Kognitionspsychologie II: Session 12

Wrap-up and Q&A

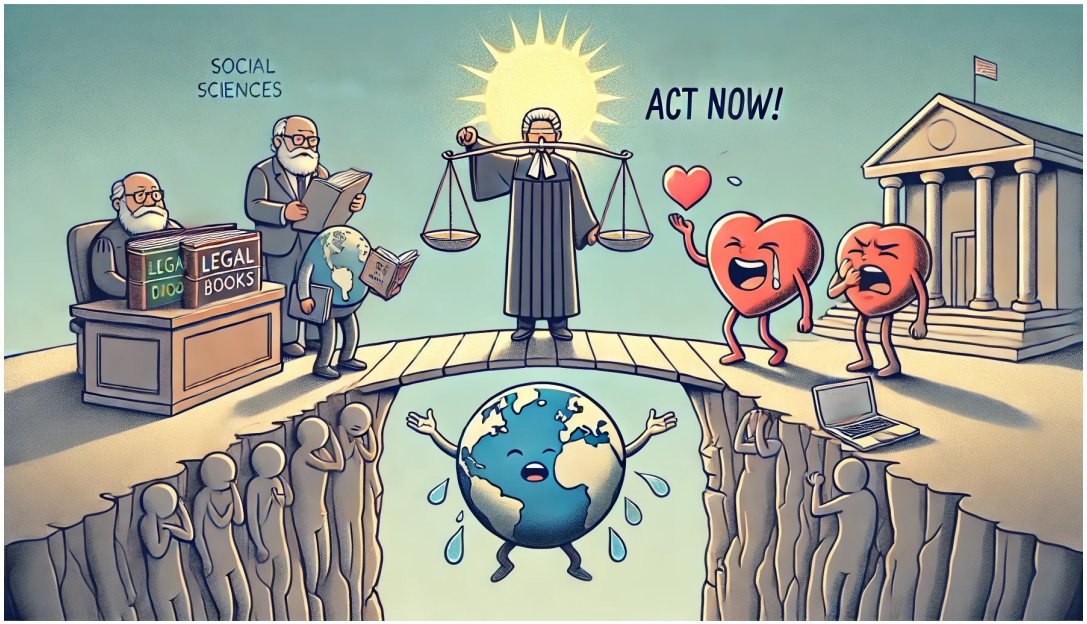
Rui Mata, FS 2026

Version: May 19, 2026

Objectives

- Wrap-up
- Q&A KOGPSY II
- Exam information

How we started: The rise of affectivism



Dukes et al. suggest that the affective sciences are reshaping key societal domains by highlighting the role of emotions in several areas. For example, legal scholars recognize how emotions influence legal decisions, while education research links emotional well-being to learning. In climate action, researchers increasingly recognize that emotions drive urgency and motivate change, and in conflict studies, emotions are seen as key forces shaping political and group behavior.

Dukes, D., Abrams, K., Adolphs, R., Ahmed, M. E., Beatty, A., Berridge, K. C., Broomhall, S., Brosch, T., Campos, J. J., Clay, Z., Clément, F., Cunningham, W. A., Damasio, A., Damasio, H., D'Arms, J., Davidson, J. W., De Gelder, B., Deonna, J., De Sousa, R., … Sander, D. (2021). The rise of affectivism. *Nature Human Behaviour, 5*(7), 816–820. https://doi.org/10.1038/s41562-021-01130-8


BUCH UND REGIE ALEX GARLAND, AUTOR VON
28 DAYS LATER
ES GIBT NICHTS MENSCHLICHERES
ALS DEN WILLEN ZU ÜBERLEBEN
EX_MACHINA
DEMNÄCHST IM KINO

AFFECTIVISM IN 2050: WILL MACHINES FEEL?

Can theories of emotion and motivation tell us whether AI feels?

Affectivism in 2050: Will machines feel?

"Our goal here is to inquire about conditions that would potentially allow machines to care about what they do or think. Under certain conditions, machines capable of implementing a process resembling homeostasis might also acquire a source of motivation and a new means to evaluate behaviour, akin to that of feelings in living organisms. Drawing on recent developments in soft robotics and multisensory abstraction, we propose a new class of machines inspired by the principles of homeostasis. The resulting machines would (1) exhibit equivalents to feeling; (2) improve their functionality across a range of environments; and (3) constitute a platform for investigating consciousness, intelligence and the feeling process itself."

