UC Merced
Proceedings of the Annual Meeting of the Cognitive Science Society

## Title
Revising the Role of Somatic Markers in the Gambling Task: A Computational Account for Neuropsychological Impairments

## Permalink
https://escholarship.org/uc/item/0p24440h

## Journal
Proceedings of the Annual Meeting of the Cognitive Science Society, 27(27)

## ISSN
1069-7977

## Authors
Fum, Danilo
Stocco, Andrea
Zalla, Tiziana

## Publication Date
2005

Peer reviewed

eScholarship.org Powered by the California Digital Library
University of California

# Revising the Role of Somatic Markers in the Gambling Task: A Computational Account for Neuropsychological Impairments

**Andrea Stocco (stocco@units.it)**
**Danilo Fum (fum@units.it)**
**Tiziana Zalla (zalla@fc.units.it)**
University of Trieste
Piazzale Europa 1, 34127 Trieste, Italy

**Abstract**

In the paper we propose a revision of the Somatic Maker Hypothesis (henceforth SMH: Damasio, 1994). Most of the corroboration for this theory, as well as some contrary results, come from an experimental decision making paradigm known as the Iowa Gambling Task. We analyze the different constituents of the SMH and argue that the discrepancy between the theory predictions and the experiments reported in the literature can be explained by modifying the theory to include a different functional role of somatic markers in high-order cognition. This revised version is in turn related to a different functional interpretation of the orbitofrontal cortex. Within this new framework we describe a detailed computational model that is able to reproduce the original experimental data, and we show that it can also account for the behavior deriving from other neuropsychological impairments.

## Introduction

The Somatic Markers Hypothesis (henceforth SMH: Damasio, 1994) provides a theory of the relations between emotion and cognition. It postulates the existence of functional interactions between bodily states, as represented in somatosensory areas, and high level cognitive processes. This interplay is mediated by the so-called *somatic markers*.

According to the hypothesis, emotions are conceived as immediate bodily reactions to environmental stimuli. Most reactions are preprogrammed in our genetic inheritance, others have to be associatively learned. They are sensed as pleasant or unpleasant through internal representations that are continuously updated in the sensory regions of the brain. These somatic representations are conveyed, through sensory pathways, to a convergence area in the orbitofrontal cortex. Within this region, they are associated with other representations conveying contextual information. In this way, the emotional reactions become somatic markers for the previously encountered stimuli that elicited them.

Once formed, somatic markers may be reactivated when the organism faces situations similar to the ones that induced the markers. The organism is then already pre-alerted and pre-disposed to react properly, and to avoid negative consequences.

### Two Components of the SMH

The SMH may be divided in two conceptually different components. The first is a theory of the nature and origin of emotions. The second is a theory of how emotions affect cognition and drive behavior.

As a theory of the nature of emotions, the SMH is basically a reenactment of the James-Lange theory (James, 1884; Lange, 1885/1912) that considers emotions as reactions to perceived changes in our bodily states. This approach was later criticized by Cannon (1927/1987), who proposed that, on the contrary, emotions anticipate and cause the somatic reactions.

A recent, striking support for the James-Lange view and, consequently, for the SMH comes from neurological studies of interoception—the ability of perceiving one's own internal body states. Individuals vary in their interoceptive capabilities, as well as in their capability to correctly perceive and report their emotions. Functional neuroimaging studies have demonstrated that a region notably associated with interoceptive capabilities is the anterior region of the right insula, that is known to be deeply involved in emotional processing (Critchley et al., 2004).

The overlapping of circuits involved in perception of both visceral states and emotions is exactly what one should expect from a James-Lange perspective (Craig, 2004). On the other hand, these findings are at odds with other theories, because they cannot postulate such a close relation, and common origin, for somatic representations and emotions.

The second component of the SMH concerns the relationship between emotions and cognition, and is less clearly identified and supported. Bechara, Damasio, Tranel, and Damasio (1997) argued that the effect of emotions is implicit: somatic markers affect behavior without people being aware of them.

