A TALK IN THREE PARTS: PART 2
BALANCING REACTIVE AND PLANNED MOVEMENTS:

How Basal Ganglia Interact with Cortical and Subcortical Networks during Learning and Action

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PLANNING VS. REACTING

Reactive unlearned behaviors

such as attending to novel events and dangers

need to be FAST

e.g., looking at a sudden peripheral movement or sound

Planned learned behaviors

are SLOW to emerge

reduce attentional salience of irrelevant events

and obey IF-THEN rules

HOW TO BALANCE REACTIVE AND PLANNED BEHAVIORS?

A Cognitive-Emotional Balancing Act

Since reactive behaviors are fast, why do they not always win?

How does the brain wait for a plan that is slow to form?

How does the brain know that a plan is being formulated before it is selected?!

How does the brain wait until competing plans settle on one winning plan?

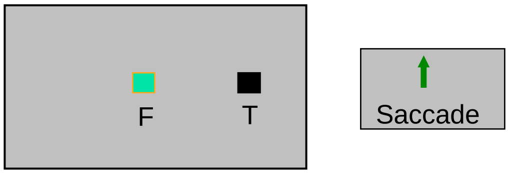
How does such a slow plan finally win over a fast reactive behavior?

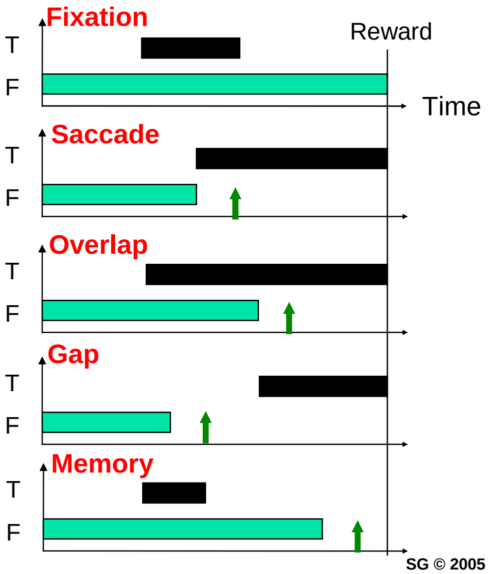
How can reactive behaviors nonetheless be performed quickly when there are no plans being formulated?

How does CogEM learning, notably reward and punishment, bias plan selection?

Basal Ganglia Gate the Selective Release of Actions

SACCADIC EYE MOVEMENT LEARNING TASKS





Develop a model that:

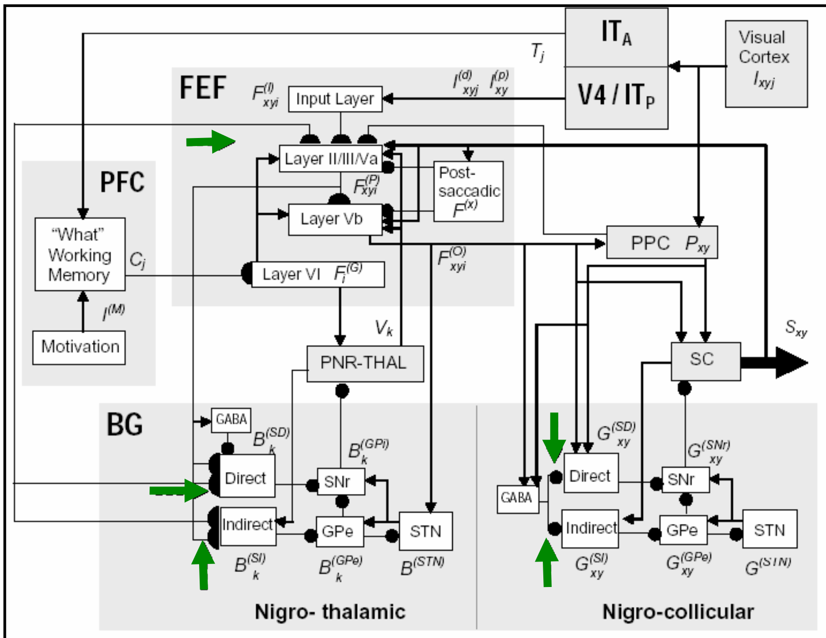
Learns these tasks and more

Does not forget previous tasks

All with the same parameters

SEVERAL TYPES OF LEARNING

Recognition	Identify	What
Reinforcement	Evaluate	Why
Timing	Synchronize	When
Spatial	Locate	Where
Motor Control	Act	How



TELOS Model

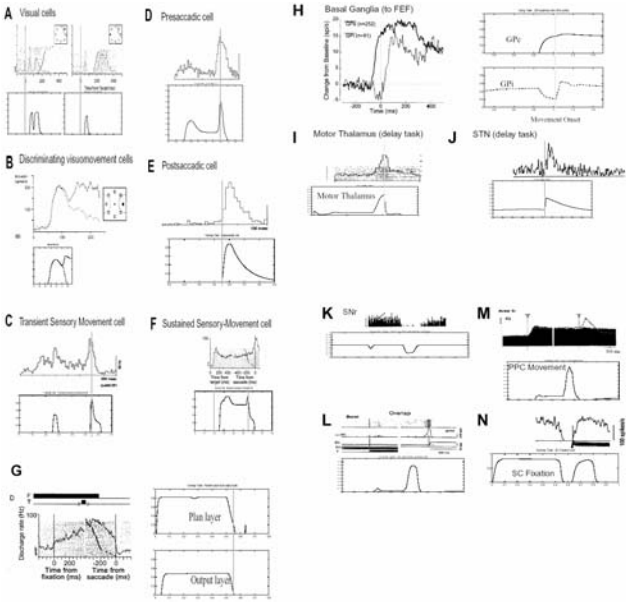
TElencephalic
Laminar
Objective
Selector

This model learns and remembers IF-THEN rules for voluntary movement in the 5 primate tasks.

→ Dopamine

Brown, Bullock, and Grossberg (2004, Neural Networks)

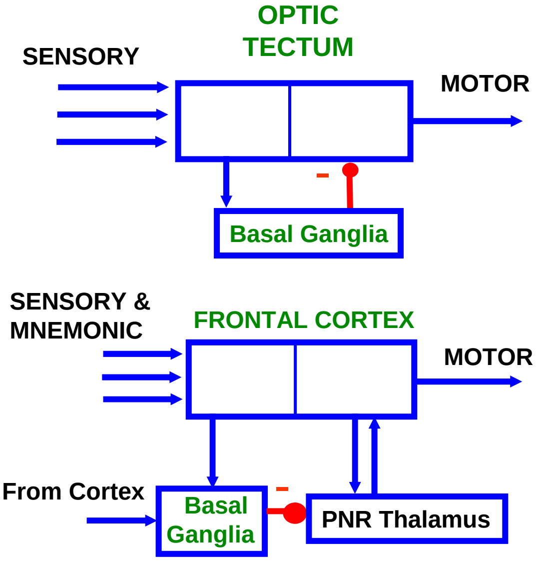
QUANTITATIVE SIMULATIONS BY TELOS OF 17 CELL TYPES



