Somatic Marker Hypothesis

Tasha Poppa

Affective Computing Guest Lecture

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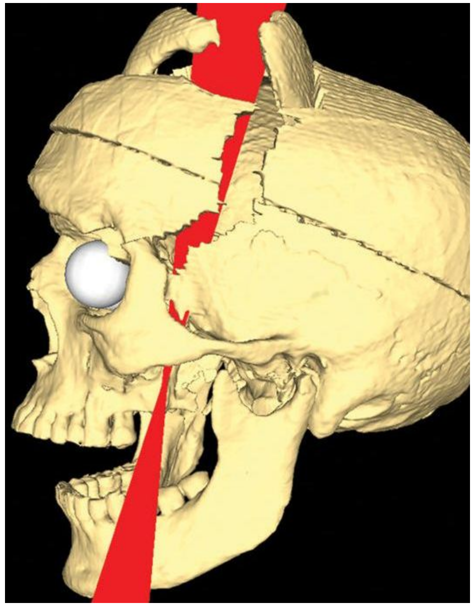
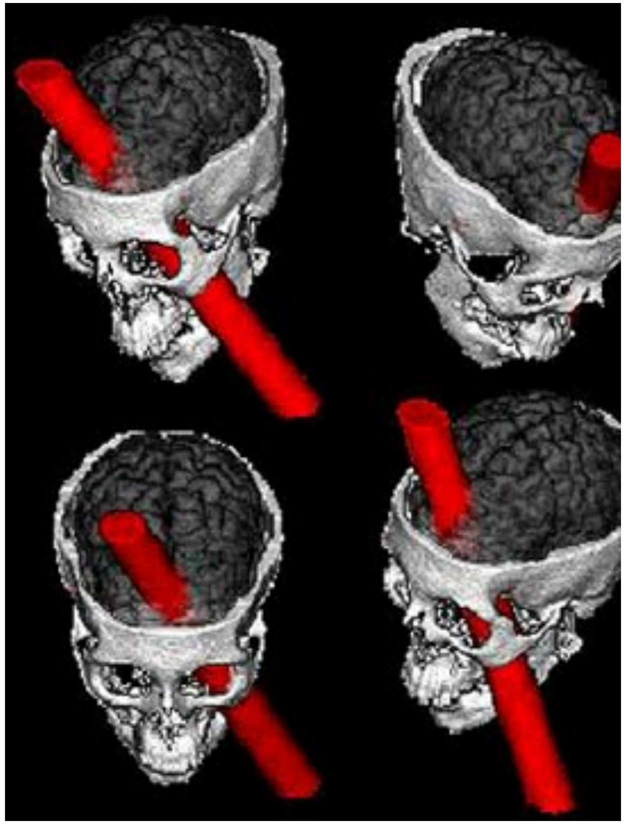
Outline

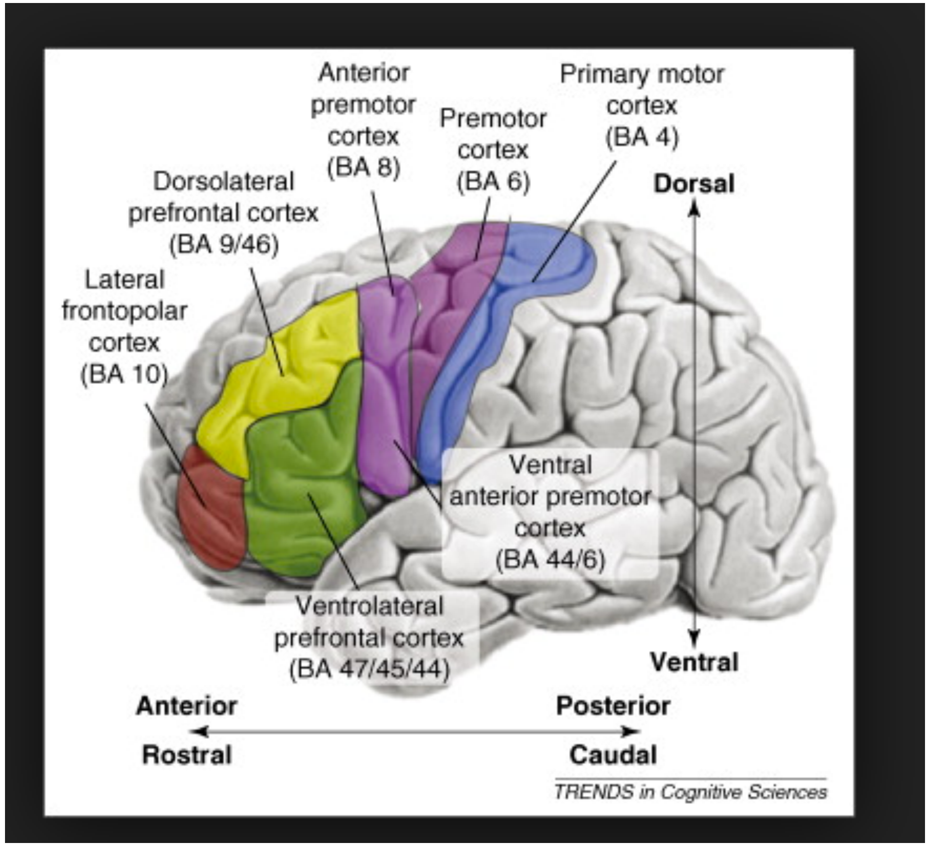
- Historical context
- Some Useful Functional Neuroanatomy
- VMPFC patients: Clinical observations
- Somatic Marker Hypothesis
- Testing The SMH
  - Iowa Gambling Task (IGT)
  - Empirical Studies
  - Computational Modeling

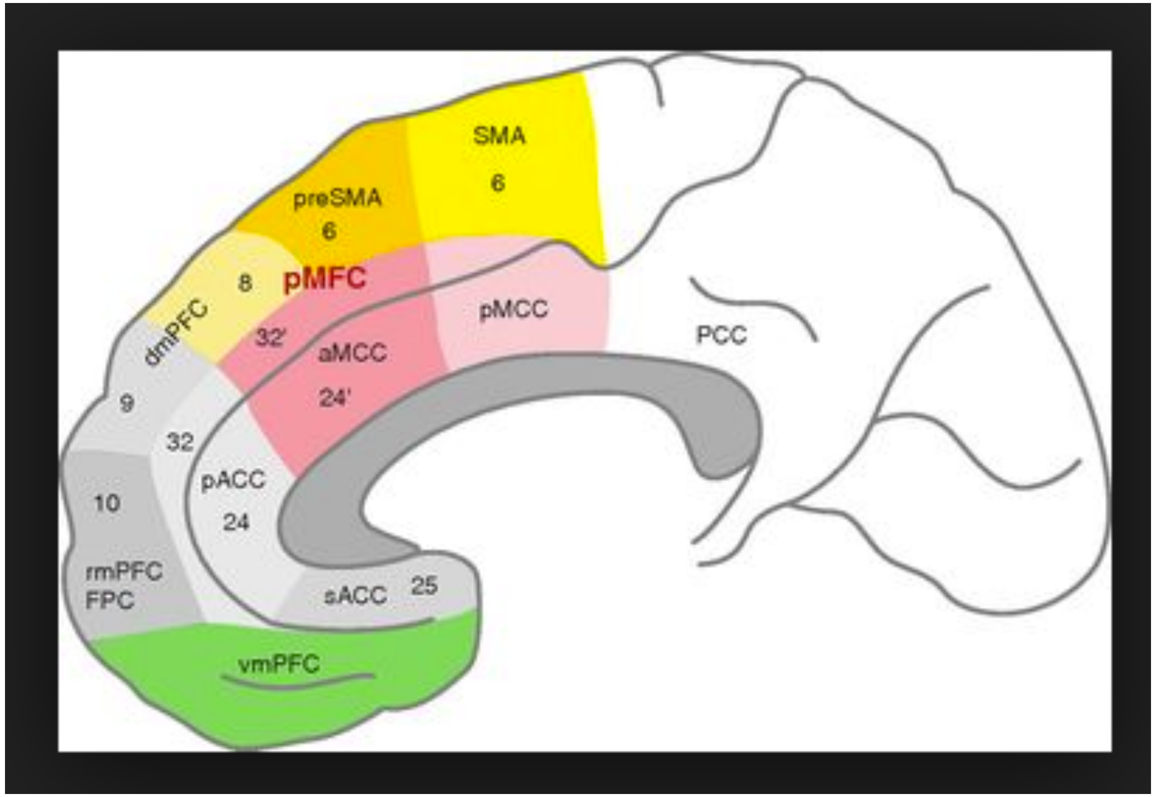
Historical Context: On the Functions of the Prefrontal Cortex

