

Kognitionspsychologie II: Session 8

Neural basis of motivation

Loreen Tisdall, FS 2025

Version: April 27, 2025

Semester overview

#	Date	Topic	Slides	Instructor
1	18.02.2025	Emotion: What is an emotion?	pdf	Mata
2	25.02.2025	Emotion: What is an emotion? (continued)	pdf	Mata
3	18.03.2025	Emotion: Neural bases	pdf	Tisdall
4	25.03.2025	Emotion: Regulation	pdf	Mata
5	01.04.2025	Emotion: Well-being	pdf	Mata
6	08.04.2025	Motivation: What is motivation?	pdf	Mata
7	15.04.2025	Motivation: Extrinsic vs intrinsic motivation	pdf	Mata
8	29.04.2025	Motivation: Neural bases		Tisdall
9	06.05.2025	Motivation: Cooperation and morality		Theisen
10	13.05.2025	Applications		Mata
11	20.05.2025	Wrap-up and Q&A		Mata
12	03.06.2025	Exam (DSBG Neubau)		
13	21.07.2025	Repeat Exam (Biozentrum)		



Learning Objectives

- Describe **different methods** (including comparative approaches) used to study the neural substrates of motivation
- Recognize the value of a **systems-level approach** to understanding the neural basis of motivation, particularly how motivation emerges from the integration of multiple brain systems
- Describe **key neural components of motivation** and explain how **interactions between neural systems** contribute to integrated motivational processes

Recap: What is motivation?

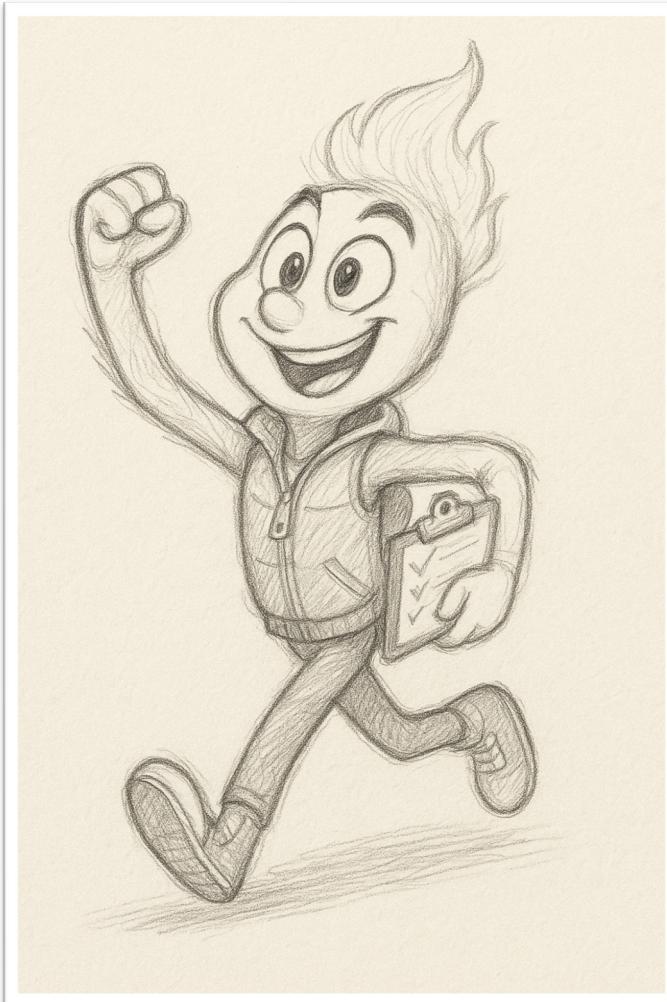
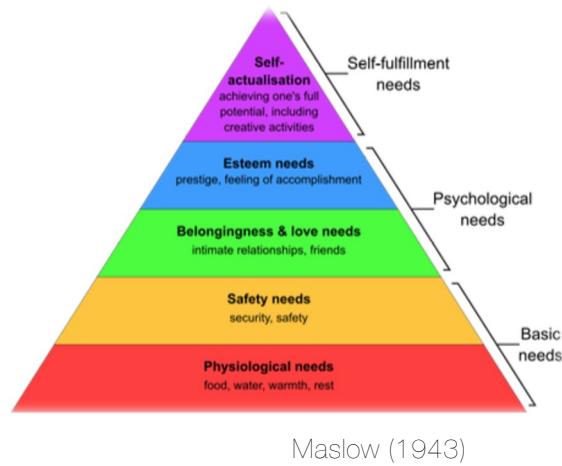


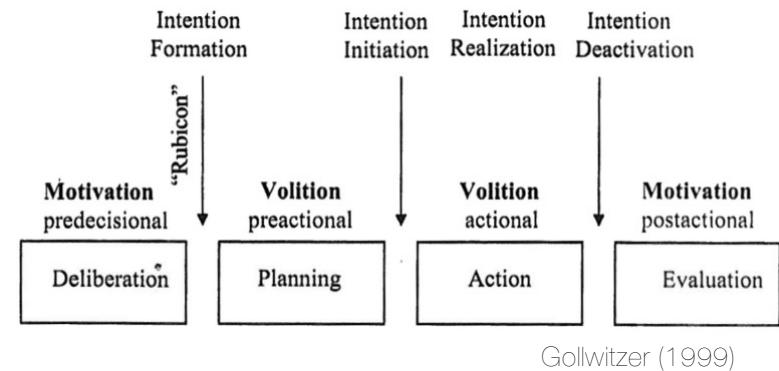
Image created with AI (ChatGPT 4o, April 2025)

- : the act or process of giving someone **a reason for doing something**; the act or process of motivating someone
- : the condition of being **eager to act or work**; the condition of being motivated
- : a force or influence that **causes someone to do something**

Recap: What are the 3 types of motivation theories?