After learning the tasks, TELOS model cell activities quantitatively simulate 17+ types of physiologically recorded cell discharge patterns

Predicts distinct functional roles for them all

AN ANCIENT BRAIN DISCOVERY: SELECTIVE GATING BY THE DORSAL BASAL GANGLIA



SENSORY

OPTIC TECTUM

MOTOR

–

Basal Ganglia

SENSORY & MNEMONIC

FRONTAL CORTEX

MOTOR

From Cortex

Basal Ganglia

–

PNR Thalamus

In amphibians, the basal ganglia control (gate) orienting movement generation by the optic tectum

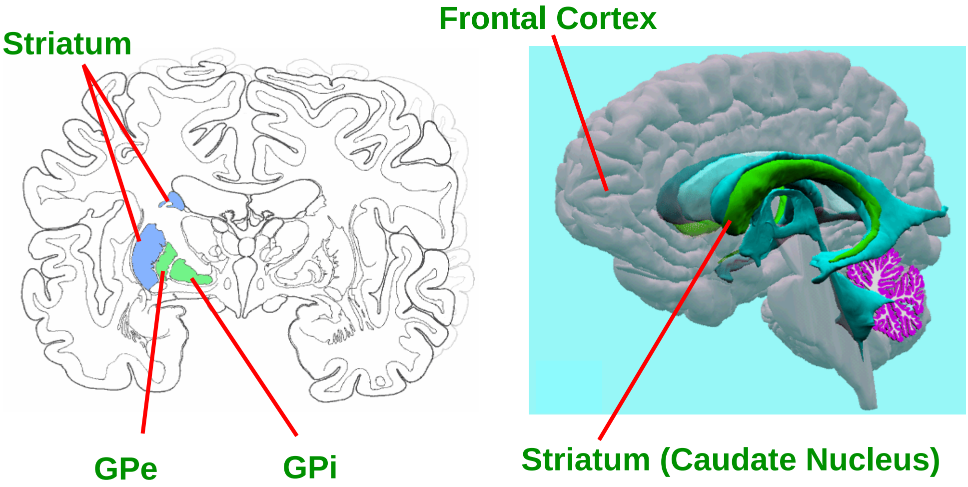
This gate is normally CLOSED (–)

Opening the normally-closed gate via transient removal of inhibition permits action

Hikosaka & Wurtz (1989)

In mammals, additional basal ganglia channels control frontal cortex areas (e.g., FEF) via the PNR (pallidum or nigra recipient) zones of the thalamus

BASAL GANGLIA AND FRONTAL CORTEX INTERACT TO CONTROL REACTIVE AND PLANNED MOVEMENTS

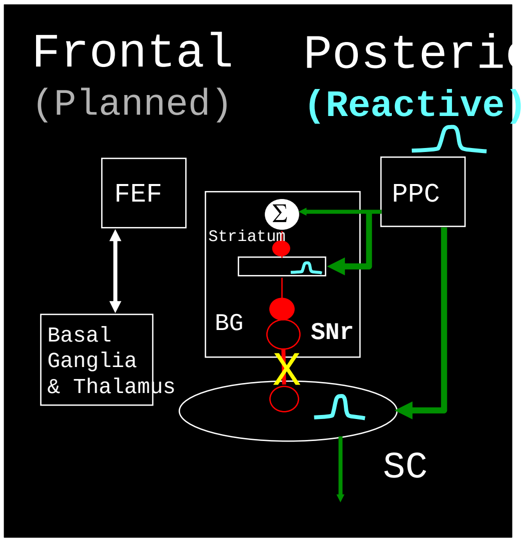


REACTIVE BASAL GANGLIA GATING

How can the oculomotor system rapidly REACT to visual targets but at other times substitute slower PLANNED responses?

TELOS: Quiet frontal cortex allows REACTIVE target to quickly open SNr gate, leading to saccade to sensory target

Reaction Time: 50-100 ms

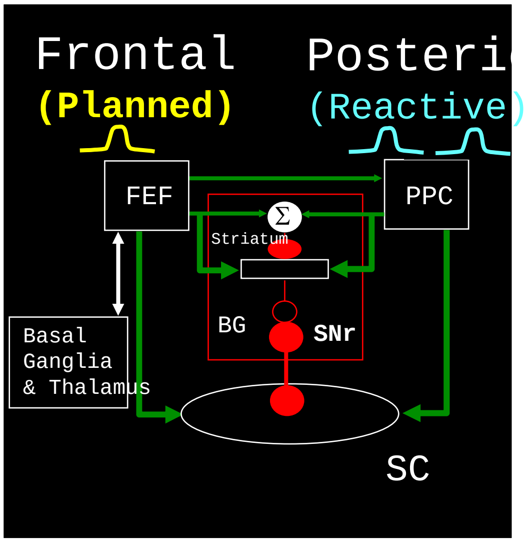


COMPETITION BETWEEN FRONTAL PLAN AND PARIETAL REACTIVE COMMANDS

When more than one reactive target and frontal plan coexist, SUMMED FEEDFORWARD INHIBITION in the striatum prevents either target from opening the SNr gate

Frontal plan can suppress reactive saccade and substitute PLANNED response to a different target

Reaction time: 200 ms or more



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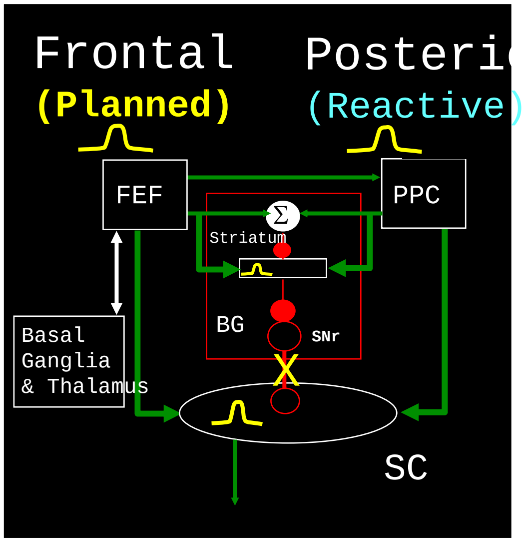
GATE OPENS AFTER PLAN RESOLVES CONFLICT

Frontal plan overrides reactive pathway and REPROGRAMS posterior cortex by selecting consistent targets and suppressing inconsistent ones

cf. ART Matching Rule

FRONTO-POSTERIOR AGREEMENT allows summed excitation of a striatal projection neuron to overcome feedforward inhibition: opens gate

Yeterian & Van Hoesen (1978)
Flaherty & Graybiel (1991)
Koos & Tepper (1999)



