Psychological Assessment
2002, Vol. 14, No. 3, 253–262

Copyright 2002 by the American Psychological Association, Inc.
1040-3590/02/$5.00 DOI: 10.1037//1040-3590.14.3.253

A Contribution of Cognitive Decision Models to Clinical Assessment: Decomposing Performance on the Bechara Gambling Task

Jerome R. Busemeyer and Julie C. Stout
Indiana University Bloomington

The Bechara simulated gambling task is a popular method of examining decision-making deficits exhibited by people with brain damage, psychopathology, antisocial personality, or drug abuse problems. However, performance on this task is confounded by complex interdependencies between cognitive, motivational, and response processes, making it difficult to sort out and identify the specific processes responsible for the observed behavioral deficits. The authors compare 3 competing cognitive decision models of the Bechara task in terms of their ability to explain the performance deficits observed in Huntington's disease patients as compared with healthy populations and people with Parkinson's disease. The parameters of the best fitting model are used to decompose the observed performance deficit of the Huntington patients into cognitive, motivational, and response sources.

The clinical neuroscience, psychopathology, and drug abuse literatures have experienced a surge of interest in the use of a laboratory-based simulated gambling paradigm developed by Bechara, Damasio, Tranel, and Anderson (1994). These tasks have been used to experimentally study the neuropsychological basis of deficits in decision making exhibited by populations with brain damage, psychopathology, antisocial personality, or drug abuse problems (Bartzokis et al., 2000; Bechara, Damasio, & Damasio, 2000; Bechara, Damasio, Damasio, & Lee, 1999; Bechara, Damasio, Tranel, & Anderson, 1998; Bechara, Damasio, Tranel, & Damasio, 1997; Grant, Contoreggi, & London, 2000; Mazas, Finn, & Steinmetz, 2000; Petry, Bickel, & Amett, 1998; Schmitt, Brinkley, & Newman, 1999; Stout, Rodawalt, & Siemers, 2001).

The Bechara task was designed to examine the complex interplay between cognitive and motivational processes. Poor performance on these tasks has been attributed to a failure to anticipate the long-term negative consequences of disadvantageous choices. A problem arises, however, when one attempts to theorize about the exact causes of these decision-making deficits. Because these decisions are produced by complex cognitive–motivational interactions, it is difficult to sort out and identify the specific processes (such as contingency learning, evaluation of consequences, and choice mechanisms) responsible for the observed behavioral deficits.

Cognitive models provide a theoretical basis for identifying and measuring the hidden processes underlying performance in complex tasks. For example, signal-detection theory (Green & Swets, 1966) provides independent measures of perceptual sensitivity and decision bias, which confound performance on detection tasks. Processing-tree theory (Batchelder & Riefer, 1999) provides independent measures of memory storage and retrieval, which confound performance on memory recall tasks. Of course, gambling tasks differ in fundamental ways from detection tasks and memory tasks, and therefore these previous two theories are inapplicable here. However, there are other cognitive decision models that are applicable to the gambling task and capable of independently measuring the cognitive, motivational, and response processes that underlie risky choice behavior.

The purpose of this article is to present an application of cognitive decision models for clinical assessment. Cognitive decision models provide a theoretical foundation for assessment of the cognitive versus motivational processes conflated in risky decision-making tasks. More specifically, we show how these models can be used to identify the source of the decision-making deficits exhibited by brain-damaged patients suffering from Huntington's disease in the Bechara gambling paradigm.

The remainder of this article is organized as follows. First, the Bechara gambling task is briefly described. Second, we summarize the decision-making deficits demonstrated on this task by people with Huntington's disease. Third, several competing models of decision making are presented. Fourth, the models are compared in terms of their ability to account for performance on the gambling task. Fifth, the best fitting model is then used to provide individual-differences assessments of the cognitive, motivational, and response processes underlying task performance. Finally, conclusions are drawn regarding the role of each of these processes for decision-making deficits in this task.

The Bechara Gambling Paradigm

The gambling task involves the use of four decks of cards (labeled here D_1, D_2, D_3, and D_4), each containing 40 cards. On each of 100 trials of the gambling task, the player is asked to choose a card from any one of the four decks. Once a card is chosen, it is turned over, the amount of money won or lost for choosing that card is revealed, and any wins and losses are included in the player's holdings. (Usually, large sums of hypothetical money are won or lost, but some studies have used small

Jerome R. Busemeyer and Julie C. Stout, Department of Psychology, Indiana University Bloomington.

This research was supported by National Institute on Drug Abuse Grant R01-DA14119.

Correspondence concerning this article should be addressed to Jerome R. Busemeyer, Department of Psychology, Indiana University, Bloomington, Indiana 47405. E-mail: jbusemey@indiana.edu

amounts of real money.) The player is never told the distribution of wins and losses associated with each deck; instead the distributions are learned from experience. The same card sequence is usually employed for all participants.

The four decks are designed in such a way that two of the decks (labeled here D_1 and D_2) are "advantageous," whereas the other two decks (labeled here D_3 and D_4) are "disadvantageous" in the following sense. Selections from advantageous decks produce an average gain of \$25; selections from disadvantageous decks produce a loss of \$25. However, the payoffs are arranged and sequenced in a manner that makes it difficult for the player to learn this fact. The advantageous decks always produce a small immediate win of \$50, whereas the disadvantageous decks always produce a large immediate win of \$100. Thus, the disadvantageous decks appear at first sight to be superior, at least with respect to the amount to win. But this is misleading because the disadvantageous decks also produce larger losses than the advantageous decks. That is, advantageous deck D_1 yields a loss of \$250 once every block of 10 cards, whereas the disadvantageous deck D_3 yields a loss of \$1,250 once every block of 10 cards. The other advantageous deck, D_2, yields five losses of \$50 within each block of 10 cards for a total loss of \$250 within each block, whereas the other disadvantageous deck, D_4, yields losses of \$150, \$200, \$250, \$300, and \$350 within each block of 10 cards for a total loss of \$1,250 within each block.

The main dependent measure is the proportion of selections from the advantageous decks as a function of training trials. A typical finding reported with this task is that healthy control participants gradually learn to favor the advantageous decks, but people with orbital frontal cortex damage persist in favoring the disadvantageous decks (see Bechara et al., 1997).

