Using Cognitive Models to Map Relations between Neuropsychological Disorders and Human Decision Making Deficits

Eldad Yechiam, Jerome R. Busemeyer, Julie C. Stout

Indiana University, Bloomington IN

And

Antoine Bechara

University of Iowa

To appear in Psychological Science.

Journal Homepage:http://www.psychologicalscience.org/journals/index.cfm?journal=ps&content=ps/home

Word count: 3,439

Mail should be addressed to the first author:

Eldad Yechiam, Department of Psychology, Indiana University, 1101 East 10th St., Bloomington, IN 47405, Phone: 812-856-0839, Fax: 812-855-4691. Email: eyehiam@indiana.edu.

Using Cognitive Models to Map Relations between Neuropsychological Disorders and Human Decision Making Deficits

Abstract:

Findings from a complex decision making task (the Iowa gambling task) show that individuals with neuropsychological disorders are characterized by decision making deficits, leading to maladaptive risk-taking behavior. The paper describes a cognitive model which distills the performance in this task into three different underlying psychological components: the first measures the relative impact of rewards and punishments on evaluations; the second estimates the rate that the contingent payoffs are learned; and the third determines the consistency between learning and responding. Findings from ten studies are organized by distilling the observed decision deficits into the three basic components, and locating the neuropsychological disorders in this component space. The results reveal a cluster of populations characterized by making risky choices despite high attention to losses, due to difficulties in creating “emotive patterns”. These findings demonstrate the contribution of cognitive models for building bridges between neuroscience and behavior.

The Iowa gambling task (Bechara, Damasio, Damasio & Anderson, 1994) is a popular method for investigating basic decision-making deficits of individuals with neuropsychological disorders. Performers in this task make a series of 100 choices from four decks of cards with the goal being to maximize their net payoff across trials (see Figure 1). Each card selection leads to monetary gains but may also lead to losses. The outcomes of each of the decks are not known to the decision makers before hand and must be learned from experience. Two of the decks are disadvantageous and risky in that they lead to relatively high gains ($100 each time) but also to occasional large losses (up to $1,250), which result in an average loss (-$25 per trial). The two other decks are advantageous, as they lead to lower gains each time (only $50 gains) but produce smaller losses, which result in an average gain (+$25 per trial).

Initially, the task was found to be effective in differentiating individuals with bilateral damage to the VentroMedial Prefrontal Cortices (VMPC) from controls (Bechara et al., 1994). VMPC lesions are associated with a syndrome in which individuals have normal IQ and reasoning ability, but their decision-making behavior consists of excessive risk taking (Bechara et al., 1994; Damasio, 1994). This deficit was reflected in the Iowa gambling task by more choices from disadvantageous decks on the part of VMPC patients. Following the initial findings with VMPC patients, poor performance in the Iowa gambling task (persistence in the selection from disadvantageous decks) was also found in several other neuropsychological disorders, including lesions in the right somatosensory and insular cortex (Bechara, Tranel & Hindes, 1999), Huntington’s disease (Stout, Rodawalt & Siemers, 2001), chronic drug abuse (Bechara et al., 2001; Stout, Busemeyer, Lin, Grant& Bonson, 2004; Yechiam et al., 2004) obsessive-compulsive disorder (Cavedini et al., 2002), and Asperger's syndrome (Johnson et al., 2004).

These results are usually interpreted as indicating that all of these disorders share a common decision making deficit. Yet it is possible that markedly different psychological processes lead to the same qualitative finding of poor performance in the gambling task. Theoretically, the decision making deficits observed in the Iowa gambling task can be broken down into three basic components: The first is a motivational factor, producing a tendency to be attracted by gains and to ignore losses; a second is a learning rate factor, producing a tendency to focus on recent events and forget or rapidly discount past losses; the third is a response factor, causing choices to be made erratically owing to factors such as loss of interest, boredom, or tiredness. Thus, to improve discriminability between different populations, the overt behavior in the Iowa gambling task must be distilled so as to reveal potential differences in more basic components.