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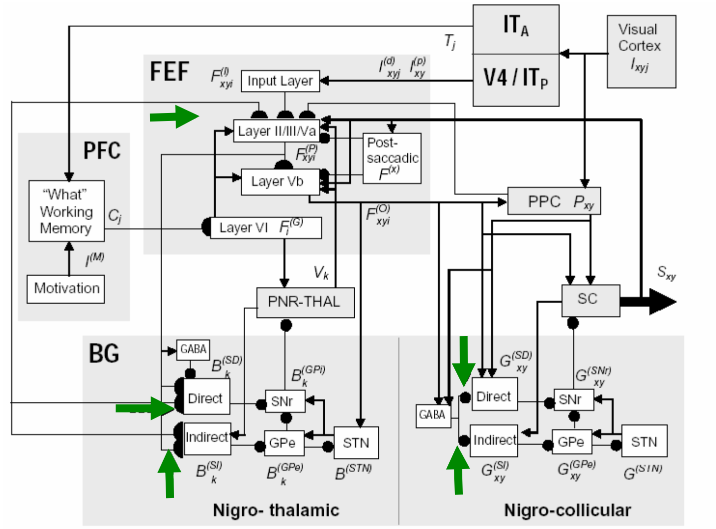
HOW ARE PLANNED RESPONSES LEARNED?

How does the brain generate reward-based signals
that can modulate learning of new
sensory - to - plan
plan - to - gate
plan - to - movement
associations?

How do rewarding experiences enable DOPAMINE to act as a reward-sensitive NOW PRINT learning signal?

MULTIPLE SITES OF DOPAMINE ACTION IN FEF AND BG



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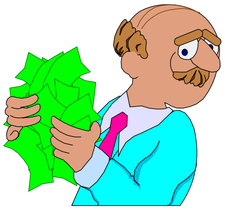
➡ Dopamine

Brown, Bullock, and Grossberg (2004, Neural Networks)

BRAIN'S COMPUTE REWARD EXPECTATION: MAGNITUDE AND TIMING



Mismatch the expected MAGNITUDE of a reward
- Waiter's Lament:
  - Tab: $50
  - Tip: $1
  - DISTRESS!

Mismatch the expected TIMING of a reward
- Late Paycheck:
  - Payday: Friday
  - Paycheck unexpectedly delayed until: Monday
  - DISTRESS! (on Friday)

UNEXPECTED REWARDS AND NON-REWARDS

How does the brain selectively detect
- unexpected rewards?
- unexpected NON-rewards?

How are brain expectations of reward
- adaptively timed?

How are reward signals translated into
- behavioral reinforcement?

REWARD EXPECTATION TRIGGERS DOPAMINE

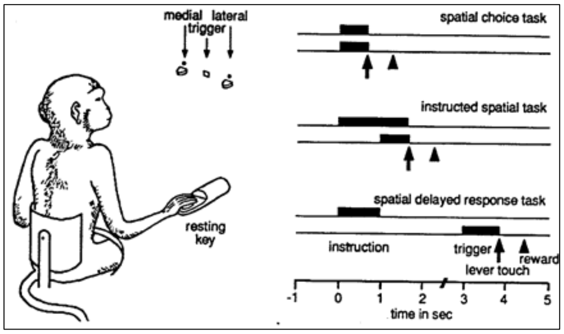


Figure from Schultz et al. (1993)

How does a monkey learn to predict the timing and magnitude of reward for a given task?

Monkey learns to push lever in response to instruction/trigger light conditioned stimuli (CS).

Apple juice given as primary reward

Dopamine cells of Substantia Nigra pars compacta (SNc) learn reward expectation

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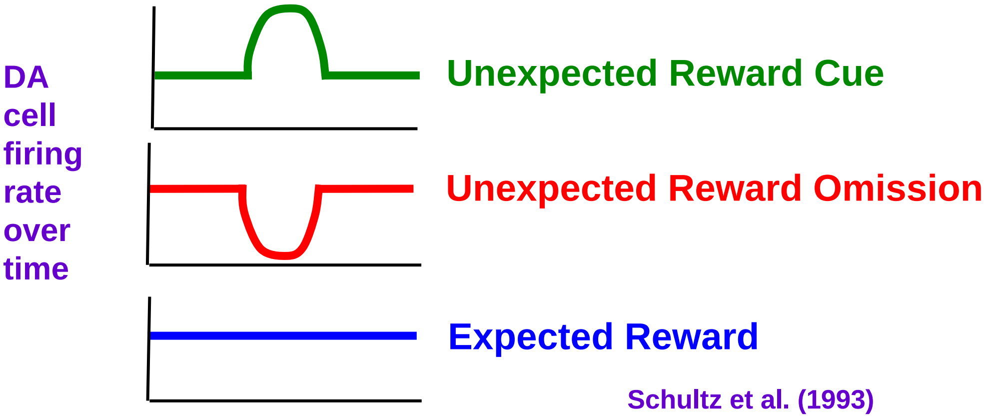
HOW DOES THE BRAIN PROCESS REWARD INFORMATION TO GUIDE LEARNING?

Mismatches with learned reward expectations lead to Bursts and Dips of a broadcast signal: DOPAMINE (DA)



DA cell firing rate over time

Unexpected Reward Cue

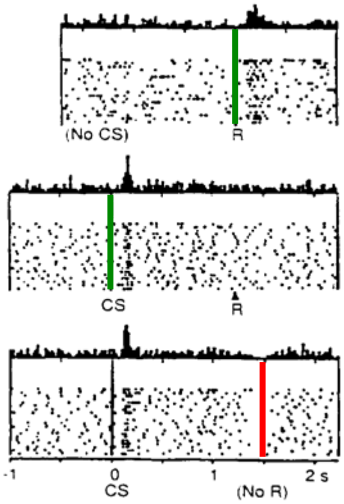
Unexpected Reward Omission

Expected Reward

Schultz et al. (1993)

SNc DOPAMINE CELL DATA

Data



Data from Schultz et al. (1997)

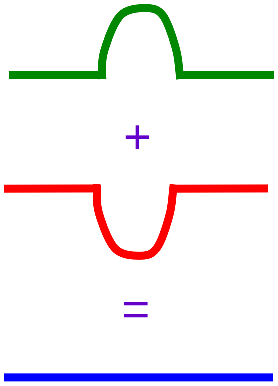
Before training, monkey does not know that instruction light predicts reward availability. Dopamine burst at reward (R) only

After training, monkey learns to expect reward. Dopamine burst at CS only

If reward is omitted, dopamine cell firing depresses when reward is expected

‡Representative of most dopamine cells

LEARNED EXPECTATION
CANCELS EXPECTED REWARD SIGNAL



Reward Signal

+

Expectation

=

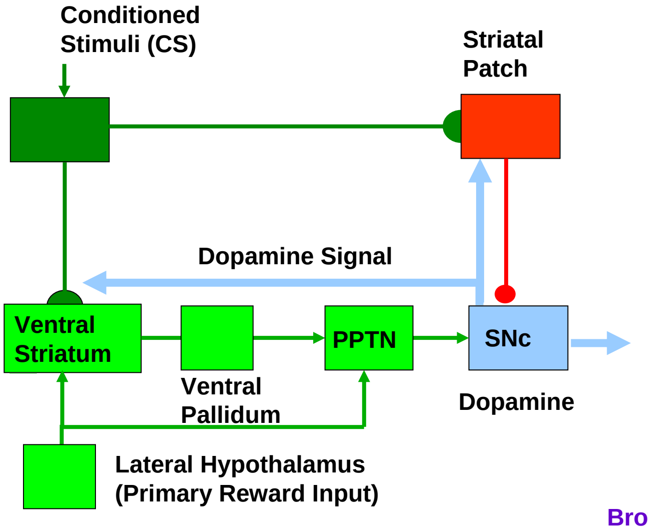
No Signal

One way to explain dopamine cell responses:

Reward cancels expectation after learning

SELECTIVE PROCESSING OF UNEXPECTED REWARDS

Balance Excitatory Learning and Adaptively Timed Inhibitory Learning



Another kind of novelty processing; cf., ART

Adaptively timed inhibitory expectations of reward mGluR

Dopamine cells signal novel events: errors in predicted

REWARD TIMING OR MAGNITUDE

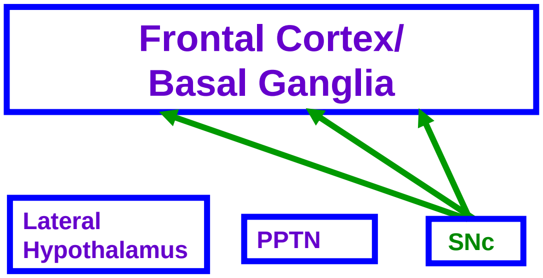
Immediate excitatory predictions of reward

Brown, Bullock & Grossberg, (1999, J. Neuroscience)

REWARD SIGNAL CIRCUITS

Dopamine cells in SNc broadcast reward-related signals to striatum and frontal cortex

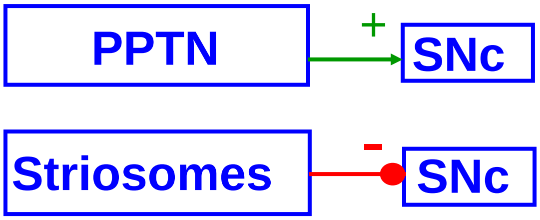
Phasic dopamine modulates LTP/LTD



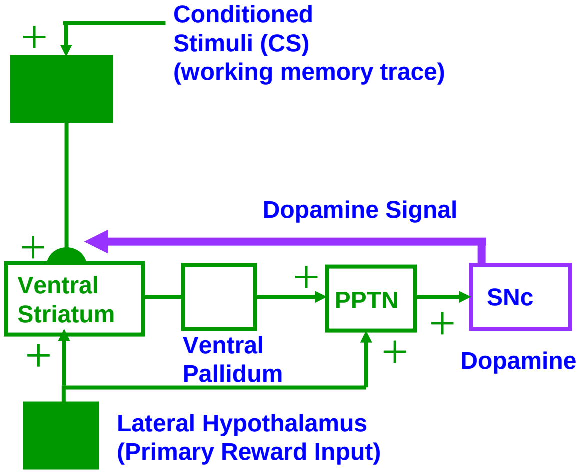
PARALLEL
EXCITATORY AND INHIBITORY PATHWAYS

Immediate excitatory reward signals via the pedunculo-pontine tegmental nucleus (PPTN)

Adaptively-timed inhibitory expectations of reward via striosomes of the striatum.



EXCITATORY PATHWAY



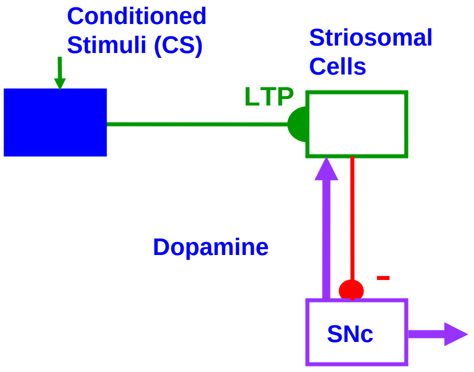
Primary reward (apple juice) briefly excites lateral hypothalamus

Hypothalamic-PPTN excitation causes SNc dopamine burst

Hypothalamic activity excites ventral striatum for training

Active CS working memory signals learn to excite ventral striatum

INHIBITORY PATHWAY

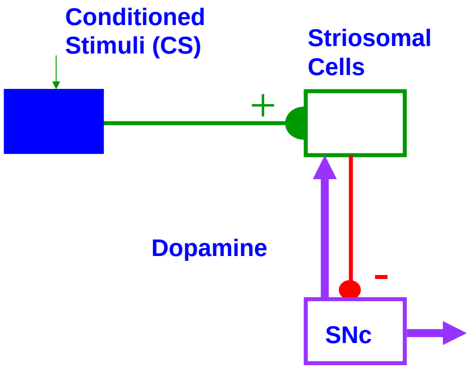


LEARNING: CS-striosomal LTP occurs due to a three-way coincidence:
- An active CS working memory input
- A Ca^{2+} spike
- A dopamine burst

SIGNALING: The delayed Ca^{2+} spike facilitates striosomal-SNc inhibition

Striosomal cells learn to predict both timing and magnitude of reward signal to cancel it: REWARD EXPECTATION

INHIBITORY PATHWAY: EXPECTATION *MAGNITUDE*



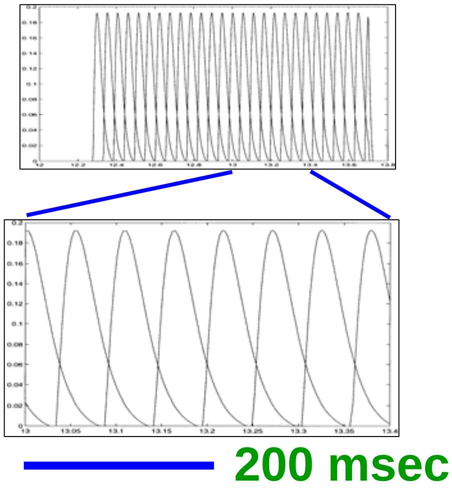
If reward is GREATER than expected, a dopamine burst causes striosomal expectation to increase

If reward is LESS than expected, a dopamine dip causes striosomal expectation to decrease

This is a negative feedback control system for learning

EXPECTATION TIMING

Timing Spectrum



CS activates a population of cells with delayed transient signals (mGluR)

Fiala, Grossberg and Bullock (1996)
Grossberg and Merrill (1996)

Each has a different delay, so that the range of delays covers the entire interval

Delayed transients gate both learning and read-out of expectations

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SECOND MESSENGERS AND SPECTRAL TIMING

A spectrum of transient, adaptively-timed striosomal Ca^{2+} spikes spans the learning interval

Ca^{2+} spike driven by mGluR

Similar mechanism proposed in the Purkinje cells of the cerebellum
Fiala, Grossberg & Bullock (1996)
and the dentate-CA3 circuit in the hippocampus
Grossberg & Schmajuk (1989), Grossberg & Merrill (1992, 1996)