## The Iowa Gambling Task

Most of the empirical evidence supporting the SMH comes from experiments performed with a paradigm known as the Iowa Gambling Task (Bechara, Damasio, Damasio, & Anderson, 1994; Bechara, Damasio, Tranel, & Damasio, 1997; Bechara, Tranel, & Damasio, 2000). This task was developed to capture, within a laboratory situation, some important aspects of real-life decision making: uncertainty about the future, lack of perfect information, and the trade-off between immediate and postponed rewards.

In the Iowa Gambling Task, participants are asked to repeatedly select a card from an array of four decks, labeled *A*, *B*, *C* and *D*. Unbeknown to them, the game would stop after 100 choices. Each selection always results in an immediate positive outcome. Decks *A* and *B* carry bigger wins, while *C* and *D* lead to smaller monetary rewards.

Unpredictably, however, a win may also be immediately followed by a subsequent negative outcome. These penalties are arranged so that selecting from *A* and *B* ("bad decks") will produce an overall loss of money. Therefore, the advan-

tageous strategy is to select from *C* and *D* ("good decks"), that yield an eventual profit.

Normal participants usually start selecting from the bad decks, but end up performing significantly more selections from the good ones.

Bechara et al. (1997) assessed the participants' knowledge during the task, and showed that the behavioral shifting to the winning strategy starts long before participants could report any verbalizable knowledge about the strategy itself or about the nature of the game they were playing.

An even more striking result came from the analysis of participants' skin conductance responses (SCR)—a common physiological correlate of emotional states (Bradley & Lang, 2002). Bechara et al. (1997) found that skin conductance increased just before a selection from a bad deck was made. These increments were predictive of a subsequent behavioral shift to the winning strategy, but they appeared long before explicit knowledge of the task. The authors claimed that SCR responses were correlates of a somatic marker activation which was driving behavior *before* (and, therefore, *without*) the participants being aware of it.

On the other hand, patients with orbitofrontal lesions do not show any SCR increase while performing the task. Correspondingly, they remain stuck to the bad decks, unable to switch to the good ones. This behavioral result is usually explained as insensitivity to future negative consequences of their actions (Bechara et al., 1994; Bechara, Tranel, & Damasio, 2000) due to an incapability of associating somatic states with the proper stimuli.

### Contrary Evidence

The strong assumption of the implicit nature of emotional impact on cognition has been criticized. One first criticism was methodological. In cognitive psychology, the access to verbalizable knowledge was initially adopted as a reasonable measure of implicitness (e.g., Berry & Broadent, 1984), but later dismissed because of its insufficient sensitivity (Shanks & St. John, 1994). More recent theories of implicit knowledge embrace indirect indicators, like the zero-correlation criterion and the guessing criterion (Dienes & Berry, 1997), or the process dissociation procedure (Jacoby, 1991).

Moreover, verbal self-reports, especially when performed in front of the experimenter, are notoriously subject to systematic distortions due to the social context. Maia and McClelland (2004) showed, by using more sensitive methods, that participants can rely on explicit knowledge of the task since its very beginning.

A similar conclusion was suggested by Tomb, Hauser, Deldin, and Caramazza (2002). They resorted to a modified version of the task, where the most advantageous decks were the ones associated with the highest immediate rewards, despite their larger monetary losses. As a result, participants showed a rational preference for those riskier decks, but preserved their increased physiological responses before selecting from *A* and *B*. Hence, the very same SCR pattern was now in anticipation of the advantageous choices, and could not be explained as an alarm signal that fired in detection of a bad choice. This finding was at odds with the original interpretation, and suggested that participants could decide without being bidden by their own inner physiological reactions.

## An Alternative Account

Fum and Stocco (2004) proposed a different interpretation of the original results from the Gamblig Task. They presented a computational model that could reproduce the original findings of Bechara et al. (1994). The main idea underlying the model was that somatic markers do not play an implicit role in cognition. On the contrary, they are necessary for fully enabling the conscious retrieval of physiological states associated with previous experiences. So, while not being in contrast with the first, the model was at odds with the second component of the SMH.