Harlow (1958)



Content:

individual motives,
traits, and motivations

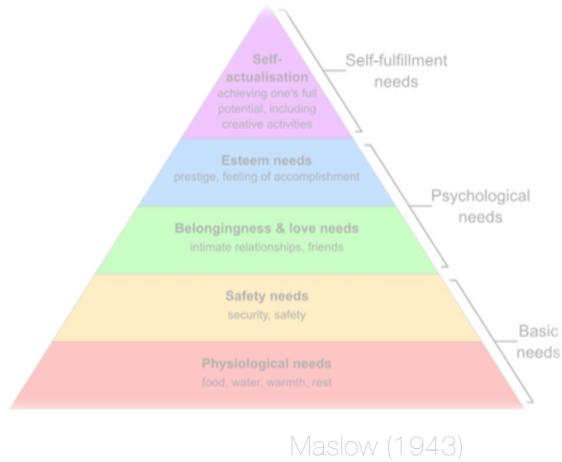
Context:

features of the job, role,
environment

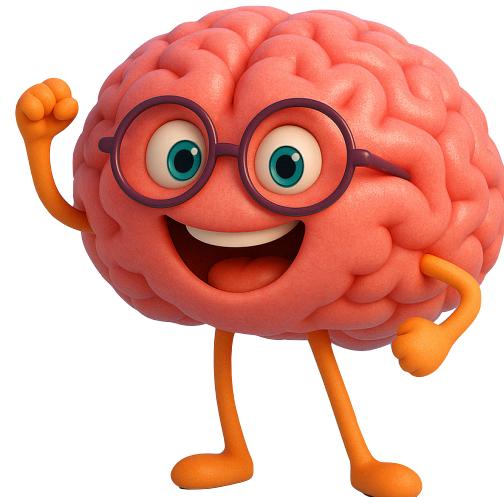
Process:

mechanisms involved in
individual choices and striving

Today: Neural substrates of motivation



Harlow (1958)



Content:

individual motives,
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Context:

features of the job, role,
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Your turn!

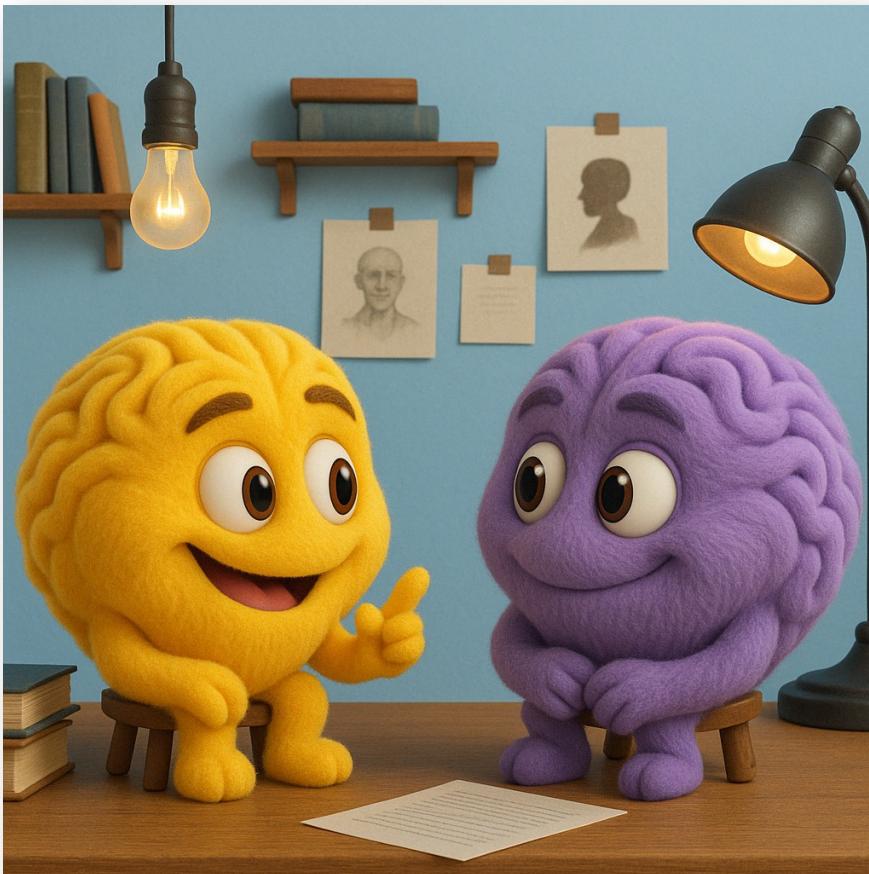


Image created with AI (ChatGPT 4o, April 2025)

What methods do (neuro)scientists have at their disposal to investigate the brain basis of motivation?

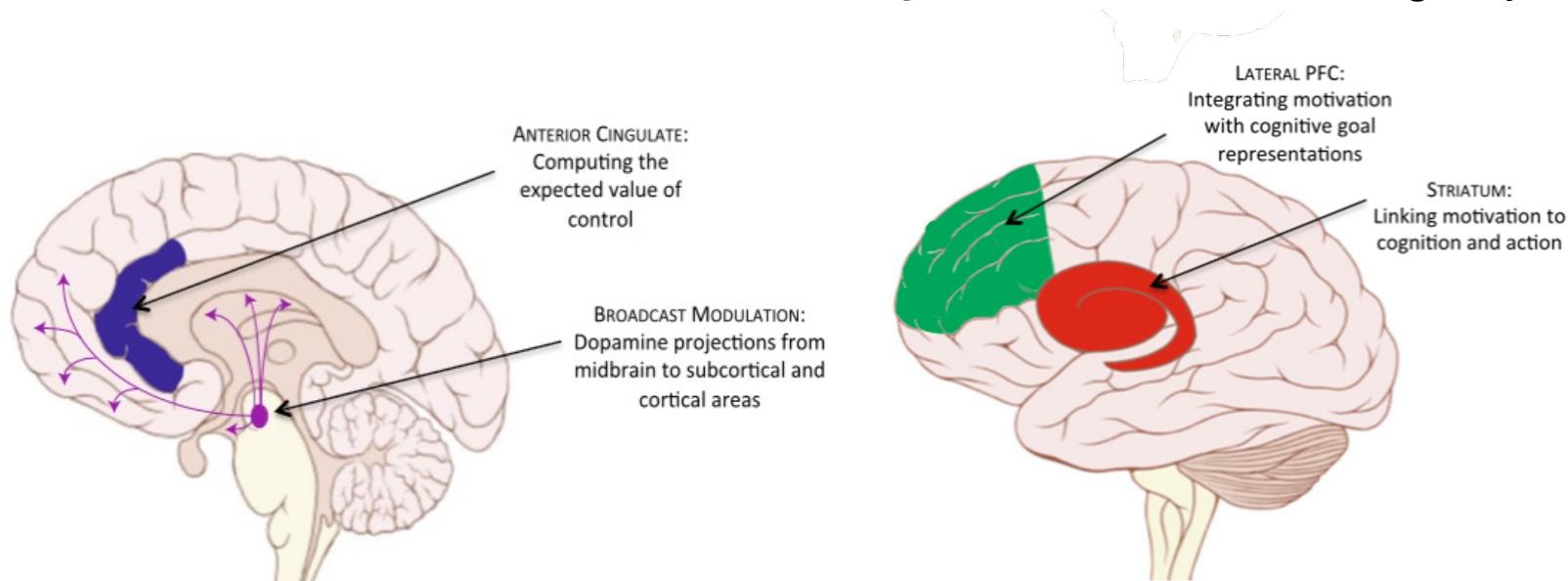
***Discuss with your neighbour(s)
~2-3 minutes***