Busemeyer and Stout (2002) developed a cognitive model that could be used to sort out these different possible explanations. This mathematical model yields quantitative parameter estimates that provide a continuous mapping of populations along the three different psychological dimensions. Note that other models could lead to different implications and conclusions, but the present model captures the essential properties of most plausible attention and memory processing interpretations for the Iowa gambling task as well as similar repeated choice tasks (see e.g., Camerer & Ho, 1999; Erev & Roth, 1998; Weber, Shafir & Blais, 2004).

This paper reviews a set of ten applications of the cognitive model to a wide variety of populations with neuropsychological disorders. The results show that poor performance in the gambling task is associated with distinct psychological components across different neuropsychological disorders.

The Expectancy-Valence model. The cognitive model is comprised of three basic assumptions.

Attention to losses vs. wins: The motivation parameter. The first parameter of the model is a motivational parameter that represents the performers' attention to gains and losses. On each trial, a deck is selected, and payoffs contingent on the selected deck are delivered. The decision maker is assumed to evaluate the immediate gains and losses experienced after making a choice by a prospect theory type utility function (Kahneman & Tversky, 1979). The valence of the payoffs experienced on trial t is denoted $v(t)$, and it is calculated as a weighted average of gains and losses option (or deck) in trial t:

$$v(t) = W \cdot win(t) - (1-W) \cdot loss(t) \qquad (1)$$

Here $win(t)$ is the amount of money won on trial t; $loss(t)$ is the amount of money lost on trial t; and W is a parameter that indicate the weights to gains versus losses. The motivation parameter is limited from 0 and 1. Small values of the parameter denote attention to losses. Higher values denote increasing attention to gains, a tendency which can increase the preference for the high-gain disadvantageous decks.

Updating expectations: The learning rate parameter. The second parameter of the model represents the attention to the most recent outcomes versus the attention to past outcomes. Performers are assumed to form expectancies for each deck, which represent the anticipated consequences of choosing a card from a deck. When a deck is chosen, the expectancy E_j for deck j is updated as a function of its previous value (which reflects the past experience), as well as on the basis of newly experienced payoffs on the current trial, as follows:

$$E_j(t) = E_j(t\text{-}1) + \phi\cdot [v(t) - E_j(t\text{-}1)] \qquad (2)$$

In other words, the new expectancy equals the previous expectancy plus an adjustment resulting from the prediction error $[v(t)–E_j(t)]$ (Rumelhart & McClelland, 1986; Busemeyer & Myung, 1992). The amount of adjustment is controlled by the learning parameter, ϕ. The parameter is limited from 0 to 1. Small parameter values produce more persistent influences across longer lags, and less discounting of past outcomes. Large values of ϕ produce rapid adjustments, strong recency effects, and rapid discounting of past outcomes. A tendency to select from the disadvantageous decks could be due to such rapid discounting because these decks produce infrequent losses.

Choice consistency: The response sensitivity parameter. The decision maker's choice on each trial is based not only on the expectancies produced by each deck, but also on the consistency with which the decision maker applies those expectancies when making choices. According to the model, the probability of choosing a deck is determined by strength of that deck relative to the sum of the strengths of all decks:

$$\Pr[G_j(t)] = \frac{e^{\theta(t)\cdot E_j(t)}}{\sum_k e^{\theta(t)\cdot E_k(t)}} \qquad (3)$$

The variable $\theta(t)$ controls the consistency between choices and the expectancies, and it is assumed to change with experience. Consistency is assumed to increase with experience, reflecting greater reliance of choice on one's expectancies. This is formalized by a power function for the sensitivity change over trials:

$$\theta(t) = (t/10)^c \qquad (4)$$

where c is the response sensitivity parameter. When the value of the response sensitivity parameter is high, choices converge towards the deck with the maximum expectancy. When the value of c is low, choices become inconsistent, random, and independent of the expectancies over time. This erratic choice pattern is a third reason for performers not to learn to choose from advantageous decks.