Performance on the Bechara task depends on multiple processes, including remembering past outcomes, learning long-term contingencies, evaluating immediate wins relative to long-term losses, and finally, choice mechanisms controlling the decision maker's impulsiveness and recklessness. Thus, the decision-making deficits exhibited by brain-damaged patients may result from individual differences on any combination of the above processes. Cognitive modeling is helpful for sorting this out and identifying which of these processes are mainly responsible for the observed deficits.

Decision Making in Huntington's Disease Patients

Recently Stout et al. (2001) examined a group of Huntington's disease patients who had only mild cognitive impairment ($n = 14$) using the Bechara gambling task. This group was compared with a healthy control group ($n = 33$) and a group of Parkinson's disease patients ($n = 20$). Individuals were excluded if they scored too low (i.e., greater impairment) on the Mattis (1988) Dementia Rating Scale (a score less than 100), and they were also excluded if they scored too high (i.e., significant depressive symptoms) on a Hamilton depression interview (score > 14; Beck, Steer, & Garbin, 1988). The average age of the Huntington's disease patients (44.6 years) was less than the Parkinson's disease patients (66.0), and the average age of the healthy controls fell in between (56.8). The average number of years of education was approximately the same across the three groups (14.5 years). For other details concerning the populations, see Stout et al. (2001).

Huntington's disease causes preferential loss of neurons in the caudate nuclei and putamen of the basal ganglia, which mediate projections into the orbital prefrontal cortex, and so this group was expected to produce the same type of decision-making deficit as patients with orbital frontal cortex damage. Parkinson's disease selectively damages the substantia nigra, which is the primary source of dopamine in the neostriatum. This group was not expected to produce the same decision-making deficit as the patients with orbital frontal cortex damage. As expected, the Huntington's disease group chose significantly less from the advantageous deck in the latter half of training as compared with the healthy controls, but there was no significant difference between the Parkinson's disease patients and the healthy controls.

Figure 1 shows a more detailed analysis of the trial-by-trial learning process exhibited by each group. The proportions reflected in the curves were computed by pooling across participants within each group and averaging across trials with a moving average window of seven trials. As can be seen in the figure, the healthy controls gradually learned to favor the advantageous deck, and to a lesser extent, so did the Parkinson's disease group, but the Huntington's disease group decreased their tendency to choose from the advantageous decks across trials. The upward displacements shown at the bottom of the left panel indicate that the control group initially suffered large punishments early in training and that these gradually diminished as they switched to the advantageous decks. The upward displacements shown in the middle panel indicate that the Huntington's disease group continued to experience large punishments throughout training.

The theoretical analysis presented below provides a way to identify the source of decision-making deficits in the Bechara task. A cognitive decision model provides a formal model of performance on the gambling task, and this model entails parameters

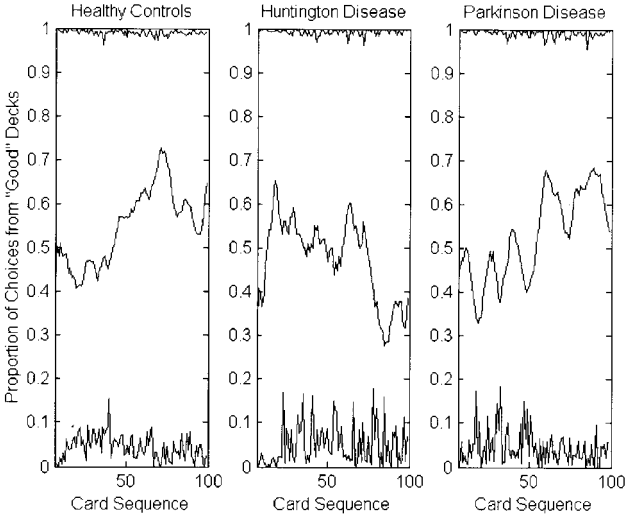

Figure 1. The smooth curve within each panel shows the proportion of advantageous choices (labeled "Good") plotted as a function of card sequence (number of trials) for each of the three groups, from Stout, Rodawalt, and Siemers (2001). The upward projections shown at the bottom of each panel indicate the losses produced by choosing the disadvantageous decks, and the downward projections shown at the top indicate the losses produced by choosing the advantageous decks.

corresponding to cognitive, motivational, and response processes. These parameters are estimated directly from the choice patterns produced by each individual participant, thus providing a distribution of estimates for each group. Finally, the parameter distributions are compared across groups to determine which parameters—cognitive, motivational, or response—are responsible for the observed group differences.

Cognitive Modeling of the Bechara Gambling Paradigm

The first step in any type of cognitive-modeling application is to compare and test competing models for the task. The best fitting model from this competition is then selected and used to provide the basis for parameter estimation and interpretation. The point is not to find a model that works but rather to establish the empirical validity of one model over others. There are many possible candidate models of decision making that one could propose and test for the Bechara gambling task. In fact, we have examined over 10 such models,[1] but to spare the reader, only 3 competing models are considered here. These 3 models were selected for consideration according to several criteria. First, we considered models that have received some general support from previous research in the decision-making literature. Second, all of the models are simple and entail only three parameters. This is needed for efficient statistical estimation of parameters. Third, the models differ fundamentally in terms of their basic processing assumptions. The purpose of the last criterion was to avoid simply testing small variations on a single idea and failing to contrast completely different ideas. Before presenting the models, we introduce some common definitions and notation.

Definitions and Notation

The two advantageous decks are labeled D_1 and D_2, and the two disadvantageous decks are labeled D_3 and D_4. The time index, *t*, is used to denote the trial number, that is, the total number of cards selected by a particular decision maker at some point during training. The deck chosen on trial *t* is denoted $D(t)$, so that, for example, $D(t) = D_2$ if the second advantageous deck is chosen on trial *t*. The reward or gain received by choosing deck *D* is denoted $R(D)$, that is, $R(D) =$ \$50 for advantageous choices, and $R(D) =$ \$100 for disadvantageous choices. The loss received from choosing deck *D* on trial *t* is denoted $L[D(t)]$; for example, if a disadvantageous deck is chosen on trial $t = 9$ and a loss of \$1,250 occurs, then $L[D(9)] = -$\$1,250.