Approaches to studying the neural substrates of motivation (comprehensive but by no means exhaustive!)

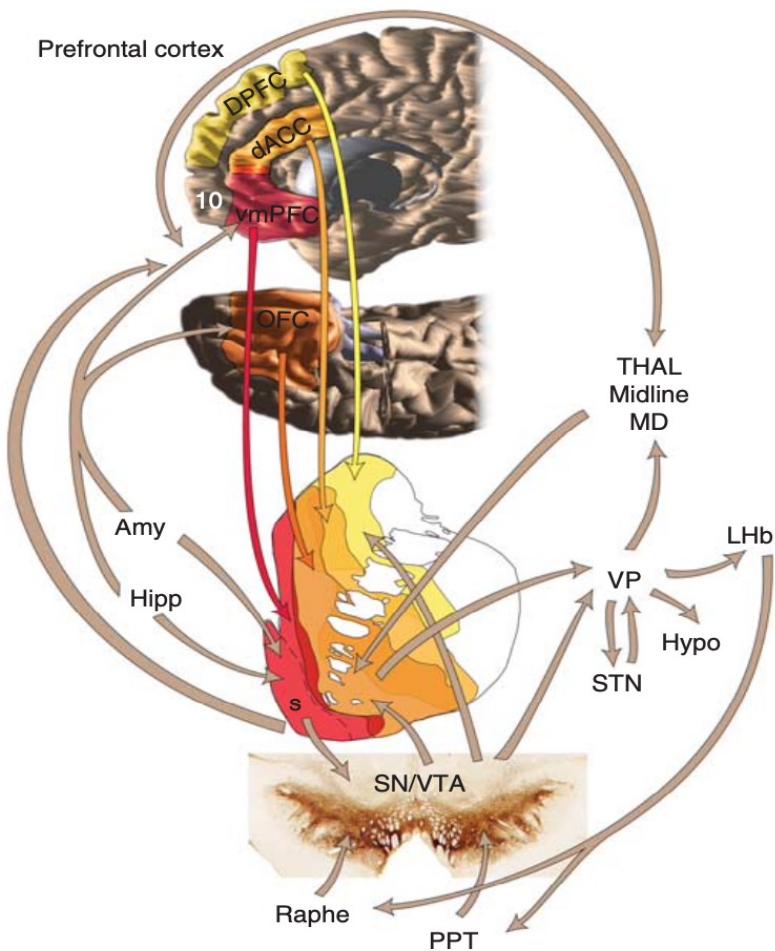
Approach	What it measures / infers / does	Strengths	Limitations	Example use
fMRI (functional Magnetic Resonance Imaging)	Brain activity via blood oxygenation (BOLD signal)	High spatial resolution; non-invasive	Low temporal resolution; indirect neural activity	NAcc activation during reward anticipation
sMRI (structural Magnetic Resonance Imaging, e.g., DWI)	Anatomical connectivity and microstructural properties of white matter tracts	Can map large-scale brain networks; non-invasive	Mechanistic interpretations rely on models of structure-function relationships	Identifying connectivity disruptions in addiction
PET (Positron Emission Tomography)	Neurochemical processes (e.g., dopamine binding)	Targets specific neurotransmitters	Minimally invasive; low temporal resolution	Mapping dopamine in reward circuits
EEG (Electroencephalography) / ERP (Event-Related Potentials)	Electrical brain signals from the scalp	High temporal resolution; non-invasive	Poor spatial resolution	Feedback-related negativity in reward tasks
Lesion studies in patient groups	Behavioral/cognitive/affective deficits after brain damage	Shows necessity of brain areas	Lesions often imprecise or diffuse	OFC damage disrupts value-based decision-making
TMS (Transcranial Magnetic Stimulation)	Temporarily disrupts or enhances activity in specific regions	Causal inference in humans; non-invasive	Limited to surface areas; moderate spatial resolution	Disrupting DLPFC affects delay discounting
Pharmacological manipulations	Alters neurotransmitter activity (e.g., dopamine agonists)	Links brain chemistry to motivational behavior	Systemic effects; limited brain specificity	Dopaminergic drugs increase effort investment
Computational modeling + imaging	Infers cognitive variables (e.g., value, prediction error)	Bridges behavior, theory, and neural activity	Complex; model-dependent	Prediction error signals in ventral striatum
Animal models (e.g., electrophysiology, optogenetics, lesions)	Direct manipulation/ recording of brain activity	Precise; allows causal inference	May not generalize to humans	Stimulating VTA increases motivated behavior

Anatomical orientation: The “motivational” brain

- Motivation emerges from the interaction of **reward systems** (what's worth pursuing), **control systems** (how to pursue it), and **interoceptive / cognitive (appraisal-based) systems** (how we feel about it)
- Think of the motivational brain as a **network of systems**, rather than a single system!