Modeling analyses. The parameters of the model were optimized separately for each individual performer by maximizing the likelihood of the observed sequence of 100 choices produced by an individual. Optimization is a process wherein the fit of the model (in log likelihood) is compared with the fit of a baseline model. The baseline model's prediction is based on the optimized proportion of the choices of the different decks. Namely, the baseline model's three parameters are the average choice proportions of decks A, B, and C (deck D's is calculated accordingly). A comparison of the fit from the learning model to the baseline model is characterized by the improvement in the fit of the learning model over the baseline model. The statistical test of this improvement is G^2, which is a model fit statistic analogous to the chi-square.

This analysis results in three parameter estimates for each individual: *W* which measures importance of gains versus losses; ϕ which measures rate of adjustment and recency effects; and *c* which measures the consistency between expectancies and choices. The distribution of parameter estimates from each neuropsychological group was summarized by computing the averages and standard deviations for each group. Corresponding to each neuropsychological group, data was obtained from a control group, matched on extraneous variables such as age, gender, and education. The differences between the neuropsychological group and corresponding control were then computed.

Modeling results. Figure 2 maps the different populations studied with the Iowa gambling task according to the difference in the parameters of the Expectancy-Valence learning model. Each mean difference score is located at the center of a circle, which is positioned along two dimensions. The horizontal dimension represents differences in the weight for gains relative to losses, and the vertical dimension represents differences in the learning rate parameter. The standard errors of the difference are denoted by a cross beginning at the center of each circle. The radius of the circle represents differences in the response sensitivity parameter. In the bottom right side of the figure appear the results of the modeling comparison (to the baseline model) and significance tests for parameter differences.

Two samples of young and relatively high-functioning adult drug abusers (Yechiam, Stout, Busemeyer, Rock & Finn, in press) and alcoholics (Mazas, Finn & Steinmetz, 2000) show very similar parameter values to controls.

The top right side of the figure denotes a cluster of populations that are high either in attention to gains or in recency compared to controls. Some populations in this cluster are characterized by behavior that focuses on gains and discounts potential losses, and also discounts past outcomes more rapidly. Among the most extreme populations in this group are chronic (5+ years) cocaine abusers (Stout et al., 2004) and cannabis abusers (Yechiam et al., 2004). Note that both populations abstained from drugs prior to the experiment. Cocaine abusers show more attention to gains while cannabis abusers exhibit more recency. The results of Huntington's patients with an average of four years since diagnosis (Busemeyer & Stout, 2002) reflect a relative greater weighting to gains and high attention to the most recent trials (although not significantly so).

The results of normal seniors between the age of 65 to 88 (average age 77) show significantly higher attention to gains than control participants between 18 to 34 years old (Wood, Busemeyer, Koling, Cox & Davis, in press)[1]. However, they also show higher sensitivity than controls (denoted by the larger size of the bubble compared to the surrounding red circle). Finally, patients with bilateral damage to the VMPC (Bechara et al., 1994) show a significant increase in recency and also display an erratic choice pattern. The finding that the main difference between VMPC patients and controls is in the learning rate parameter fits well with recent results showing that in decision tasks that involve no learning (i.e., description based tasks) the differences between VMPC patients and controls disappear (Leland & Grafman, in press).

As a stark contrast to this first cluster, there are three populations at the left hand side of the figure whose decision making style is characterized mostly by *high attention*

[1] Note that these results differ somewhat from Wood et al.'s (in press) analysis. The present study uses a three parameter model which was found to be more robust (see Yechiam, Veinott, Busemeyer & Stout, in press).

to losses. The first such population is Parkinson's patients (whose average age was 66, with an average of eight years following diagnosis; Busemeyer & Stout, 2002). This is consistant with previous studies showing that Parkinson's patients score high on harm avoidance tests (Kaasinen et al., 2001), and also with the curious finding that individuals who do not smoke regularly have been found to be more prone to have Parkinson's disease (Louis, Luchsinger, Tang & Mayeux, 2003). Two populations in the second cluster display sensitivity to losses coupled with erratic choices: individuals with lesions in the right somatosensory and insular cortex (RSIC; Bechara et al., 1999) and adolescents with Asperger's syndrome (Johnson et al., 2004). Potentially, extremely low sensitivity can lead to disadvantageous choices despite high attention to losses due to continuous rejection of advantageous alternatives (which also contain small losses). This is consistant with the trial-to-trial pattern of behavior in both these populations syndrome, which is characterized by an extreme tendency to shift and change their prior choices (Johnson et al., 2004).

