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# Risky decision making in Huntington's disease

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**Abstract**

In the clinical setting, Huntington's disease is associated with problems in judgment and decision making, however, the extent of these problems and their association with clinical characteristics have not been assessed. Recently, a laboratory-based simulated gambling task has been used to quantify similar decision-making deficits in ventromedial frontal lobe damaged participants. We hypothesized that participants with Huntington's disease (HD) would show deficits on this gambling task. For this study, 14 HD participants were asked to make 100 selections from four decks of cards with varied payoffs in order to maximize winnings of play money. They were compared to 22 participants with Parkinson's disease (PD) and 33 healthy controls. After an initial period in which participants had to learn contingencies of the decks, the HD group made fewer advantageous selections than the PD and control groups. In HD, the number of advantageous selections in the gambling task was correlated with measures of memory and conceptualization but not disinhibition. Thus, people with HD may have had difficulties learning or remembering win/loss contingencies of the decks, or they may have failed to consistently take these into account in their card selections. These findings are consistent with current models of frontal-subcortical brain circuits and behavior. (*JINS*, 2001, *7*, 92–101.)

**Keywords:** Huntington's disease, Parkinson's disease, Decision making

## INTRODUCTION

Huntington's disease (HD) is an autosomal dominant neurodegenerative disease that causes preferential loss of neurons in the caudate nucleus and putamen due to the destruction of medium spiny neurons (Albin et al., 1989). Because the HD mutation has complete penetrance, each offspring of a person with the HD gene has a 50% chance of inheriting the abnormal allele and developing the progressive motor, cognitive, and personality symptoms that are associated with the disease (Brandt & Butters, 1996). HD has an impact on all individuals in a family because a person either develops HD or experiences the presence of HD in multiple relatives. A frequent complaint expressed in the clinical setting is that people with HD, regardless of their premorbid personalities, display poor judgment as well as various other changes in their personalities and social behavior.

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There is a growing body of empirical evidence dating back more than 30 years that documents a variety of behavioral disturbances in HD. Early studies have reported that the majority of people with HD presented to mental health or general medical settings with personality disturbance or psychiatric problems prior to manifesting the choreiform movements necessary to obtain a diagnosis of HD (Dewhurst et al., 1970). Among the symptoms noted to occur in HD are impulsivity, erratic behavior, aggression, irritability, apathy, emotional lability, reduced initiative, depression, anxiety, psychosis, and others (Burns et al., 1990; Cummings, 1995; Dewhurst et al., 1970; Hulvershorn et al., 1999; Jacobs & Huber, 1992; Litvan et al., 1998; Martin & Gusella, 1986; Mayeux, 1984; Paulsen et al., 1996). Perhaps related to these behavioral disturbances, an increase in levels of nonviolent crime has also been identified in men with HD compared to their nonaffected first degree relatives (Jensen et al., 1998). Personality and psychiatric functioning in HD have received far less attention than motor and cognitive functions; however, there is ample evidence of a wide variety of such behavioral disturbances in HD.

Parallels are often drawn between the behavioral disturbances in HD and those observed with damage to the frontal lobes (Cummings, 1993; Jacobs & Huber, 1992; Mega & Cummings, 1994). To the extent that similar behavioral disturbances occur in HD and frontal lobe damage, an anatomical basis for these similarities does exist. Cummings (1993) and others have suggested that the similarities between behavior after frontal cortex damage and behavior after damage to the basal ganglia are due to the connectivity of these anatomic structures within several frontal-subcortical circuits. These circuits have been described in nonhuman primates as projections from the frontal cortex to the caudate nuclei and putamen, which project to the globus pallidus and substantia nigra, which project to several thalamic nuclei, which then project back onto the frontal cortex at or near the location of origin for that circuit (Alexander & Crutcher, 1990; Alexander et al., 1986, 1990). Using this model, disruption of circuits *via* damage to subcortical structures would be sufficient to produce disturbances in the behaviors subserved by that circuit. Thus, in HD, damage to the basal ganglia may be responsible for creating frontal-like behavioral disturbances.

One difficulty in assessing the behavioral disturbances in people with damage to the frontal lobes is that standardized laboratory-based testing has been insensitive to many of the behavioral disturbances that occur in the course of everyday functioning. For example, E.V.R., the well-studied patient with ventromedial brain damage, reportedly exhibited deficits in judgment and decision making in his natural, everyday setting despite normal performance on judgment tasks in the laboratory (Eslinger & Damasio, 1985). Recently, Bechara and Damasio and their colleagues have reported a series of studies using a simulated gambling task in the laboratory which they believe may reflect the kind of impulsivity and judgment deficits reported to occur in daily life in people with frontal lobe damage (Bechara et al., 1994, 1996, 1997, 1998). Given the reported similarities between behavioral disturbances in frontal lobe damage and HD, we hypothesized that people with HD would also show deficits in performance on this simulated gambling task.