A closer look: Reward circuitry



“Although cells in many brain regions respond to reward, the cortical-basal ganglia circuit is at the heart of the reward system. The key structures in this network are the **anterior cingulate cortex**, the **orbital prefrontal cortex**, the **ventral striatum**, the **ventral pallidum**, and the **midbrain dopamine neurons**.

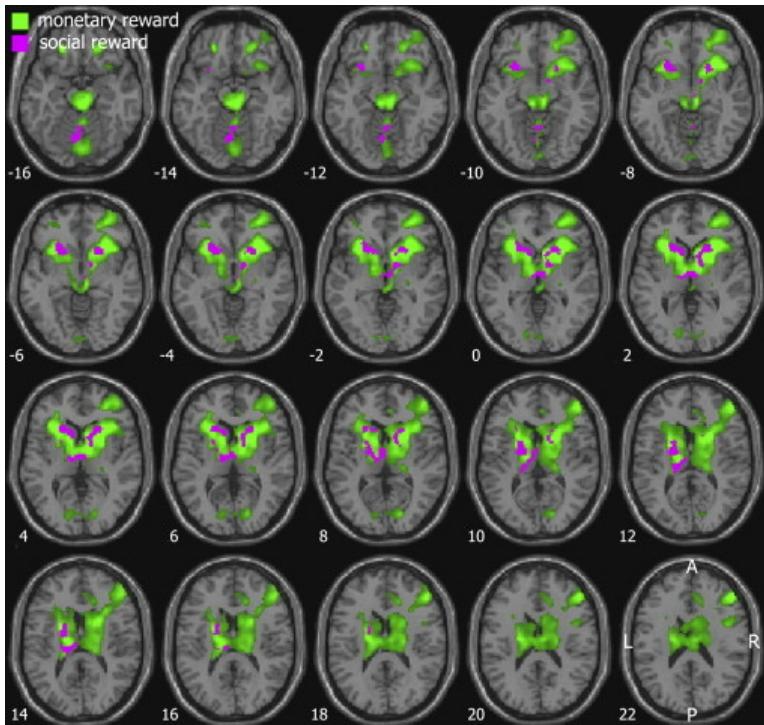
In addition, other structures, including the **dorsal prefrontal cortex**, **amygdala**, **hippocampus**, **thalamus**, and **lateral habenular nucleus**, and specific **brainstem structures** such as the pedunculopontine nucleus, and the raphe nucleus, are **key components in regulating the reward circuit**. [...]

Advances in neuroimaging techniques allow better spatial and temporal resolution. **These studies now demonstrate that human functional and structural imaging results map increasingly close to primate anatomy.”**

A closer look: Are all rewards processed equally?



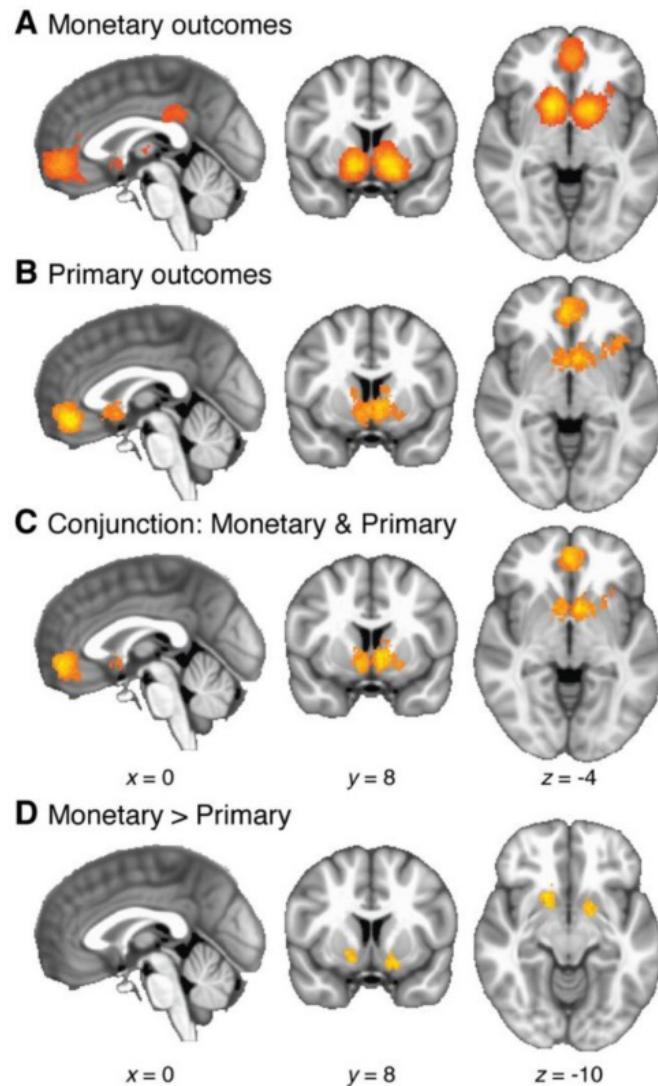
links back to
content theories



Single study results:

"The acquisition of one's good reputation robustly activated reward-related brain areas, notably the **striatum**, and these **overlapped with the areas activated by monetary rewards**. Our findings **support the idea of a “common neural currency” for rewards** and represent an important first step toward a neural explanation for complex human social behaviors."

A closer look: Are all rewards processed equally?



