and making wrong turns.
- Zipped through the maze much faster.

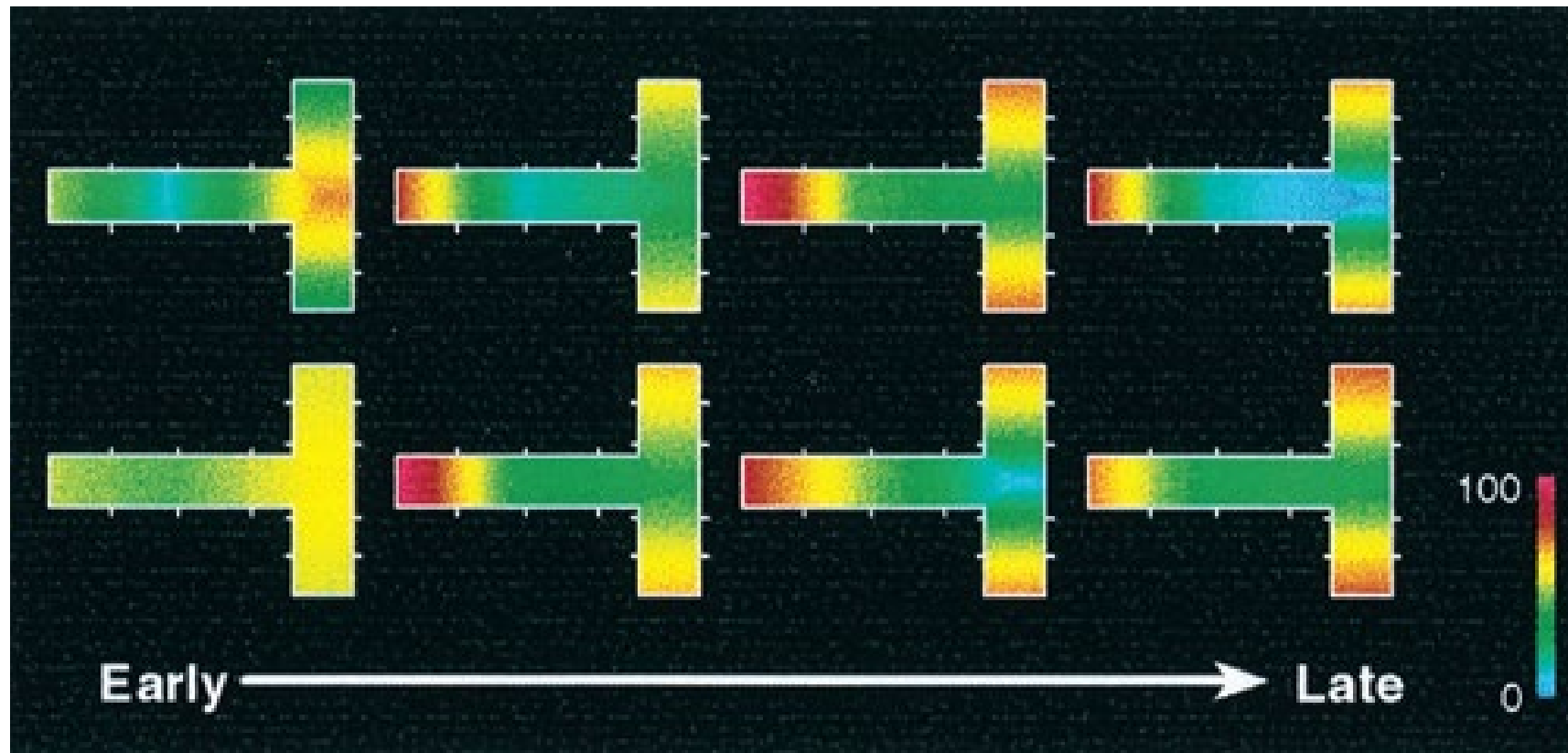
"As each rat learned how to navigate the maze, its mental activity *decreased*. As the route became more and more automatic, each rat started thinking less and less."

Experimental paradigm for assessing habit learning.



Event-related neuronal firing patterns were recorded in relation to start, tone, turn, and goal reaching from electrodes placed in the sensorimotor striatum (dorsolateral caudo-putamen).

Reorganization of neuronal activity in the sensorimotor striatum during habit learning.



Schematic activity maps representing the average proportion of task-related units responsive to different parts of the task from early to late in training.