- Phineas Gage’s famous tamping rod accident of 1848
- Damage did not result in loss of intellect, motor, language abilities
- Instead, demonstrated loss of social inhibitions/inappropriate behavior
- Exact location of the damage is uncertain
  - Ventral vs. Dorsal?
  - H. and A. Damasio revived interest in the study of the prefrontal cortex









Brief Tour of the Frontal Lobes

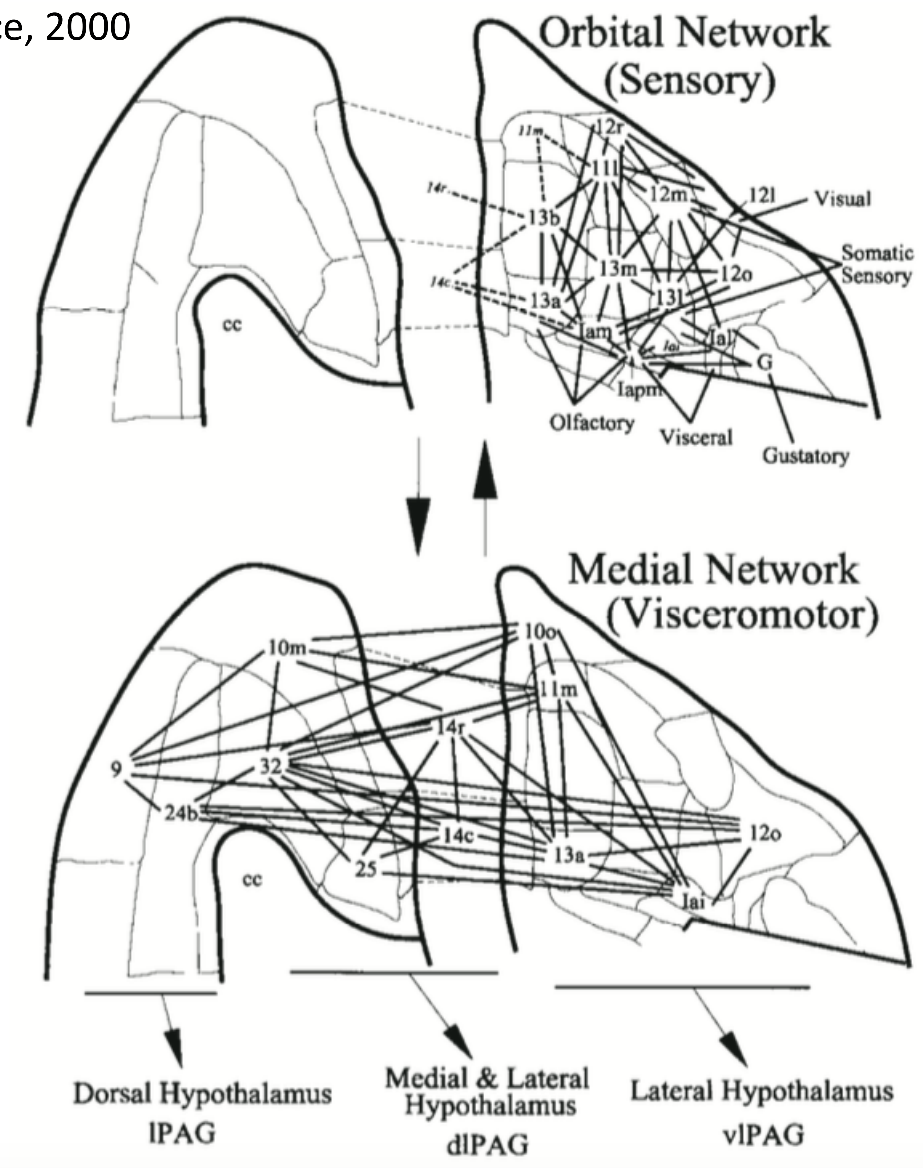
A Ventral Perspective

- The orbitofrontal cortex
  - Lateral and Medial divisions
  - Sulcus/Gyrus rectus defines the boundary

A Ventral Perspective

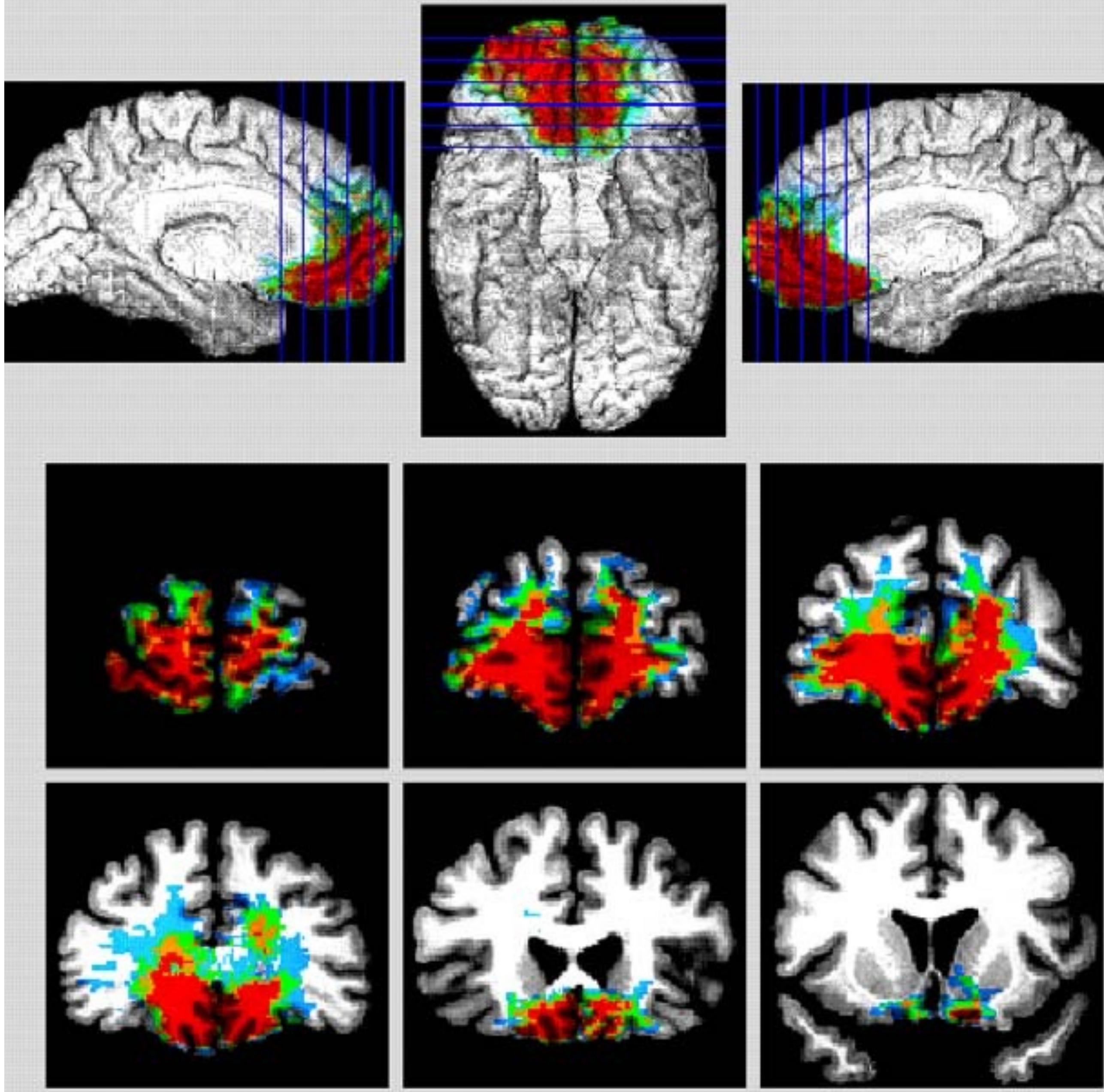
- Lateral orbital regions:
    - Multimodal sensory integration
    - Esp. sensitive to reward value of sensory stimuli
    - Associative
- Medial orbital regions:
    - Motor, but of visceral outputs
    - PAG & Hypothalamus
- Medial network overlaps with lateral network
    - Sensory-visceromotor integration