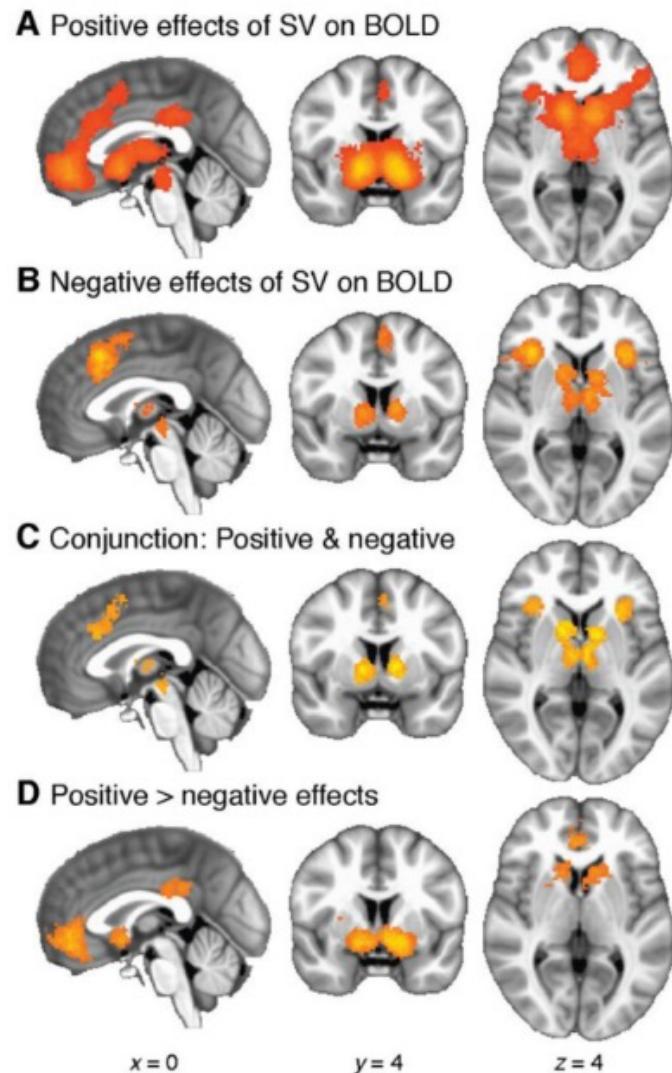
links back to
content theories

Meta-analytic consensus:

"Both primary and monetary incentives elicited SV responses in the same brain regions. This aspect of our results aligns with an emerging consensus that a **unitary neural system**, including regions of striatum and VMPFC, represents SV across different categories of goods."

"[...] suggests that a reward that is merely signaled (e.g., money) is evaluated similarly to one that is actually consumed (e.g., juice) [...]"

A closer look: Subjective value signal (“want/not want”?)



Meta-analytic consensus:

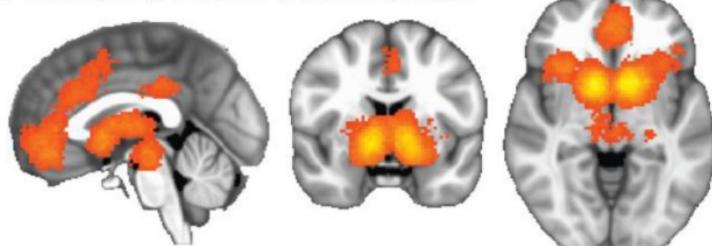
“An effect was coded as positive if a greater BOLD response was observed for more rewarding (or less aversive) outcomes; it was coded as negative if a greater BOLD response was observed for less rewarding (or more aversive) outcomes.”

“We observed a greater density of positive than negative effects in the VMPFC, PCC, and striatum. We observed overlapping significant densities for both positive and negative effects in DMPFC, thalamus, striatum, and bilateral anterior insula.”

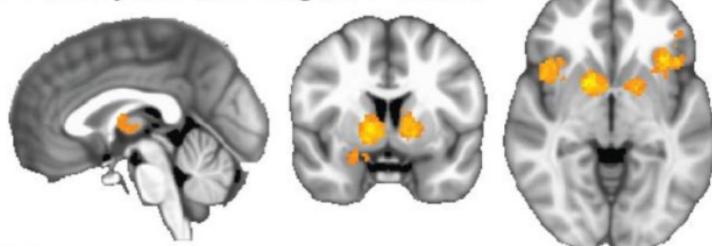
“In summary, some brain regions showed both positively and negatively signed effects of SV on BOLD across studies, while other regions showed positive effects only.”

A closer look: Reward and punishment?

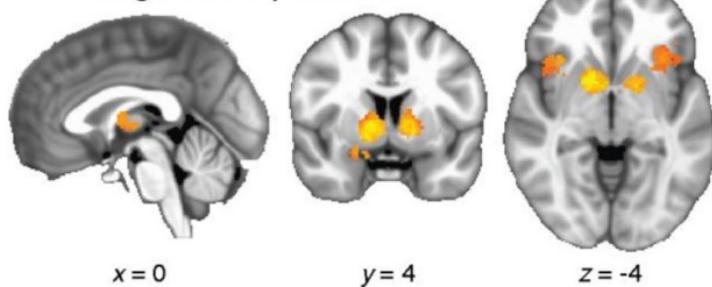
A Reward domain: Positive effects



B Penalty domain: Negative effects



C Conjunction: Positive for rewards & negative for penalties



Can you think of
methodological confounds
that need to be ruled out to
support the result of
overlapping brain regions for
gains and losses?

Meta-analytic consensus:

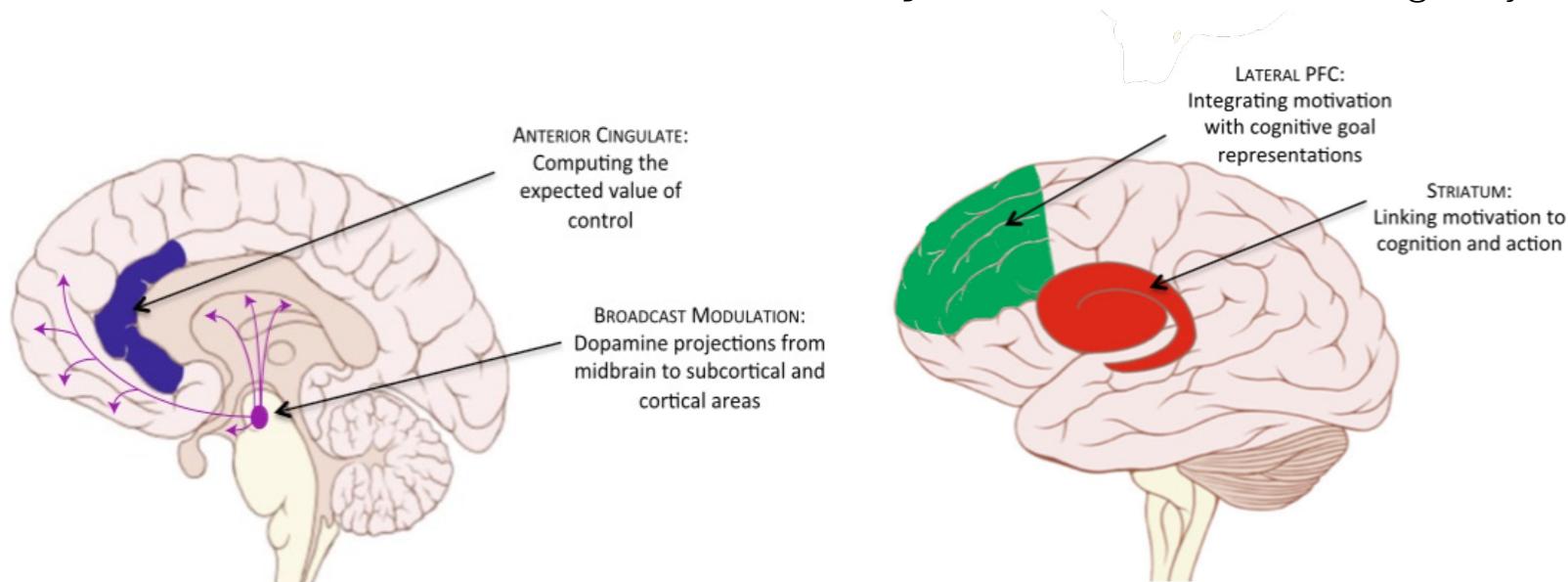
"The co-occurrence of positive and negative effects in the same brain regions may initially appear counterintuitive."

"Pleasant experiences and monetary gains counted as rewards, while aversive experiences and monetary losses counted as penalties."

"A brain region with a nonlinear SV response (pattern B in Figure 1) would be expected to show both positive effects for rewards and negative effects for penalties. Consistent with this, regions of bilateral insula and striatum were significant in a conjunction test evaluating overlap between these two categories."

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- Ok, but **how do these systems interact** to lead to goal-directed behavior?

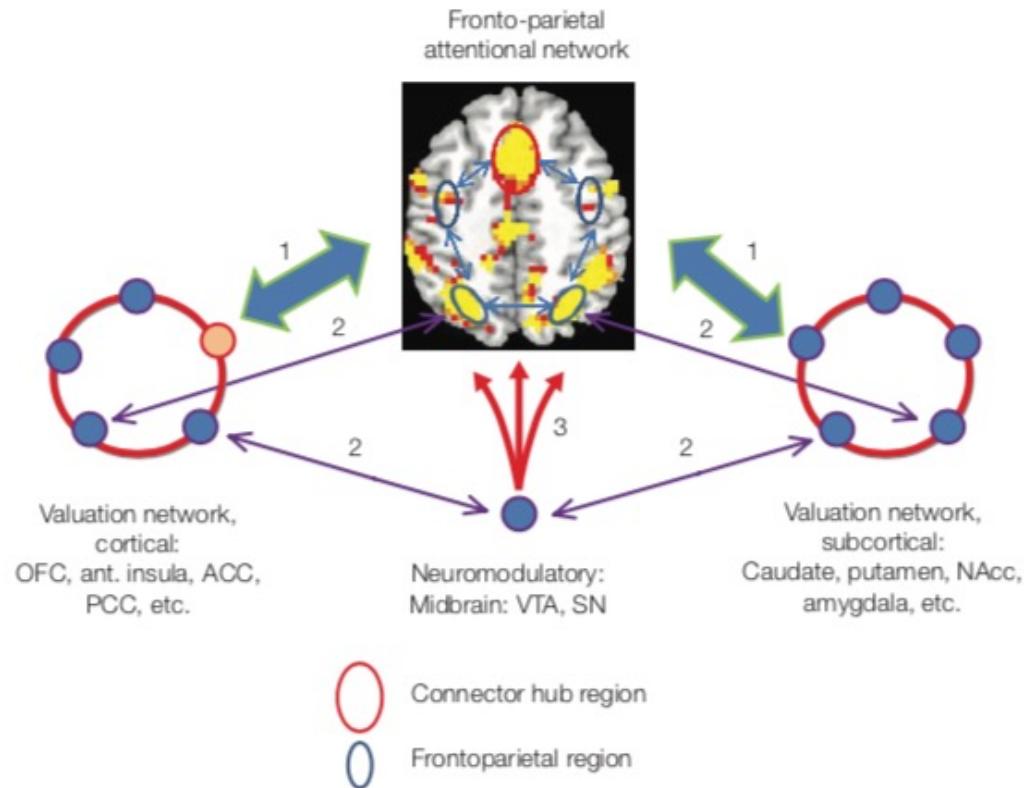
Mechanisms of motivation–cognition interaction

Modes of communication between cognitive and motivation networks illustrated for attentional-motivational interactions.

(1) Interactions rely on **connector “hub” regions**, such as the anterior cingulate cortex, which are part of both attentional and motivational networks (indicated via the red outline in both the valuation-cortical and attentional networks).

(2) In addition, **specific regions may link the two networks**, either directly or via the thalamus.

(3) Finally, motivational signals are embedded within cognitive mechanisms via the action of **diffuse neuromodulatory systems**.



The link between models of motivation, emotion, and cognition is important because it emphasizes the idea that cognition is not value neutral!

