

## PDF hosted at the Radboud Repository of the Radboud University Nijmegen

The following full text is a publisher's version.

For additional information about this publication click this link.

<http://hdl.handle.net/2066/159086>

Please be advised that this information was generated on 2017-12-05 and may be subject to change.

# Neural Mechanisms Underlying Compensatory and Noncompensatory Strategies in Risky Choice

Anna C. K. Van Duijvenvoorde<sup>1,2,3</sup>, Bernd Figner<sup>4</sup>, Wouter D. Weeda<sup>2,3</sup>,  
Maurits W. Van der Molen<sup>1</sup>, Brenda R. J. Jansen<sup>1,4,5</sup>, and Hilde M. Huizenga<sup>1,4,5</sup>

## Abstract

■ Individuals may differ systematically in their applied decision strategies, which has critical implications for decision neuroscience but is yet scarcely studied. Our study's main focus was therefore to investigate the neural mechanisms underlying compensatory versus noncompensatory strategies in risky choice. Here, we compared people using a compensatory expected value maximization with people using a simplified noncompensatory loss-minimizing choice strategy. To this end, we used a two-choice paradigm including a set of "simple" items (e.g., simple condition), in which one option was superior on all attributes, and a set of "conflict" items, in which one option was superior on one attribute but inferior on other attributes. A binomial mixture analysis of the decisions elicited by these items differentiated between decision-makers using either a compensatory or a noncompensatory strategy. Behavioral

differences were particularly pronounced in the conflict condition, and these were paralleled by neural results. That is, we expected compensatory decision-makers to use an integrated value comparison during choice in the conflict condition. Accordingly, the compensatory group tracked the difference in expected value between choice options reflected in neural activation in the parietal cortex. Furthermore, we expected noncompensatory, compared with compensatory, decision-makers to experience increased conflict when attributes provided conflicting information. Accordingly, the noncompensatory group showed greater dorsomedial PFC activation only in the conflict condition. These pronounced behavioral and neural differences indicate the need for decision neuroscience to account for individual differences in risky choice strategies and to broaden its scope to noncompensatory risky choice strategies. ■

## INTRODUCTION

An important goal of decision neuroscience is to identify the neural mechanisms underlying individuals' choices (Smith & Huettel, 2010; Sanfey, 2004, 2007). The dominant view in this field (Vlaev, Chater, Stewart, & Brown, 2011; Trepel, Fox, & Poldrack, 2005) is that of compensatory decision strategies (Tversky & Kahneman, 1974; Von Neumann & Morgenstern, 1944). Using these strategies, the decision-maker integrates attributes of options, namely gains, losses, and their probabilities into an overall index of value (often referred to as utility) and chooses the option with the highest integrated value. These decision strategies are "compensatory," because an option's weaknesses on one attribute can be compensated by strengths on other attributes. Behavioral decision research, however, has highlighted the role of individual differences in decision strategies and has shown that people may resort to a variety of noncompensatory strategies to simplify the decision process (Gigerenzer & Goldstein, 1996; Russo & Doshier, 1983; Tversky & Kahneman, 1974). Using a noncompensatory decision strategy, the decision-maker does not compare options on their integrated value but makes attribute-wise comparisons on a

limited set of attributes. These strategies are "noncompensatory," because they do not necessarily allow weaknesses on one attribute to be compensated by strengths on other attributes. Individuals may differ systematically in the propensity to use compensatory versus noncompensatory decision strategies (Scheibehenne, Rieskamp, & Wagenmakers, 2013; Jansen, van Duijvenvoorde, & Huizenga, 2012; Huizenga, Crone, & Jansen, 2007). These individual differences in decision strategies have critical implications for decision neuroscience and are therefore the main focus of the current study.

As decision neuroscience has focused on compensatory decision-making, it has identified neural substrates representing decision-making based on integrated value. Representations of such integrated value signals in risky choice are, for instance, expected value (EV), that is, an objective integration of probability and amount, and expected utility (EU), that is, a subjective integration of probability and amount. Classic decision theories predicted and explained choice behavior as aimed toward maximizing EV or EU. Consistently, neuroimaging studies have observed neural correlates of EV and EU in cortical and subcortical brain regions, including regions innervated by mesolimbic dopamine projections, such as the ventral striatum (VStr; Rolls, McCabe, & Redoute, 2008; Knutson, Taylor, Kaufman, Peterson, & Glover, 2005) and the ventromedial PFC (vmPFC), but also in regions such as the posterior cingulate