In this alternative view, the orbitofrontal cortex enables the contextual retrieval and the active maintenance of emotional experiences. Therefore, the role of this region is functionally similar to that played by the remaining areas of prefrontal cortex (Schoenbaum & Setlow, 2001), but specialized on different content. In particular, it is involved in creating associations and retrieving memories about somatic states.

Such traces carry information about one's own subjective evaluation of different options. When this information is made available, individuals may take it into consideration in their decision process. However, they may also choose to ignore it, and voluntarily make decisions that are contrary to their immediate somatic reactions. The latter is probably the case for the participants in Tomb et al. (2002).

On the other hand, individuals with focal damage to the orbitofrontal cortex appear to be unable to properly recollect, and focus their attention on, possible negative events. Their control over behavior is restricted, these patients being completely attracted by the positive outcomes.

Our explanation is also more general than the original one (Bechara et al, 1994). Indeed, it has been recently shown that orbitofrontal patients are not only oblivious of future negative outcomes, but also of possible *alternative* aversive ones, resulting impaired in experiencing regret for decisions they did not made (Camille et al., 2004).

### An Overview of the Model

The model proposed in Fum & Stocco (2004) is functional, rather that structural. That is, it simulates the function of different brain areas instead of reproducing their structural properties. Since we are interested in the specific role of the different circuits, we think that this implementation allows us a more specific control over our predictions. An overview of the different components of the model and of their relations is given in Figure 1.

The core of the model is its declarative memory system that is based on the ACT-R theory (Anderson & Lebiere, 1998). In ACT-R, memory traces are encoded in form of chunks—elementary constituents of the declarative knowledge, made of several individual atomic features. Chunks have an associated value of *activation*, which determines their own availability to retrieval.

Interference stems out as the result of the concurrent activation of chunks competing for retrieval. Since activation is subject to progressive decay, proactive interference decreases over time (Altmann & Gray, 2002).

Chunks are also linked to each other by *associative strengths*, which carry additional activation in presence of the previously co-experienced context, and permit cued re-

trieval. Residual associative activation increases the retrieval probability of the information even when the target itself is no longer accessible. This fact captures implicit memory effects.

More formally, the activation of a chunk $i$ is defined as the log of the probability of that chunk being retrieved. This probability is calculated on the basis of a rational Bayesian estimate of the needed information (Anderson, 1990). Activation is the sum of two components: a base level $B_i$, and a contextual constituent $C_i$.

The base component only depends on the past history of $i$, and, in particular, on how frequently and recently $i$ has been retrieved or recoded. In our implementation, it is computed as in Altmann & Trafton (2002):

$$B_i = \ln\left(\frac{n_i}{\sqrt{T_i}}\right)$$

where $n_i$ is the number of times the trace was accessed, and $T_i$ is time elapsed since the its first encoding.

The contextual component is the sum of the associative strengths $S_{ji}$ spreading from $j$, that is supposed to be the current focus of attention, to the chunk $i$. It captures the increase of probability of retrieving $i$ when $j$ is attended. These strengths are weighted by a parameter, $W$, that establishes the amount of attentional resources. As a result, $W$ permits the sustained activation of elements beyond the interference coming from intrusive traces. This enables working memory, goal maintenance over time, and, in turn, voluntary control (Anderson, Reder, & Lebiere, 1996; Altmann & Trafton, 2002).

## Outcome Evaluation

In the model, whenever an outcome following a selection is encoded, it produces an emotional impact $V$. This evaluation reflects the immediate contribution of several brain structures that are known to be sensitive to the absolute magnitude of a reward. In particular we think that this reflects the contribution of the amygdala.