Braver, T. S., Krug, M. K., Chiew, K. S., Kool, W., Westbrook, J. A., Clement, N. J., et al. (2014). Mechanisms of motivation-cognition interaction: challenges and opportunities. *Cognitive, Affective, & Behavioral Neuroscience*, 14(2), 443–472. <http://doi.org/10.3758/s13415-014-0300-0>

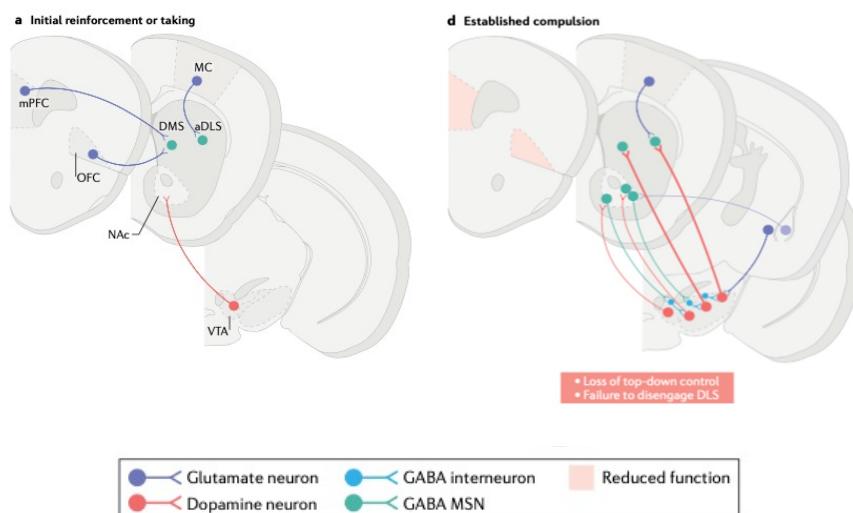
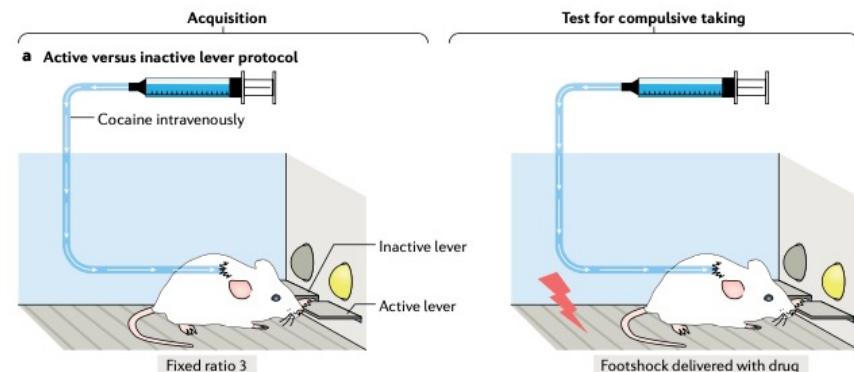
Pessoa, L., & Engelmann, J. B. (2010). Embedding reward signals into perception and cognition. *Frontiers in Neuroscience*, 4, 17.

Dysregulated motivation: Habit formation in addiction

Experimental setup of animal models:

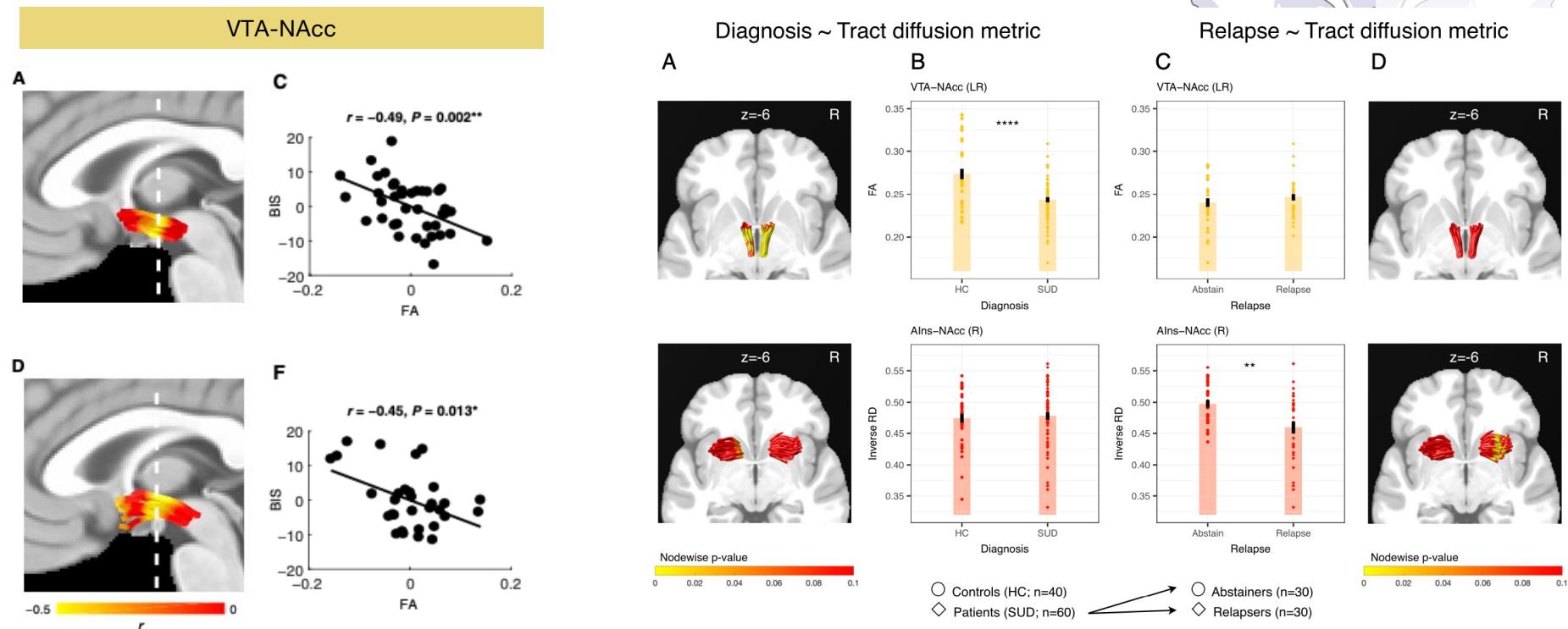
Operant chamber with an active lever and an inactive lever

- Responding on the active lever results in drug infusion (drug taking), and a presented light stimulus becomes a drug conditioned stimulus through Pavlovian conditioning (left panel)
- Compulsive drug taking is defined as persistent responding when the lever press is punished at the same time as drug infusion (right panel).