Ongur & Price, 2000



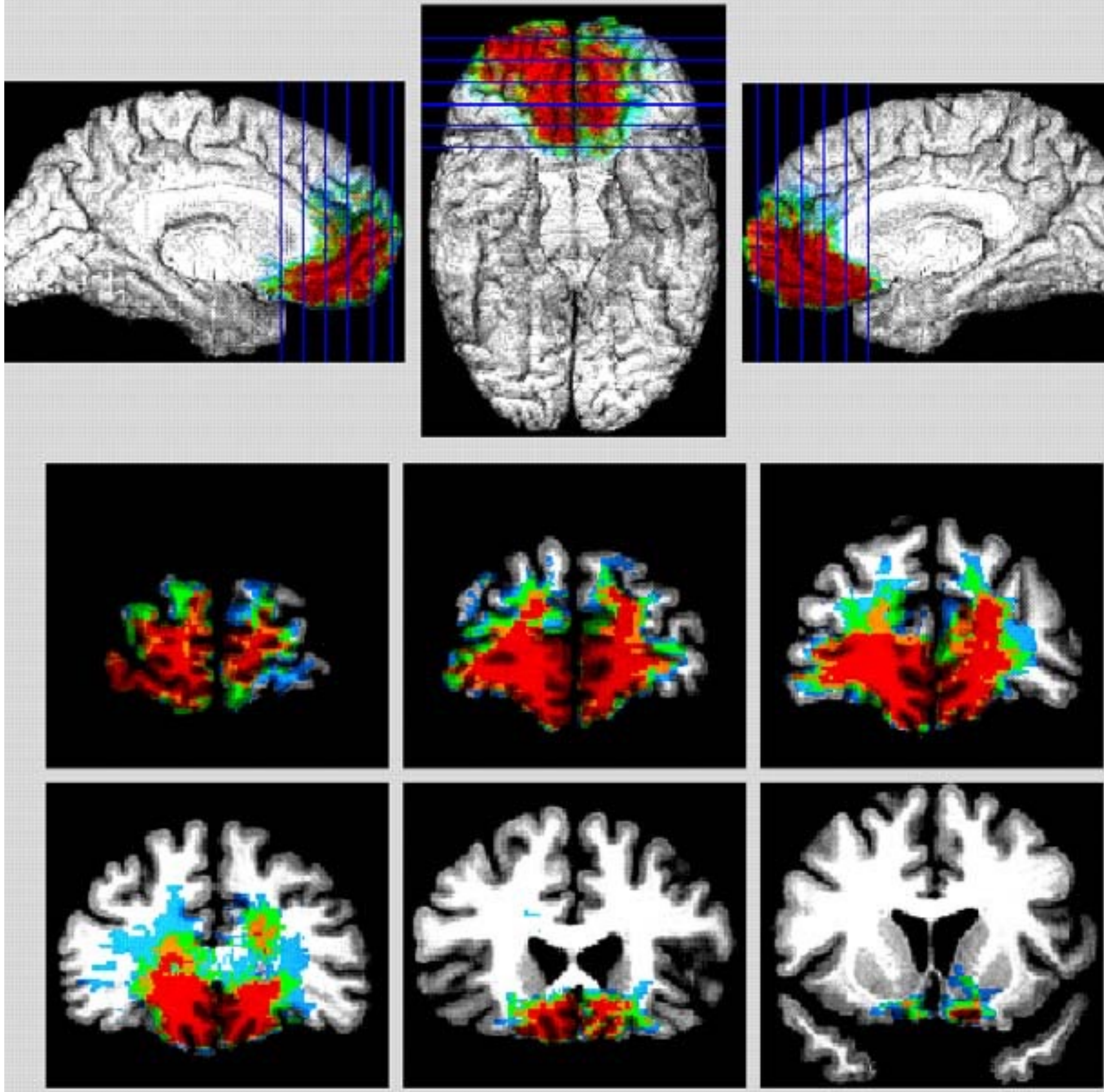
VMPFC Patients: Clinical Observations

- Normal:
  - Semantic/factual knowledge
  - Intellect (IQ)
  - Reasoning (incl. about morals)
  - Language
  - Working Memory
  - Attention



VMPFC Patients: Clinical Observations

- Abnormal:
  - Difficulty planning daily activities
  - Future-oriented planning
  - Emotionally labile/blunted
  - Behaviors/choices lead to financial losses, loss of friends/family/social stature
  - Acquired psychopathy/sociopathy



Somatic Marker Hypothesis (Damasio, 1991; Damasio, et al., 1994)

- A systems-level neuroanatomical and cognitive framework for decision-making and its influence by emotion

- SMH part of a tradition of models of emotion which posit that emotion/feelings are representations of embodied changes in visceral/musculoskeletal states

  - William James & Carl Lange (late 1800s)

  - The model also echoes with the writings of Nauta (1971)
    - Affective reference points, “navigational markers”, assist adaptive behavior wrt. frontal lobe function

Somatic Marker Hypothesis (Damasio, 1991; Damasio, et al., 1994)

- Emotion:
  - "…changes in body and brain states triggered by a dedicated brain system that responds to specific contents of one's perceptions, actual or recalled relative to a particular object or event" (Bechara and Damasio, 2005)

- Feelings:
  - Concerns how bodily states are represented in somatosensing cortices (S1, S2, operculum, insula)

Somatic Marker Hypothesis (Damasio, 1991; Damasio, et al., 1994)

BODY

- Endocrine release:
  - Epinephrine/Adrenaline
  - Oxytocin
- Autonomic modifications:
  - Cardiac
  - Vascular
  - Sweating
- Musculoskeletal:
  - Posture
  - Facial expression
  - Freeze/Flight/Flight