Zalla et al. (2000) provided functional evidence that the human amygdala differentially responds to changes in magnitude of positive or negative reinforcement. They used lexical stimuli to convey positive "win" or negative "lose" feedback. The frequency of positive and negative trials was parametrically varied independently from the subject's actual performance and unbeknownst to them. The results showed that

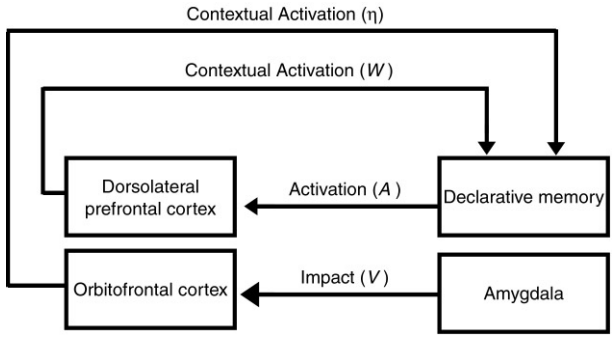


Figure 1: Architecture of Fum & Stocco's (2004) model of the Iowa Gambling Task

.

the parametric increase of winning was associated with left amygdala activation whereas the parametric increase of losing was associated with right amygdala activation.

In general, the emotional impact may depend on different attributes of the stimulus. However, in the case of monetary values, it is obviously proportional to its numerical magnitude. Large numerical values are known to be processed by a specialized circuit for approximate quantities, and their perceived magnitude is best captured by a logarithmic scale representation (Feigenson, Dehaene, & Spelke, 2004). In our case, the emotional impact $V_O$ of an outcome $O$ was implemented as:

$$V_O = \frac{\ln(|O|)}{\ln(|O_{max}|)}$$

where $O_{max}$ is the maximum absolute value amongst the possible results, needed to return a normalized value for $V_O$. Since humans are more sensible to losses than gains, and wins are more frequent than losses, positive outcomes are also multiplied by a discounting factor. The final result of this processing is a representantion of the abstract rewarding value of a stimulus, which we suppose to be held in the orbitofronal cortex. Brain imaging studies indeed proved that the activation of this region is proportional to the magnitude of monetary amount used in other experimental paradigms (O'Doherty, Kringelbach, Rolls, Hornak, & Andrews, 2001).

In the model, the emotional impact of an outcome is used to create and reinforce further associative strengths between the trace representing the outcome and that representing the deck it came from. A similar functional role for the orbitofrontal region had already been advocated by Rolls (2000).

This new associative strength is computed as $\eta V_O$, and adds up to the original, frequency-based component of contextual activation. Here, $\eta$ is a parameter reflecting the amount of contribution of orbitofrontal cortex in creating associations.

As a result, the overall contextual activation $\bar{S}_{D,O}$ between an outcome $O$ and the deck $D$ which it derived from is calculated as follows:

$$\bar{S}_{DO} = WS_{DO} + \eta V_O$$

where $S_{DO}$ is the usual associative strength, computed over a frequency-base estimate as in Anderson & Lebiere (1998).

Although both the dorsolateral and the orbitofrontal regions perform similar activities, the use of two different parameter ($W$ and $\eta$) reflects their different biological properties. These two regions receive connections from different areas of the brain, making the orbitofrontal cortex more suitable for processing information about emotional events (Mesulam, 2002).

## Deck Selection

When required to choose a card, the model evaluates the decks by sampling its own memory for their previous outcomes, and retrieving the most active one. Since outcomes are associatively linked to the deck they come from, each deck cues its own experienced results. After each of the decks has been evaluated, the model chooses the deck that is associated with the most favorable value of $V_O$. Outcome sampling