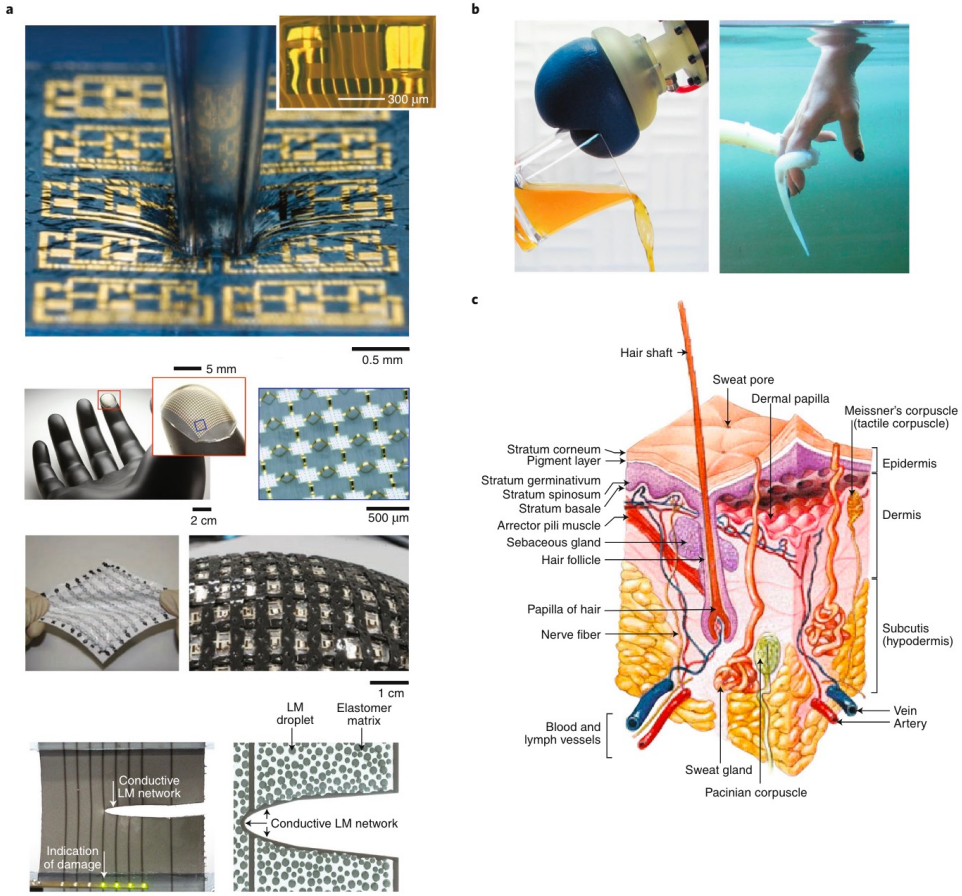


Fig. 2 | Artificial and natural soft materials. a, Soft electronics can be embedded on flexible and stretchable substrates. LM, liquid metal. **b**, Soft robotic effectors grip by conforming to the object. **c**, Human skin contains dense embeddings of sensors and effectors for the maintenance of its own integrity. Reproduced from ref. [23], AAAS (**a**, top three rows); ref. [29], Wiley (**a**, bottom row); and ref. [24], Elsevier (**b**). Credit: National Cancer Institute (**c**).

Homeostasis: process by which a system maintains internal stability despite external changes.

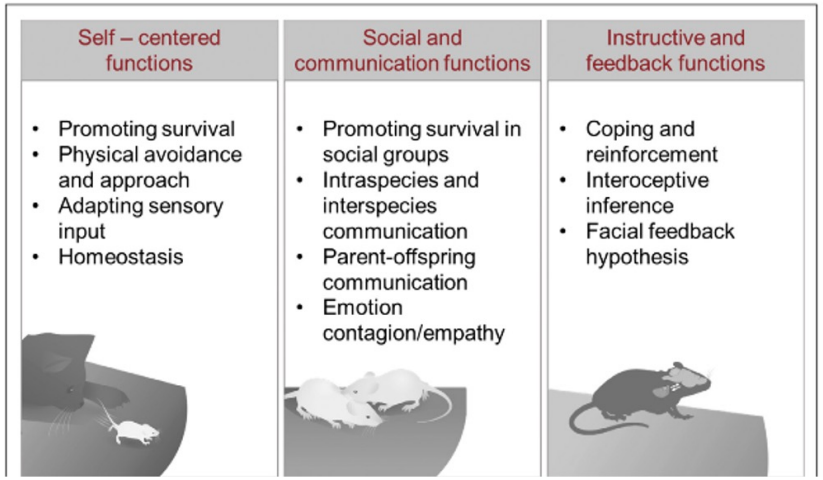
Man, K., & Damasio, A. (2019). Homeostasis and soft robotics in the design of feeling machines. Nature Machine Intelligence, 1, 446–452. https://doi.org/10.1038/s42256-019-0103-7