In the current study, we examined the performance of people with HD, who had only relatively mild cognitive impairment, on the gambling task described by Bechara et al. (1994). For this task, research participants were presented with four decks of cards and told to try to maximize their profits on a $2000 loan of play money by selecting cards from the decks. Each card selection resulted in either winning money or winning money coupled with losing money. The four decks were designed such that over the course of several selections, two of the decks resulted in a net win and the other two decks resulted in a net loss. To test the hypothesis that HD is associated with deficits on the simulated gambling task, we compared the performance of people with HD to similarly aged and educated healthy control participants.

In addition to the HD and healthy control groups, we also studied a group of people with Parkinson's disease (PD) and an older healthy control group that was of similar age, education, and degree of cognitive impairment to the PD group. Like HD, PD is associated with preferential damage at a subcortical level of the frontal-striatal brain circuits. The specific areas of preferential damage in the basal ganglia differ, however, in HD and PD. In HD the initial loss of striatal cells occurs primarily in the caudate nuclei, whereas, in PD cell loss preferentially affects the dopaminergic neurons of the substantia nigra pars compacta, which project primarily to the putamen (Albin et al., 1989; Olanow et al., 1998). HD and PD also differ in some of their typical behavioral features. In contrast to HD, PD is not typically associated with disinhibited, impulsive behavior and poor judgment. Rather, PD is associated with a cautious, conservative nature (Levin & Katzen, 1995). Thus, if gambling-task performance is largely affected by impulsive behavior in which future consequences have little impact, we would hypothesize that HD, but not PD, would be associated with poorer gambling-task performance.

There were three main goals in the current study. First, we wanted to determine whether people with HD perform more poorly on the simulated gambling task than do healthy and PD comparison groups. Second, we were interested in examining relationships between the number of cards selected from winning decks and the severity of cognitive decline in HD and PD. Finally, we wished to examine relationships between selection of cards from winning decks and a family member's rating of the participant's frontal lobe-type behavioral disturbance and disinhibition.

## METHOD

### Participants

Seventy-three adult research volunteers were tested with the gambling task as part of their participation in a larger neuropsychological study of HD and PD. Participants with HD were recruited from the Huntington's Disease and Movement Disorder Clinics in the Department of Neurology at Indiana University School of Medicine (Indianapolis, IN). Participants with PD were recruited from the Movement Disorders Clinic at Indiana University School of Medicine and from neurologists and primary care physicians and support groups in Bloomington, Evansville, and Indianapolis, IN. The majority of the healthy controls were spouses, family members, or friends of the PD and HD participants, and the remainder were recruited through efforts in the community such as clubs frequented by older adults. The study was approved by Institutional Review Boards at the Indiana University School of Medicine in Indianapolis and Indiana University in Bloomington. Written informed consent was obtained for all participants.

Potential participants were excluded if they reported current drug or alcohol abuse, less than an eighth grade education, an inability to write or communicate, a major neurological diagnosis other than HD or PD (e.g., stroke,

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epilepsy, head injury with loss of consciousness for greater than 30 min or requiring medical attention), or reported a major psychiatric diagnosis such as schizophrenia or bipolar disorder. Because of the level of difficulty of the testing, individuals with more severe dementia as indicated by a Mattis Dementia Rating Scale (MDRS) Score $< 100$ (Mattis, 1988) were also excluded. Additionally, all examinees were interviewed with the Hamilton Depression Interview (Hamilton, 1967) and were excluded if they scored greater than 14 (mild depressive symptoms). Four potential participants were excluded on the basis of dementia severity and/or depression. This left a total of 69 participants, including 14 with HD, 22 with PD, and 33 healthy controls.

As expected, given the characteristic ages of onset in HD and PD, the two groups differed significantly in age, $t(34) = -6.42$, $p < .001$ (see Table 1). Therefore, separate overlapping control groups were constructed from the 33 healthy control participants to provide age-matched comparison groups for each patient group. There were no significant differences between the younger healthy control group (YHC) and HD on age, $t(30) = 0.16$, $p = .87$, education, $t(30) = -0.96$, $p = .32$, or sex, $\chi^2$ (3, $N = 32$) $= 0.65$, $p = .42$. Similarly for the older healthy control (OHC) and PD groups, age, $t(44) = 0.37$, $p = .85$ and education, $t(44) = 0.32$, $p = .58$ were not significantly different. The OHC group included more males than did the PD group, $\chi^2(3, N = 46) = 4.18$, $p = .04$. PD and HD did not significantly differ from one another on education, $t(34) = 1.17$, $p = .25$, sex, $\chi^2$ (3, $N = 36$) $= .10$, $p = .76$, or MDRS total, $t(35) = -0.38$, $p = .70$. According to Shay et al.'s criteria (1991) for the MDRS, 42% of the HD sample and 32% of the PD sample met criteria for mild dementia (MDRS scores between 103 and 130) while the remainder of each sample scored in the normal range on the MDRS (MDRS scores between 131–144). Participants did not receive a formal clinical evaluation for dementia, and thus only the MDRS cutoffs were available for allocating participants to demented and nondemented groups. Participants with HD and PD estimated their number of years since diagnosis which were then used to compute estimated age at diagnosis (see Table 1). One participant with HD had juvenile onset (age 15), while the remainder had adult onset. The juvenile onset participant also had the lowest MDRS score (104 points). For the PD group, the mean rating on the Hoehn and Yahr scale of disease severity (Hoehn & Yahr, 1967) was 2.6 (mild to moderate impairments; see Table 1), with a range from 2 (mild impairments) to 4 (severe impairments).