BRAIN

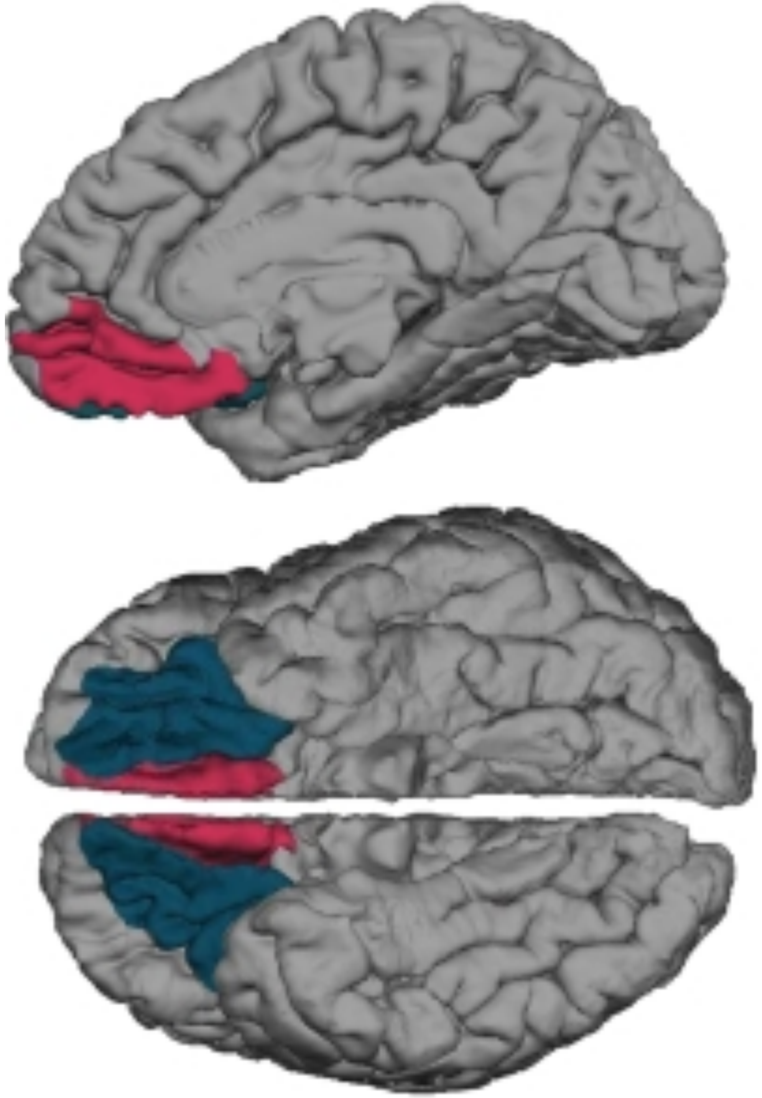
- Neurotransmitter systems:
  - Dopamine
  - Serotonin
  - Acetylcholine
- Alteration in somatosensory maps
  - Esp. insular cortex
- Afferent feedback to brain from body
  - Vagus nerve
  - Spinal pathways

Somatic Marker Hypothesis (Damasio, 1991; Damasio, et al., 1994)

- Induction of somatic states:
  - Primary Inducers
    - Innate or learned stimuli that generate pleasurable or aversive feeling states
    - Automatically, obligatorily elicit bodily response
    - Amygdala
  - Secondary Inducers
    - Generated by recall of personal or hypothetical event
    - i.e. thoughts about/memories of a primary inducer
    - VMPFC
  - Secondary induction relies on intact mechanisms of primary induction
    - After a somatic state has been triggered by a primary inducer and experienced at least once, a neural pattern for this somatic state is formed

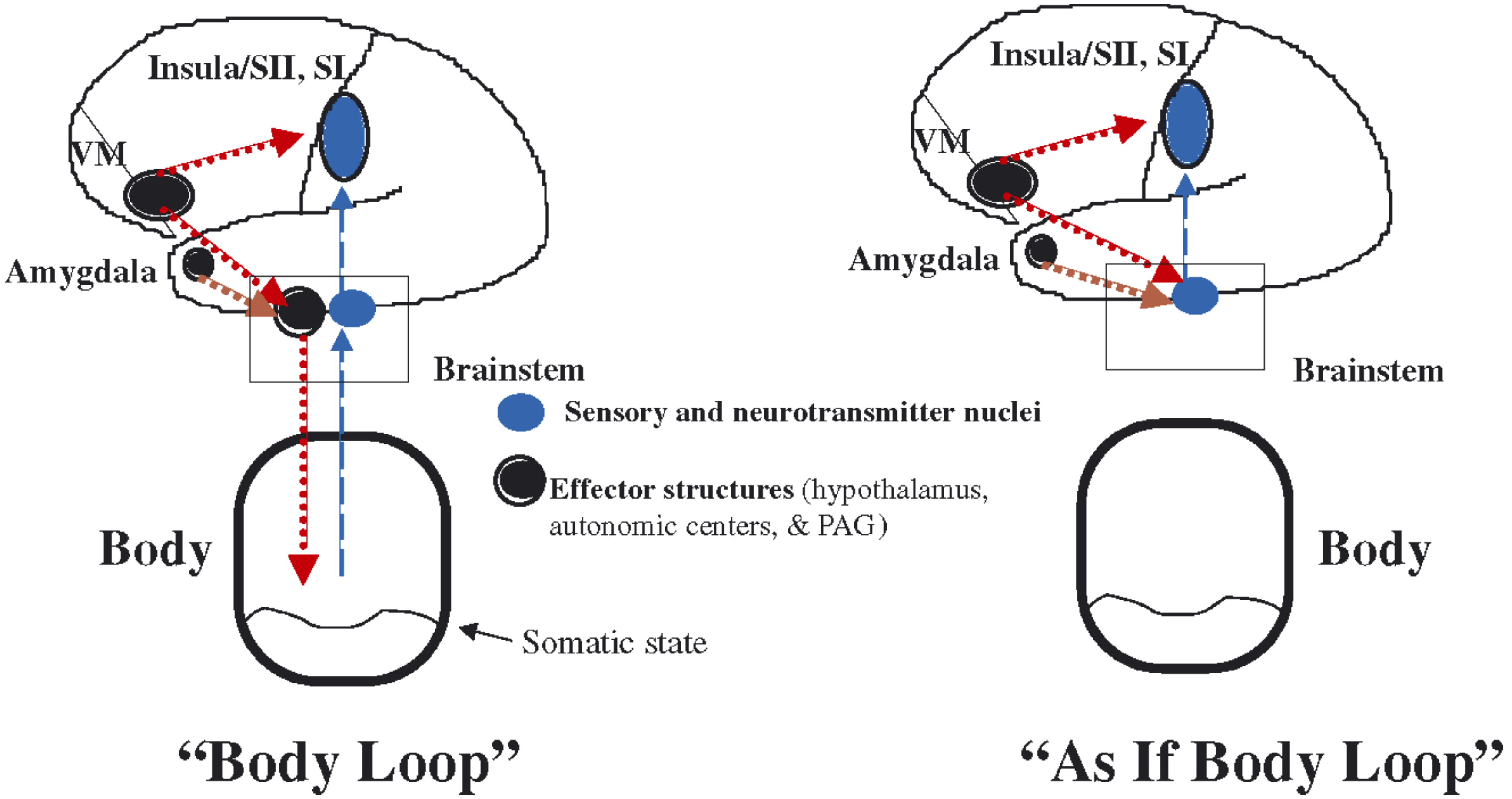
Somatic Marker Hypothesis (Damasio, 1991; Damasio, et al., 1994)

- VMPFC:
  - Trigger structure for somatic states from secondary inducers
  - VMPFC neuron ensembles couple:
    - Memories/sensory input from association cortices
    - **Effector structures to actualize a somatic state**
    - **Neural patterns related to feeling states**
  - In sum: “what it feels like to be in a given situation”



Somatic Marker Hypothesis (Damasio, 1991; Damasio, et al., 1994)