Your questions concerning KOGPSY II
(paraphrased and selected)

Session 2: What is an emotion? (continued)

- Would it be correct to say that self-centered functions are about immediate survival reactions, whereas instructive/feedback functions are about how bodily or emotional responses provide feedback and influence future behaviour?

The adaptive value of emotions



Evolutionary approaches suggest that emotion (expression) serves different functions: **Self-centered functions** promote survival directly (for example, blood flow prepares fight-or-flight responses, facial expressions associated with fear enhance sensory input, disgust reduce it). **Social and communication functions** help maintain survival in social groups by enabling emotional recognition, empathy, and emotion contagion within and across species, influenced by factors such as familiarity, experience, and social dynamics. **Instructive and feedback functions** involve emotional expressions like freezing or heartbeat changes that feedback modulate emotional states and future behavior.

Zych, A. D., & Gogolla, N. (2021). Expressions of emotions across species. *Current Opinion in Neurobiology, 68*, 57–66. https://doi.org/10.1016/j.conb.2021.01.003

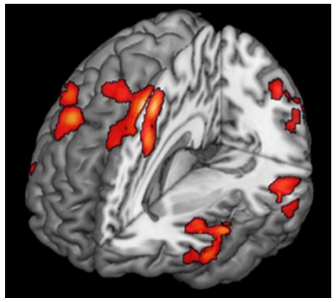
Session 4: Regulation

- Could you explain the neural basis of emotion regulation?

The neural bases of emotion regulation: Meta-analytic evidence

Example data

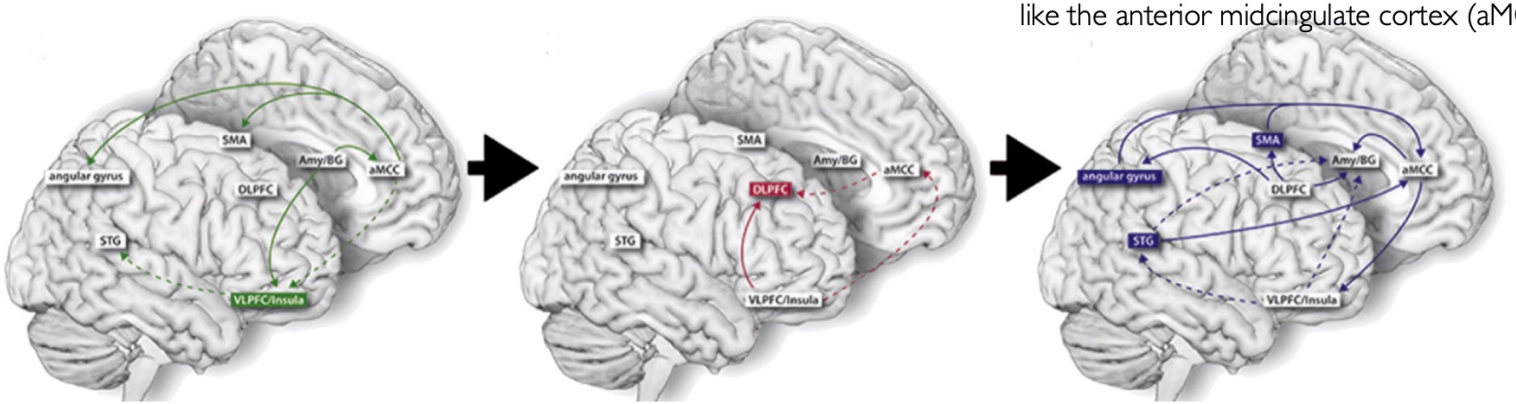
Authors	Number of subjects	Gender ratio (f/m)	Stimulus material	Contrasts
Campbell-Sills et al. (2011)	26	22/4	IAPS, negative	Reduce > baseline
Delgado et al. (2008)	12	6/6	Fear conditioning paradigm with instruction	Decrease > attend
Domes et al. (2010)	33	17/16	IAPS, negative	Decrease > maintain Increase > maintain
Eippert et al. (2007)	24	24/0	IAPS, neutral and negative (fear)	Decrease > view Increase > view
Goldin et al. (2008)	17	17/0	Disgust-inducing and neutral film clips	Reappraise > watch negative (early, middle, late) Suppress > watch negative (early, middle, late)
Harenski et Hamann (2006)	10	10/0	IAPS and popular media, moral vs non-moral, social unpleasant scenes	Decrease moral > odd–even baseline Decrease non-moral > odd–even baseline Decrease moral > watch moral Decrease non-moral > watch non-moral
Hayes et al. (2010)	25	11/14	IAPS and in-house pictures, negative	Reappraise > view Suppress > view