In the HD group, six participants were using antidepressants (fluoxetine hydrochloride), three were on anticonvulsants (phenytoin or clonazepam), and two were receiving neuroleptics (haloperidol). In the PD group, three participants were using antidepressants (fluoxetine hydrochloride, amitriptyline hydrochloride, nortriptyline hydrochloride), and two were receiving neuroleptics (haloperidol or thioridazine hydrochloride). Additionally, 16 of the PD participants were on carbidopa/levodopa along with other anti-Parkinsonian medications (selegiline hydrochloride or

**Table 1.** Demographic and clinical characteristics

| | YHC[a] ($n = 18$) | HD ($n = 14$) | OHC[a] ($n = 24$) | PD ($n = 22$) |
|---|---|---|---|---|
| Age (years) | | | | |
| Mean | 45.3 | 44.6 | 65.5 | 66.0 |
| *SD* | 10.6 | 11.7 | 10.7 | 8.3 |
| Range | 21–62 | 21–62 | 47–82 | 47–78 |
| Education (years) | | | | |
| Mean | 14.3 | 15.3 | 14.7 | 14.2 |
| *SD* | 2.1 | 2.3 | 2.4 | 2.9 |
| Range | 12–18 | 12–18 | 12–20 | 12–20 |
| Sex (male/female) | 9/9 | 9/5 | 7/17 | 13/9 |
| MDRS Total | | | | |
| Mean | 139.5 | 130.6 | 138.0 | 131.7 |
| *SD* | 2.2 | 10.1 | 4.6 | 7.6 |
| Range | 136–143 | 104–141 | 126–143 | 111–143 |
| Years since diagnosis | | | | |
| Mean | N/A | 4.1 | N/A | 7.7 |
| *SD* | N/A | 2.8 | N/A | 5.5 |
| Range | N/A | 0–10 | N/A | 1–27 |
| Estimated age at diagnosis | | | | |
| Mean | N/A | 40.5 | N/A | 58.3 |
| *SD* | N/A | 10.8 | N/A | 7.6 |
| Range | N/A | 15–55 | N/A | 46–71 |
| Hoehn and Yahr scores[b] | | | | |
| Mean | N/A | N/A | N/A | 2.6 |
| *SD* | N/A | N/A | N/A | .7 |
| Range | N/A | N/A | N/A | 2–4 |
| FLOPS[c,d,e,f] | | | | |
| Total | | | | |
| Mean | 72.4 | 102.4 | 74.1 | 83.6 |
| *SD* | 12.9 | 23.6 | 15.8 | 20.4 |
| Range | 59–102 | 50–138 | 57–125 | 54–126 |
| FLOPS Scale A | | | | |
| Mean | 22.1 | 33.6 | 23.4 | 27.5 |
| *SD* | 5.4 | 8.6 | 7.2 | 6.6 |
| Range | 14–34 | 14–42 | 14–46 | 16–39 |
| FLOPS Scale D | | | | |
| Mean | 21.7 | 27.5 | 21.3 | 21.7 |
| *SD* | 4.8 | 7.3 | 3.7 | 5.8 |
| Range | 15–31 | 16–41 | 15–26 | 15–33 |
| FLOPS Scale E | | | | |
| Mean | 28.7 | 41.3 | 29.3 | 34.8 |
| *SD* | 8.1 | 11.6 | 8.2 | 11.1 |
| Range | 20–46 | 20–55 | 20–54 | 21–64 |

*Note*. YHC: Younger Healthy Controls; HD: Huntington's disease; OHC: Older Healthy Controls; PD: Parkinson's disease; FLOPS: Frontal Lobe Personality Scale; MDRS: Mattis Dementia Rating Scale; and *SD*: standard deviation.
[a]YHC and OHC are overlapping groups constructed from a pool of 33 healthy control participants.
[b]Hoehn and Yahr data were unavailable for two participants ($n = 20$).
[c]For YHC, $n = 13$ as FLOPS data were missing for five participants.
[d]For HD, $n = 11$ as FLOPS data were missing for three participants.
[e]For OHC, $n = 19$ as FLOPS data were missing for five participants.
[f]For PD, $n = 17$ as FLOPS data were missing for five participants.