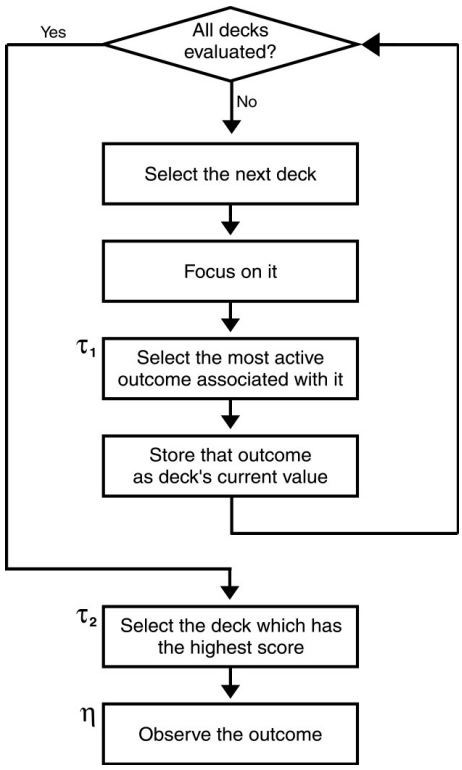


Figure 2: Flow chart of the deck selection process in Fum & Stocco's (2004) model of the Iowa Gambling Task. Greek letters refer to model parameters.

and deck selection are ruled by two Boltzmann equations regulated by the two temperatures $\tau_1$ and $\tau_2$. The process of deck evaluation is represented in Figure 2.

In short, normal participants could correctly recall, and therefore evaluate, the risks following a bad deck choice. When sampling for negative consequences, their retrieval process is not undermined by interference from other material, and has the opportunity to take into consideration a broader range of possible results.

On the contrary, orbitofrontal patients cannot take advantage of the additive contextual activation that flows from the associative paths created by the orbitofrontal cortex. This fact makes them prone to perseverations over the intruding positive outcomes, and makes them less aware of the possible aversive upshots.

It may be argued that, contrary to our hypothesis, a task as simple and repetitive as the Iowa Gambling Task could be performed procedurally, with the action of choosing from the good decks being routinized.

However, Stout, Rodawalt, & Siemers (2001) have shown that patients suffering from Parkinson's disease, that disrupts the capability of acquiring procedural knowledge (Knowlton, Mangels, & Squire, 1996), are not impaired in the Iowa Gambling Task.

## Simulations

Fum & Stocco (2004) showed that the model could reproduce the performance of normal participants and patients in the Gambling Task. They fitted one parameter only ($\eta = 2.0$) in the normal version of their model, and set it to zero to simulate a lesion in the orbitofrontal cortex. The basic findings of the simulations are summarized in Figure 3.

In this work, we intend to further test the robustness of our model by providing simulations of two other neuropsychological findings. One of them, in particular, is critical since it seems to support the original strong hypothesis (Bechara et al., 1997) about the implicit nature of somatic markers in decision making.

It should be noted that, in our simulations, we did not fit any parameter of the original model. We simply performed a virtual impairment of the component corrisponding to the function of the region which was damaged in patients.

### Simulating a Lesion in the Amygdala

Bechara, Damasio, Damasio, & Lee (1999) found that patients with a bilateral damage to the amygdala were also impaired in the Iowa Gambling Task. These patients exhibited a flat pattern of SCR responses similar to orbitofrontal patients. However, did not perseverate in selecting the bad cards, showing only a slight preference for them.

In our model, a damage to the amygdala may be simulated by damaging the early processing stage that produces the emotional impact $V$. We obtained this by making the $V_O$ function return a value of zero for each *negative* outcome, independent of its numerical magnitude (that is indeed preserved in encoding). Consequently, the orbitofrontal module cannot distribute additional contextual association on encoded losses. Since positive values remain easier to retrieve, the model maintains a slight bias towards disadvantageous choices.

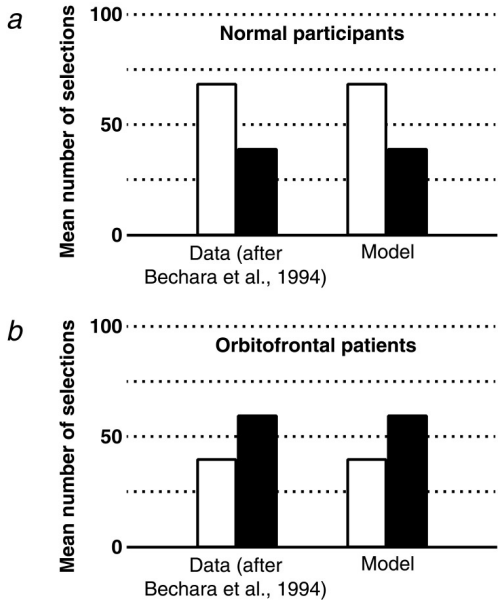


Figure 3: Mean number of selections from good (white bars) and bad decks (black bars). Results from model's simulations are compared with the original data collected from normal participants (*a*), and from orbitofrontal patients (*b*).