Strategy-Switching Heuristic Choice Model

One major approach to human decision making is based on the idea that decision makers use simple heuristic strategies, and they learn to adapt or switch strategies depending on the decision environment and task demands (see Payne, Bettman, & Johnson, 1993, for a review of this approach). The following model is an application of these ideas to the Bechara task.

According to this model, the decision maker initially hypothesizes that the high immediate payoff decks are best and thus starts with a tendency to choose from the disadvantageous decks. But after experiencing a series of large losses produced by the disadvantageous decks, he or she switches hypotheses and changes toward a tendency to choose more from the advantageous decks. This general idea is formalized as follows.

Initial tendencies. The symbol p_1 stands for the probability of choosing from a disadvantageous deck during the initial stage of training. Note that both disadvantageous decks produce the same immediate reward and average losses, and so we further assume that the two disadvantageous decks, D_2 and D_3, are chosen equally often (but see footnote 1). Similarly, the two advantageous decks produce the same immediate reward and average losses, and so we again assume that D_1 and D_2 are chosen equally often. The free parameter, p_1, must be estimated from the choice data.

Switching tendencies. After experiencing a series of losses, the decision maker switches tendencies, so that $p_2 = (1 - p_1)$ becomes the probability of choosing from the disadvantageous decks at the later stage of training. The probability of switching depends on the losses the decision maker incurs from the disadvantageous decks during training. The symbol $S(t)$ represents the sum of all the losses produced by choosing from the disadvantageous decks up to and including *t*. The probability of switching tendencies on trial *t*, denoted $c(t)$, is determined from this sum as follows:

$$c(t) = \frac{e^{aS(t)}}{e^{aS(t)} + e^{b}}. \tag{1a}$$

Equation 1a is the logistic distribution function, which produces a smooth S-shaped curve that is an increasing function of the loss sum, $S(t)$. The coefficients *a* and *b* control the slope and location of the logistic curve. These two free parameters must be estimated from the choice data also.

Choice mechanism. The symbol $Pr[D_i|t + 1]$ denotes the probability that deck D_i is chosen on trial $t + 1$. Figure 2 illustrates the choice process. First a strategy is selected with probabilities associated with the first pair of branches, and then a deck is chosen with probabilities associated with the second pair of branches. Multiplying probabilities along the paths and summing the paths that lead to a choice of each kind of deck (labeled "good" for

[1] The 10 models were variations on the 3 models that are presented here. We chose the best variation of each type for this presentation. For example, we also fit a Bayesian–expected utility model that allowed the guessing probability to change over time in a manner similar to the expectancy–valence model. But this produced poorer fits, and so we present the better fitting version with constant guessing probability. As another example, we also tried a version of the heuristic choice model that allowed the probabilities of choosing between the two disadvantageous decks to differ from each other during the first stage, and this version also allowed the probabilities of choosing between the two advantageous decks to differ from each other during the second stage. However, this version did not fit any better than the heuristic choice model that we present here. All of the models are limited to three parameters, which is advisable given the models are fit to individuals' data for a relatively small number of trials. More complex models may be examined in the future provided that more extensive training is used, producing more data to support efficient estimation. Note, however, that equating the number of parameters used by each model does not guarantee that model complexity has been equated (see Myung & Pitt, 1997).

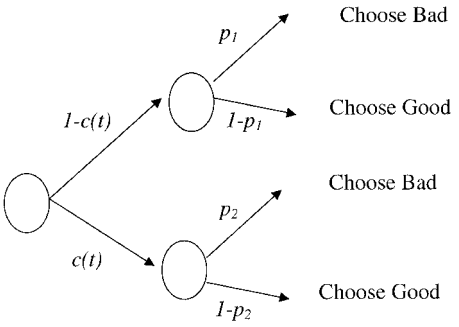

Figure 2. Diagram of the choice mechanism for the heuristic choice model. The first stage represents the choice of strategy, and the second stage represents the choice of deck on the basis of a selected strategy.

advantageous and "bad" for disadvantageous in the figure) produces the following choice probabilities:

$$Pr[\mathrm{D}_i|t+1] = \{[1 - c(t)] \cdot (1 - p_1) + c(t) \cdot (1 - p_2)\}/2 \quad \text{for } \mathrm{D}_i = \mathrm{D}_1 \text{ or } \mathrm{D}_2$$

$$Pr[\mathrm{D}_i|t+1] = \{[1 - c(t)] \cdot p_1 + c(t) \cdot p_2\}/2 \quad \text{for } \mathrm{D}_i = \mathrm{D}_3 \text{ or } \mathrm{D}_4. \quad (1b)$$

In sum, the heuristic choice model requires the estimation of three parameters from the data: p_1 represents the initial tendency to choose from the disadvantageous decks, and *a* and *b* determine the shape of the logistic function for switching based on the total losses produced by the disadvantageous decks. Of course, it is possible to formulate other models, but we examined alternative three-parameter versions (see Footnote 1), and this version produced the best results among the versions that we examined. We could also add more parameters to this model, but we need to restrict the model to three parameters for reasons mentioned earlier. First, fits to individual data do not support large numbers of parameters, and all the remaining models were also constrained to three parameters.

Bayesian–Expected Utility Model

Another major approach to human decision making is based on the idea that people use bounded rational decision strategies (see Luce, 1999, for a review of this approach). That is, they attempt to optimize their decisions under constraints imposed by human information-processing limitations. The following model is an application of these ideas to the Bechara task.

According to this model, the decision maker uses Bayesian updating to change prior estimates of the payoff probabilities into posterior probabilities on the basis of experience with the outcome observed by the choice made on each trial. With these probability estimates, the expected utility of each deck is computed on each trial. Finally, the deck that maximizes expected utility is chosen on each trial.

Probabilities. The symbol $P_D(t)$ denotes the decision maker's estimate of the probability of a loss given that deck *D* is chosen on trial *t.* Bayesian theorists normally use what is called a *beta prior distribution* for estimating relative frequencies (see Berry, 1996, for a detailed presentation). Using the beta prior distribution, the rule for updating the expected probability of a loss is stated as follows:

$$P_D(t) = \frac{f_D(t) + f(0)}{n_D(t) + n(0)}. \quad (2a)$$

In the above equation, $f_D(t)$ symbolizes the number of cards producing a loss experienced by choosing deck *D* up to and including trial *t;* $n_D(t)$, symbolizes the total number of trials that deck *D* was chosen up to and including trial *t;* and $f(0)$ and $n(0)$ reflect the prior estimates before any experience.[2] Note this updating rule provides a computationally simple and intuitively reasonable model for learning the probabilities.

