



Neural Network Modeling of Developmental and Attentional Biases in Decision Making



Daniel S. Levine, Department of Psychology, University of Texas at Arlington, Arlington, TX 76019-0528, levine@uta.edu

Abstract

Preference decisions between probabilistic alternatives are modeled by a neural network including several brain regions. The network reproduces data whereby small probabilities are most overweighted for affectively rich outcomes. The network also reproduces data whereby framing effects increase when possible zero outcomes are salient. Model equations contain randomly varying parameters to simulate individual differences.

The network classifies options via unequally weighted attributes, with non-numerical attributes emphasized under emotional arousal. Weights emerge in development via a supervised algorithm identifying attributes that most clearly differentiate consequences. The neural transmitter acetylcholine sets the classification criterion. The transmitter dopamine mediates emotional value learning.

Some of this work is published in Levine, *Cognitive Systems Research*, 15-16, 57-72, 2012, and AlQaudi, Levine, and Lewis, *Proceedings of IJCNN 2015*.

Neural Network Structure

The model structure combines roles of several brain regions and parts of existing theories: **adaptive resonance theory** of categorization (Carpenter & Grossberg, 1987); emotion-influenced **selective attention** (Grossberg & Levine, 1975); **gated dipole theory** of affective contrasts (Grossberg & Gutowski, 1987); **fuzzy trace theory** of memory (Reyna & Brainerd, 2008).

Fuzzy trace theory: information is encoded in two ways: verbatim encoding (literal storage of facts) vs. gist encoding (storing intuitive meanings). Gist encoding of probabilities tends toward all-or-none representations of risk. That is, the gist encoding perceives gambles as “certainty,” “no chance,” or “some chance” of a particular gain or loss, largely neglecting precise probabilities of that gain or loss. Hence, gist encoding tends to reduce the relative attractiveness of sure losses and enhance the relative attractiveness of sure gains in comparison with risky alternatives.

Amygdala and orbitofrontal cortex (OFC) are both involved in emotional encoding and are connected by reciprocal pathways. Stimulus-reinforcement associations can be more rapidly learned and reversed by OFC neurons than by amygdala neurons (Rolls, 2006). This suggests a hierarchical relationship: OFC representations of emotional stimuli are more influenced by higher-order cognition than are amygdalar representations.

Adaptive resonance theory (ART) models hierarchical relationships between two layers. Nodes at the “higher” level F2 (OFC here) encode categories of activity patterns at “lower” level F1 (amygdala here). ART includes a “reset” that becomes active when a pattern does not match a previously stored prototype. Matching is across unequally weighted attributes. Reset is identified with anterior cingulate (ACC), which is active in conflict or error detection.

Gated dipole theory: to get OFC reset, each F2 category is coded by a *gated dipole*, an opponent processing network with two channels of opposite meaning (e.g., positive and negative affect, or category activity and inactivity). Habituating neurotransmitters make one channel transiently active (rebound) when activity of the other channel decreases. Rebound requires nonspecific arousal. Amygdala dipoles encode attributes. Affect toward an option’s attribute level depends on counterfactual comparisons with other options.

Action network. Signals carrying information that particular actions have good or bad consequences pass from the amygdala to the action gate in the basal ganglia. Appetitive signals facilitate the direct pathway, which excites the motor cortex. Aversive signals facilitate the indirect pathway, which inhibits the motor cortex. The gate opens when the direct pathway input sufficiently counteracts the indirect pathway input.

Network and Simulations

Two options are represented by here are two inputs A and B to the network. Each run is assumed to represent a different experimental participant. The inputs A and B each have three attributes: *Possibility of gain* (value of 1 or 0); *Possibility of no gain* (value of 1 or 0); *Probability of gain* (continuum of values from 0 to 1). Each attribute is represented by a separate dipole at the amygdala. Each amygdalar dipole includes positive and negative input nodes x_{1i}^+ and x_{1i}^- ; depletable transmitters z_i^+ and z_i^- ; next stage nodes x_{2i}^+ and x_{2i}^- , and output nodes x_{3i}^+ and x_{3i}^- .

There are five categories at the F₂ (OFC) level, each with its own gated dipole. The five categories represent: (1) *sure gain*; (2) *sure non-gain*; (3) *tossup between gain and non-gain*; (4) *almost impossible gain*; (5) *almost certain gain*.

2012 article reproduced data of Rottenstreich and Hsee (2001) on risky choices of kiss versus money. 2015 conference paper reproduced data of Reyna and Brainerd (1991) and of Kühberger and Tanner (2010) on **framing in the Asian Disease Problem: increased framing if nonzero complements removed, decreased or no framing if zero complements removed**.