We had the model run 1,000 times in this conditions, then we averaged the data for good and bad deck selections. The results are reported in Figure 4*a*, together with experimental data derived from Bechara et al. (1999). As could be expected, the model exhibits the same results obtained from the patients.

### Simulating a Working Memory Impairment

An important indirect evidence in support of the implicit role of emotion in driving cognition come from an experiment by Bechara, Tranel, Damasio, & Anderson (1998). They contrasted performance of two groups of frontal patients. One group was composed by individuals with focal lesions to the dorsolateral prefrontal cortex (DLPFC), while the other comprised only patients with an injury in the ventromedial part of prefrontal cortex, having the dorsolateral region spared. Both groups were tested on two paradigms: a working memory task and the Gambling Task.

Results evidenced a double dissociation, with DLPFC patients selectively impaired in the working memory task but not with the Gambling Task, while the others exhibited the opposite pattern. A similar dissociation was taken as evidence that somatic markers drive behavior regardless of the support of higher-level cognitive processes, which are usually compromised by DLPFC lesions. Indeed, the authors claimed that working memory can be dissociated from decision making *tout court*.

This finding is apparently in contrast with our account, that claims the existence of a collaborative integration of two processes, with maintenance of information in working memory resulting from two sources of contextual activation. However, we will argue and illustrate by means of simulations, that our account indeed *predicts* such an apparent dissociation.

In our model, a damage closely similar to a dorsolateral prefrontal lesion in humans can be simulated by lowering or zeroing the *W* parameter. As previously discussed, this parameter regulates selective attention, and disrupts the contextual spreading of activation to memory traces.

A model with an abnormally lower *W* parameter exhibits a behavior that closely resembles the frontal syndrome. Such a model is necessarily impaired in working memory tasks, since the *W* parameter's most important function is to preserve attended material from interference, resulting in functional sustenance of information. Working memory disorders are amongst the most important signatures of the dysexecutive syndrome (Baddeley, 1986).

Therefore, we lowered the *W* parameter in our model, setting it to 1.0 (half of the original value), and then had it perform the Iowa Gambling Task. Again, we run the model 1,000 times and averaged the choices from good and bad decks. The results of our simulations are presented in Figure 4*b*, together with the original data from Bechara et al. (1998).

Surprisingly, the model's performance was not affected by our manipulation. We repeated our simulations by setting *W* to 1.5 and 0.5, and found the same qualitative pattern of results, with the model correctly preferring the good decks.

The rationale behind this results is the following. Lesioning the $\eta$ parameter causes the immediate rewards to become more salient because of their greatest frequency, that is exalted by the contextual activation. On the contrary, reducing the *W* parameter increases the interference of past experiences only over the most frequent (i.e., positive only) outcomes. But the negative ones, still actively sustained by the $\eta$ parameter, become more distinguished, and benefit from a reduced interference from the most frequent ones. As our simulations clearly demonstrate, this mechanisms is sufficient to allow dysexecutive patients outperform orbitofrontal ones in the limited domain of the Iowa Gambling Task.