Utilities. On each trial, the decision maker receives a gain and possibly a loss, denoted $R[D(t)]$ and $L[D(t)]$ for trial *t,* where $L[D(t)]$ may be zero on some trials. The net payoff, denoted $x(t)$, for trial *t* is the sum of the gains and losses, $x(t) = R[D(t)] + L[D(t)]$. The decision maker's subjective evaluation of this net payoff, denoted $u[x(t)]$, is called the utility of the net payoff. In general, the utility function is a possibly nonlinear but monotonically increasing function of the net payoff. A standard assumption made by decision theorists (see Luce, 1999) is to represent the utility function by a pair of power functions. This assumption will be used here as well, in which case the utilities of the gains and losses are computed as follows:

$$u[x] = x^a \text{ if } x > 0 \text{ and } u[x] = -|x|^b \text{ if } x < 0. \quad (2b)$$

The exponents of the power functions, *a* and *b,* are parameters to be estimated from the choice data.

Choice rule. The choice on each trial is made on the basis of a comparison of the expected utilities estimated for each deck, and the deck producing the maximum expected utility is chosen. The expected utility of choosing deck *D* on trial *t,* denoted $Eu[D|t]$, is computed as follows:

$$Eu[D|t] = [1 - P_D(t)] \cdot u\{R[D(t)]\} + P_D(t) \cdot u\{R[D(t)] + L[D(t)]\}. \quad (2c)$$

Finally, deck D_i is chosen on trial $t + 1$ if

$$Eu[\mathrm{D}_i|t] = \max\{Eu[\mathrm{D}_1|t], Eu[\mathrm{D}_2|t], Eu[\mathrm{D}_3|t], Eu[\mathrm{D}_4|t]\}. \quad (2d)$$

To allow for occasional random guessing, it is assumed that the decision maker chooses the maximum expected utility option on a trial with probability *p,* and otherwise guesses randomly among the four decks with equal probability. In sum, the Bayesian–expected utility model requires the estimation of three parameters from the

[2] The beta prior states that the prior expected probability of a loss, before any experience, starts at $f(0)/n(0)$. We set $f(0) = .10$ and $n(0) = 1$, which implies that the prior expected probability of a loss starts at .10. After each feedback trial, this expected probability is changed according to Equation 2a. These priors are rapidly changed by new experience, so that the expected posterior probabilities are primarily determined by the observed relative frequencies. Note that Bayesian updating is considered the optimal way to learn probabilities from experience.

data: *a* and *b* determine the shape of the utility function for gains and losses, and *p* allows for some proportion of random guessing.

Expectancy–Valence Learning Model

The third model is based on previous theoretical developments that were specifically designed to integrate learning and decision-making processes into a unified model (Busemeyer & Myung, 1992; Erev & Roth, 1998). The following model applies these ideas to the Bechara task.

According to this model, the decision maker integrates the gains and losses experienced on each trial into a single affective reaction called a valence. Expectancies about the valence produced by each deck are learned by an adaptive learning mechanism. Finally, these expectancies serve as the inputs into a probabilistic choice mechanism that selects the choice on each trial.

Valences. The gains and losses experienced after making a choice produce an affective reaction in the decision maker called a valence. The valence experienced after choosing deck *D* on trial *t,* denoted $v(t)$, is represented as a weighted average of the gains and losses:

$$v(t) = \{(1 - w) \cdot R[D(t)] + w \cdot L[D(t)]\}. \quad (3a)$$

The attention weight parameter, $0 < w < 1$, allows for the possibility that different amounts of attention are given to the losses as compared to the gains.

Expectancy learning. The decision maker learns expectancies about the valences produced by choosing each deck during training. The expected valence for deck D_i on trial t is denoted $Ev[D_i|t]$. These expectancies are updated by an adaptive learning mechanism. If deck D_i is chosen on trial t, and the valence $v(t)$ is experienced, then the expectancy for this deck is updated as follows:

$$Ev[D_i|t] = (1 - a) \cdot Ev[D_i|t - 1] + a \cdot v(t). \quad (3b)$$

If deck D_i was not chosen, then its expectancy remains unchanged. This learning model produces expectancies that are a weighted average of the past valences. The weight given to each past valence decreases as a function of the lag (number of choices back in time) of the experience. Recently experienced valences receive more weight than more remotely experienced valences. In Equation 3b, the parameter *a* represents the updating rate ($0 < a < 1$). Large rates produce fast changes, strong recency effects, short associative memories, and rapid forgetting. Small rates produce slow changes, weak recency effects, long associative memories, and slow forgetting.[3]

Probabilistic choice. The choice made on each trial is a probabilistic function of the expectancies associated with each deck. The probability of choosing deck D_i is an increasing function of the expectancy for that deck and a decreasing function of the expectancies for the other decks. This principle is captured by the following ratio-of-strengths rule for choice probabilities (cf. Luce, 1959):

$$Pr[D_i|t + 1] = \frac{e^{Ev[D_i|t]\theta(t)}}{\sum_{j=1}^{4} e^{Ev[D_j|t]\theta(t)}}. \quad (3c)$$

The parameter $\theta(t)$ in Equation 3c is called the sensitivity parameter. It determines the sensitivity of the choice probabilities to the expectancies. If the sensitivity parameter is set to zero, then choices are completely random and independent of the expectancies. As the sensitivity parameter increases in magnitude, the choices become more strongly dependent on the expectancies. For very large values of the sensitivity parameter, choice becomes deterministic and the deck producing the largest expectancy is chosen with certainty. We assume that the decision maker's sensitivity to the expectancies changes with experience. For healthy individuals, the sensitivity may initially start out at a low value, so that choice is almost random, but as training progresses, their sensitivity may increase so that choice becomes more strongly influenced by expectancies. Brain-damaged patients may get tired and lose concentration as training progresses, and thus their sensitivity may actually decrease with training. This hypothesis is formalized by assuming that sensitivity changes over trials according to the following power function

$$\theta(t) = (t/10)^c. \quad (3d)$$

Positive values of *c* indicate increasing sensitivity (less random), and negative values indicate decreasing sensitivity (due to boredom or fatigue). In sum, the expectancy–valence model has three parameters: one is the attention weight, *w,* given to losses as opposed to gains; the second is the learning rate parameter, *a;* and the third is the parameter, *c,* that controls the changes in the sensitivity over training.