Affective arousal is processed in subcortical regions such as the amygdala, which project to cortical regions including the VLPFC

Activity in the VLPFC is associated with the valuation of emotional stimuli and engagement of regulatory processes, including recruitment of the DLPFC.

The DLPFC is involved in implementing regulation strategies (e.g., reappraisal), and modulates activity in subcortical regions—including the amygdala and basal ganglia—either directly or via intermediary structures like the anterior midcingulate cortex (aMCC)



Kohn, N., Eickhoff, S. B., Scheller, M., Laird, A. R., Fox, P. T., & Habel, U. (2014). Neural network of cognitive emotion regulation: An ALE meta-analysis and MACM analysis. ***NeuroImage, 87***(C), 345–355. **23**

Session 4: Regulation

- Is it important to know the details of the table on slide 25? Especially the clinical condition and description part? Or is it meant for us to have a more general understanding of the implementations?

Implications: Understanding of psychopathology

Table 1 The extended process model of emotion regulation and psychopathology[1]

Regulatory stages and dynamics	Regulatory element	Clinical condition and description
Identification	Perception	**Panic attacks:** overrepresenting subtle signs of current emotional states **Disengagement bias in anxiety:** overrepresenting threatening information for an extended time **Alexithymia:** underrepresenting emotional states
	Valuation	**Experiential avoidance:** overvaluating the costs of emotional states **Clinging behavior in dependent personality disorder:** undervaluating the benefits of intrinsic regulation
	Action	**Learned helplessness in depression:** failing to translate a general regulatory goal into action
Selection	Perception	**Escape from self in binge eating and suicide behavior:** overrepresenting maladaptive regulatory options
	Valuation	**Nonsuicidal self-injury and substance abuse:** positively valuing general maladaptive regulatory categories
	Action	**Cognitive change in autism:** impaired ability to activate general adaptive regulatory categories
Implementation	Perception	**Long-term tactics in ADHD:** underrepresenting adaptive regulatory tactics
	Valuation	**Worry in GAD:** positively valuing maladaptive regulatory tactics
	Action	**Positive distraction in major depression:** impaired ability to activate adaptive regulatory tactics
Monitoring	Stopping	**Rumination in depression:** stopping a maladaptive regulatory tactic too late **Low regulatory self-efficacy in SAD:** stopping an adaptive regulatory tactic too early
	Switching	**Depression, anxiety, and OCPD:** switching from an inefficient implemented tactic too late **Manic states in bipolar disorder:** switching between regulatory categories too early

[1]A summary of clinical conditions that represent potential impairments in specific elements of regulatory stages according to the extended process model of emotion regulation. Examples of clinical conditions are not necessarily characterized by difficulties at a single emotion-regulation stage. Rather, each clinical condition may involve failures at multiple stages (see text for details). Abbreviations: ADHD, attention-deficit/hyperactivity disorder; GAD, generalized anxiety disorder; OCPD, obsessive-compulsive personality disorder; SAD, social anxiety disorder.

"Importantly, clinical conditions are not necessarily characterized by difficulties at a single emotion-regulation stage. Rather, a clinical condition may involve failures at multiple stages. At the same time, a clinical condition that is associated with difficulties in one emotion-regulation stage may not be related to difficulties in another regulation stage."

Sheppes, G., Suri, G., & Gross, J. J. (2015). Emotion regulation and psychopathology. *Annual Review of Clinical Psychology, 11*(1), 379–405. https://doi.org/10.1146/annurev-clinpsy-032814-112739

Session 4: Regulation

- Could explain the theory integration part of emotional well-being?