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pergolide), and two patients were maintained on carbidopa/levodopa alone. Six HD and four PD participants were receiving no psychotropic or neurologic medications.

## Materials

Four decks of cards with 40 cards in each deck were constructed from laminated plain white card stock for the simulated gambling task. In order to assure that no win or loss information could be viewed from the back of the card, a masking design was printed on the back side. Dollar amounts of the winnings and losses were printed on the center of the face of each card. Cards were printed at the top with either "WIN $100" or "WIN $50". On some of the cards, losing amounts from $0 to $1250 (e.g., "LOSE $1250") were printed below the winnings. Thus, on cards with both winnings and losses printed on them, the net gain or loss for that card was based on the difference between the win and loss amounts.

For each deck, cards were arranged in a predetermined order. Two of the decks of cards yielded a $100 win with each card chosen, and the other two decks yielded a $50 win for each choice. Despite having a higher card-by-card winning amount, the decks yielding the $100 win for each card were disadvantageous across a number of selections. That is, across ten choices from the $100 decks, participants would lose $1250 while winning only $1000, resulting in a net loss of $250. In contrast, the $50 decks were advantageous overall. Across ten choices in these decks, participants would lose $250 while winning $500, for a net win of $250.

The two advantageous decks differed from each other in the frequency of losses as did the two disadvantageous decks. That is, one of the advantageous and one of the disadvantageous decks had high frequency losses (5 of 10 draws) while the other two decks had infrequent losses (1 of 10 draws; see Table 2). This frequency of loss factor was not analyzed in tests of the main hypotheses of the study. To our knowledge, none of the previously published studies using this task reported analyses of the frequency of loss factor (Bechara et al., 1994, 1996, 1997, 1998). We believe that this factor was included when the task was developed only to enhance the complexity of the deck contingencies and overall task difficulty. For the sake of completeness, we did examine whether the frequency of losses was associated with diagnosis in our study and also characterized the overall effect of loss frequency in our sample. We found no relationship between diagnosis and loss frequency, but did observe that our participants showed a preference for the larger, infrequent losses than for the smaller, more frequent losses.

For the task, play money was exchanged with the participant after each card selection on the basis of the amount of winnings and losses printed on that card. The play money, which we constructed in the laboratory, consisted of $25, $50, and $100 bills, each printed on a different color paper (white, yellow, and pink, respectively). The dollar value of the play money was printed on all four corners as well as being spelled out on the top and bottom of the bill. Bills were printed on both the front and back.

## Design and Procedure

The four decks of cards were placed face down in a row in front of the participant. A randomly generated participant number determined the position of the various decks from left to right with four possible orders. A preliminary data analysis indicated no significant effect of deck order, therefore, we did not further address deck order in our analyses. A standard set of instructions was then read to the participant. Participants were told that they would be given $2000 in play money, and that the object of the game was to make as much money as possible. They were instructed to make a series of card selections from the decks in front of them and told that they would either win or lose some money with each selection. Participants were allowed to draw only the

**Table 2.** Winnings and losses associated with the first ten cards from each deck[a]

| Card # | Deck A | Deck B | Deck C | Deck D |
|---|---|---|---|---|
| 1 | Win $100 | Win $100 / Lose $0 | Win $50 | Win $50 / Lose $0 |
| 2 | Win $100 | Win $100 / Lose $0 | Win $50 | Win $50 |
| 3 | Win $100 / Lose $150 | Win $100 | Win $50 / Lose $50 | Win $50 / Lose $0 |
| 4 | Win $100 | Win $100 / Lose $0 | Win $50 | Win $50 |
| 5 | Win $100 / Lose $300 | Win $100 | Win $50 / Lose $50 | Win $50 |
| 6 | Win $100 | Win $100 / Lose $0 | Win $50 | Win $50 / Lose $0 |
| 7 | Win $100 / Lose $200 | Win $100 | Win $50 / Lose $50 | Win $50 / Lose $0 |
| 8 | Win $100 | Win $100 | Win $50 | Win $50 |
| 9 | Win $100 / Lose $250 | Win $100 / Lose $1250 | Win $50 / Lose $50 | Win $50 |
| 10 | Win $100 / Lose $350 | Win $100 | Win $50 / Lose $50 | Win $50 / Lose $250 |
| Net Win/Loss | Lose $250 | Lose $250 | Win $250 | Win $250 |

*Note*. Net Win/Loss refers to the total of the winnings and losses over ten selections from a particular deck.
[a]For a complete table of contingencies for the gambling task, see Bechara et al. (1994).

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topmost cards in the decks, and were told that the examiner would inform them when the task was completed (after 100 selections). The examiner recorded selections card by card for later analysis.