Supported by recent calcium imaging studies
Takechi et al. (1998)
Finch & Augustine (1998)

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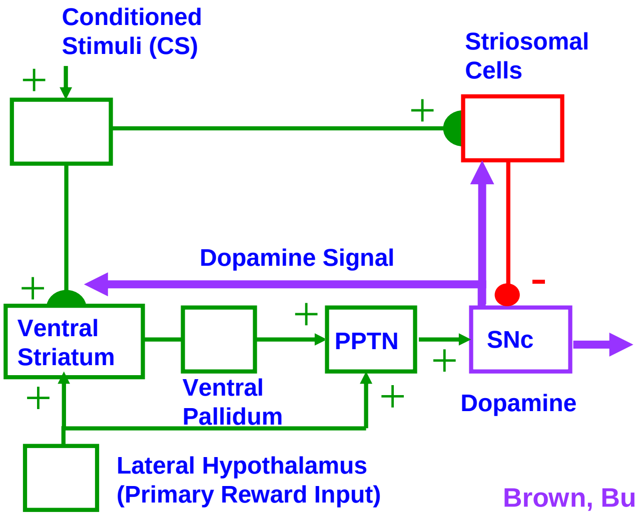
MODEL OF SPECTRALLY TIMED SNc LEARNING



Delayed inhibitory expectations of reward

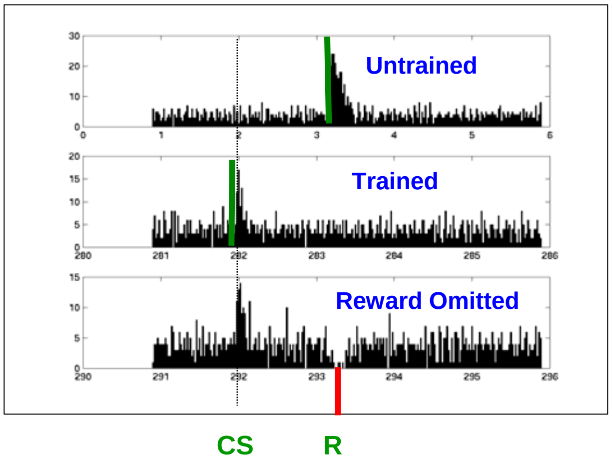
Dopamine cells signal an error in reward prediction timing or magnitude

Immediate excitatory predictions of reward

Brown, Bullock, and Grossberg (1999)

MODEL DOPAMINE CELL

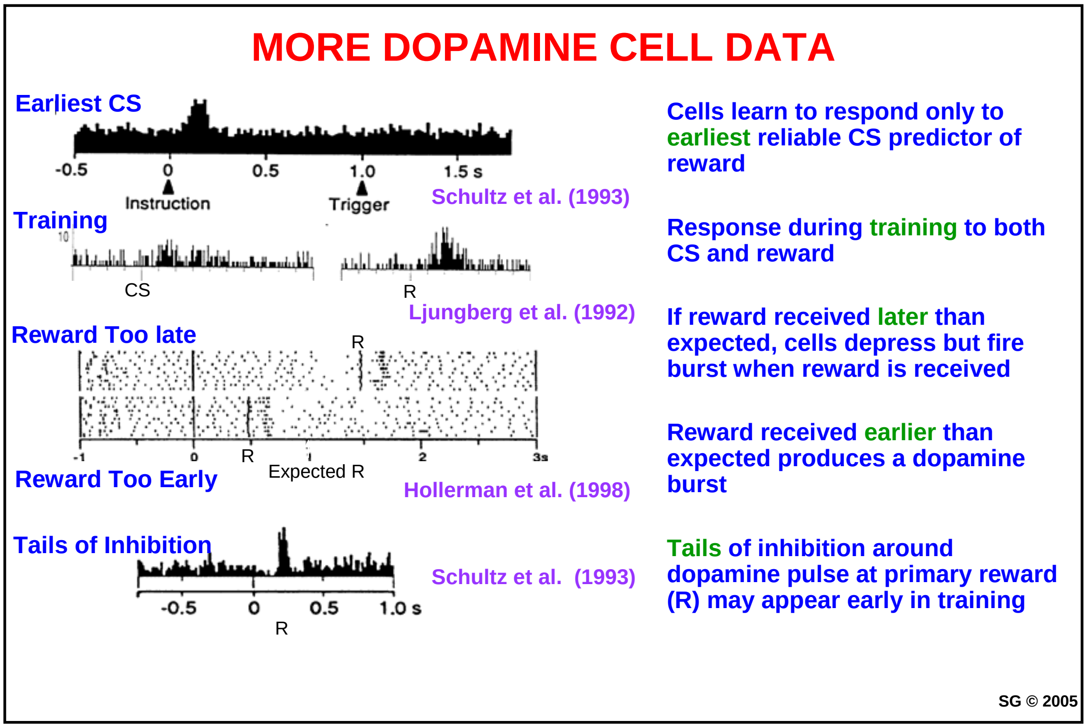
Model



Untrained model responds only to primary reward

Model learns response to CS

Omission of expected reward leads to depression of dopamine signal



MORE DOPAMINE CELL DATA

Schultz et al. (1993)

Ljungberg et al. (1992)

Hollerman et al. (1998)

Schultz et al. (1993)

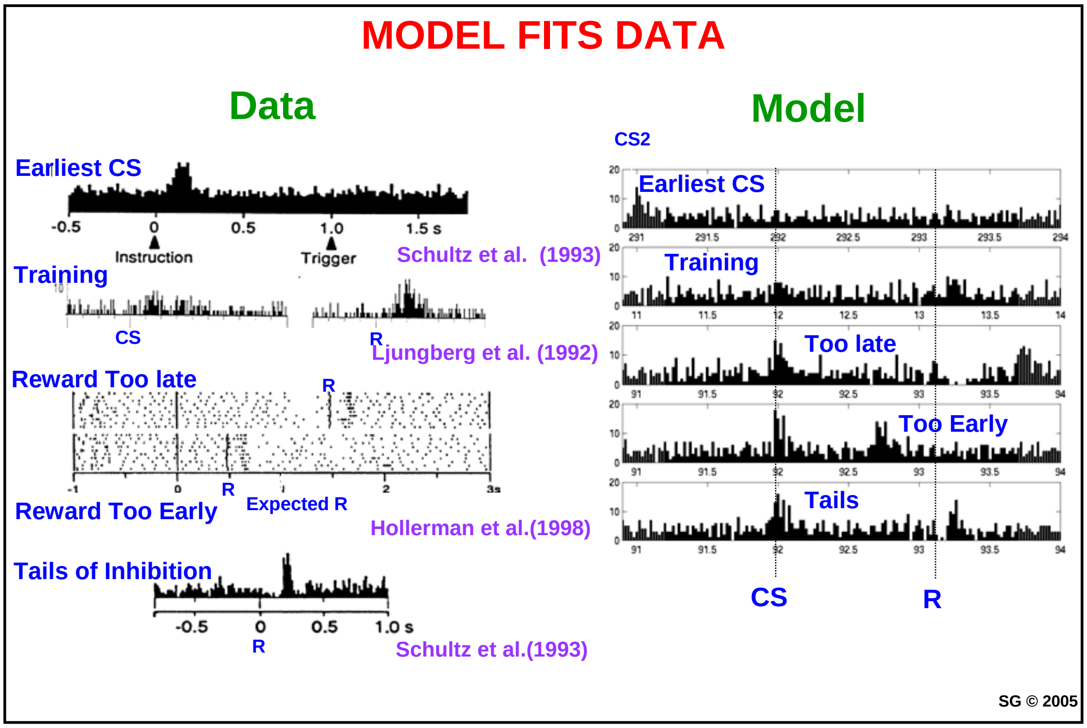
Cells learn to respond only to earliest reliable CS predictor of reward