The findings that chronic drug abusers in the first cluster demonstrate a motivational bias for immediate gains is consistant with theories of the behavior of drug abusers in choice tasks (see reviews in Finn, 2002; Gorenstein & Newman, 1980), which postulate that for drug abusers, signals of positive reward may carry larger weight over signals of potential risk due to stronger appetitive processes and weaker disinhibitory mechanisms.

The first cluster showing that both VMPC patients and chronic drug abusers demonstrate a degree of "myopia" for distant consequences is consistent with prior observations (Bechara et al., 1994; Bechara et al., 1999; Damasio, 1994). Huntington's patients also belong to this cluster. Given the close anatomical and functional links between the striatum and prefrontal cortex, finding similar decision-making profiles in Huntington’s and VMPC patients is not surprising. In addition, the findings of high recency in drug abusers and Huntington’s patients can be due to a memory deficit. The relative increase in recency in chronic cannabis abusers (as compared to cocaine abusers) is consistant with the known effect of cannabis abuse on cannabinoids, which have regional binding specificity within the caudate nucleus and putamen, and within the hippocampus, brain areas important in memory (Bolla, Brown, Eldreth, Tate & Cadet, 2002). Huntington’s patients are likewise known to have memory impairments (see e.g., Huber & Paulson, 1987; Stout et al., 2001).

The second cluster shows that patients with right somatosensory and insular lesions and individuals with Asperger’s syndrome show low attention to gains (or pay relatively more attention to losses), but most importantly, they have a pronounced erratic choice pattern (i.e., low sensitivity parameter). This is consistent with the notion that a deficit in the neural systems subserving emotions and feelings may be the source of this choice pattern (Bechara et al., 1999; Damasio, 1994). “Feeling” the pleasure of gain, or the pain of loss, may be dependent on neural processes within the right insular and somatosensory cortices (Damasio, 1994). Patients with RSIC lesions can generate physiological responses to gains and losses, but their subjective ratings of how good or bad they felt when they won or lost is severely reduced (Bechara et al., 1999). Perhaps this feeling deficit translates into a cognitive deficit, in that the subjects may never learn how to win because they never “care” about winning. As such, the subject would adopt a simple “win-stay” or “lose-shift” strategy in all decks, thus producing a tendency to oscillate between alternatives. Consequently, performers do not learn to choose the better decks.

In summary, the results of the analysis using our cognitive model show that rather than a single common decision making deficit, poor choices tend to be associated with different component processes which reflect the continuous influence of attention to gains and losses, the degree of recency, and response sensitivity. Cognitive neuroscience is just beginning to unravel the brain mystery of human decision-making. In the past, the predominant approach to studying this complex function has focused on specific component processes of decision-making, such as learning reversal, working memory, and other executive functions. Unfortunately, this approach did not lead to a satisfactory understanding, for example, of the decision-making impairments observed in patients with VMPC lesions (Bechara et al., 1994). One successful attempt in capturing key aspects of human decision-making and its disorder was the use of complex laboratory tasks, such as the Iowa gambling task, that mimics real-life choices in the way it factors reward and punishment, and the uncertainties of their occurrence. This has led to the revival of old interest in the relationship between emotion and cognition. Although the Iowa gambling task succeeded in capturing many of the critical elements of decision-making that were missed by the component process approach, the relative complexity of this task still prohibits a finer resolution of its underlying neural processes. However, the cognitive model described in this study provides a novel way for circumventing this problem, thus building a new bridge between cognitive neuroscience and complex human behaviors.

References

Bechara, A., Dolan, S., Denburg, N., Hindes, A., Anderson, S.W., & Nathan, P.E. (2001). Decision-making deficits, linked to a dysfunctional ventromedial prefrontal cortex, revealed in alcohol and stimulant abusers. *Neuropsychologia*, *9*, 376-389.

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

Bechara, A., Tranel, D., & Hindes, A. (1999). Roles of the amygdala, pre-frontal cortex, and right somatosensory/insular cortex in decision making and emotional processing. *The Journal of Neuropsychiatry and Clinical Neurosciences*, *11*, 157.