### Additional Measures

The MDRS was administered and scored according to standard procedures, except that all test items were administered to every patient. The MDRS is a measure of global cognitive function designed for screening of patients with possible dementia. Dementia severity is assessed using a composite score from five domains of cognitive function including attention, initiation and perseveration, construction, conceptualization, and memory for a total of 144 possible points. *T* tests comparing HD and PD groups on MDRS total and each of the five subscales indicated no significant differences between the groups (for all, $p > .10$).

The Frontal Lobe Personality Scale (FLOPS; Grace et al., 1999; Paulsen et al., 1996) is a 46-item behavior rating scale designed to identify and quantify behavioral syndromes associated with frontal lobe brain damage. The FLOPS Family Form was filled out at home after the testing session by a close family member of the participant, and then was returned by mail to the investigators. The return rate for the questionnaires was 78%. A five-point Likert Scale is used for rating the frequency of behaviors on items allocated into three subscales corresponding to three frontal behavior syndromes, apathy, disinhibition and emotional dysregulation, and executive dysfunction. The total possible score is 230 based on the sum of the three subscales. HD participants differed from PD participants on the FLOPS total, $t(34) = 2.21$, $p = .03$ as well as on the apathy subscale, $t(34) = 2.08$, $p = .04$, and the disinhibition subscale, $t(34) = 2.23$, $p = .03$, but not on the executive dysfunction subscale, $t(34) = 1.59$, $p = .12$, with HD participants rated as having more severe behavioral disturbances (higher scores; see Table 1).

### Data Analysis

To compare the performance of HD to YHC and PD to OHC, two separate one-way repeated-measures analyses of variance (ANOVAs) were computed using the number of choices from advantageous decks as the dependent measure. The factors were diagnosis (DIAG) and blocks of 25 trials (BLOCK). To test the main hypotheses of the study, that HD would perform more poorly than YHC and PD would perform similarly to OHC, we examined the main effects of DIAG in the latter two blocks of the task. So that HD and PD could be compared directly on gambling-task performance, we computed a repeated-measures analysis of covariance (ANCOVA). Age and education were used as covariates because the average ages the HD and PD groups were significantly different and education levels tended to be slightly lower in PD. Only the latter two blocks were considered for ascertaining group differences because in a preliminary analysis of the data looking at performance across the four blocks and across the four groups, we noted that nearly all participants performed near the chance level in the first two blocks. We attributed this effect to learning the contingencies of the decks. Only in the third and fourth blocks were participants showing evidence of having identified advantageous decks.

In a set of secondary analyses, we examined the relationship between the number of advantageous card selections summed across the third and fourth blocks and the severity of cognitive impairment on the MDRS using Pearson correlations. HD, PD, and the total healthy groups were examined separately. Similarly, we examined the relationship between the sum of advantageous card selections in the blocks 3 and 4 and the FLOPS total and subscale scores.

## RESULTS

The HD group performed worse on the gambling task than did the PD group or the healthy controls. The major findings from the ANOVA are presented first for HD and YHC, then for PD and OHC, and finally, comparing HD to PD. An alpha level of .05 was used for determining significance of all statistical tests.

### Patient Groups Compared to Matched Control Groups

In the ANOVA comparing HD to YHC, results indicated no significant main effect of BLOCK, $F(1,30) = 2.46$, $p = .07$, or DIAG, $F(1,30) = 2.44$, $p = .13$; however, consistent with our hypothesis, there was a significant interaction of BLOCK × DIAG, $F(1,30) = 4.88$, $p < .04$, which indicated that as the task progressed the YHC participants chose more cards from advantageous decks than did HD participants (see Figure 1). We examined this finding further by comparing HD and YHC on the total number of advantageous choices in blocks 3 and 4. There was a trend in block 3, $t(30) = 1.74$, $p < .10$, and a significant effect in block 4, $t(30) = 2.12$, $p < .05$, indicating that participants with HD chose fewer cards from advantageous decks than did YHC during the final two trial blocks.

In the ANOVA comparing PD to OHC, results indicated a significant main effect for BLOCK, $F(1,44) = 17.26$, $p = .001$, indicating that both PD and OHC participants learned to take increasing numbers of cards from advantageous decks. There was no significant effect for DIAG, $F(1,44) = 0.15$, $p = .70$, nor any significant BLOCK × DIAG interaction, $F(1,44) = 0.60$, $p = .44$. Thus, the PD group did not perform differently from the OHC group, even when considering only the third and fourth blocks (see Figure 1). This was consistent with our main hypothesis regarding PD.