Response during training to both CS and reward

If reward received later than expected, cells depress but fire burst when reward is received

Reward received earlier than expected produces a dopamine burst

Tails of inhibition around dopamine pulse at primary reward (R) may appear early in training



MODEL FITS DATA

Data

Model

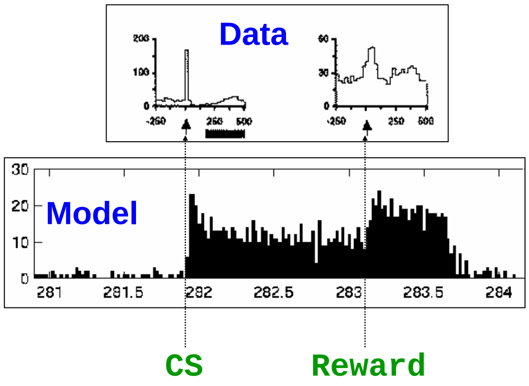
Schultz et al. (1993)

Ljungberg et al. (1992)

Hollerman et al.(1998)

Schultz et al.(1993)

PPTN CELLS



Some PPTN cells excite the SNc monosynaptically

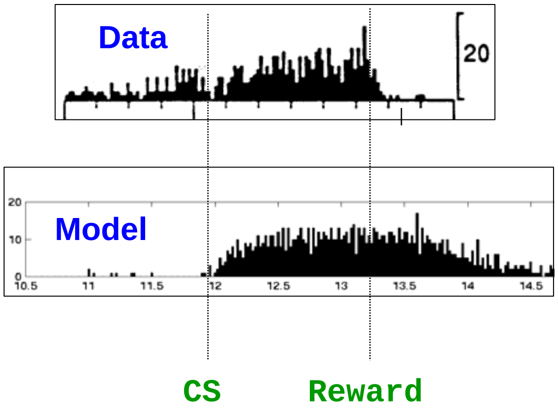
PPTN cells fire transiently in response to both primary reward (from lateral hypothalamus) and learned CS predictors of reward (from ventral striatum)

Accomodation accounts for transient nature of burst

Takakusaki et al. (1997)

Data (cat) from Dormont et al. (1998)

STRIOSOMAL CELLS



Firing rate ramps up toward Ca^{2+} spike peak

Trained cells transiently inhibit their SNc target cells

Rapid falloff after Ca^{2+} spike

Data (macaque) from Schultz et al. (1992)

SUPPORT FOR MODEL ANATOMY

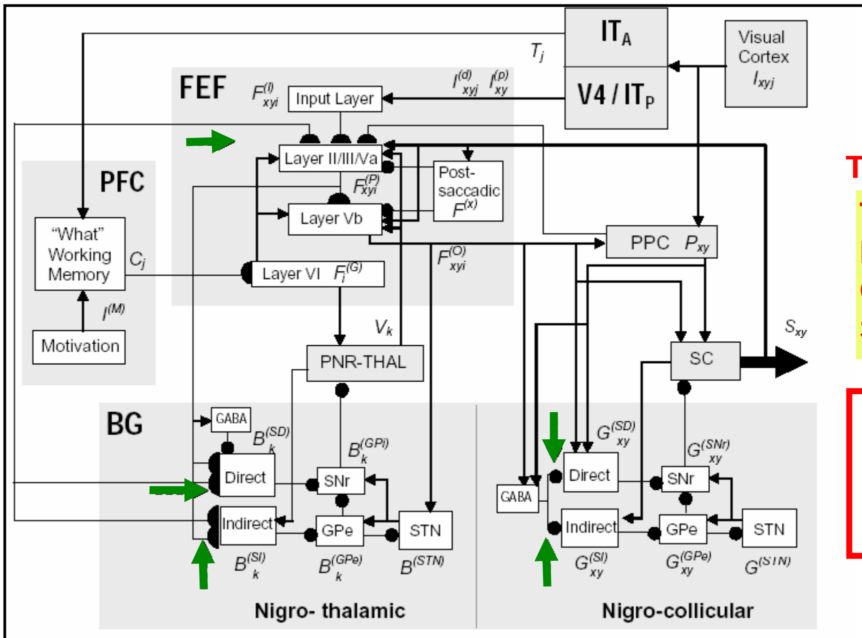
From	To	References
• Striosomes (GABA)	Ventral SNc	Gerfen (1992)
• PPTN (Glut., ACh)	SNC	Conde (1992)
• Lateral Hypothalamus	PPTN	Semba & Fibiger (1992)
• Lateral Hypothalamus	Ventral Striatum	Brog et al. (1993)
• Limbic cortex	Ventral Striatum	Yang & Mogenson (1987), Brog et al. (1993)
• Ventral Striatum	Ventral Pallidum	Yang & Mogenson (1987)
• Ventral Pallidum	PPTN	Yang & Mogenson (1987)
• SNc (ventral) • (Dopamine)	Striosomes	Gerfen (1992)
• Limbic cortex	Striosomes	Gerfen (1992)

MODEL SUMMARY

Model shows how two parallel circuits, excitatory and inhibitory, enable SNc dopamine cells to respond selectively to unexpected reward-predicting signals

Shows how the basal ganglia learns to predict both the magnitude and timing of reward, and generate a learning signal if expectations are violated

Real-time computational model bridges the gap between neurobiology and cognition



TELOS Model

TElencephalic
Laminar
Objective
Selector

The SNc generates the Dopamine bursts and dips in TELOS

➡ Dopamine

Brown, Bullock, and Grossberg (2004, Neural Networks)

FRONTAL MAPS AS GATED CORTICAL ZONES:

Multiplexing Features and Movements

How to simultaneously store many alternative plans without forgetting? How to learn and choose among task strategies?