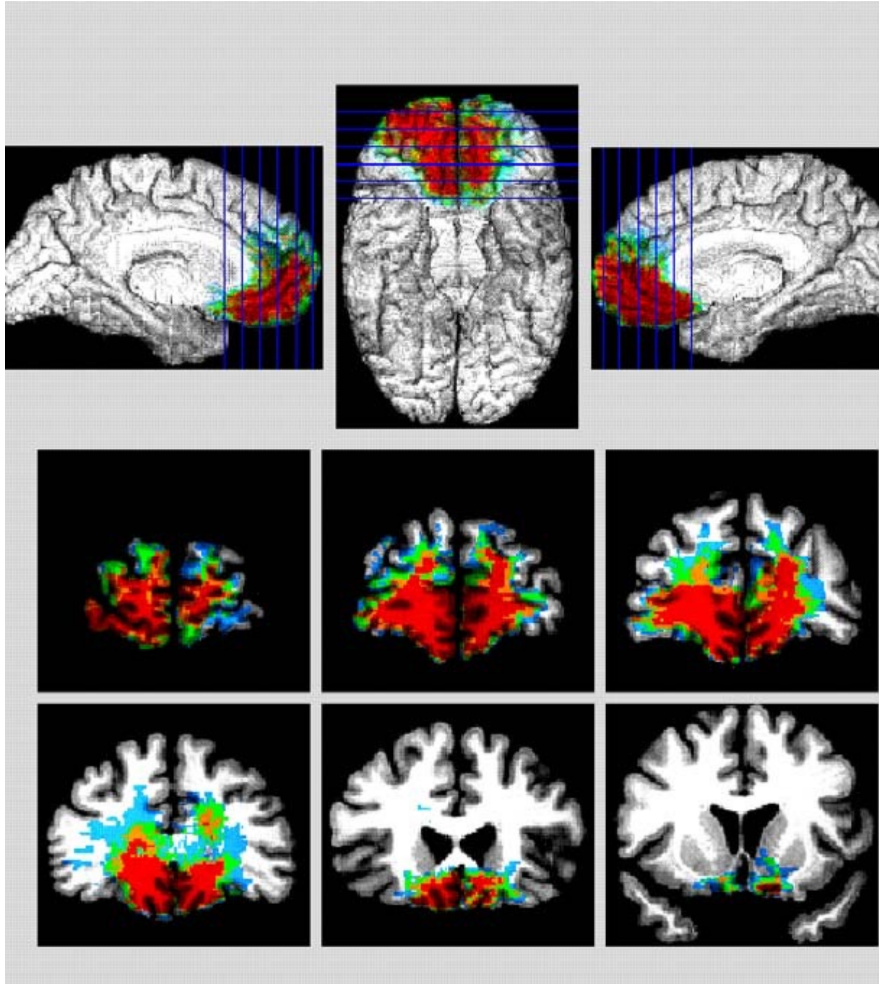
- The "body loop" mechanism of somatic markers



Somatic Marker Hypothesis (Damasio, 1991; Damasio, et al., 1994)

- In sum:
- Reasoning and knowledge are insufficient for advantageous choices under uncertainty

- VMPFC patients' inability to make advantageous decisions due to defect in emotional mechanism that signals prospective consequences of an action/assists in selection of choices



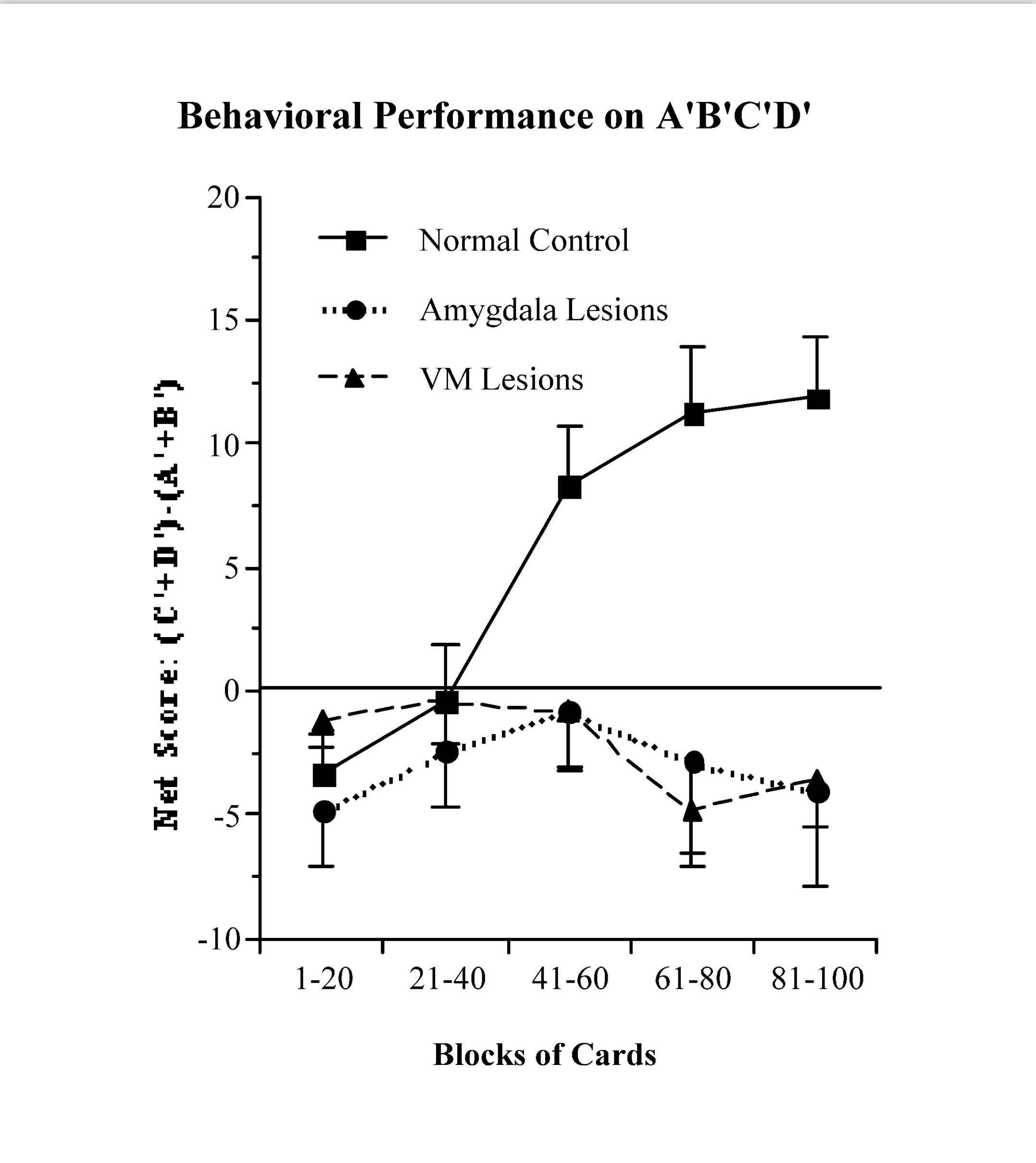
Testing the SMH: IGT

- 4-armed bandit
- Decks A & B → Negative Expected Value
- Decks C & D → Positive Expected Value