Restructuring
striatal neural
response
during habit
formation

1



Activity
became
centered
around the
beginning and
end of the task

2



Dopamine
neurons shifted
their firing pattern
to respond to the
earliest indicator
of reward

3



Dopamine –
containing
neurons fired
predictively.

4

Associated with habits

- Receives the most input from the cortex

striatum

Decision to perform an action

- Automatic habit response

caudate

Dopamine input

- Receives (DA) input from substantia nigra (pc)

putamen

Output

- Global Pallidus (i/e)



Is pathological gambling a brain disorder?

*NEUROBEHAVIORAL EVIDENCE FOR THE “NEAR-MISS” EFFECT IN
PATHOLOGICAL GAMBLERS*

REZA HABIB AND MARK R. DIXON

SOUTHERN ILLINOIS UNIVERSITY

The purpose of this translational study was twofold: (1) to contrast behavioral and brain activity between pathological and nonpathological gamblers, and (2) to examine differences as a function of the outcome of the spin of a slot machine, focusing predominately on the “Near-Miss”—when two reels stop on the same symbol, and that symbol is just above or below the payoff line on the third reel. Twenty-two participants (11 nonpathological; 11 pathological) completed the study by rating the closeness of various outcomes of slot machine displays (wins, losses, and near-misses) to a win. No behavioral differences were observed between groups of participants, however, differences in brain activity were found in the left midbrain, near the substantia nigra and ventral tegmental area (SN / VTA). Near-miss outcomes uniquely activated brain regions associated with wins for the pathological gamblers and regions associated with losses for the nonpathological gamblers. Thus, near-miss outcomes on slot machines may contain both functional and neurological properties of wins for pathological gamblers. Such a translational approach to the study of gambling behavior may be considered an example that gives life to B. F. Skinner’s conceptualization of the physiologist of the future.

Key words: pathological gambling, fMRI, near-miss, slot machine, addiction

Pathological Gamblers

People who have lied to their families about their gambling,
Missed work to gamble
Bounced checks at casinos

Social Gamblers

No problematic behaviors with gambling.

LUCKY 7s 2014

7

7

7

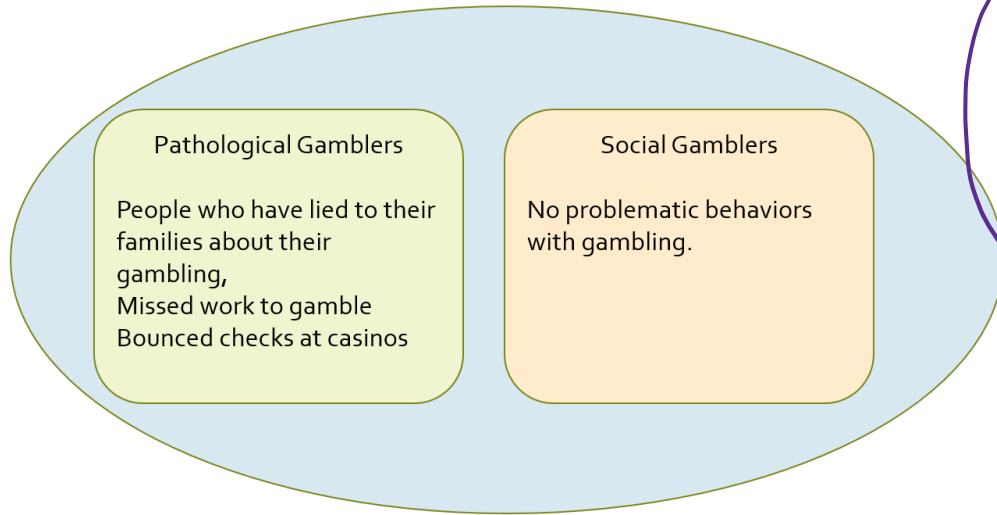
The slot machine was programmed to deliver three outcomes:

win

loss

near miss

slots almost matched up but, at the last moment, failed to align



SAME BRAIN SYSTEM
MAY BE INVOLVED
IN HABITS & ADDICTIONS.

- Pathological gamblers got more excited about winning
- Brain areas associated with emotion and reward much more active
- Near misses looked like wins
- **Brains reacted the same for a near miss as a win.**
- Pathological gamblers got a "mental high" from a near miss.
- Social gamblers → near miss = a loss
 - "I should quit now before I lose more"

Non pathological gamblers – near misses looked like a loss.