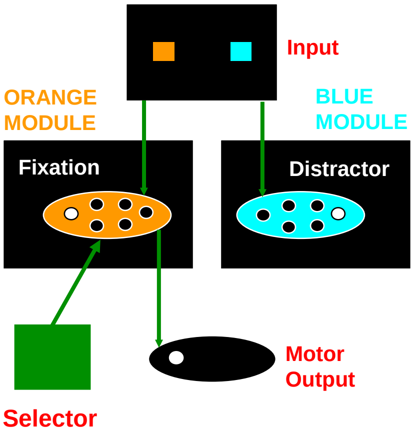
~ 300,000 pallidal basal ganglia outputs in humans (fewer in other animals) may imply a limited number of gateable cortical zones (GCZs)

Model: Frontal GCZs are a basis for functional mappings; e.g., cells selective for common features but different movement vectors can be collectively activated by a given output from PNR-Thalamus

Cells within a GCZ can FILTER the scene to promote foveation of rewarded features (e.g. color, motion, shape) wherever they appear

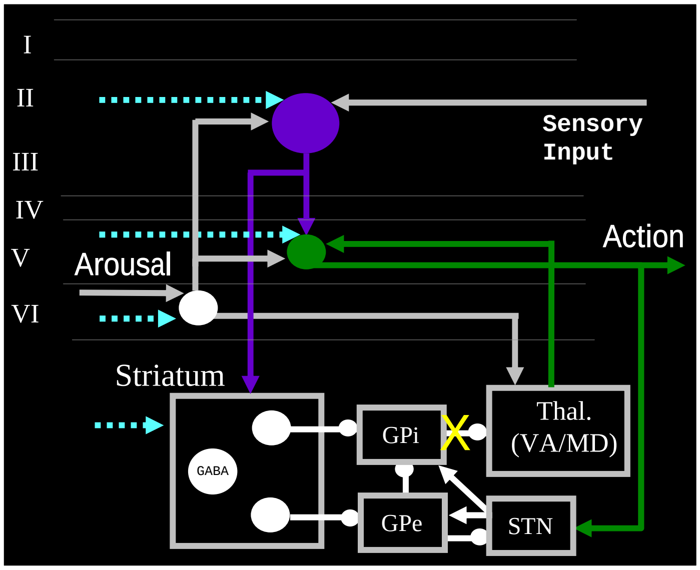
Key Point: Basal ganglia efficiently select actions by gating cortical zones

Foveate Orange or Blue?



cf. Redgrave et al. (1999)

CORTICAL LAYERS SEPARATE PLANNING AND ACTION



····▸ Dopamine

Model dissociates planning (layer II/III/Va) from

action (layer Vb)

Model learning and performance need dual cortical output projections:

plan layers to striatum

output layer to STN

Model learning and performance need dual BG output projections: DA-ergic and gating signals

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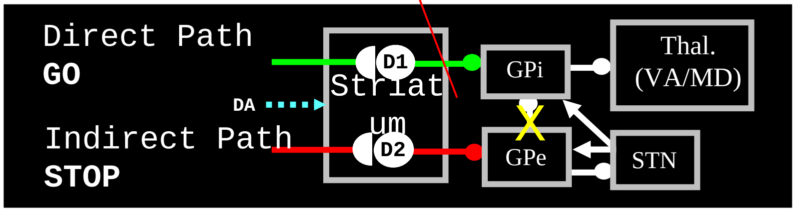
PREFRONTAL CORTEX CONNECTS TO DIRECT AND INDIRECT BASAL GANGLIA PATHS

DIRECT pathway (Striatum-GPi/SNr-Thalamus/SC) actively opens the gate for a planned action

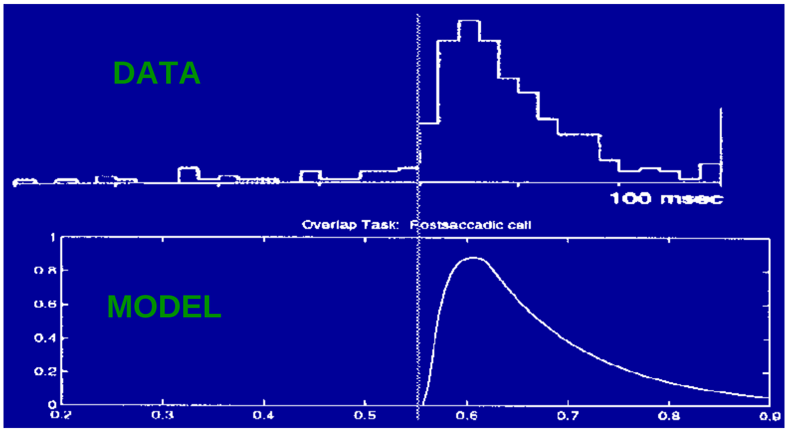
INDIRECT pathway (Striatum-GPe-GPi/SNr) actively suppresses planned action by disinhibiting GPi/SNr

LEARNING:
DA burst strengthens inputs to direct path
DA dip strengthens inputs to indirect path

PERFORMANCE:
Indirect pathway can STOP
direct pathway GO signal



MODEL FITS PHYSIOLOGY



FEF Postsaccadic Cells Schall (1991)

Activity immediately after saccade onset
In the model, it shuts off saccade plan

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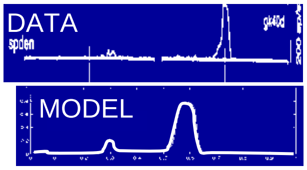
MODEL FITS PHYSIOLOGY

Model fits cell types in major oculomotor structures

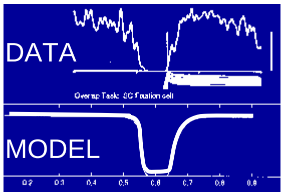
SC

Saccade Cells



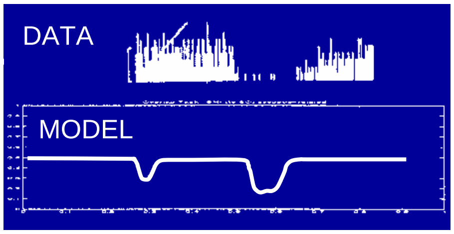
Munoz & Wurtz (1995)

Fixation Cells



Munoz & Wurtz (1993)

SNr



Hikosaka & Wurtz (1989)

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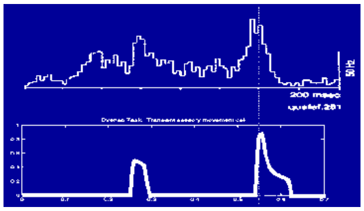
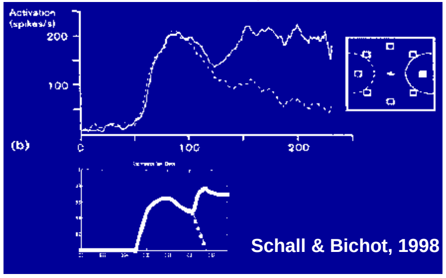
MODEL FITS PHYSIOLOGY

Transient Sensory-Movement Cell



Schall (1991)