Baseline Model

In addition to the three cognitive models above, we used a baseline model as a standard for comparison with each model. The baseline model is a statistical model rather than a cognitive model, and it assumes that a multinomial process generates choices with constant probabilities across trials. The probability of choosing from deck D_1 is denoted p_1, the probability of choosing from deck D_2 is denoted p_2, the probability of choosing from deck D_3 is denoted p_3, and finally the probability of choosing from deck D_4 is $p_4 = 1 - (p_1 + p_2 + p_3)$. Like the three cognitive models described above, the baseline model has three parameters that must be estimated from the data, p_1, p_2, and p_3. Unlike the cognitive models, this model simply assumes that the choices are independently and identically distributed across trials. Nevertheless, the baseline model is a strong competitor because it can perfectly reproduce the marginal choice probabilities, pooled across trials. Thus, a cognitive model can perform better than this model only if it succeeds in explaining how the choices depend on the sequence of trial-by-trial feedback.

[3] Note that "ideal" learning as implemented by the optimal Bayesian–expected utility model assumes equal weighting of all past events and no forgetting of past experiences. The expectancy valence model can approximate this ideal learning by using very small updating rates, which would produce approximately equal weighting of past experiences and a long memory for past outcomes. Large updating rates give current experiences too much weight and cause rapid forgetting of past outcomes.

Model Comparison Analyses

Maximum Likelihood Estimation

The three parameters from each of the four models described above were estimated separately for each decision maker using maximum likelihood methods. First we need to define the maximum likelihood criteria. Define $[y_1(t)y_2(t)y_3(t)y_4(t)]$ as a vector representing the observed choice by a decision maker on trial t. If deck D_i was chosen on trial t, then the coordinate $y_i(t) = 1$; else $y_i(t) = 0$. Similarly, define $[P_1(t)P_2(t)P_3(t)P_4(t)]$ as a vector representing the corresponding predicted choice probabilities from a given model. Then the log likelihood of the observed sequence given the model predictions is defined as follows:

$$L_M = \sum_{t=1}^{100} \{y_1(t) \cdot \ln[P_1(t)] + y_2(t) \cdot \ln[P_2(t)] + y_3(t) \cdot \ln[P_3(t)] + y_4(t) \cdot \ln[P_4(t)]\}. \quad (4a)$$

The subscript M attached to the log likelihood, L_M, indicates the model that is used to compute the predictions.

The three parameters of each model are selected separately for each decision maker to produce the maximum log likelihood (maximizing Equation 4a) for that individual's choices. For the baseline model, the maximum likelihood estimates are simply the sample proportion of card choices from each deck for each decision maker. For the cognitive models, it is necessary to use a nonlinear parameter search program to find the parameters that maximize the log likelihood. In this case, we used the Nelder–Meade simplex algorithm available in the Matlab (MathWorks Inc., 2000) programming language. This algorithm is slower than some other nonlinear estimation algorithms, but it is more robust and less sensitive to local maxima than other algorithms, which is important for fitting individual choice data. The three parameters for each model were estimated for each individual using this method.

Model Comparisons Using the G^2 Criterion

The parameters that maximize the log likelihood for each model and person are used to make model comparisons. Each of the three cognitive models can be compared with the baseline model by computing differences in log likelihood as follows:

$$G^2 = 2(L_{\text{cognitive}} - L_{\text{baseline}}). \quad (4b)$$

If the two models being compared were nested so that one was a special case of the other, then G^2 would be chi-square distributed with a degree of freedom equal to the difference in the number of parameters. However, in this case the models are not nested and they contain the same number of parameters, so that standard chi-square tests are not possible. Furthermore, standard nonnested model comparison measures, such as the Akaike information criterion and the Schwartz Bayesian information criterion produce exactly the same result as Equation 4b, because there are no differences in number of parameters. Nevertheless, we can continue to use the G^2 statistic as a descriptive index of model performance. Positive values of the G^2 statistic indicate that a given cognitive model performs better than the baseline model. Furthermore, if the G^2 for Cognitive Model A exceeds the G^2 for Cognitive Model B, then Cognitive Model A performs better than Cognitive Model B for a particular individual.

Table 1 provides the means, medians, and standard deviations of the G^2 statistics for each model and group. First, note that the expectancy–valence model outperformed the baseline model for all groups. Additionally, the expectancy–valence model produced a positive G^2 for 78% of the individuals. However, the heuristic choice model and the Bayesian–expected utility model generally did no better or much worse than the baseline model.

Table 1 also shows that the expectancy–valence model performed much better than either of the other two cognitive models, with respect to the means and medians for each group. In addition, the expectancy–valence model produced larger G^2 values than the heuristic choice model for 76% of the individuals, and it produced larger G^2 values than the Bayesian–expected utility rule for 85% of the individuals, and both of these percentages are significantly different from 50% according to a z test of proportions ($z > 4.0$, $p < .01$). On the basis of these fairly clear cut results, we conclude that the expectancy–valence model is superior to both the heuristic choice model and the Bayesian–expected utility model.

Many other model comparisons were made to check the robustness of these conclusions (see Footnote 1). For example, the Bayesian–expected utility model was modified to allow the guessing probability to change as a function of training trials, but this produced inferior results as compared with the model with fixed probability of guessing. The heuristic choice model was modified to allow different probabilities of choosing between the disadvantageous decks, but this modification still performed much worse than the expectancy–valence model.