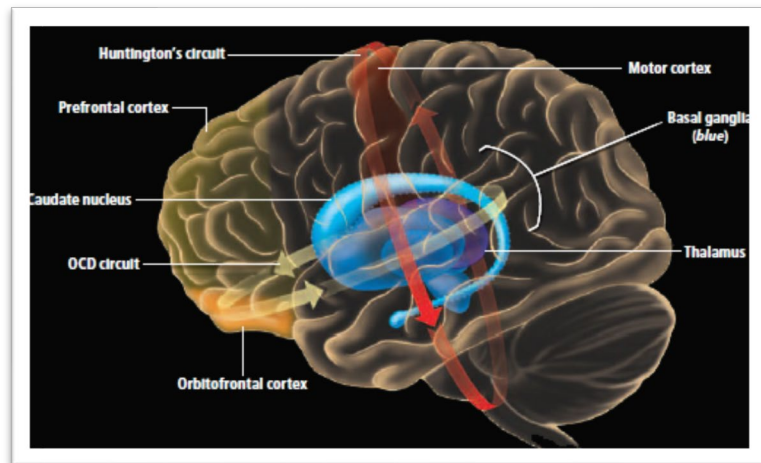
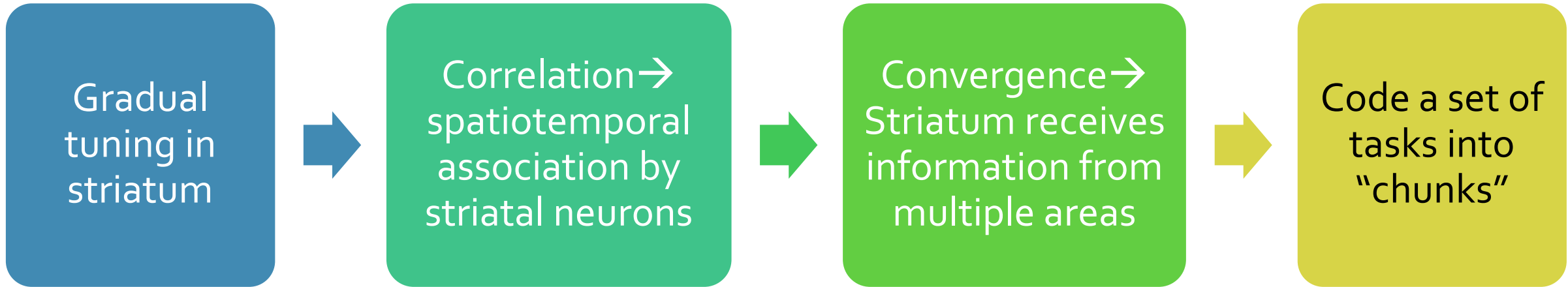
Harrah's Entertainment "Pavlovian Marketing Scheme"

- Leader in sophisticated customer-tracking systems
- computer programs that studied gamblers' habits
- Harrah's tried to figure out how to persuade them to spend more money and time at the casino
- Players were assigned a "predicted lifetime value" score
- Software created calendars that anticipated how often gamblers would visit and how much they would spend
- The company tracked customers through loyalty cards
- In addition they mailed out coupons for free meals and cash vouchers
- Telemarketers called people at home



Adding a near miss to a lottery is like pouring jet fuel on a fire," said a state lottery consultant who spoke to me on the condition of anonymity. "You want to know why sales have exploded? Every other scratch-off ticket is designed to make you feel like you almost won."

How is the template developed?

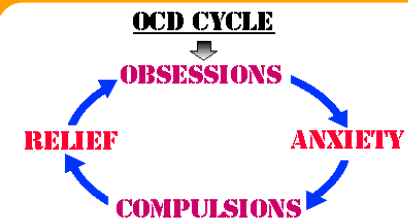


OCD?



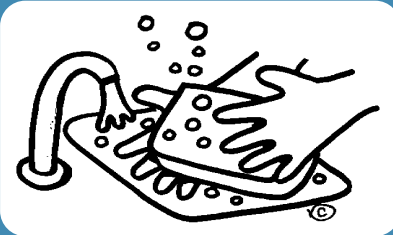
Obsessive-compulsive disorder

- OCD
- Once considered as neurosis – psychic conflict



Characteristics:

- Intrusive, repetitive thoughts -- obsessions
- Impaired by the need to perform stereotypic, repetitive rituals – compulsions



Examples

- Contaminated – wash hands repetitively
- Failed responsibility – keep checking stove