Discriminating Cell

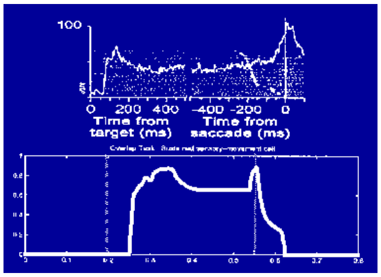


Schall & Bichot, 1998

Plan Cells respond to visual input and corollary discharge of motor output

Plan activity sustained via local recurrent excitation

Sustained Sensory-Movement Cell



Hanes et al. (1998)

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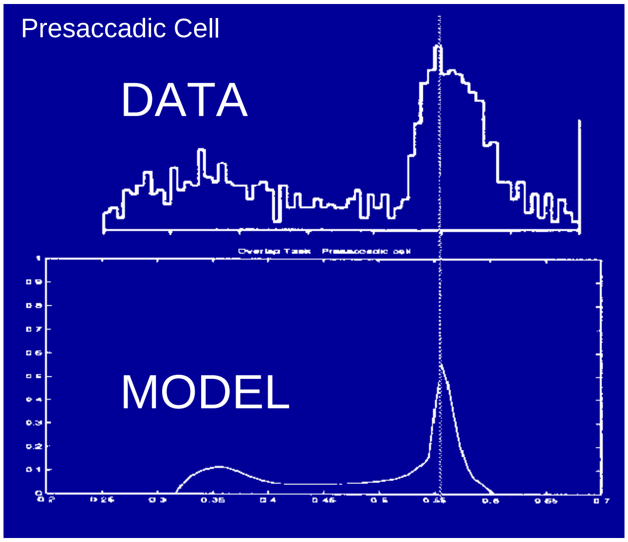
MODEL FITS PHYSIOLOGY



FEF Output Cells

Weak response to stimulus onset

Strong response peaks at saccade initiation

Schall (1991)

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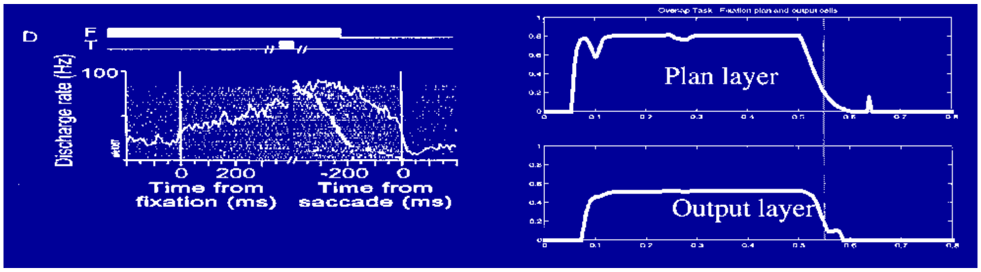
MODEL FITS PHYSIOLOGY

DATA **MODEL**



Schall et al. (1995)

FEF Fixation Cells

Both FEF Plan and Output layers have Fixation-related cells, as distinct from Saccade-related cells

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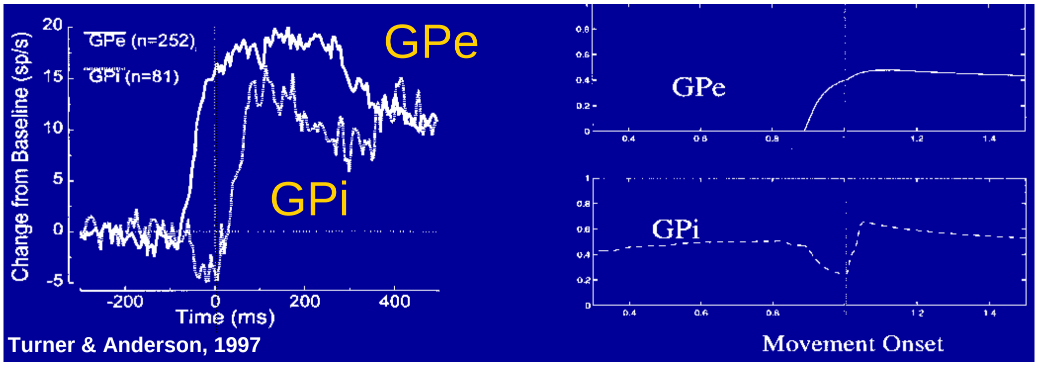
MODEL FITS PHYSIOLOGY

DATA **MODEL**



Turner & Anderson, 1997

Basal Ganglia

Indirect path activity ceases (GPe)
Direct path activity increases (GPi)
After movement begins, STN excites GP

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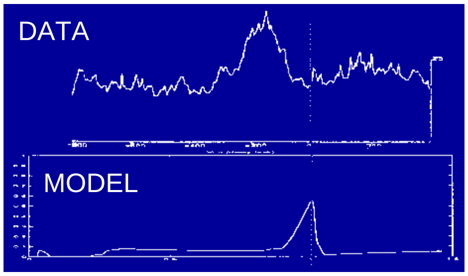
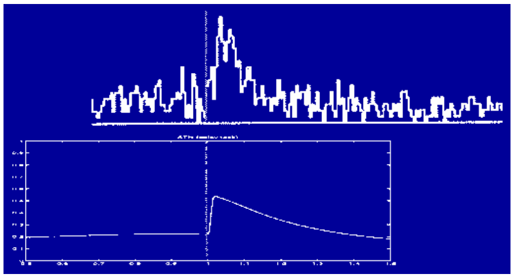
Thalamus **STN**





Turner & Anderson (1997)

Wichmann et al. (1994)

Thalamus burst immediately *before* movement begins
STN activity burst immediately *after* movement begins

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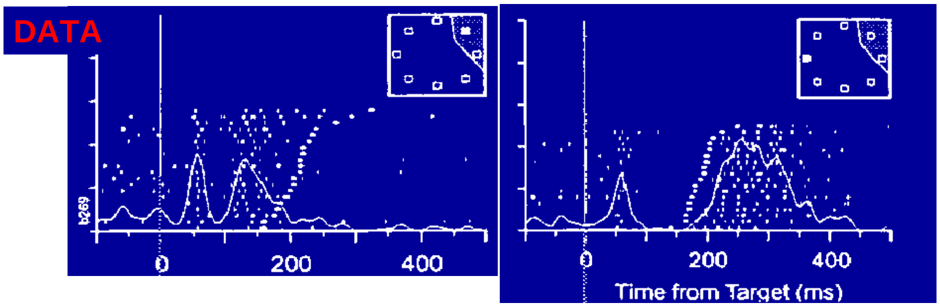
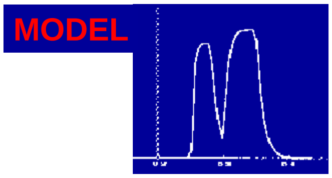
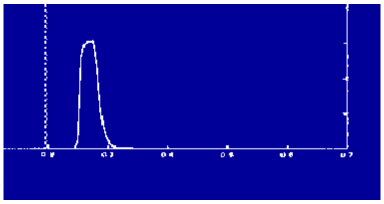
FEF Visual Cells





Schall et al. (1995)



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