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Memory, Emotion, and Rationality: An ACT-R interpretation for Gambling Task results

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Abstract

A new interpretation for the results of several experiments carried out using the Gambling Task paradigm is presented that differs from that put forward by Damasio (1994). While Damasio grounds his analysis on the somatic markers hypothesis, we propose a perspective based on the functional integration of emotion with memory that is sympathetic with the view of the prefrontal cortex endorsed by Rolls (1999, 2000). Our interpretation is supported by the development of a computational model implemented in the ACT-R cognitive architecture. The model highlights some limitations of the architecture that arise when dealing with situations charged with emotional significance. In particular, the model—when developed according to the standard ACT-R theory—produces a perseverating behavior that replicates the performance exhibited by participants with lesions in the orbitofrontal cortex. An adjustment to the ACT-R equations describing the activation of elements in declarative memory is suggested that allows modeling the performance of both normal controls and orbitofrontal patients.

Introduction

The idea that emotions could play a critical role in some cognitive processes—like reasoning, planning, and decision making—that were traditionally considered as "cold" has been put forward in the last years by several researchers (LeDoux, 1996; Panksepp, 1998; Rolls, 1999, 2000). The arousal of interest for these topics should be possibly credited to Antonio Damasio who, in his *Descartes' error* (Damasio, 1994), strongly emphasized the link between emotion and rational action, and highlighted the functional role played by the former in determining the latter.

According to Damasio, completely rational decisions (i.e., emotionless decisions based on cost-benefit analysis, and on the maximization of subjective utility) are insufficient when dealing with personal and social affairs. Making a good decision in such domains means in fact to select an option that will prove advantageous for the survival of the organism, and for the quality of such a survival. Making a good decision often means to decide fast, because the environment asks for quick choices. In the best cases, deciding on a purely rational basis will require too much time. In the worst cases, it will lead to bad choices, or to the impossibility of making any choice, because the cognitive resources brought to bear in the decision process do not allow to take into account all the information needed for an optimal conclusion.

While authors who have acknowledged these limitations have spoken of *bounded rationality* (Simon, 1978), *heuristics and biases* (Kahneman, Slovic, & Tversky, 1982) or *adaptive decision making* (Payne, Bettman, & Johnson, 1993), and tried to solve the problem of sub-optimal reasoning within a purely cognitive approach, Damasio takes a different path.

His basic idea is that, in considering the alternatives for a course of action, we base our decision on the representation of similar events experienced in the past. These memories, however, are not neutral entities but are charged with emotional dispositions resulting from the positive or negative outcomes they have been associated with. In contrast with what happens in more abstract domains, when the topic at issue has personal significance, our reaction is not purely intellectual but involves a physical component. According to Damasio, affective memories generate *somatic markers*, bodily sensations activated by situations analogous to the current one.

Somatic marker are thus triggers of (positive and negative) emotions that have been associated, through learning, with the outcomes obtained in previously experienced situations. The markers make the decision process more precise and efficient. For instance, when a negative somatic marker is associated with a particular outcome, it plays the role of an alarm signal. By making the reasoner dump immediately the option associated with the outcome, it allows the discarding of unacceptable choices to promote a rational, in-depth analysis of more promising courses of action.

The acquisition of the somatic markers is mediated, according to Damasio, by the orbitofrontal cortex (henceforth OFC). This area plays, in fact, a critical role "in regulating our abilities to inhibit, evaluate, and act on social and emotional information" (Gazzaniga, Ivry, & Mangun, p. 547). It receives signals from all the sensitive regions of the brain, and it is directly involved in every chemical or motor response. A damage in this area causes an impairment in personal and social decisions in spite of the fact that intellectual capacities are generally preserved. In particular, patients with lesions in the OFC seem oblivious to the consequences of their actions, and guided only by immediate prospects. They are generally unable to learn from their mistakes, and persist in making decisions that lead to negative conseguences. According to Damasio, OFC lesions can damage the emotional processing of affective memories, disrupt the delivery of somatic markers and, consequently, worsen the quality of decision making.