Bolla, K.I., Brown, K., Eldreth, D., Tate, K., & Cadet, J.L. (2002). Dose-related neurocognitive effects of marijuana use. *Neurology*, *59*, 1337-1343.

Busemeyer, J.R., & Myung, I.J. (1992). An adaptive approach to human decision-making: learning theory, decision theory, and human performance. *Journal of Experimental Psychology: General*, *121*, 177-194.

Busemeyer, J.R., & Stout, J.C. (2002). A contribution of cognitive decision models to clinical assessment: Decomposing performance on the Bechara gambling task. *Psychological Assessment*, *14*, 253-262.

Camerer, C., & Ho, T. –H (1999). Experience-weighted attraction learning in normal form games. *Econometrica*, *67*, 827-874.

Cavedini, P., Riboldi, G., D'Annucci, A., Belotti, P., Cisima, M., & Bellodi, L. (2002). Decision-making heterogeneity in obsessive-compulsive disorder: Ventromedial prefrontal cortex function predicts different treatment outcomes. *Neuropsychologia*, *40*, 205-211.

Damasio, A.R. (1994). *Descartes' error: Emotion, reason, and the human brain*. Avon Books, NY.

Erev, I., & Roth, A. E. (1998). Predicting how people play games: Reinforcement learning in experimental games with unique, mixed strategy equilibria. *American Economic Review*, *88*, 848-881.

Finn, P.R. (2002). Motivation, working memory, and decision making: A cognitive motivational theory of vulnerability to alcoholism. *Behavioral and Cognitive Neuroscience Reviews*, *1*, 181-203.

Gorenstein, E.E., & Newman, J.P. (1980). Disinhibitory psychopathology: A new perspective and a model for research. *Psychological Review*, *87*, 301-315.

Huber, S.J., & Paulson, G.W. (1987). Memory impairment associated with progression of Huntington's disease. *Cortex*, *23*, 275-83.

Johnson, S.A., Yechiam, E., Murphy, R., Stout, J.C., & Busemeyer, J.R. (2004). A formal decision model: Application to Asperger's disorder. Paper presented at the International Meeting for Autism Research (IMFAR), Sacramento, CA, May 2004.

Kaasinen, V., Nurmi, E., Bergman, J., Eskola, O., Solin, O., Sonninen, P., & Rinne, J.O. (2001). Personality traits and brain dopaminergic function in Parkinson's disease. *Proceedings of the National Academy of Sciences*, *98*, 13272-13277.

Kahneman, D., & Tversky, A. (1979). Prospect theory: An analysis of decision under risk. *Econometrica*, 47, 263-291.

Leland, J.W., & Grafman, J. (in press). Experimental tests of the Somatic Marker hypothesis. *Games and Economic Behavior*.

Louis, E.D., Luchsinger, J.A., Tang, M.X., & Mayeux, R. (2003). Parkinsonian signs in older people: Prevalence and associations with smoking and coffee. *Neurology*, 61, 24-28.

Mazas, C.A., Finn, P.R., & Steinmetz, J.E. (2000). Decision-making biases, antisocial personality, and early-onset alcoholism. *Alcoholism: Clinical and Experimental Research, 24*, 1036-1040.

Rumelhart, D.E., & McClelland J.L. (1986). *Parallel distributed processing: Explorations in the microstructure of cognition, volumes 1 and 2*. MIT Press, Cambridge, MA.

Stout, J.C., Busemeyer, J.R., Lin, A., Grant, S.R., & Bonson, K.R. (2004). Cognitive modeling analysis of the decision-making processes used by cocaine abusers. *Psychonomic Bulletin and Review*, *11*, 742-747.

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.

Weber, E.U., Shafir, S., & Blais, A-R. (2004). Predicting risk sensitivity in humans and lower animals: Risk as variance or coefficient of variation. *Psychological Review*, *111*, 430-445

Wood, S., Busemeyer, J.R., Koling, A., Cox, C., & Davis, H. (in press). Older Adults as adaptive decision-makers: Evidence from the gambling task. *Psychology and aging*

Yechiam, E., Stout, J.C., Busemeyer, J.R., Rock, S.L., & Finn, P.R. (in press). Individual differences in the response to forgone payoffs: An examination of high functioning drug abusers. *Journal of Behavioral Decision Making*.