Theory Integration

Fig. 1 Associations between theories of SWB

- Fulfillment and engagement theories focus on explaining the influences of goals, needs, and activities on SWB.
- Personal orientation theories focus on explaining the influence of temperament on SWB by dynamically affecting the process of fulfillment and engagement as well as how the dynamic process leads to the readjustment of personal orientation.
- Evaluative theories focus on how personal evaluations of life (i.e., the cognitive aspect of SWB) are interconnected with the process of fulfillment and emotions.
- Emotion theories focus on how experiences of emotions (i.e., the affective aspect of SWB) are interconnected with the process of fulfillment, engagement, and evaluations.

Das, K. V., Jones-Harrell, C., Fan, Y., Ramaswami, A., Orlove, B., & Botchwey, N. (2020). Understanding subjective well-being: Perspectives from psychology and public health. *Public Health Reviews, 41*(1), 25. https://doi.org/10.1186/s40985-020-00142-5

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Session 5: Well-being

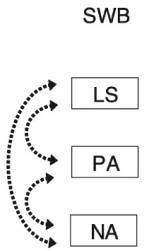
- One theory sees the LS, PA, and NA measures as interconnected manifestations of an overarching factor: SWB. Can SBW be compared to the *g* factor?
- With LS, PA, NA being interconnected manifestations does that also mean that the SWB factor can only exist when all the three measures are included?
- Is an SWB factor less meaningful under the view that treats LS, PS, and NA as independent constructs?

The measurement of subjective well-being

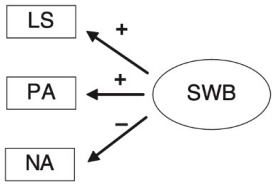
The measurement of subjective well-being (SWB) varies widely but typically consists of one or more measures of: 1) life satisfaction (LS, i.e. cognitive evaluation of one's life); 2) positive affect (PA, i.e., frequency/intensity of pleasant emotional experiences; 3) negative affect (NA, i.e., frequency/intensity of unpleasant emotional experiences). The psychometric structure of SWB is, however, still debated and unresolved (see two examples below – additional ones can be found in Busseri and Sadava, 2011).

	NA	LS
PA	-.49	.53
LS	-.37	

Meta-analytic correlation matrix (cf. Busseri, 2018)



This model treats LS, PA, and NA as independent constructs. SWB is seen as a broad research domain, not a unified psychological construct.



SWB conceptualized as a unified latent construct. LS, PA, and NA are interconnected manifestations of an overarching psychological factor: SWB.

Busseri, M. A., & Sadava, S. W. (2011). A review of the tripartite structure of subjective well-being: Implications for conceptualization, operationalization, analysis, and synthesis. *Personality and Social Psychology Review, 15*(3), 290–314. https://doi.org/10.1177/1088868310391271

Busseri, M. A. (2018). Examining the structure of subjective well-being through meta-analysis of the associations among positive affect, negative affect, and life satisfaction. *Personality and Individual Differences, 122*, 68–71. https://doi.org/10.1016/j.paid.2017.10.003

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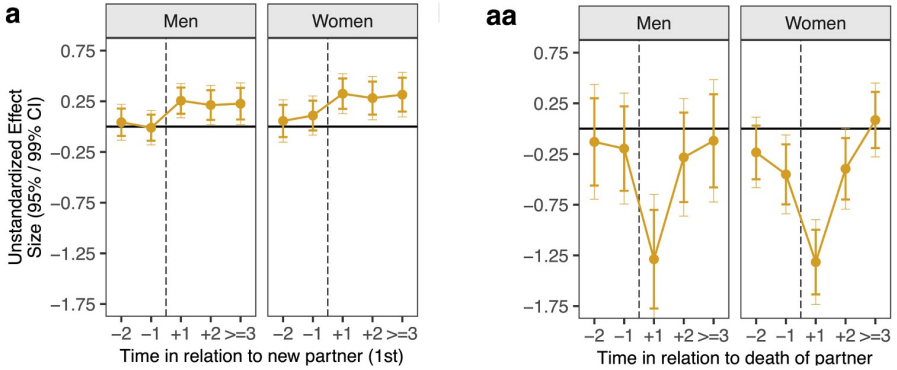
Session 5: Well-being

- Please explain how life events affect cognitive vs. emotional subjective well-being and how this differentiates between positive and negative life events.
- Is it right that when asked about life satisfaction, people rely more on a cognitive representation of SWB and in this concept a more stable change is experienced after a life event? And in emotional SWB (e.g. happiness) people experience a change which will revert back to a baseline in most cases after a while (Adaptation).
- Is the change in cognitive SWB stable (strongly and consistently affected) in both positive and negative life events? And does emotional SWB go through adaptation in both positive and negative life events?