Gain frame:

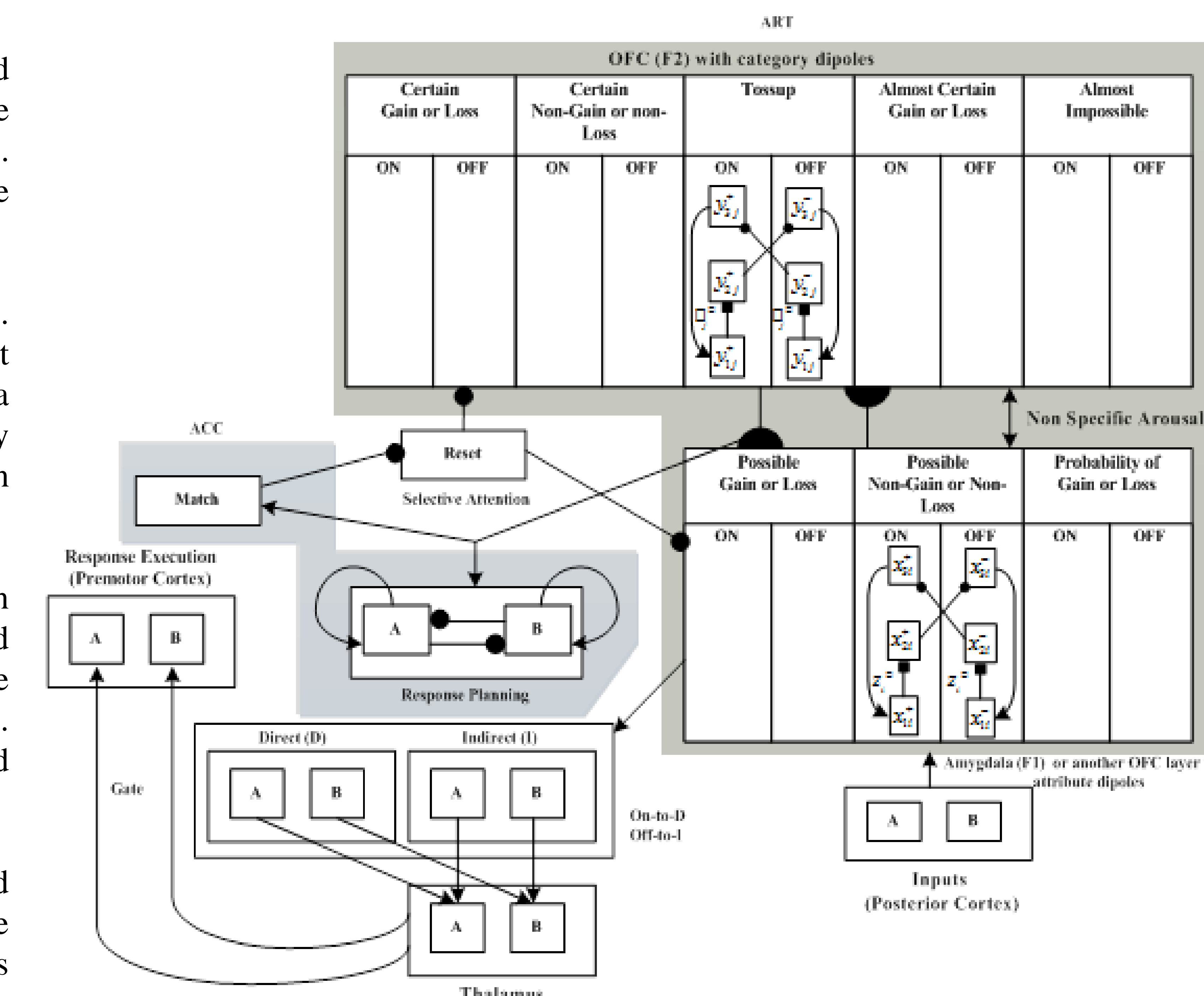
If Program A is adopted, 200 people will be saved.

If Program B is adopted, there is a one-third probability that 600 people will be saved (nonzero complement) and a two-thirds probability that no people will be saved (zero complement).