The best empirical corroboration for the somatic markers hypothesis comes from a group of experiments (Bechara, Damasio, Damasio, & Anderson, 1994; Bechara, Damasio, Damasio, & Lee, 1999; Bechara, Damasio, Tranel, & Damasio, 1997; Bechara, Tranel, & Damasio, 2000) carried out with the so-called Gambling Task (henceforth, GT) paradigm. This task simulates some of the features of daily-life deci-

sions, that occur in real time, have personal consequences, are based on uncertain premises, and could end up with rewards or punishments. While normal participants in this task are able to show an adaptive behavior, patients with OFC lesions repeatedly engage in decisions leading to negative consequences. The difference in performance between these two groups, according to Damasio, makes a case for the role of somatic markers in shaping rational behavior.

In the paper we offer an interpretation for the GT findings that avoids the somatic markers hypothesis and that is based on the functional integration of emotion with memory. Our interpretation is supported by the development of a computational model implemented in the ACT-R (Anderson & Lebiere, 1998) cognitive architecture. The following section illustrates in detail the GT paradigm, presents the data to be modeled, and contrasts Damasio's interpretation with other positions. The essential assumptions of our approach are presented in section 3, where the main features of the model are also described. The model highlights some limitations of the ACT-R architecture that arise when dealing with situations charged with emotional significance. Section 4 shows how the model—when developed according to the standard theory—produces a perseverating behavior that replicates the performance exhibited by participants with lesions in the OFC. An adjustment to the ACT-R equations describing the activation of elements in declarative memory is suggested that allows modeling the performance of both normal controls and OFC patients. The final section illustrates the significance of our work for the ACT-R architecture and for neuropsychological data modeling.

The Gambling Task

The paradigm

In a typical experiment with the GT paradigm, participants are put in front of four decks of cards, labeled A, B, C, and D, respectively. Each deck is composed of 40 cards and, because the backs of the cards all look the same, the decks are distinguishable only by their position. Participants are initially given an amount of play money, and they are said their goal is to maximize the money they will hold at the end of the game. Participants are then asked to choose one card at a time from any of the four decks until told to stop. They are free to switch from any deck to another, but they are not informed about how many selections they have to make. Unbeknown to participants, the experiment is stopped after 100 trials.

Once a card is chosen, it is turned over and it allows participants to gain some money whose amount varies with the deck. After turning some cards, however, the participant is both given money *and* asked to pay a penalty whose amount is revealed only after the card is turned, and that also varies with the deck—and with the card position within the deck—according to the fixed schedule reported in Figure 1.

Turning any card from deck A or deck B yields \$100 to the participant; turning any card from deck C or deck D yields \$50. However, the net yielding of each deck varies because the penalties also vary with the decks. In particular, decks A and B assure high wins but are also associated with higher losses (up to \$1250); decks C and D are associated with both small wins and small penalties (for a maximum of \$100). The payoff sequence, however, is unknown to the participants, and is arranged in a manner that makes it difficult to learn the long term effects of each deck.

For instance, after turning ten cards from deck A, the participants earn \$1000 but they also receive five punishments—ranging from \$150 to \$350—that will bring the total cost to \$1250, resulting in a net loss of \$250. Ten cards from deck B will gain \$1000, but they will produce a penalty of \$1250. Decks A and B are thus equivalent in terms of the overall net loss over trials. The difference is that in deck A the punishment is more frequent but of smaller magnitude, whereas in deck B the punishment is less frequent but of higher magnitude. On the other hand, after turning ten cards from decks C or D, participants earn \$500 but the total of their punishments is only \$250 (ranging from \$25 to \$75 in deck C, and resulting from a single \$250 loss in deck D, respectively), bringing a net gain of \$250. Again, one deck (C) leads to more frequent and lower magnitude punishments while the other (D) produces the opposite pattern.

Taking the overall effects into account, however, it is clear that decks A and B are disadvantageous, and that choosing from them will lead in the long run to financial disaster. On the other hand, decks C and D are advantageous because they eventually result in an overall gain.