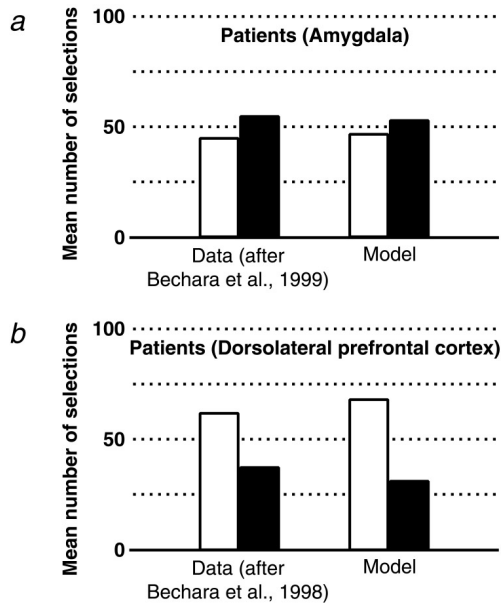


Figure 4: Mean number of selections from good (white bars) and bad decks (black bars). Model's simulations are shown together with performance of patients with a lesion in the amygdala (*a*, after Bechara et al., 1999) and with a lesion in the dorsolateral prefrontal cortex (*b*, after Bechara et al., 1998).

## Conclusions

In this work we provided further neuropsychological support for the Fum & Stocco (2004) model by testing its predictions over two new datasets from patients with brain damages. The model could simulate the performance in the Iowa Gambling Task after a bilateral lesion in the amygdala and after a lesion in the dorsolateral prefrontal cortex. The success in capturing the impaired performance of different classes of patients constitutes a confirmation for the neuropsychological grounding of the model itself, corroborating the hypothesized functional role that different regions play in the circuits connecting emotion and cognition.

Our simulations provide support also for the key tenet that inspired the model, i.e. the fact somatic markers do not need to perform an unconscious evaluation of long-term strategies. On the contrary, we think that their most important effect is to facilitate the retrieval and overt evalution of the possible, emotionally significant, consequences. In particular, we proved that the double dissociation reported in Bechara et al. (1998), that could be taken as evidence againts our claims, is indeed a quantitative prediction of our account.

In fact, one of the most important advantages of the computational models is that they allow for a finer grained analysis of behavior, and that they can account for more subtle interactions between processes than those allowed by a coarse-grained identification of dissociations.

## References


Altmann, E. M., & Gray, W. D. (2002). Forgetting to remember: The functional relationship of decay and interference. *Psychological Science*, *13*, 27–33.

Altmann, E. M., & Trafton, J. G. (2002). Memory for goals: An activation-based model. *Cognitive Science*, *26*, 39–83.

Anderson, J. R. (1990). *The adaptive character of thought*. Mahwah, NJ: Lawrence Erlbaum.

Anderson, J. R., & Lebiere, C. (1998). *The atomic components of thought*. Mahwah, NJ: Lawrence Erlbaum.

Anderson, J. R., Reder, L. M., & Lebiere, C. (1996). Working memory: Activation limitations on retrieval. *Cognitive Psychology*, *30*, 221–256.

Baddeley, A. D. (1986). *Working memory*. Oxford, UK: Clarendon Press.

Bechara, A., Tranel, D., & Damasio, H., (2000). Characterization of the decision-making deficit of patients with ventromedial prefrontal cortex lesions. *Brain*, *123*, 2189–2202.

Bechara, A., Damasio, A. R., Damasio, H., & Anderson, S. W. (1994). Insensitivity to future consequences following damage to human prefrontal cortex. *Cognition*, *50*, 7–15.

Bechara, A., Damasio, H., Damasio, A. R., & Lee, G. P (1999). Different contributions of the human amygdala and ventromedial prefrontal cortex to decision-making. *Journal of Neuroscience*, *19*, 5473–5481.

Bechara, A., Damasio, H., Tranel, D., & Damasio, A. R. (1997). Deciding advantageously before knowing the advantageous strategy. *Science*, *275*, 1293–1295.

Bechara, A., Damasio, H., Tranel, D., & Anderson, S. W. (1998). Dissociation of working memory from decision making within the human prefrontal cortex. *Journal of Neuroscience*, *18*, 428–437

Berry, D. C., & Broadbent, D. E. (1984) On the relationship between taks performance and associated verbalizable knowledge. *Quarterly Journal of Experimental Psychology*, *36A*, 209–231.

Bradley, M. M., & Lang, P. J. (2002). Measuring emotion: Behavior, feeling, and physiology. In R. D. Lane & L. Nadel (Eds.), *Cognitive neuroscience of emotion*. Oxford, UK: Oxford University Press.