Yechiam, E., Stout, J., Lamborn, C., Mussat-Whitlow, B.J, Liguori, A., & Porrino, L.J. (2004). Differential influence of marijuana on decision processes in the Bechara gambling task. Poster presented at the 11th annual Cognitive Neuroscience Society (CNS) meeting, San Francisco, California, April 2004.

Yechiam, E., Veinott, E.S., Busemeyer, J.R., & Stout, J.C . (In press). Cognitive models for evaluating basic decision processes in clinical populations. In R. Neufeld (Ed.). *Advances in clinical cognitive science: Formal modeling and assessment of processes and symptoms*. APA Publications, Washington DC.

Author note

We thank Peter Finn, Steve Grant, Shannon Johnson, Linda Porrino, and Stacey Wood for sharing their results with the authors in previous works summarized here. Thanks due to Igor Weinberg for his helpful comments. This research was supported by a grant (R01-DA014119) from the National Institute on Drug Abuse.

Figure 1: Left – A screen-shot from the computerized version of the The Iowa gambling task. Right – the available alternatives in their task and their outcomes. In the illustration on the right hand side the top two decks (A,B) are disadvantageous and the bottom two (C, D) are advantageous. Note that as in the example on the left, gains and losses can occur simultaneously on the same trial.

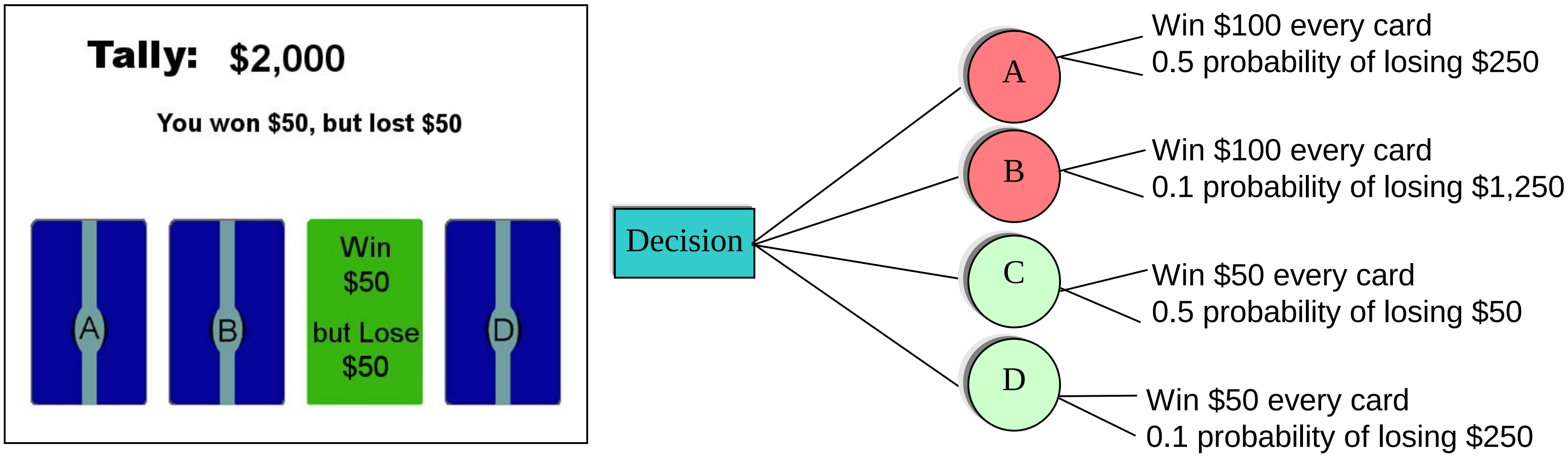
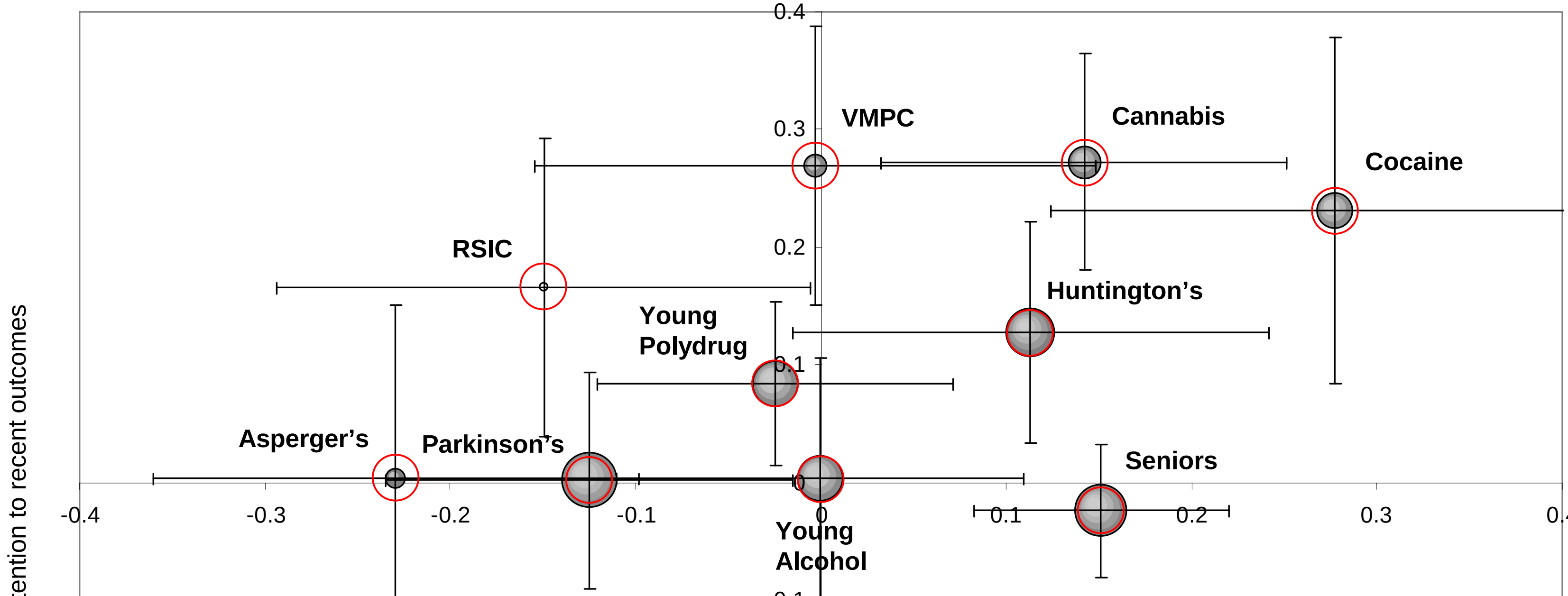


Figure 2: Mapping of studied populations according to the differences in attention to loss/gain parameter and recency parameter compared to controls (averages and standard errors of the difference). The volume of bubbles is proportional to the difference in the choice consistency parameter. The red ring around bubbles denotes the zero difference boundary (bubbles smaller than the ring denote populations with low sensitivity). The table at the bottom right side presents the percent of positive G^2 values, indicating an improvement of the adaptive learning model over the baseline model, and the results of significance tests for the different parameters.



Samples (left to right)	Sample (n)	Controls (n)	$\%G^2>0$	p<.05
Asperger's syndrome	15	14	66%	$W^{\dagger}$, *c*
RSIC lesion	22	12	62%	*c*
Parkinson's disease	20	33	75%*	
Young Polydrug abusers	39	37	49%	
Young Alcohol abusers	27	32	67%*	
VMPC lesion	21	12	76%*	ϕ, *c*
Normal Seniors	63	87	61%*	*W*, *c*
Huntington's disease	14	33	75%*	
Chronic Cannabis abusers	25	16	24%	ϕ, *W*
Chronic Cocaine abusers	12	14	69%*	*W*

* = p<. 05 in a binomial test ; † -sig. after 150 trials.