The results

Experiments carried out with the GT paradigm produce findings that exhibit essentially the same pattern. As the task progresses, normal participants gradually concentrate their choices on the good decks avoiding the bad ones. Patients with OFC lesions, on the other hand, fail to demonstrate this shift, and persevere in choosing from riskier decks even though these decks lead to negative consequences. Card selection profiles reveal that controls initially sample all decks and make quite a few selections from the bad ones. Eventually, however, they make more and more selections from good decks, with only occasional returns to the alternative options. OFC patients behave like normal controls only in the very first trials while in the long run, even though they make occasional selections from good decks, they stick more frequently and more systematically to bad choices.

Figure 2 presents the number of cards selected from each deck by normal controls and OFC patients in the experiment carried out by Bechara et al. (1994). Statistical analysis revealed that the number of cards selected by normal controls from bad decks was significantly less than the number of cards selected by OFC patients from the same decks. On the contrary, the cards selected from good decks by normals were significantly more numerous than those selected by patients.

Normal controls and OFC patients differed also in the pattern of results shown at the SCR (skin conductance response), an effect mediated by the autonomic nervous system that is used as an index of somatic state activation. On turning over a card, both groups displayed a transient increase in SCR, and hence an autonomic response to reward and penalty. After encountering a few losses (usually by card 10), however, normal participants began to generate anticipatory SCR to bad decks: i.e., their SCR showed a peak immediately before choosing a card from these decks. None of the OFC patients, however, revealed this anticipatory reaction, their only SCR responses being exclusively reactive.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40
A (+100)			-150		-300		-200		-250	-350		-350		-250	-200		-300	-150			-300		-350		-200	-250	-150			-350	-200	-250				-150	-200		
B (+100)									-1250					-1250							-1250										-1250								
C (+50)			-50		-50		-50		-50	-50		-25	-75				-25	-75		-50			-50	-25	-50			-75	-50				-25	-25		-75		-50	-75
D (+50)										-250										-250									-250						-250					

Figure 1: The payoff schedule used by Bechara et al. (1994).

The interpretations

According to Damasio, the anticipatory SCRs show evidence for a process of nonconscious signaling (the somatic markers) produced by traces of previous experiences, and their associated emotional states. Damages to OFC act by precluding the access to these individual experiences and, consequently, by disabling the possibility for emotions to bias the decision making process. The performance offered by OFC patients, with their repeated engagement in decisions leading to negative consequences, emphasizes thus the role of somatic markers as mediators for the influence of emotions on cognition.

This claim has been questioned by Tomb, Hauser, Deldin, & Caramazza (2002) who observed how GT findings could be explained by simply sticking to the rewards and punishments schedule. Because, on the average, the amount of money that is both win and lost is greater for the disadvantageous than for the advantageous decks, the anticipatory SCRs produced by normal participants could be due to the higher activation aroused by the former. The authors built an experimental situation in which the *advantageous* decks were the ones associated with higher rewards and higher punishments. (Normal) participants in their experiment not only picked more cards but also showed higher anticipatory SCRs for these decks. Although this result is not necessary incompatible with Damasio's hypothesis (that posits the existence of both negative and positive somatic markers) it could be reconciled with it only by assuming that the modified task produced an inversion of the marker signal.

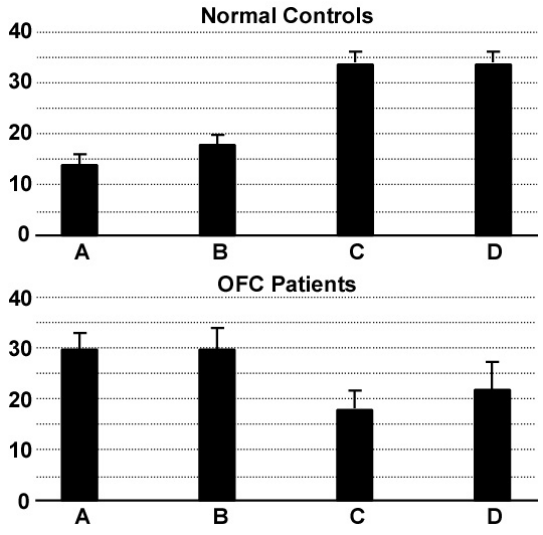

Figure 2: Number of cards chosen from the different decks (after Bechara et al., 1994).