- Decks A & C → High Variance
- Decks B & D → Low Variance

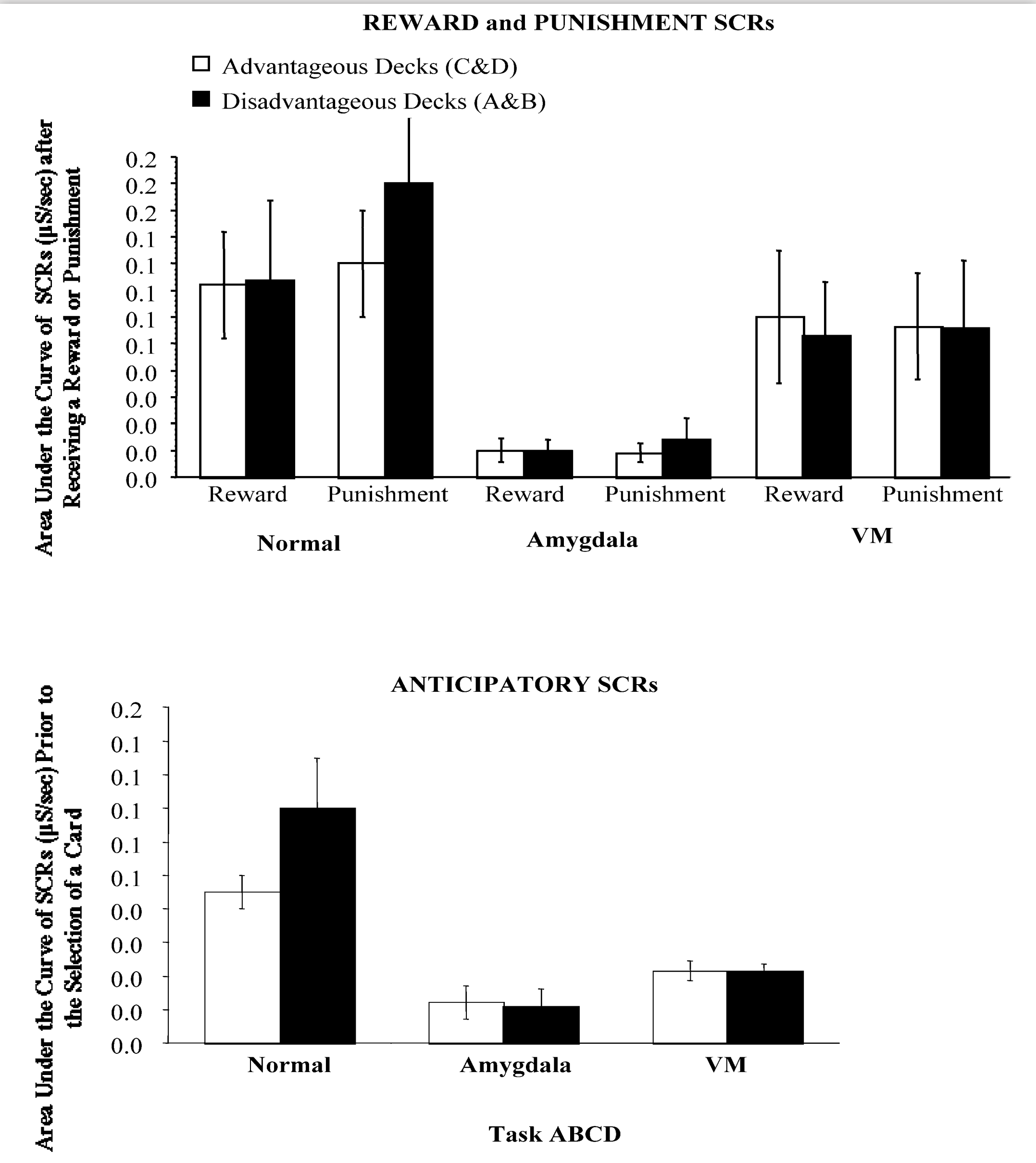
Testing the SMH: IGT

- Patients with lesions to VMPFC and Amygdala do not learn to associate decks with outcomes
- But through different mechanisms?



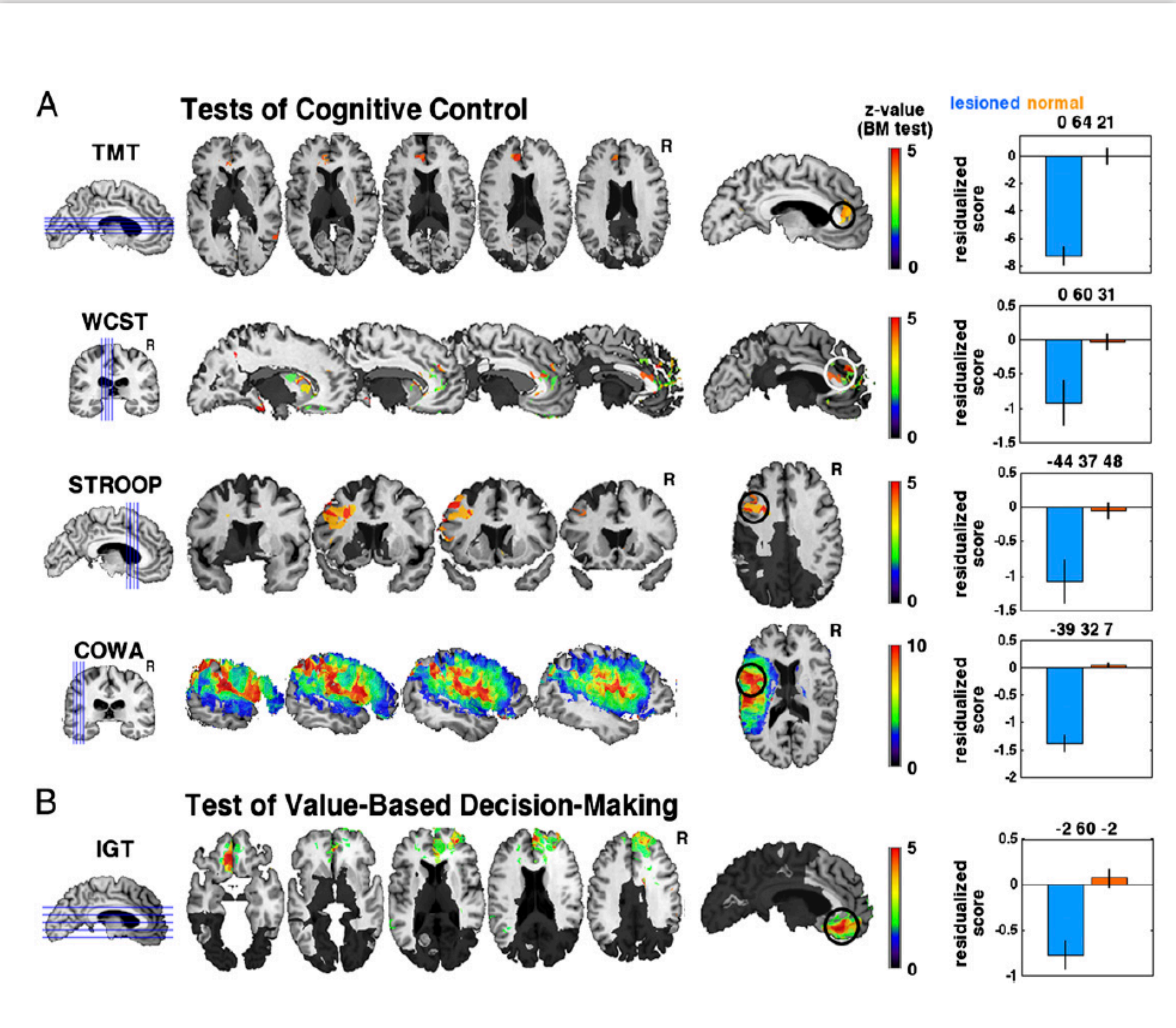
Testing the SMH: IGT

- Skin Conductance Responses (SCR)
  - Pure sympathetic innervation to eccrine glands found on palmar surface
  - Correlates with arousal (but not valence)
- Amygdala damage:
  - No arousal to reward/punishment
  - No anticipatory response
- VMPFC damage:
  - Arousal to reward/punishment
  - No anticipatory response



IGT sensitive to VMPFC lesions

- N = 344 patients
- Various neuropsychological tests of executive function:
    - Trail Making Test (TMT)
    - Wisconsin Card Sorting Task (WCST)
    - STROOP
    - Controlled Oral Word Association (COWA)
    - IGT
- Only damage to VMPFC is significantly linked to IGT performance



Additional Empirical Studies

- Applications to clinical populations:
  - Greater dopamine release in ventral striatum in pathological gamblers while playing IGT (Linnet, et al., 2010)
  - Opiate users tend to prefer deck C (frequent reward/negative EV deck) (Upton, et al., 2012)
  - Worse performance in Obsessive Compulsive Disorder (Filardi da Rocha, et al., 2011)

Reinforcement Learning Model of IGT:
Expectancy–Valence Learning Model (Busemeyer and Stout, 2002)

- Lesions, various psychiatric, other neurological conditions can generate impairments on the IGT
- IGT is a complex task that may require involvement of a number of latent cognitive processes
- Scoring criterion of [(C + D) – (A + B)] is not very sensitive at distinguishing the underlying source of the deficit
- Structural models that formalize latent processes in mathematical terms may reveal where the impairment originates

Reinforcement Learning Model of IGT:
Expectancy–Valence Learning Model (Busemeyer and Stout, 2002)

- EVL model has 3 free parameters *(w*, *c*, and *a*) that control choice behavior in the IGT
- Valence – parameter *w* describes the relative weight given to gains vs. losses in a given trial

$$v(t) = \{(1 - w) \cdot R[D(t)] + w \cdot L[D(t)]\}$$

Reinforcement Learning Model of IGT:
Expectancy–Valence Learning Model (Busemeyer and Stout, 2002)