Figure 3 provides a comparison of the predictions derived from the expectancy–valence model with the observed proportion of choices averaged over subjects within each group. The thin line represents the observed data and the thick solid line represents the model predictions. As can be seen in this figure, the expectancy–valence model tracks the observed trends fairly closely, although there are some notable discrepancies in the fits for the Parkinson's disease group. The percentage of variance predicted by the model for the results shown in Figure 3 are 83%, 61%, and 51% for the

Table 1
Model Comparisons Based on G^2 Statistics

	G^2		
Group and model	*M*	*Mdn*	*SD*
Healthy			
Heuristic	6.55	0.78	13.59
Bayes–EU	8.19	0.12	21.20
Expectancy	21.72	11.30	25.79
Huntington			
Heuristic	0.69	−0.49	8.01
Bayes–EU	−6.70	−2.30	16.50
Expectancy	11.66	9.01	13.20
Parkinson			
Heuristic	4.32	1.94	16.55
Bayes–EU	0.96	−0.89	12.80
Expectancy	10.80	3.69	24.83

Note. Heuristic = heuristic choice; Bayes–EU = Bayesian–expected utility; Expectancy = expectancy–valence.

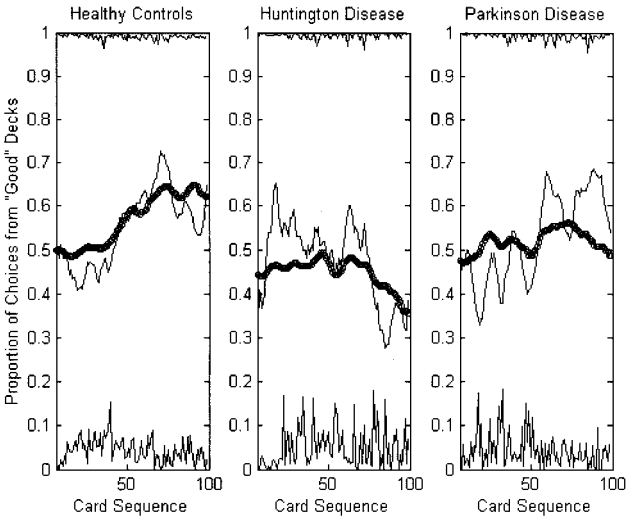

Figure 3. The thick smooth curve represents the predicted choice proportions derived from the expectancy–valence model, and the thin smooth curve represents the observed choice proportions, plotted as a function of training separately for each group.

healthy controls, Huntington's disease, and Parkinson's disease groups, respectively.

It is worth noting that although the expectancy–valence model was superior for all three groups, the fit of the model was best for the healthy controls and much lower for the other two patient groups. This partly reflects the fact that the latter two groups responded more randomly (reflected by choice probabilities closer to .50 in Figure 1) as compared with the healthy controls, which makes their behavior more difficult to predict.

Analysis of Parameter Estimates

The first issue of model selection has been resolved, and now it is appropriate to consider the second issue: interpretation of the model parameters. Recall that the expectancy–valence model has three model parameters: an updating rate parameter, *a*, which determines the memory for past consequences produced by each deck; a weight parameter, *w*, which determines the amount of attention allocated to gains as opposed to losses; and a threshold parameter, *c*, which determines the sensitivity of the choice mechanism to the expectancies.

On the one hand, differences among the groups in the updating rate parameter would indicate that performance deficits on the Bechara task result from the cognitive system rather than the motivational system. On the other hand, differences among the groups in the attention weight parameter would indicate that the performance deficits result from the motivational system. Finally, differences in the sensitivity parameter would indicate that response mechanisms (recklessness, impulsiveness, or both) are responsible for the performance deficit rather than cognitive or motivational mechanisms.

Table 2 presents the means, medians, and standard deviations for each parameter and group. Note that the updating rate is larger for the Huntington group as compared with the healthy controls and the Parkinson groups (the means are .58, .34, and .29, respectively). This means that the Huntington group was more reactive to recent information and forgot old information more rapidly.

The weight parameters do not differ very much among the three groups (the means are .40, .35, and .28 for the Huntington, healthy, and Parkinson groups, respectively). This indicates that the three groups allocated approximately the same amount of attention to losses.

The sensitivity parameter for the Huntington group was negative, as compared with the positive values for the healthy control and Parkinson groups (the means are −.89, .32, and .36, respectively). This means that the choices of the healthy control and Parkinson groups became more sensitive to their expectancies as training progressed. But the Huntington group became less sensitive with training, producing more random behavior, possibly because of fatigue and loss of concentration on the task.[4]

The differences observed in Table 2 were assessed more rigorously by computing confidence interval estimates of the group differences. The most important comparison is between the Huntington group and the other two groups (healthy control and Parkinson groups). The Huntington group had brain damage in a location that was predicted to affect decision processes, the Parkinson group had brain damage that was not predicted to affect decision processes, and, of course, the healthy controls had no special deficits.

Table 3 provides 95% confidence interval estimates of the differences between the Huntington's disease group and the average of all of the individuals in the other two control groups, separately for each parameter. As can be seen in this table, the difference on the updating rate parameter between the Huntington's disease group and the other two controls is positive (mean difference is .25). The 95% confidence interval for this difference lies entirely within the positive region, indicating that the difference is significant. The larger updating rate parameter for the Huntington's disease group indicates that these individuals were more reactive to recent information and forgot remote experiences more rapidly as compared to healthy individuals. Or in other words, healthy individuals, and to a lesser extent, the Parkinson group, had longer memories for past outcomes as compared with the Huntington's disease individuals.

The Huntington's disease group differs very little in terms of the attention weight parameter (mean difference is .08). The 95% confidence interval covers zero, and so the difference is not sig-

[4] In particular, for the healthy control group, the mean sensitivity started at $\theta(1) = (1/10)^{.32} = .48$ and ended at $\theta(100) = (100/10)^{.32} = 2.09$. If these values are inserted into Equation 3c, a valence difference of 1 unit favoring the advantageous deck would produce a choice probability for the advantageous deck equal to .62 at the beginning of training and a choice probability equal to .90 at the end of training. For the Huntington patient group, the mean sensitivity started at $\theta(1) = (1/10)^{-.89} = 7.76$ and ended at $\theta(100) = (100/10)^{-.89} = 0.13$. If these values are inserted into Equation 3c, a valence difference of 1 unit favoring the disadvantageous deck would produce a choice probability for the disadvantageous deck equal to .99 at the beginning of training and a choice probability equal to .53 at the end of training. Note, however, that the expected valence for the advantageous deck is not necessarily predicted to be near 1 at the beginning for the healthy controls, nor is it necessarily predicted to be near −1 for the Huntington patients. These numbers were used only to illustrate the effects of changing sensitivities on choice probabilities.