A different perspective has been put forward by Rolls (1999, 2000). According to his point of view, the OFC is critical for a rapid, on-line evaluation of the reinforcement properties of a stimulus or of the outcome associated with a response. The point is that these properties may change as we interact with the environment, and therefore a correction of the inappropriate associations may be required. Rolls (2000, p. 284) noticed that humans with frontal lobe damage can show impairment in a number of tasks in which an alteration of behavior is required as a response to a change in environmental reinforcement strategies. For example, in the Wisconsin Card Sorting Task frontal patients have difficulty in shifting to a second sorting principle when required to do so. In stylus maze tasks it is hard for them to change direction when a sound indicates that the correct path has been left. In the same vein, it is tough for patients with OFC damage to move from disadvantageous to advantageous decks.

In summary, the idea that memories charged with emotional associations could help in guiding our actions is uncontroversial, and equally well accepted is the idea that patients with lesions in the OFC, who generally experience emotional problems, show an impairment in their decision making. The fundamental problem, however, consists in determining whether their defective behavior depends on the incapacity to link a stimulus or an action to their reinforcing properties (Rolls' point of view), or to the incapacity to generate adequate somatic markers (Damasio's position).

An Alternative Approach

The interpretation we suggest for the results of the GT deals with the problem of emotion within the general framework of cognition. It avoids taking into account the concept of somatic markers, and proposes instead a functional integration of emotion with memory.

We start by the rather uncontentious assumption that participants, following the instructions they have received, try to pursue the options that will maximize their gains and minimize their losses. In doing so, they rely on their past experiences to inform their future choices. From this point of view, the GT could be considered as a memory-based task: when

participants figure out which card to pick up, they try to remember the typical outcomes associated with each deck, and restrict their choices to the most promising alternatives.

The details of this process are specified in a computational model framed in the ACT-R cognitive architecture. ACT-R (Anderson & Lebiere, 1998) is grounded on the idea that cognition depends on the interaction between two knowledge sources, i.e, declarative knowledge—expressed through *chunks*, frame-like structures composed of labeled slots with their associated filler values—and procedural knowledge—represented by *productions*, rule-like elements that subordinate the execution of their actions to the existence of particular conditions represented through declarative chunks. Productions retrieve and transform declarative knowledge.

Another assumption is that the kind of memory that plays a critical role in the GT is the declarative one. We believe that the performance of normal participants in the GT experiments, who gradually shift their choices from disadvantageous to more advantageous decks, reflects a type of learning that could not be assimilated to the acquisition of more effective procedural strategies, but that is grounded on the memory of previous events, i.e. on remembering the outcomes associated with the various decks.

The reasons why we think it would be unacceptable to model the GT in ACT-R by relying mainly on the procedural knowledge are essentially two. First, as reported by Bechara et al. (1997), all normal participants—and no orbitofrontal patient—are able to reach, approximately at the middle of the experiment, a phase in which they begin to express the "hunch" that decks A and B are riskier than C and D, and most of them are able to explicitly verbalize, before the end, why it is so. The only explicit knowledge in ACT-R is that represented through declarative chunks that can be retrieved and inspected. Procedural knowledge (and the subsymbolic processes that control the retrieval of declarative chunks) are not accessible to consciousness. The fact that participants are able to verbalize the reasons underlying their choices constitutes therefore a case for the declarative nature of the elements their behavior is based upon.

Second, ACT-R assumes that productions are implemented, at the neural level, by basal ganglia and associated connections. The quality of the performance obtained in pure implicit learning experiments, like those employing the serial reaction time paradigm, is correlated with the integrity of these structures. Patients with damages at the basal ganglia, e.g., suffering from Parkinson's disease, fail in these as well as in other tasks identified as "habit learning" (Knowlton, Mangels & Squire, 1996). Apparently, however, these patients behave like normal controls in the GT (Stout, Rodawalt & Siemers, 2001), a fact that supports the idea that the main responsible for their performance is the declarative component.