Individual differences: Life events



Longitudinal studies of the effects of life events suggest systematic effects on subjective well-being (SWB), albeit these vary significantly by event. Cognitive well-being (life satisfaction) appears more strongly and consistently affected than emotional well-being (positive/negative affect). Adaptation (regression to baseline) is often but not always observed.

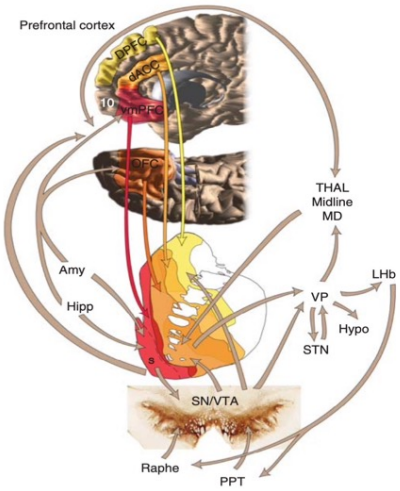
Krämer, M. D., Rohrer, J. M., Lucas, R. E., & Richter, D. (2025). Life events and life satisfaction: Estimating effects of multiple life events in combined models. *European Journal of Personality, 39*(1), 3–23. https://doi.org/10.1177/08902070241231017

For a meta-analysis: Luhmann, M., Hofmann, W., Eid, M., & Lucas, R. E. (2012). Subjective well-being and adaptation to life events: A meta-analysis. *Journal of Personality and Social Psychology, 102*(3), 592–615. https://doi.org/10.1037/a0025948

Session 8

- Could you explain the main idea of the reward circuitry slide again, especially the cortical–basal ganglia circuit and the role of dopamine pathways? I understand that reward processing is distributed across several regions, but I'm still confused about how the cortical, subcortical, and dopaminergic systems interact functionally within the circuit. I'm also having trouble understanding which brain regions are the most important to focus on and what their specific roles are within reward processing.

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**."

Haber, S. N., & Knutson, B. (2010). The reward circuit: linking primate anatomy and human imaging. ***Neuropsychopharmacology, 35***(1), 4-26.

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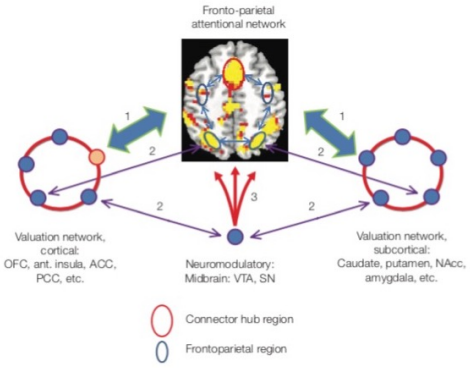
Mechanisms of motivation–cognition interaction

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.

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Session 8

- My question is, whether animal models in conditioning psychology can provide a clear understanding of motivation. On one hand, Session 8 mentioned that a drawback of animal models is their lack of generalizability to humans. On the other hand, we looked at devalued motivation and habit formation in addiction; for instance, page 18 states that this is due to the prefrontal cortex no longer exercising top-down control. Since this can be proven neurologically, are animal models actually effective for understanding motivation, or just not to a human context?

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 neuro-transmitters	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

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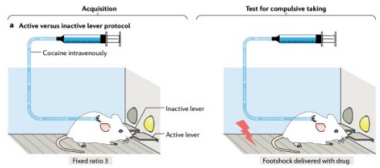
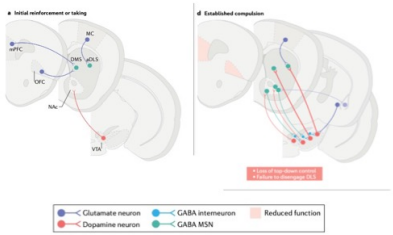
Dysregulated motivation: Habit formation in addiction

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).