- Expectancy learning: a decision maker learns expectancies about each deck over the course of experience

- Updating mechanism with a recency parameter *a*:

$$Ev[\mathrm{D}_i|t] = (1 - a) \cdot Ev[\mathrm{D}_i|t - 1] + a \cdot v(t)$$

Reinforcement Learning Model of IGT:
Expectancy–Valence Learning Model (Busemeyer and Stout, 2002)

- The choice made on each trial is a probabilistic function of deck expectancies based on Luce's ratio of strength rule (1959)

$$Pr[D_i|t+1] = \frac{e^{Ev[D_i|t]\theta(t)}}{\sum_{j=1}^{4} e^{Ev[D_j|t]\theta(t)}} \qquad \theta(t) = (t/10)^c$$

- c is the sensitivity parameter. Determines sensitivity of choice probabilities to the expectancies
- The way Θ is defined allows for sensitivity to change over trials
  - Explore vs. exploit

Reinforcement Learning Model of IGT:
Expectancy–Valence Learning Model (Busemeyer and Stout, 2002)

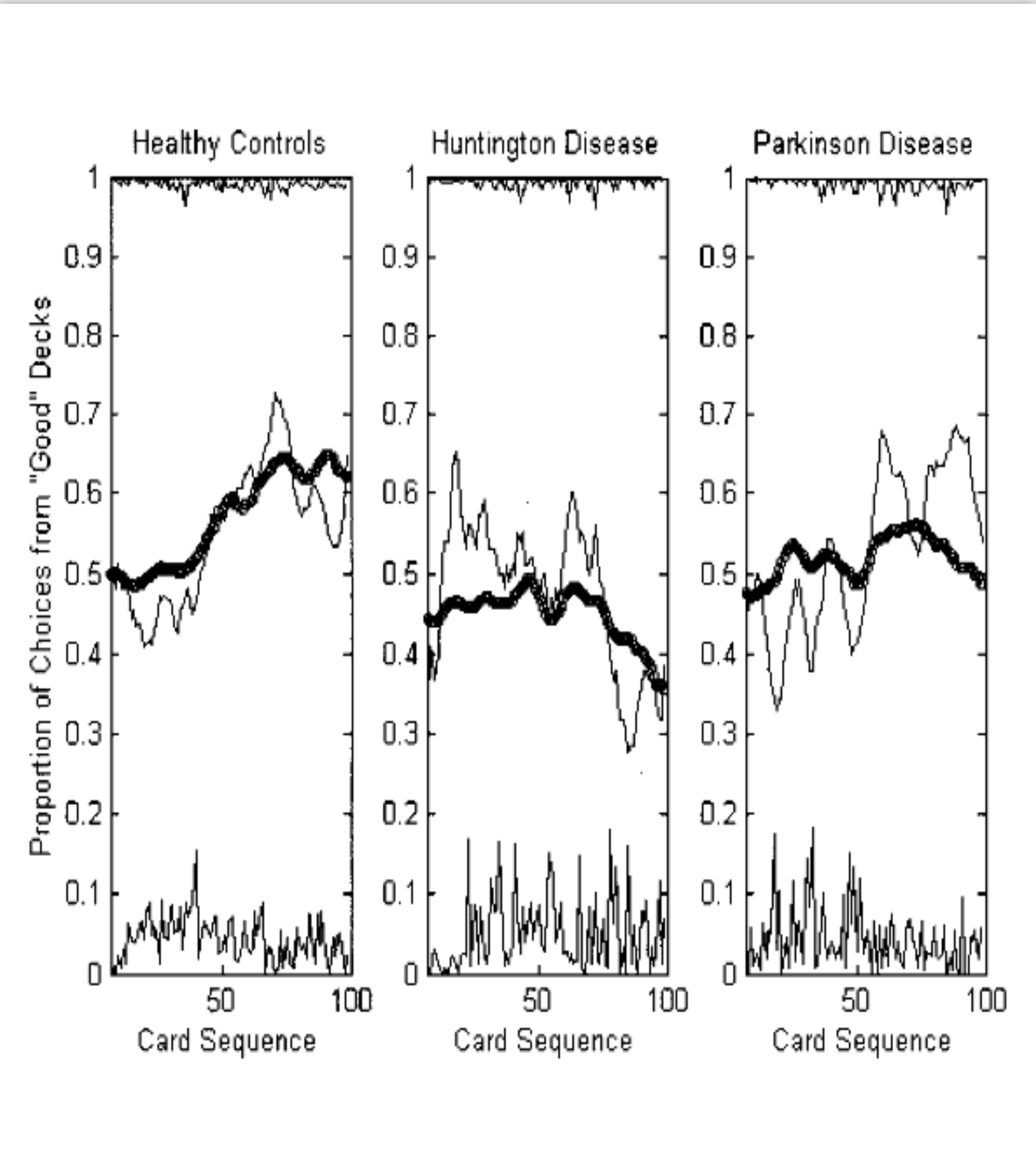
Free parameters: *w*, *c*, and *a*

- *1)* $v(t) = \{(1 - w) \cdot R[D(t)] + w \cdot L[D(t)]\}$
- *2)* $Ev[D_i|t] = (1 - a) \cdot Ev[D_i|t - 1] + a \cdot v(t)$
- 3) $Pr[D_i|t + 1] = \dfrac{e^{Ev[D_i|t]\theta(t)}}{\sum_{j=1}^{4} e^{Ev[D_j|t]\theta(t)}}$ ← $\theta(t) = (t/10)^c$

Reinforcement Learning Models of IGT

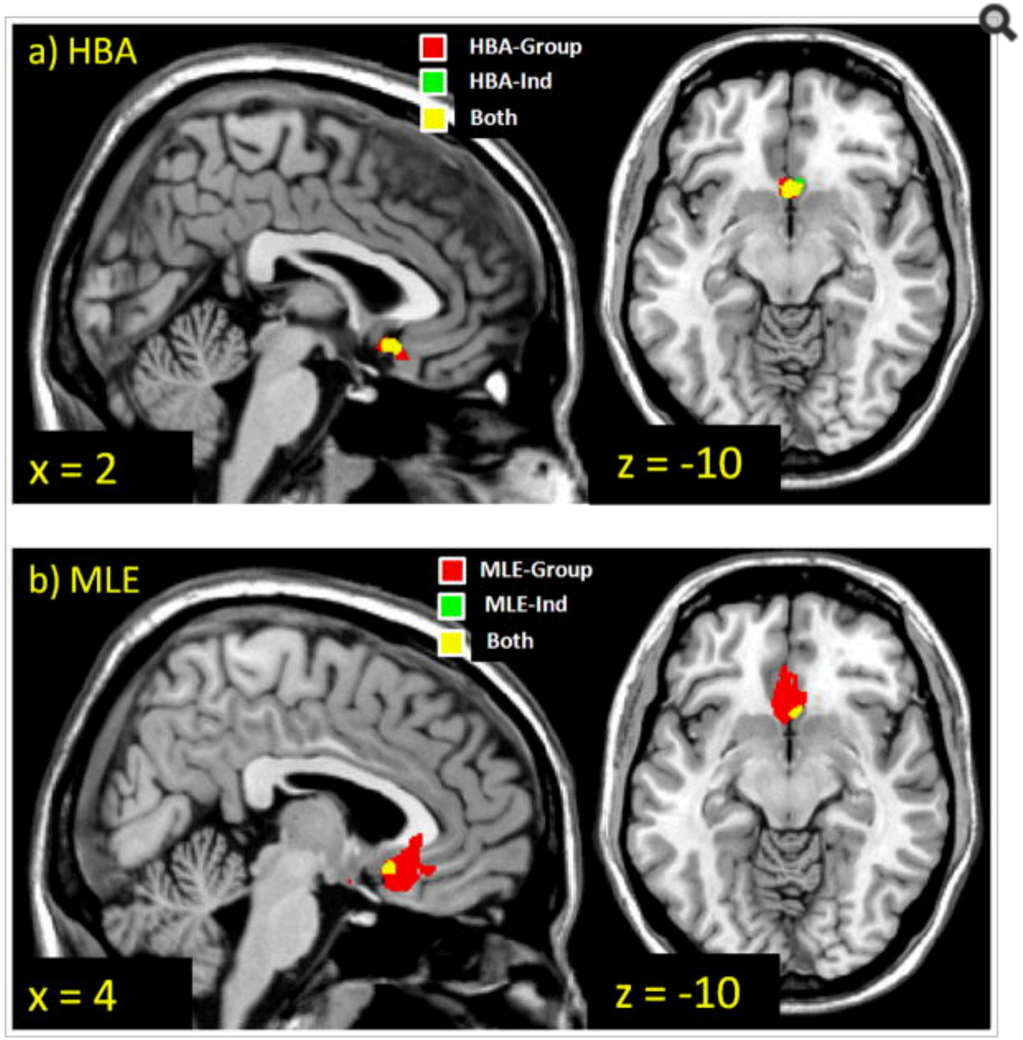
- Model performance:
  - Thick line = predicted choices
  - Thin line = observed choice proportions