In ACT-R the key factor determining the accessibility of the declarative knowledge is represented by chunk activation: the more active a chunk, the higher its probability of being retrieved, and the higher the speed of its retrieval. The activation A_i of a generic chunk i is determined by two components:

$$A_i = B_i + \sum_j W_j S_{ji} \quad (1)$$

where B_i constitutes the base component of the activation while the other term constitues the contextual, or associative, component. The base component reflects the general usefulness of a chunk: its value is higher the more times the chunk has been retrieved in the past and it decays as a function of the time passed by the last retrieval. Frequency and recency of use are thus the elements that control the base activation.

The associative component reflects the probability that a given chunk will be used in the current context. It depends on the multiplicative combination of two factors. The first, is represented by a parameter, W_j, expressing attentional weighing, i.e., the variable degree of attention different individuals are able to dedicate to the elements of the current context. The second, S_{ji}, reflects the degree in which the given chunk is related or associated to the elements of the current context (more precisely: to the different attributes, or *slots*, of the chunk representing the goal being currently pursued). The associative strength S_{ji} defines thus an estimate of the degree in which the presence of an item j in a slot of the goal chunk increases the utilization probability of the chunk i.

Banning some fluctuations due to stochastic noise, when an attempt is made to access a chunk by specifying some of its feature, the most active chunk sharing those features is retrieved from declarative memory. The activation of a chunk is determined, as we have seen, essentially by frequency-based constraints that are not affected by the content of the item being retrieved, nor by any other non cognitive factor, such as the emotional dispositions associated with the chunk.

A model of the GT based on the ACT-R theory, in trying to establish the typical outcome of each deck, will retrieve the most active chunk associated with it. Because the most frequently retrieved chunks are those with the highest activation, it is possible to predict that the disadvantageous decks A and B will remind of the numerous and frequently repeated positive-only outcomes, being the punishments relatively rare and not representative of the typical result obtained by choosing a card from those decks. Moreover, because the value of the positive-only outcomes of the disadvantageous decks outweighs that of the advantageous ones, the model, trying to be rational, will concentrate its choices mainly on the former decks, not taking into account the fact that, in the long run, they will lead to an awful performance. In other words, a typical ACT-R model will behave like an OFC patient.

To be able to replicate the performance of normal participants, taking advantage from the emotional effect aroused by receiving a punishment, it is necessary to allow the chunks encoding these relatively rare, but emotionally salient, events to have an activation sufficient to be retrieved, and to be considered as the typical outcomes associated with a given deck.

According to our point of view, the emotional effect of an event should be restricted to the contextual part of the activation. While the base component reflects the statistical structure of the environment, as revealed by the outcomes of past events, the contextual one could be considered as mirroring the biological, or evolutionary, usefulness of a given memory, helping to establish the cases in which a given context could profitably facilitate its retrieval. The emotion contributes to this process by charging declarative memories with biologically significant pleasant or unpleasant feelings, thus influencing their contextual activation, and therefore enhancing

their memorability. An ACT-R model having its activation equation modified to take into account these emotional aspects should therefore be able to model the behavior of normal participants in the GT.

An alternative approach to take into account the fact that memory retrieval is influenced not only by the frequency of an event but also by the quality of the outcome associated with it would be to devise a mechanism capable of explicitly evaluating, and updating, the deck values by computing the average outcome associated with each deck. Even if it could be possible to build such a mechanism adhering strictly to the principles of the ACT-R theory, and even if the mechanism would replicate the shift from disadvantageous to advantageous decks shown by normal participants, it would certainly fail in explaining why OFC patients keep on choosing from the disadvantageous decks when the total yielding has become negative.

Implementation and results

Relying on the above reported considerations, we developed two versions of a GT model. To keep a better control of the memory retrieval processes, and of the parameters underlying them, we did not use the customary release of the ACT-R architecture but we reimplemented its required parts.