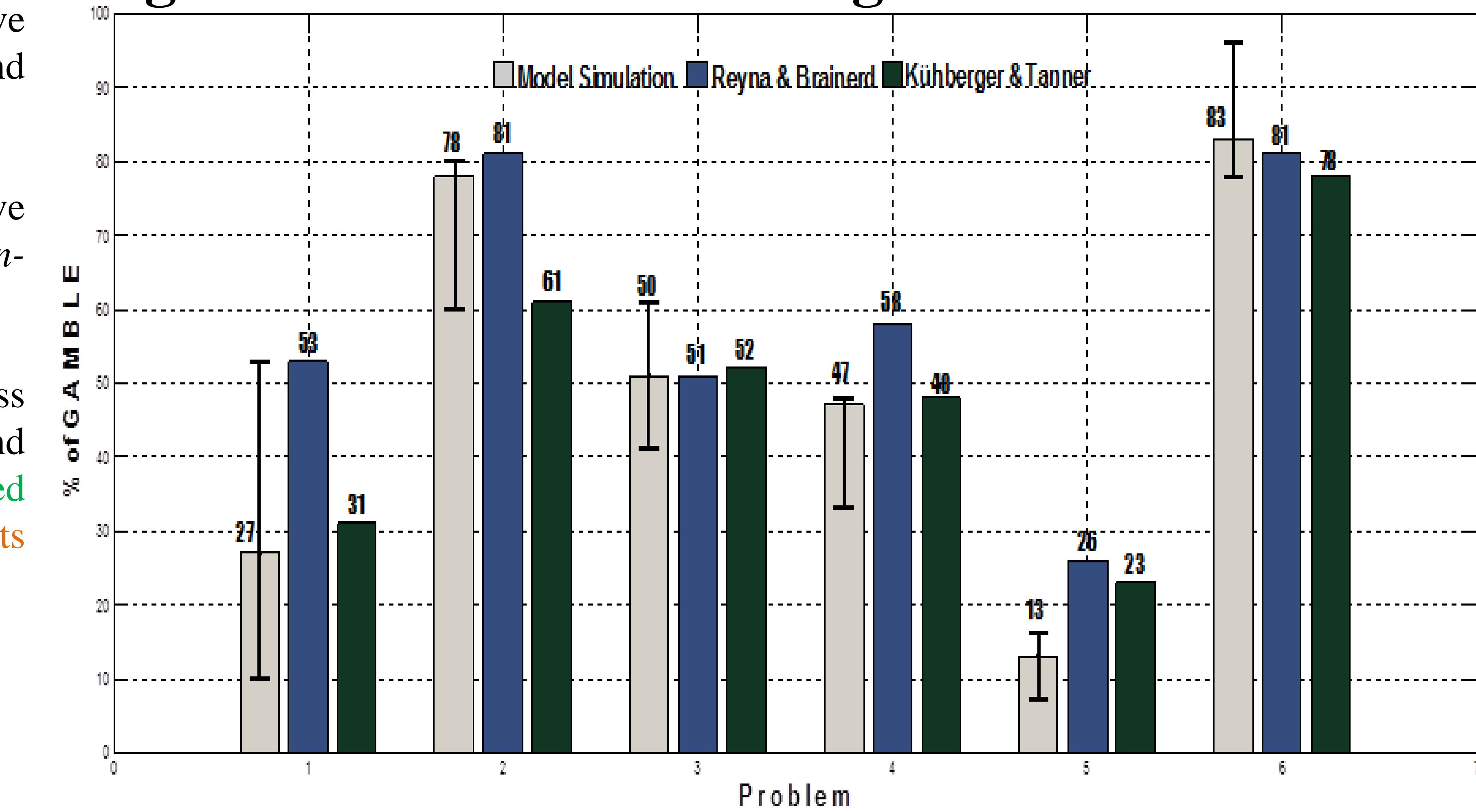
Loss frame:

If Program C is adopted, 400 people will die.

If Program D is adopted, there is a one-third probability that nobody will die (zero complement) and a two-thirds probability that 600 people will die (nonzero complement).



Reyna-Brainerd and Kühberger-Tanner data and simulations. Vertical axis is percentage of choices of the gamble over the sure thing:



How might the gist categories of options be learned over the course of development (speculation):

Hippocampus encodes *episodes* in which a category is presented. This leads to representations of the associations between (linguistic) inputs and their consequences.

Then how do some attributes get selectively emphasized over others?

If change in one attribute leads to a change in consequences, a **dopamine error signal** enhances the weight of that attribute.

That enhanced weight is carried to DLPFC, which enhances **acetylcholine attentional signals** to that attribute – both via septum to hippocampus and via nucleus basalis to cortex.

“Gist” is the original input with the attributes thus attentionally weighted.

(Related models: ARTMAP (Carpenter, Grossberg, & Reynolds, 1991), the supervised version of ART, includes a map field that encodes associations, which could be identified with part of the hippocampus. O’Reilly, Bhattacharyya, Howard, & Ketz (2014) lists roles in episodic-to-semantic memory transition for regions of hippocampus – entorhinal, dentate gyrus, CA3, CA1.)

References

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