A repeated-measures ANCOVA comparing HD to PD using number of advantageous choices over the four blocks as the dependent measure and designating age and education level as covariates indicated no significant main effect

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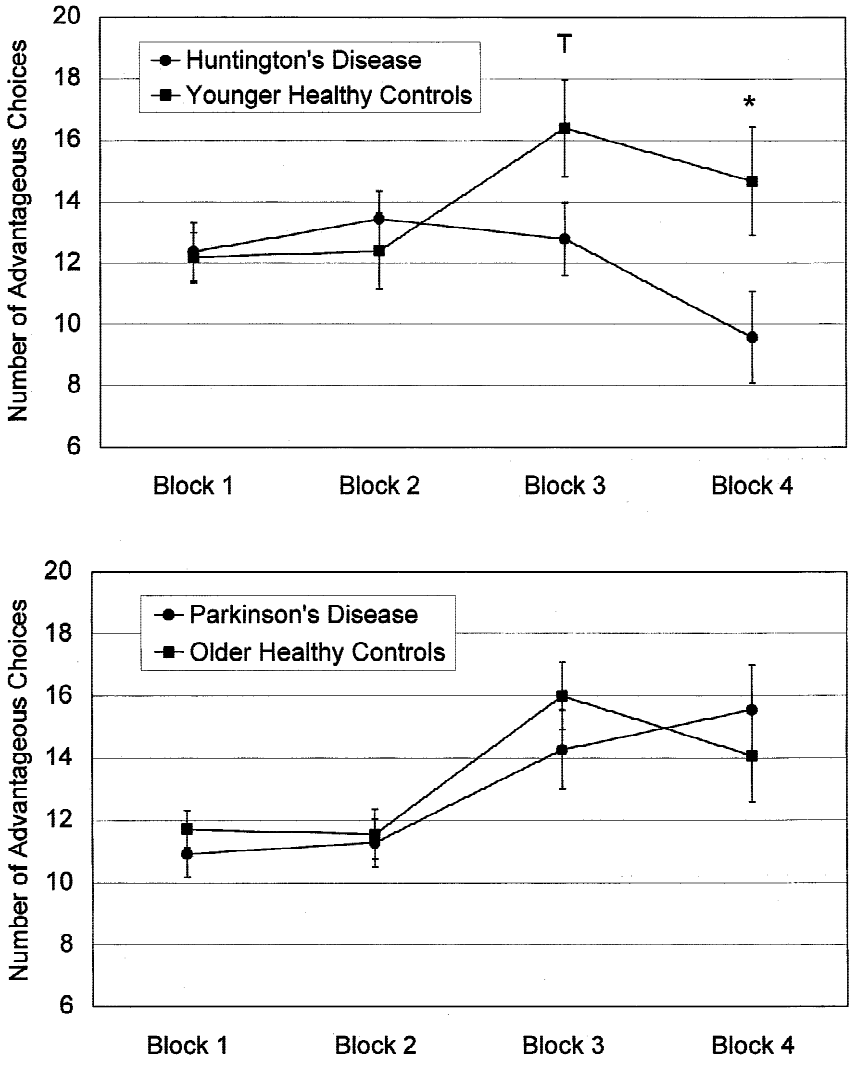


**Fig. 1.** The sum of choices made from advantageous decks (Decks C & D) over four blocks of 25 trials. T indicates a statistical trend at the $p < .10$ level, and "*" indicates significance at the $p < .05$ level.

for DIAG, $F(1,32) = 1.37$, $p = .25$; AGE, $F(1,32) = 1.36$, $p = .25$; or EDUCATION LEVEL, $F(1,32) = .21$, $p = .65$. There was also no significant effect of BLOCK, $F(1,32) = 0.00$, $p = .99$, indicating that when collapsed across subject groups there appeared to be no indication that participants learned to take increasing numbers of cards from advantageous decks. However, there was a significant BLOCK × DIAG interaction, $F(1,32) = 14.97$, $p = .001$, indicating that while the PD group selected more advantageous cards as the task proceeded, this was not true for the HD group.

In order to determine whether the findings above were influenced unduly by the two participants with HD and two participants with PD who were receiving neuroleptic treatment at the time of testing, the above analyses were repeated excluding data from these four participants. For all three contrasts (HD *vs*. YHC, PD *vs*. OHC, HD *vs*. PD), the pattern of results was identical to the analyses including the neuroleptic-treated participants. We also examined the data for the participant with HD who had juvenile onset to ascertain whether it was an outlier or extreme case, and may therefore be having a large effect on the results. We found that this case was not an extreme in the gambling data, and re-analysis with this case excluded yielded an identical pattern of results.

## Correlations of Simulated Gambling to General Intellectual Functioning and Frontal Behavioral Ratings

In HD, the number of advantageous choices summed across blocks 3 and 4 was not significantly correlated with severity of global cognitive dysfunction measured by MDRS total score, although a trend was present for higher advantageous choices correlating with less severe cognitive dysfunction (see Table 3). Examination of MDRS subscales indicated that performance in blocks 3 and 4 was significantly correlated with the MDRS conceptualization and MDRS memory subscales (see Table 3). These findings sug-