The first version of the model was based on the standard ACT-R retrieval mechanisms, as described in Anderson & Lebiere (1998). In our implementation we adopted the equation of Altmann & Trafton (2002) which sets the basic activation B_i of a chunk i to:

$$B_i = \log\left(\frac{n_i}{\sqrt{T_i}}\right) \tag{2}$$

where n_i is the number of past retrievals of the chunk and T_i is the time passed since the creation of the chunk in memory.

The association between the chunk representing the outcome of a card (C_i) and the deck it comes from (D_j) was implemented in the link S_{ji}. The value of S_{ji} is computed as the log ratio between the conditioned probability of the outcome, given that the card has been selected from the deck, and the unconditioned probability of the outcome:

$$S_{ji} = \log\left(\frac{P(C_i|D_j)}{P(C_i)}\right) \tag{3}$$

S_{ji} represents thus the increment in the likelihood that a particular outcome associated with a deck will be retrieved as a consequence of trying to retrieve the outcomes of that deck.

The model utilizes the most active outcome associated with a deck as an estimate of the typical outcome that could be obtained from that deck. At each trial the model then selects, following a Boltzmann soft-max rule, the card on the top of the deck with the best estimate. This version of the model was used to simulate the performance of OFC patients.

To replicate the performance of normal participants, we modified the contextual component of the activation equation by introducing a new parameter η taking into account the emotional impact of an event. The basic idea is that different situations could be more or less emotionally charged. For instance, if participants in the GT would gain and loss real money instead of playing with simulated dollar bills, the emotional effect of an outcome on memory would be enhanced, and this fact would be reflected in a higher value of the η parameter. In most cases, however, the emotional impact of an event is related also to its magnitude. For instance, losing $1250 (of both real and simulated money) would provoke a more negative feeling that losing only $150. This fact is captured by a a normalized factor V representing a situation-independent evaluation of the magnitude of an emotional effect. Being the representation of numerical magnitudes subjected to distortion effects, we represent the magnitude of an outcome i through its logarithm. To obtain values of V_i comprised between 0 and 1 we normalized the absolute value of each outcome i on the absolute value of the maximum possible outcome:

$$V_i = \frac{\log(|i|)}{\log(|\max(\mathbf{i})|)} \tag{4}$$

The value thus obtained is then multiplied by η, and added to the original value of S_{ji} [1]

In summary, the activation equation (1) in the case of normal participants becomes now:

$$A_i = B_i + \sum_j W_j \bar{S}_{ji} \tag{5}$$

where:

$$\bar{S}_{ji} = S_{ji} + \eta V_i \tag{6}$$

Figure 3 reports the comparison between the results of the simulations with the two versions of the model (the normal version using a value of η = 2), and the data of Bechara et al. (1994).

[1] More precisely, the original likelihood ratio of S_{ji} is multiplied by $e^{\eta V_i}$. Taking the logarithm of this value, the two terms are simply added.

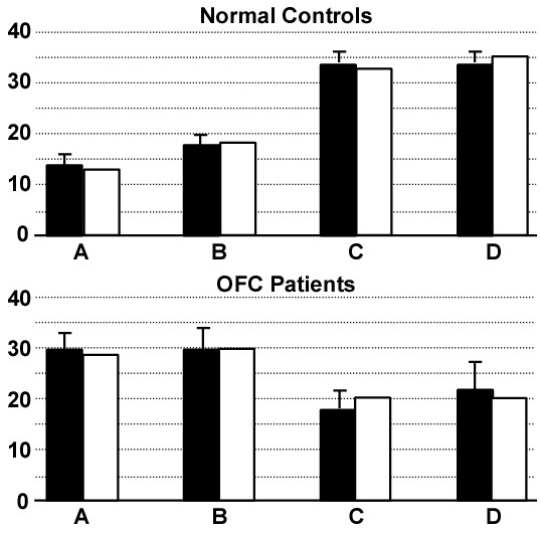

Figure 3: A comparison between participants (black) and model (white) choices.