Camille, N., Coricelli, G., Sallet, J., Pradat-Diehl, P., Duhamel, J.-R., & Sirigu, A. (2004). The involvement of the orbitofrontal cortex in the experience of regret. *Science*, *204*, 1167–1170.

Cannon, W. B. (1927/1987). The James–Lange theory of emotions: A critical examination and an alternative theory. *American Journal of Psychology*, *100*, 567-586

Craig, A. D. (2004). Human feelings: Why are some more aware than others? *Trends in Cognitive Sciences*, *8*, 239–241.

Critchley, H. D., Wiens, S., Rotshtein, P., Öhman, A., & Dolan, R. J. (2004). Neural systems supporting interoceptive awareness. *Nature Neuroscience*, *7*, 189–195.

Damasio, A. R. (1994). *Descartes' Error: Emotion, Reason, and the Human Brain*. New York, NY: Gosset/Putnam Press.

Dienes, Z., & Berry, D. C. (1997). Implicit learning: Below the subjective threshold. *Psychonomic Bulletin and Review*, *4*, 3–23.

Feigenson, L., Dehaene, S., & Spelke, E. S. (2004). Core systems of number. *Trends in Cognitive Sciences*, *8*, 307–314.

Fum, D., & Stocco, A. (2004). Memory, emotion, and rationality: An ACT-R interpretation for Gambling Task results. In C. D. Schunn, M. C. Lovett, C. Lebiere & P. Munro (Eds.), *Proceedings of the Sixth International Conference on Cognitive Modelling*. Mahwah, NJ: Lawrence Erlbaum.

Jacoby, L. L. (1991). A process dissociation framework: Separating automatic from intentional uses of memory. *Journal of Memory & Language*, *30*, 513–541.

James, W. (1884). What is an emotion? *Mind*, *9*, 188–205.

Knowlton, B. J., Mangels, J. A., & Squire, L. R. (1996). A neostriatal habit learning system in humans. *Science*, *273*, 1399–1402.

Lange, C. G. (1885/1912). The mechanism of the emotions. In B. Rand (Ed.), *The Classical Psychologists*. Boston: Houghton Mifflin.

Maia, T. V., & McClelland, J. L. (2004). A reexamination of the evidence for the somatic marker hypothesis: What participants really know in the Iowa gambling task. *Proceedings of the National Academy of Sciences*, *101*, 16075-16080.

Mesulam, M. M. (2002). The human frontal lobes: Trascending the default mode through contingent encoding. In D. T. Stuss & R. T. Knight (Eds.), *Principles of prefrontal lobe function*. Oxford, UK: Oxford University Press.

O'Doherty, J., Kringelbach, M. L., Rolls, E. T., Hornak, J., & Andrews, C. (2001). Abstract reward and punishment representations in the human orbitofrontal cortex. *Nature Neuroscience*, *4*, 95–102.

Rolls, E. T. (2000). *The brain and emotion*. Oxford, UK: Oxford University Press.

Schoenbaum, G., & Setlow, B. (2001). Integrating orbitofrontal cortex into prefrontal theory: Common processing themes across species and subdivisions. *Learning & Memory*, *8*, 134-147.

Shanks, D. R., & St.John, M. F. (1994). Characteristics of dissociable human learning systems. *Behavioral and Brain Sciences*, *17*, 367–447.

Stout, J. C., Rodawalt, W. C., & Siemers, E. R. (2001). Risky decision making in Huntington's disease. *Journal of the International Neuropsychological Society*, *7*, 92-101.

Tomb, I., Hauser, M., Deldin, P., & Caramazza, A. (2002). Do somatic markers mediate decisions on the gambling task? *Nature Neuroscience*, *5*, 1103–1104.

Zalla, T., Koechlin, E., Pietrini, P., Basso, G., Aquino, P., Sirigu, A., & Grafman, J. (2000). Differential amygdala responses to winning and losing: A functional magnetic resonance study in humans. *European Journal of Neuroscience*, *12*, 1764–1770.