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**Table 3.** Correlations of the sum of advantageous selections in the third and fourth blocks with Mattis Dementia Rating Scale and Frontal Lobe Personality Scale

| | HD ($n$ = 14) | PD ($n$ = 22) | HC ($n$ = 33) |
|---|---|---|---|
| MDRS | | | |
| Total | .47[d] | .17 | −.12 |
| Attention | .10 | −.29 | .13 |
| Initiation | .02 | .30 | .05 |
| Construction | −.24 | .06 | −.19 |
| Conceptualization | .63[e] | .07 | −.23 |
| Memory | .61[e] | .05 | .16 |
| FLOPS[a,b,c] | | | |
| Total | −.33 | −.07 | −.01 |
| Scale A | −.37 | −.12 | −.26 |
| Scale D | .04 | −.05 | −.31 |
| Scale E | −.42 | −.03 | .03 |

*Note*. MDRS: Mattis Dementia Rating Scale; FLOPS: Frontal Lobe Personality Scale; HD: Huntington's disease; PD: Parkinson's disease; HC: Healthy Control.
[a]For HD $n$ = 11, data missing for three participants.
[b]For PD $n$ = 17, data missing for five participants.
[c]For HC $n$ = 24, data missing for nine participants.
[d]$p < .10$.
[e]$p < .05$.

gest that those participants with HD who perform relatively better on the gambling task also have relatively better conceptual and memory abilities. There were no significant associations with the attention, construction, or initiation and perseveration subscales of the MDRS (all $p > .10$; see Table 3). In contrast, in PD, the number of advantageous choices summed across blocks 3 and 4 was not significantly associated with severity of global cognitive dysfunction measured by MDRS total score or any of the five MDRS subscales (all $p > .10$; see Table 3).

In HD, the number of advantageous choices summed across blocks 3 and 4 was not significantly correlated with FLOPS total or any of the FLOPS subscales, including apathy, disinhibition, and executive function (see Table 3). Similarly, in PD, the number of advantageous choices in the blocks 3 and 4 did not correlate with FLOPS total or any of the FLOPS subscales, including apathy, disinhibition, and executive function (see Table 3).

## DISCUSSION

Results of this study indicated poorer performance on the simulated gambling task in a group of mildly-to-moderately demented research participants with HD. These findings were similar to results reported in participants with ventromedial frontal lobe damage (Bechara et al., 1994, 1996, 1997, 1998). In contrast, the PD group performed similarly to healthy comparison groups despite having a similar level of cognitive impairment to the HD group. These findings are consistent with the major hypotheses of the study and with previous findings that damage at the subcortical level of the frontal-subcortical brain circuits can produce behavioral changes similar to those observed when damage is located within frontal cortex itself.

The pattern of results across four blocks of 25 card selections suggests that the healthy control and PD participants learned which were the advantageous decks and that they acted according to this knowledge as the task progressed. Research on decision making has shown that once an advantageous pattern of behavior is established, it is common for participants to continue to sample alternatives (Busemeyer & Myung, 1992; Busemeyer & Townsend, 1993). Consistent with this idea, we observed that our PD, healthy control, and HD participants continued to sample from disadvantageous decks throughout the task. Also consistent with Busemeyer and colleagues, our findings in PD and healthy controls indicated that these sampling behaviors tended to be brief. That is, when negative reinforcement was encountered, PD and healthy control participants quickly returned to the choices that were known to be "safe". In contrast, findings from the HD group suggested that HD participants either did not learn which decks were advantageous, or despite knowledge of which decks were advantageous, they continued to make frequent selections from disadvantageous decks. Interestingly, in administering the gambling task, we noted that several participants with HD indicated during the task that one or more of the decks were "bad", yet they continued to make repeated selections from that deck.

There are several possible interpretations of our findings. The results of correlations between gambling-task performance and MDRS performance suggest that memory and conceptual deficits may underlie the poor performance of HD participants. In HD, MDRS memory and conceptualization subscale scores were significantly correlated with the number of advantageous selections in the gambling task. In contrast, performance on the gambling task in the PD group was not correlated with any of the measures of cognitive impairment from the MDRS. HD participants may have acted primarily according to the knowledge that two of the decks offered a card-by-card win of $100 while the other two decks offered consistent winning amounts of only $50. These deck contingencies occur consistently and would therefore be more easily learned and remembered than would the long-term consequences of selection from a particular deck. An inability to learn or remember which decks are advantageous may be caused by any of several forms of explicit and implicit learning and memory which are known to be impaired in HD (Brandt & Butters, 1996). For example, given the high level of complexity of the task and the impossibility for participants to recall the precise results of each card selection, participants who perform well in the task may rely on some form of implicit learning to guide response selection. This is consistent with a report by Bechara et al. (1997) that normal research participants selected at higher rates from advantageous decks even before they are able to verbalize which of the decks were advantageous.