Table 2
Parameter Estimates From Expectancy–Valence Model

	Updating rate			Attention weight			Sensitivity		
Group	*M*	*Mdn*	*SD*	*M*	*Mdn*	*SD*	*M*	*Mdn*	*SD*
Healthy	0.34	0.16	0.39	0.35	0.32	0.30	0.32	0.68	1.58
Huntington	0.58	0.78	0.46	0.40	0.17	0.47	−0.89	−0.78	2.13
Parkinson	0.29	0.09	0.40	0.28	0.20	0.32	0.36	0.01	1.99

nificant. This indicates that the difference in performance on the Bechara task for these groups was not due to the Huntington's disease group placing too little weight on the losses or being insensitive to losses as compared with the other two groups.

The difference between the Huntington's disease group and the other two control groups is most pronounced for the sensitivity parameter (mean difference is −1.22). The 95% confidence interval for this difference lies entirely within the negative region, and so the difference is statistically significant. The positive average estimate of the sensitivity parameter for the healthy individuals indicates that their choices became more sensitive to their expectancies as training progressed. The negative average estimate of the sensitivity parameter for the Huntington's disease group indicates that they became less sensitive to their expectancies as training progressed. Apparently, the Huntington patients became tired or bored, lost concentration in the task, and randomly guessed more as training progressed.

It is also informative to compare a typical estimate of each parameter estimate obtained from the Huntington group to the distribution of estimates obtained from the healthy control and Parkinson groups. To do this, we took the median estimate of the Huntington group as the typical estimate. Then we computed the percentile for this typical value with respect to the estimates produced by the individuals from the healthy control and Parkinson groups. For the updating rate parameter, the percentile of a typical Huntington group member with respect to the control distribution was 77%. This means that a typical Huntington patient lies at the upper end of the distribution of updating rates produced by control participants. For the attention weight parameter, the percentile of a typical Huntington patient with respect to the control distribution was 34%. In other words, a typical Huntington patient lies at the lower end of the distribution of weights for losses produced by the control participants. Finally, for the sensitivity parameter, the percentile of a typical Huntington patient with respect to the control distribution was 19%. In other words, the typical Huntington patient lies near the bottom of the distribution of sensitivity parameters produced by the control participants.

Table 3
Confidence Intervals for Mean of Huntington Participants Minus Average of Healthy Control and Parkinson Participants

Parameter	Lower bound	Mean difference	Upper bound
Update rate	0.01	0.25	0.50
Weight	−0.12	0.08	0.29
Threshold	−2.31	−1.22	−0.14

Note. Table shows 95% confidence intervals. The middle column shows the mean difference between the two groups or, in other words, the center of the confidence interval. The left column indicates the lower bound of the 95% confidence interval, and the right column indicates the upper bound of the 95% confidence interval.

General Discussion

This article described the use of cognitive decision models for the assessment of the component processes responsible for performance deficits on the Bechara gambling task. This analysis was applied to a study reported by Stout et al. (2001), who examined performance deficits of Huntington's disease patients in comparison with healthy individuals and Parkinson's disease patients on the Bechara gambling task.

The first step in the cognitive analysis was to compare competing models for the task. This is a crucial first step for purposes of model validation. It is meaningless to evaluate a model in isolation, and the only way to build confidence in a model is to compare it with reasonable competitors. Three models were compared—a heuristic choice model, a Bayesian–expected utility model, and an expectancy–valence model—and the last clearly outperformed the first two models. This does not imply that no other versions of the former two exist. However, we attempted to explore a variety of different versions of each model, and the expectancy–valence model was superior among all of the versions that we examined.

The second step in the cognitive analysis was to estimate the three parameters of the expectancy–valence model separately for each individual. The updating rate parameter is a cognitive parameter representing the memory for past experiences; the weight parameter is a motivational parameter representing the amount of attention given to gains as compared with losses; and the sensitivity parameter is a response parameter representing the dependence of the choice mechanism on the decision maker's expectancies.

Differences in updating rate were found between the Huntington's disease group and the individuals from the healthy and Parkinson's disease groups. These results indicated that performance deficits partly result from the cognitive system. The Huntington patients were more reactive to recently experienced payoffs and forgot past experiences more rapidly than healthy controls or the Parkinson patients.

No differences between these groups were observed in the attention weights for gains and losses. This suggests that the performance deficits do not result directly from this part of the motivational system for Huntington patients.

Finally, the largest difference appeared in the sensitivity between the Huntington group and the individuals from the healthy and Parkinson groups. This indicates that the choice response mechanism (recklessness, impulsiveness, or both) is also partly

responsible for the performance deficit. The choices of Huntington patients became less sensitive to expectancies and more random as training progressed, suggesting that participants may have become tired and lost concentration on the task.

Stout et al. (2001) attempted to identify the source of the performance deficit observed with Huntington patients by correlating gambling task performance with measures derived from a standard clinical assessment tool called the Mattis Dementia Rating Scale (Mattis, 1988). They found that only memory deficits on the Mattis were correlated with decision deficits on this gambling task. This finding suggests that learning and memory processes were partly responsible for the deficit observed with the Huntington's disease group, in agreement with the present cognitive analysis.[5]

Bechara et al. (1997) also examined the relation between memory and performance on their gambling task. In contrast to the more long-term associative memory measure used by Stout et al. (2001), however, they specifically examined working memory. They reported dissociations between memory and decision deficits with patients who had suffered orbital frontal cortex damage. One obvious explanation for these contradictory findings is that the neurological source of the deficit for Huntington's disease individuals differs from that for individuals with orbital frontal cortex damage. Alternatively, it may be that the relation between memory and decision deficits depends on the type of assessment tool used to measure cognitive functioning. If the wrong tools are used, then dissociations may be observed.

These examples point to a limitation of traditional assessment tools to uncover the source of deficits in complex cognitive tasks—the type of cognitive process tapped by the assessment tool may not match the type of cognitive process required to perform the target task. On the one hand, Stout et al. (2001) used subscales from the Mattis Dementia Rating Scale that reflect long-term associative memory. On the other hand, Bechara et al. (1998) used a delayed matching test that primarily reflects working memory. Although both types of memory are probably involved to some extent, one type may be much more important than the other for determining performance on the gambling task.