Conclusions

In the paper we presented an ACT-R model capable of replicating the behavior of normal and OFC patients in the GT. In addition to clarifying the findings obtained with that paradigm, our work presents some implications for the interpretation of the role of the frontal cortex and for the development of the ACT-R cognitive architecture.

At the best of our knowledge, the present model is the first computational account of the GT. Previous investigations, in fact, were focused more on the discussion of the neurological issues than on providing a precise explanation for the experimental results. The only approach comparable to ours is that of Busemeyer & Stout (2002) who, however, pursue purely statistical modeling and are able to fit their models to data but cannot use them to make predictions about the behavior of participants in untested situations. After fixing the relevant parameters, on the other hand, our model is able to anticipate the participants performance for practically every variation of the GT.

The model takes a stance on the role of the OFC, and on the effects of injures in this area. It follows essentially Rolls' (1999, 2000) position according to which OFC patients are unable to exploit the emotional effect associated to outcomes because they are unable to associate an event with the emotion it produces.

A non trivial consequence of this position is that OFC patients should not differ from normal participants in experiencing emotive reactions that follow immediately their actions, while they should be disadvantaged when the task requires further processing that depends on the retrieval of emotional outcomes. An example of this distinction is given by the difference between *disappointment* (i.e, the simple reaction to an unpleasant outcome) and *regret* (that requires a form of counterfactual thinking comparing what was obtained to what would have been obtained by making a different decision). We predict that OFC patients should be impaired with their regret reactions but not with disappointment. Giorgio Coricelli from the University of Siena (personal communication, March 11, 2004) has tested the difference between disappointment and regret in frontal patients, and his data are congruent with our approach.

The present work has some implications for the ACT-R architecture, too. As far as we know, our model constitutes the first attempt to apply ACT-R to the explanation of neuropsychological results, being previous ACT-R neuroscience related work (e.g., Fincham, Carter, van Veen, Stenger, & Anderson 2002) focused exclusively on the interpretation of fMRI data. It should be noted that the behavior of the OFC patients could be considered as rational because, regarding the most frequent results as the typical outcomes of a deck, they stick to them ignoring the less frequent events that are kept in shadow by an inadequate contextual activation. To replicate the performance of normal participants in the GT, we propose a change to the ACT-R retrieval mechanism that takes into account the role of emotions in memory. The proposed modification makes ACT-R more sensible to this important situational factor, and is essentially backword compatible with previous modeling work that has dealt mainly with emotionally neutral tasks.

References

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

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

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

Busemeyer, J. R. & Stout, J. (2002). A contribution to cognitive decision models to clinical assessment: Decomposing performance on the Bechara Gambling Task. *Psychological Assessment, 14*, 253–262.

Damasio, A. R. (1994). *Descartes' error: Emotion, reason, and the human brain.* New York: Grosset/Putnam.

Fincham, J. M., Carter, C. S., van Veen, V., Stenger, V. A., & Anderson, J. R. (2002). Neural mechanisms of planning: A computational analysis using event-related fMRI. *Proceedings of the National Academy of Sciences, 99*, 3346–3351.

Gazzaniga, M. S., Ivry, R. B., & Mangun, G. R. (2002). *Cognitive neuroscience: The biology of the mind.* (2nd. ed.) New York: Norton & Company.

Kahneman, D., Slovic, P., & Tversky, A. (1982). *Judgment under uncertainty: Heuristics and biases*. New York: Cambridge University Press.

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

LeDoux, J. E. (1996). *The emotional brain*. New York: Simon & Shuster.

Panksepp, J. (1998). *Affective neuroscience*. New York: Oxford University Press.

Payne, J. W., Bettman, J. R., & Johnson, E. J. (1993). *The adaptive decision maker*. New York: Cambridge University Press.

Rolls, E. T. (1999). *The brain and emotion*. Oxford, UK: Oxford, University Press.

Rolls, E. T. (2000). The orbitofrontal cortex and reward. *Cerebral Cortex, 10*, 284–294.

Simon, H. A. (1978). Rationality as a process and as a product of thought. *American Economic Review, 68*, 1–16.

Stout, J. R., 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.