- **(a)** Addictive drugs have a common initial effect of increasing levels of dopamine in the nucleus accumbens (NAc) — particularly dopamine released by neurons projecting from the ventral tegmental area (VTA) → **crucial for initial drug reinforcement**
- Drug taking depends on plasticity of projections from the medial prefrontal cortex (mPFC) and orbitofrontal cortex (OFC) to the dorsomedial striatum (DMS)
- **Initially, drug seeking is goal-directed** and depends on the DMS and afferents from the mPFC and OFC.
- **(d)** Compulsive drug seeking depends on **the loss of prefrontal cortical ‘top-down’ control over the striatal mechanisms** underlying drug-seeking habits (denoted by shading of the DLS and grey shading of the mPFC and OFC).

Impulsivity versus compulsivity in human stimulant addiction



“[...] reduced diffusion metrics of a tract projecting from the right anterior insula to the NAcc were associated with subsequent relapse to stimulant use, but not with previous diagnosis. These findings highlight a structural target for predicting relapse to stimulant use and further suggest that distinct connections to the NAcc may confer risk for relapse versus diagnosis.”

MacNiven, K. H., Leong, J. K., & Knutson, B. (2020). Medial forebrain bundle structure is linked to human impulsivity. *Science Advances*, 6(38), eaba4788.

Tisdall, L., MacNiven, K. H., Padula, C. B., Leong, J. K., & Knutson, B. (2022). Brain tract structure predicts relapse to stimulant drug use. *Proceedings of the National Academy of Sciences*, 119(26), e2116703119.

Summary

- **Systems approaches to motivation** view it as an emergent property of dynamic interactions between cognitive, emotional, social, and environmental factors. These models often require multi-level, interdisciplinary to fully capture the complexity of motivated behavior.
- **Cognitive and neural models:** Cognitive models have focused on distinguishing phases such as deliberation and action phases; neural models of motivation overlap with neural models of cognition (e.g., attention) and emotional processing, involving aspects of information integration (prefrontal cortex) and valuation (cortical/subcortical).
- **Comparative approaches:** Animal models have been (and still are) instrumental in helping understand hierarchy of needs and the neural basis of simple motivational processes, such as habit formation (e.g., drug addiction).

Key (mandatory) reading

- Braver, T. S., Krug, M. K., Chiew, K. S., Kool, W., Westbrook, J. A., Clement, N. J., ... & Momca Group. (2014). Mechanisms of motivation–cognition interaction: challenges and opportunities. *Cognitive, Affective, & Behavioral Neuroscience*, 14, 443-472. <https://link.springer.com/content/pdf/10.3758/s13415-014-0300-0.pdf>

Cogn Affect Behav Neurosci (2014) 14:443–472
DOI 10.3758/s13415-014-0300-0

Mechanisms of motivation–cognition interaction: challenges and opportunities

Todd S. Braver · Marie K. Krug · Kimberly S. Chiew · Wouter Kool · J. Andrew Westbrook · Nathan J. Clement · R. Alison Adcock · Deanna M. Barch · Matthew M. Botvinick · Charles S. Carver · Roshan Cools · Ruud Custers · Anthony Dickinson · Carol S. Dweck · Ayelet Fishbach · Peter M. Gollwitzer · Thomas M. Hess · Derek M. Isaacowitz · Mara Mather · Kou Murayama · Luiz Pessoa · Gregory R. Samanez-Larkin · Leah H. Somerville · for the MOMCAI group

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Abstract Recent years have seen a rejuvenation of interest in studies of motivation–cognition interactions arising from many different areas of psychology and neuroscience. The present issue of *Cognitive, Affective, & Behavioral Neuroscience* provides a sampling of some of the latest research from a number of these different areas. In this introductory article, we provide an overview of the current state of the field, in terms of key research developments and candidate neural mechanisms receiving focused investigation as potential sources of motivation–cognition interaction. However, our primary goal is conceptual: to highlight the distinct perspectives taken by different research areas, in terms of how motivation is defined, the relevant dimensions and dissociations that are emphasized, and the theoretical questions being

The *MOMCAI group* refers to the attendees of the Mechanisms of Motivation, Cognition, and Aging Interactions conference, held in Washington, D.C., on May 2–4, 2013, who were directly involved in specifying the content of this article from conference discussions. The attendees, beyond the listed authors, were Stan Floresco, Michael Frank, Ulrich Mayr, Erik Asp, Sarah Barber, Brittany Cassidy, Jong Moon Choi, Michael Cohen, Reka Daniel, Kathryn Dickerson, Natalie Ebner, Tammy English, Natasha Fourquet, Nichole Lighthall, Brenton McMenamin, Srikanth Padmala, Angela Radulescu, Kendra Seaman, Brian Smith, Mieke van Holstein, Steven Stanton, Isha Vicaria, Tara Queen, and Lisa Zaval.

T. S. Braver (✉) · M. K. Krug · J. A. Westbrook · D. M. Barch
Department of Psychology, Washington University in St. Louis,
CB1125, One Brookings Drive, St. Louis, MO 63130, USA
e-mail: tbraver@artsy.wustl.edu

K. S. Chiew · N. J. Clement · R. A. Adcock
Center for Cognitive Neuroscience, Duke University, Durham, NC,
USA

W. Kool · M. M. Botvinick
Department of Psychology, Princeton University, Princeton, NJ,
USA

C. S. Carver
Department of Psychology, University of Miami, Miami, FL, USA

R. Cools
Radboud University Nijmegen Medical Center, Nijmegen, The
Netherlands

R. Custers
Cognitive, Perceptual, and Brain Sciences, University College
London, London, UK

A. Dickinson
Experimental Psychology, University of Cambridge, Cambridge, UK

C. S. Dweck
Department of Psychology, Stanford University, Stanford, CA, USA

A. Fishbach
Booth School of Business, University of Chicago, Chicago, IL, USA

P. M. Gollwitzer
Department of Psychology, New York University, New York, NY,
USA

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