Patients know

- Their thoughts are senseless
- Cannot control the obsession or compulsion – “mental tics”

obsessions

Highly
negative
thoughts

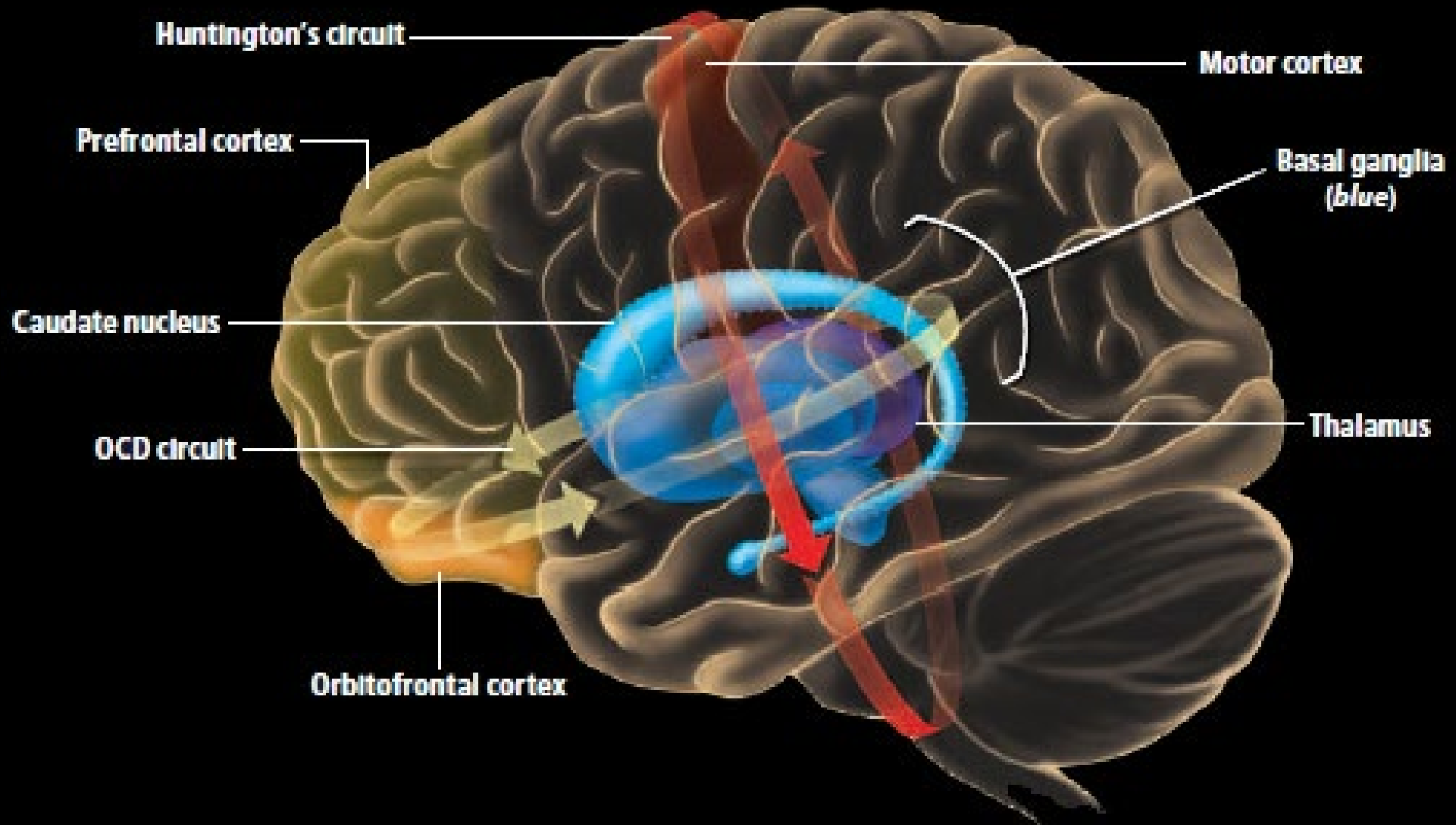


compulsions

Rituals
designed to
lessen anxiety



OCD CIRCUIT



Orbitofrontal cortex

- Involved in complex tasks
- e.g. decision making

Ventral caudate nucleus

- Located within the basal ganglia
- Basal ganglia: centers for initiating and coordinating various aspects of movement (including involuntary tics)

Thalamus

- Relays and integrates sensory information

Brain Lock Unlocked

USING PLASTICITY TO STOP WORRIES, OBSESSIONS,
COMPULSIONS, AND BAD HABITS



BY NORMAN DOIDGE

Recall the
following
information from
the OCD reading:

// We detect mistakes with our *orbital frontal cortex*, part of the frontal lobe, on the underside of the brain, just behind our eyes. Scans show that the more obsessive a person is, the more activated the orbital frontal cortex is.