The use of additional assessment instruments to decompose the basic processes responsible for deficient performance of a complex cognitive task is also subject to other sources of unreliability. First, complex tasks may rely on the interplay of several basic processes, but measurement of those basic processes alone may fail to elicit a deficiency in the basic process that occurs in more complex circumstances. Second, ancillary tasks may be ineffective in explaining behavior in a central complex task because the range and degree of sensitivity within the population being studied may vary dramatically for the tasks. This problem is especially likely when one task originated in the neuropsychological literature in conjunction with the behavior of people with brain damage and the other originated for research in cognitive neuroscience. Additionally, many ancillary tasks are quite complex in their own right, and the use of any single measure derived from a complex task may not be easily interpretable in the context of the basic process that it was chosen to represent.

Decomposition of complex tasks into basic processes has been a long-standing approach to identifying sources of poor performance. Neuropsychological research has clearly favored an approach in which performance relationships across tasks are studied to identify possible sources of deficit that may account for poor performance in complex tasks. This article has outlined a potential alternative, the use of cognitive modeling. A primary advantage of the cognitive modeling approach presented in this article is that the model provides parameter estimates of cognitive, motivational, and response processes that are directly estimated from the target choice behavior rather than from ancillary assessment instruments that have ill-specified relevance to the complex target task.

Another important advantage of the cognitive modeling approach is its potential application to very small samples. For example, cognitive neuroscientists often are forced to work with very small numbers of individuals who satisfy highly restrictive brain-damage specifications. The small sample size prohibits the use of traditional statistical analyses such as correlation analyses or *t* tests. However, the present approach remains applicable in this case. A model parameter is estimated from the choice pattern of a single brain-damaged individual. This same parameter is also estimated separately for each of a large sample of healthy controls. Then the estimate obtained from the brain-damaged individual can be referred to the distribution obtained from the healthy controls, and the likelihood of the brain damaged value arising from the healthy distribution can be computed.

In general, it is clear that the use of complex decision tasks to emulate real-world decision making has the potential to uncover the complex and fascinating interplay among various cognitive, motivational, and response processes. This is an area of intense interest to scientists and the general public alike. Much additional research will be necessary to flush out the advantages and limitations of using multiple assessment tools compared with cognitive modeling approaches to best discover how cognitive, motivational, and response processes interact in complex real-world decisions.

[5] It is important to point out that although the present analysis provides converging support for Stout's initial finding that memory plays a role in the performance deficit of Huntington patients, it also goes several important steps further. In addition, we found that the choice mechanism also differed between the Huntington group and the healthy controls: Healthy controls became more sensitive to expectancies whereas the Huntington patients became less sensitive. Furthermore, we found no differences in the amount of attention weight for losses given by healthy controls and Huntington patients. The instruments used by Stout provided no information regarding the latter two processes involved in the task.

References

Bartzokis, G., Lu, P., Beckson, M., Rapoport, B., Grant, S., Wiseman, E., & Londond, E. (2000). Abstinence from cocaine reduces high-risk responses on a gambling task. *Neuropsychopharmacology, 22,* 102–103.

Batchelder, W. H., & Riefer, D. M. (1999). Theoretical and empirical review of multinomial process tree modeling. *Psychonomic Bulletin and Review, 6,* 57–86.

Bechara, A., Damasio, H., & Damasio, A. R. (2000). Emotion, decision making and the orbitofrontal cortex. *Cerebral Cortex, 10,* 295–307.

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., & Anderson, S. (1994). Insensitivity to future consequences following damage to human prefrontal cortex. *Cognition, 50,* 7–15.

Bechara, A., Damasio, H., Tranel, D., & Anderson, S. W. (1998). Disso-

ciation of working memory from decision making within the human prefrontal cortex. *Journal of Neuroscience, 18,* 428–437.

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

Beck, A. T., Steer, R. A., & Garbin, M. G. (1988). Psychometric properties of the Beck Depression Inventory: Twenty-five years of evaluation. *Clinical Psychology Review, 8,* 77–100.

Berry, D. A. (1996). *Statistics: A Bayesian perspective.* New York: Duxbury Press.

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.

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.

Grant, S., Contoreggi, C., & London, E. D. (2000). Drug abusers show impaired performance in a laboratory test of decision making. *Neuropsychologia, 38,* 1180–1187.

Green, D. M., & Swets, J. A. (1966). *Signal detection theory and psychophysics.* New York: Wiley.

Luce, R. D. (1959). *Individual choice behavior: A theoretical analysis.* New York: Wiley.

Luce, R. D. (1999). *Utility of gains and losses: Measurement—theoretical and experimental approaches.* Hillsdale, NJ: Erlbaum.

MathWorks Inc. (2000). Matlab: The language of technical computing [Computer software]. Natick, MA: Mathworks Inc.

Mattis, S. (1988). *Dementia Rating Scale: Professional manual.* Odessa, FL: Psychological Assessment Resources.

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

Myung, I. J., & Pitt, M. A. (1997). Applying Occam's razor in modeling cognition: A Bayesian approach. *Psychonomic Bulletin and Review, 4,* 79–95.

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

Petry, N., Bickel, W., & Amett, M. (1998). Shortened time horizons and insensitivity to future consequences in heroin addicts. *Addiction, 93,* 729–738.

Schmitt, W. A., Brinkley, C. A., & Newman, J. P. (1999). Testing Damasio's somatic marker hypothesis with psychopathic individuals: Risk takers or risk averse? *Journal of Abnormal Psychology, 108,* 538–543.

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.

Received April 2, 2001
Revision received September 24, 2001
Accepted March 27, 2002 ■

Correction to Patrick et al. (2002)

The article "Development and Validation of a Brief Form of the Multidimensional Personality Questionnaire," by Christopher J. Patrick, John J. Curtin, and Auke Tellegen (*Psychological Assessment,* 2002, Vol. 14, No. 2, pp. 150–163), contained an error.

On page 154, in Table 1, the MPQ–BF column for the "Has a happy disposition" item (second row) under the Wellbeing scale incorrectly reads "26, 97, *31*, *104.*" The correct numbers are 26, 97, *32*, *104.*