- Model performance evaluated against a baseline model that simulates choices based on observed choice proportions using log-likelihoods



Reinforcement Learning Models in the Brain

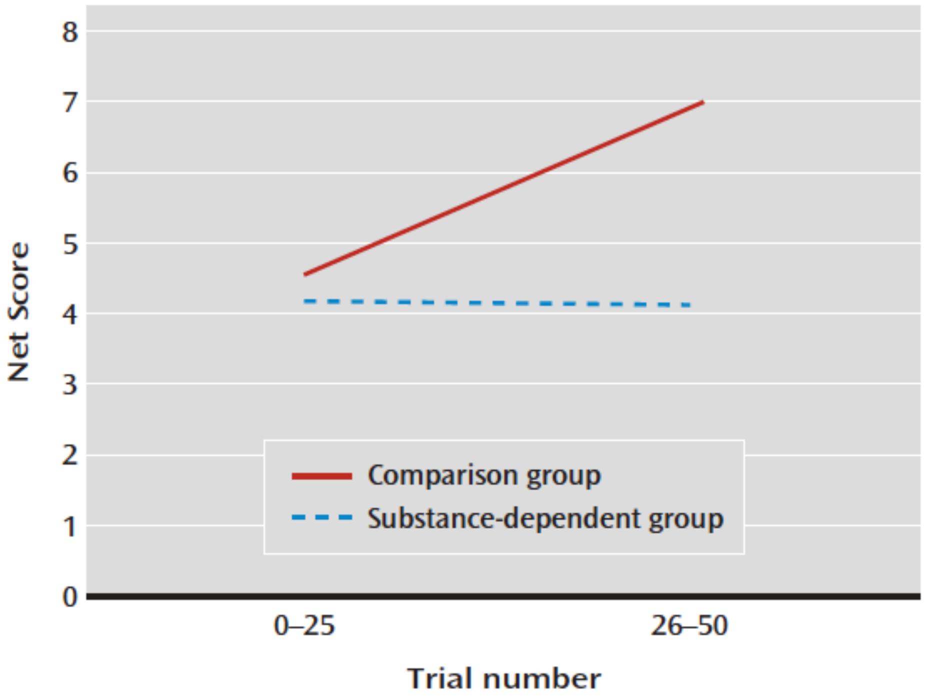
- Uses individually or group identified parameters to generate trial-by-trial values relevant to the stage of decision-making
- Typically: at decision period (D) & after feedback (F) from a choice
- At decision period, weight trials by expected values for the subsequently chosen deck
- At outcome stage, weight trial by difference between received – expected reward (i.e. Prediction Error)
- BOLD = $D_1 * w_1 + F_1 * z_1 + \ldots. + D_n * w_n + F_n * z_n$ + Error

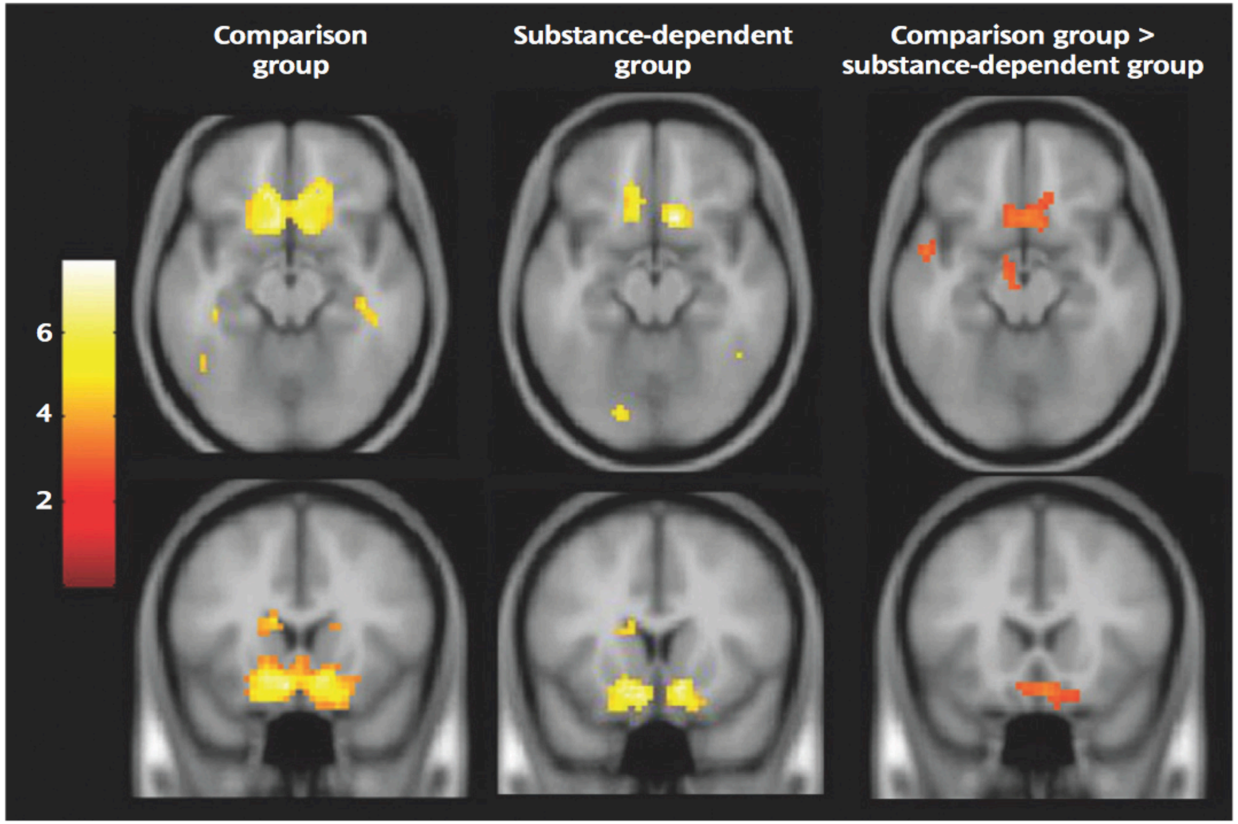


Ahn, et al., 2011

Reinforcement Learning Models in the Brain

FIGURE 1. Decision Making on a Modified Iowa Gambling Task[a]





Tanabe, et al., 2013

Reinforcement Learning Models and the SMH

- They are behavioral models with assumptions about latent processes

  How do these latent processes actually map onto brain function?

- They do not directly incorporate relevant biological signals – e.g. from brain and body

- Don't say much about the SMH this way nor that way
  - Just models IGT as a basic reinforcement learning process
  - From neuroimaging perspective, model-derived regressors tend to correlate with ventral striatum and posterior VMPFC

Reinforcement Learning Models and the SMH

- Incorporating biological signals directly into predictive models may be more insightful
- Correspond more directly to the theoretical processes described in SMH

Questions?

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