Lüscher, C., Robbins, T. W., & Everitt, B. J. (2020). The transition to compulsion in addiction. *Nature Reviews Neuroscience*, 1–17. http://doi.org/10.1038/s41583-020-0289-z

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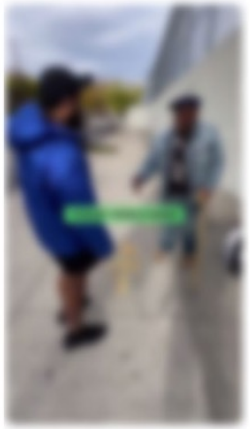
Session 9

- In the lecture you stated that if we wanted to falsify Psychological Egoism, we would have to prove that an ultimate desire exists, that is not selfish, but this is obviously not possible. However, in the summary is says that Psychological Egoism is an empirical claim. How can a theory be empirical if it is neither falsifiable nor operational?

Psychological Egoism: Summary

- Psychological egoism is the empirical claim that all voluntary action is ultimately selfish
- Difficulty: How do we know a desire is ultimate?
- Relationship between empathy and helping suggests motivating role of other-concerned emotions



Surprising Homeless Man With $3,000!
266.072 Aufrufe

Session 9: Cooperation

- Do we need to learn the traits and their definitions on slides 24-26 for the exam? Or should we just know that there are 58 different traits but mainly know D (dark factor of personality)?

Caring as a Trait: Some More Candidates…

Table 1. Candidate Traits Along With Their Definitions, Operationalizations, and Measurement Occasion, and Whether They Were Also Assigned to the Strict Set of Traits Involving Unequivocal Conceptual Overlap With the Criterion

Construct	Definition	Questionnaire	Number of items (ω)	Example item	Measurement occasion	Strict set
Basic/broad traits						
Antagonism	Behaviors that put the individual at odds with other people, including an exaggerated sense of self-importance and a concomitant expectation of special treatment, as well as a callous antipathy toward others, encompassing both unawareness of others' needs and feelings, and a readiness to use others in the service of self-enhancement (American Psychiatric Association, 2013)	PID-5 (Maples et al., 2015)	20 (.90)	I'll stretch the truth if it's to my advantage.	T2	Yes
Big Five Agreeableness	[Individual] differences in the motivation to cooperate (vs. acting selfishly) in resource conflicts (Denissen & Penke, 2008); motivation to maintain positive relations with others (Graziano & Tobin, 2009)	NEO Five Factor Inventory (Costa & McCrae, 1992); Big Five Aspects Scales (DeYoung et al., 2007); Big Five Inventory–2 (Soto & John, 2017); IPIP-50 (Goldberg, 1992)	52 (.95)	I am someone who takes advantage of others.	T1	Yes
HEXACO Agreeableness	The tendency to be forgiving and tolerant of others, in the sense of cooperating with others even when one might be suffering exploitation by them (Ashton & Lee, 2007)	HEXACO-60 (Ashton & Lee, 2009)	10 (.75)	People think of me as someone who has a quick temper.	T1	No
HEXACO Honesty-Humility	The tendency to be fair and genuine in dealing with others, in the sense of cooperating with others even when one might exploit them without suffering retaliation (Ashton & Lee, 2007)	HEXACO-60 (Ashton & Lee, 2009)	10 (.76)	I think that I am entitled to more respect than the average person is.	T1	Yes
Detachment	Avoidance of socioemotional experience, including both withdrawal from interpersonal interactions, ranging from casual daily interactions to friendships to intimate relationships, as well as restricted affective experience and expression, particularly limited hedonic capacity (APA, 2013)	PID-5 (Maples et al., 2015)	20 (.91)	I don't like spending time with others.	T2	No

(continued)

Hilbig et al. (2023)

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Exam

- Multiple-choice: 20 questions Kognitionspsychologie I, 20 questions Kognitionspsychologie II; 50% A, 50% K-prim
- Language: English (dictionary can be used)
- Time & location: Wednesday, June 3rd, 8:00-10:00, DSBG Neubau, Sporthalle 1 (we aim to let people in at ca. 7:30 so we can start at 8:00)

Exam

- Once you entered the room, you would need to wait to leave. If needed, use the bathrooms before entering the exam room.
- There will be 4 registration desks (rather than 2, as in the “Verhaltensregeln” document), organised by name (1st letter of 1st last name)
- At the registration desk, students must present their ID and DICTIONARY (book format only) for checking.
- Bags should be placed under own table (not at the front or sides of the exam room), phones turned off.
- Doors close at 07:55, to allow time for announcements and punctual start.
- First 30 minutes: No hand-in between 08:00 and 08:30.
- Final 15 minutes: No hand-in between 09:45-10:00.