Once the orbital frontal cortex has fired the “mistake feeling,” it sends a signal to the *cingulate gyrus*, located in the deepest part of the cortex. The cingulate triggers the dreadful anxiety that something bad is going to happen unless we correct the mistake and sends signals to both the gut and the heart, causing the physical sensations we associate with dread.

The “automatic gearshift” the *caudate nucleus*, sits deep in the center of the brain and allows our thoughts to flow from one to the next unless, as happens in OCD, the caudate becomes extremely “sticky.”⁶

Brain scans of OCD patients show that all three brain areas are hyperactive. The orbital frontal cortex and the cingulate turn on and stay on as though locked in the “on position” together—one reason that Schwartz calls OCD “brain lock.” Because the caudate doesn’t “shift the gear” automatically, the orbital frontal cortex and the cingulate continue to fire off their signals, increasing the mistake feeling and the anxiety. Because the person has already corrected the mistake, these are, of course, false alarms. The malfunctioning caudate is probably overactive because it is stuck and is still being inundated with signals from the orbital frontal cortex.

The causes of severe OCD brain lock vary. In many cases it runs in families and may be genetic, but it can also be caused by infections that swell the caudate.⁷ And, as we shall see, learning also plays a role in its development. //

Dopaminergic Network Differences in Human Impulsivity

Joshua W. Buckholtz,^{1,2,*†} Michael T. Treadway,^{1†} Ronald L. Cowan,^{3,4} Neil D. Woodward,^{3,4}
Rui Li,⁵ M. Sib Ansari,⁵ Ronald M. Baldwin,⁵ Ashley N. Schwartzman,¹ Evan S. Shelby,¹
Clarence E. Smith,³ Robert M. Kessler,⁵ David H. Zald^{1,3}

Buckholtz J. et al. July 30, 2010 Science

Impulsivity
variation among
individuals

Ability to deliberate
on consequences of
an action.

Impulsivity linked
to dopamine

Learning and
reward.

OCD and the striatum



Abnormal activity in
OCD patients

Changes in
abnormal activity
with treatment

OCD symptoms:
sequential repetitive
behaviors driven by
compulsions

Behaviors are
performed as
"chunks"

NEUROBIOLOGY OF LEARNING AND MEMORY **70**, 119–136 (1998)

ARTICLE NO. NL983843

The Basal Ganglia and Chunking of Action Repertoires

Ann M. Graybiel

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Cambridge, Massachusetts 02139*

The basal ganglia have been shown to contribute to habit and stimulus–response (S–R) learning. These forms of learning have the property of slow acquisition and, in humans, can occur without conscious awareness. This paper proposes that one aspect of basal ganglia-based learning is the recoding of cortically derived information within the striatum. Modular corticostriatal projection patterns, demonstrated experimentally, are viewed as producing recoded templates suitable for the gradual selection of new input–output relations in cortico-basal ganglia loops. Recordings from striatal projection neurons and interneurons show that activity patterns in the striatum are modified gradually during the course of S–R learning. It is proposed that this recoding within the striatum can chunk the representations of motor and cognitive action sequences so that they can be implemented as performance units. This scheme generalizes Miller’s notion of information chunking to action control. The formation and the efficient implementation of action chunks are viewed as being based on predictive signals. It is suggested that information chunking provides a mechanism for the acquisition and the expression of action repertoires that, without such information compression would be biologically unwieldy or difficult to implement. The learning and memory functions of the basal ganglia are thus seen as core features of the basal ganglia’s influence on motor and cognitive pattern generators. © 1998 Academic Press

Post surgery: the reward was given one step earlier

Training:
Two monkeys trained in 3-step task
Reward always came at end.

Lesion before training

Stopped after 2nd step!

Prevented the chunking

Lesion after training

Continued with the 3 steps

Bound the behaviors into a chunk

It was a habit!

unilateral lesion of the nigrostriatal system

Graybiel, A. (1998)

Slow learning and chunking

slow learning is
necessary

NOT paying
attention to task
is essential

repeated over
time

“the predictive firing of dopamine-containing neurons and the chunking of habit steps makes it especially difficult to break a habit once it is formed.”