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In addition to problems with learning and memory, poor task performance may occur because of poor judgment. That is, our HD participants may have learned contingencies normally but failed to apply that knowledge. Such behavior could occur because of difficulty in simultaneously considering both the short- and long-term outcomes of the task or because there was a relatively greater attraction to short-term outcomes than long-term outcomes. In this task, higher short-term rewards were disadvantageous over the long-term, while lower short-term rewards were advantageous over the long term. Thus, the design of this task confounds short-term and long-term outcomes, precluding a direct test of the contributions of rewards and punishments in the short- and long-term contexts. Bechara et al. have suggested that in their ventromedial frontal lobe damaged sample, poor gambling-task performance was due to a disregard of future outcomes (Bechara et al., 1994, 1996, 1997, 1998). However, this issue must be tested empirically in future studies using a modified gambling-task design.

Alternatively, HD participants may have performed poorly on the gambling task because of disinhibition. That is, they may have acted on impulse to the short-term or immediate outcomes rather than carefully considering the long-term consequences. The HD group was rated on the FLOPS as being significantly more disinhibited than the PD group, although gambling-task performance was not correlated in either group with disinhibition levels measured on the FLOPS. Thus, these data do not provide support for a role of disinhibition in gambling-task decrements in HD. In future studies, it may be useful to select a subset of items from the FLOPS and other personality scales that are more specifically relevant to behavioral disturbances that could lead to faulty decision making. In addition, the use of neuropsychological rather than behavioral rating instruments to measure disinhibition, such as a "go–no-go" task, may be helpful for examining possible relationships between gambling task performance and disinhibition. Finally, a larger sample size may reveal additional small effects and thus help to clarify relationships among these behaviors.

One caveat for interpreting the results from the current study is that because each deck contained only 40 cards, it was possible for participants to deplete the cards in their most preferred decks and be forced to make the remainder of card selections from nonpreferred decks. In our data, 36% of the HD, 39% of the PD, and 48% of the healthy control participants depleted at least one of the decks. The majority of the healthy controls and PD who depleted cards in one of the decks depleted an advantageous deck (75% and 63%, respectively), while in contrast, 80% of HD who depleted one deck depleted a disadvantageous deck. These findings suggest that given an unlimited supply of cards in each deck, even greater differences between the performance of HD participants and those of healthy controls and PD might be found.

One limitation of the current study is the relatively small number of demented participants and mild degree of dementia in both groups. Addition of more severely demented participants in both groups would allow greater generalization of these data to HD and PD populations, and may also reveal a significant effect in the PD group. Nonetheless, using MDRS equivalent groups, the current data do suggest that, relatively speaking, mild dementia in HD is more likely than in PD to impair gambling-task performance.

Given the focus of the study on describing gambling-task performance in HD and its relationship to dementia severity and the neurobehavioral syndrome of disinhibition, data collection for the current study was not optimized for describing associations of implicit memory, explicit memory, and conceptualization, to gambling-task performance. However, *post-hoc* analyses of the data that we did collect strengthen the findings of the association of memory and conceptualization from the MDRS. For example, using a test of explicit memory using a verbal-list learning task, the California Verbal Learning Test (Delis et al., 1987), there was a significant correlation between word-list learning (number of words recalled over five learning trials) and the number of advantageous card selections in the gambling task (Stout, unpublished data). Similarly, the number of advantageous card selections was also associated with a measure of strategy and concept formation on the Twenty Questions Task (Laine & Butters, 1982) in which participants must try to identify which of 42 pictures presented on a stimulus card is the correct target by asking a series of "yes" or "no" questions (Stout, unpublished data). Further studies will be necessary to delineate the relative influences of different aspects of memory, response styles, and conceptual abilities on gambling-task performance in HD and other populations. The outcomes of such studies will have important implications for understanding decision processes and their consequences in people with cerebral damage.

Both HD and PD are associated with many documented abnormalities occurring at both subcortical and cortical sites (Forno, 1981; Olanow et al., 1998; Penney & Young, 1998). However, there are notable differences between HD and PD in the regional distributions of cerebral abnormalities and in the specific ways in which motor, cognitive, and personality symptoms manifest. Future studies that incorporate both behavioral and neural measures will allow inferences to be made about more specific neural correlates of performance decrements in the simulated gambling task.

In conclusion, the results of this study suggest that the HD participants had problems maintaining a pattern of advantageous choices across the task, similar to those experienced by participants with ventromedial frontal brain damage studied by Bechara et al. (1994). While definitive interpretations of this finding await further studies using different task conditions, our findings are consistent with current views of frontal-subcortical brain circuits and behavior in general, and with the idea that damage at any of several levels of these circuits may disrupt behavior in a risky decision-making context. The findings also suggest that, similar to the ventromedial frontal lobe damaged participants studied by Bechara et al. (1994), assessment of the "real-life" decision-making problems in HD may be enhanced by using

a simulated gambling task. However, preliminary findings in the current study suggest the possibility that poor performance in HD may be more related to problems in learning and concept formation rather than a propensity for risk-taking behavior. Further studies will be essential for understanding the possible roles of learning, conceptualization, propensity for risk taking, or other factors in this simulated gambling task in HD.

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