<sup>1</sup>University of Amsterdam, <sup>2</sup>Leiden University, <sup>3</sup>Leiden Institute for Brain & Cognition, <sup>4</sup>Radboud University Nijmegen, <sup>5</sup>Amsterdam Brain & Cognition Center

cortex (PCC; Mc Kell Carter, Meyer, & Huettel, 2010; Kable & Glimcher, 2007; Tom, Fox, Trepel, & Poldrack, 2007; Blair et al., 2006; see for a recent meta-analysis Bartra, McGuire, & Kable, 2013). Additionally, the lateral prefrontal and the parietal cortex have been implicated in value coding in macaques (Sugrue, Corrado, & Newsome, 2004; Platt & Glimcher, 1999), in value comparisons in humans (Hunt et al., 2012), and in numerical computations (Arsalidou & Taylor, 2011). However, it is unknown whether such integrated value representations are also present when people use noncompensatory decision strategies.

An integrated value signal may not be apparent in individuals using a noncompensatory strategy, because such a strategy does not entail the compensatory process of weighing and summing but instead may include an attribute-wise comparison. During the evaluation of options, the decision-maker may notice that some attributes make one option more attractive, whereas other attributes make the other option more attractive; thus, the different attributes may provide conflicting information (Rao et al., 2011; De Neys, Vartanian, & Goel, 2008; Tversky, Sattath, & Slovic, 1988). More specifically, as highlighted by Tversky and colleagues, a first step for a decision-maker is to examine whether one option dominates the other (i.e., is superior on all attributes). If dominance emerges, the decision-maker easily chooses the dominant alternative. However, if attributes provide conflicting information (e.g., an option is favorable on one attribute but is unfavorable on another), a common procedure for resolving the apparent decision conflict is to select the option that is most favorable on the most important attribute. Alternatively, one might argue that in a noncompensatory decision strategy the decision-maker always only focuses on the most important decision attribute, experiencing no decision conflict at all. These interpretations generate opposing hypotheses. That is, in one interpretation, a noncompensatory strategy results in experienced decision conflict when there is no dominant option (e.g., conflict condition). The second interpretation, however, predicts no experienced decision conflict irrespective of whether there is, or is not, a dominating option.

Experienced decision conflict has been linked to neural activation in the dorsomedial prefrontal cortex (dmPFC). That is, the dmPFC is one of the key regions that has been related to conflict detection (Pochon, Riis, Sanfey, Nystrom, & Cohen, 2008; Liston, Matalon, Hare, Davidson, & Casey, 2006; Badre & Wagner, 2004) and to the selection between mutually incompatible responses (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Carter et al., 1998). Indeed, a study of De Neys et al. (2008) using a reasoning problem showed that heuristic (i.e., stereotypical) responses led to greater activation in the dmPFC. This was interpreted as a signal of experienced decision conflict, indicating that people detect their heuristic biases. Moreover, Venkatraman, Rosati, Taren, and Huettel (2009) showed that activation in the dmPFC presented an anterior-to-posterior topography based on varying control demands. That is, particularly a middle dmPFC region was

associated with decision-related control and was shown to exhibit activation increasing with the difficulty in making decisions. Taken together, neural activation in the dmPFC seems a prominent candidate for tracking decision conflict. Here, we expect that if decision conflict occurs, this will be particularly present in the conflict condition and in participants using a noncompensatory decision strategy.

The current study explicitly aims to test the neural correlates underlying individual differences in decision strategy. That is, even when two individuals make ultimately identical choices, the strategies used to arrive at these decisions may differ. To identify and investigate individuals' decision strategies and the underlying neural correlates, we used the Gambling Machine Task (GMT). In the GMT, participants have to choose between two options, each characterized by a sure gain amount, a loss amount, and a loss probability. An advantage of the GMT is that it, in combination with a binomial mixture analysis of the choice data, allows for classification of individuals into groups characterized by distinct decision strategies (Jansen et al., 2012; Van Duijvenvoorde, Jansen, Visser, & Huizenga, 2010).

We investigated strategies located at quite opposite ends of the possible range of compensatory versus noncompensatory risky choice strategies: a compensatory strategy that maximizes EV (EV-maximizing) versus a noncompensatory strategy in which loss amount is minimized. In EV-maximizing, decision-makers weigh the amount of gains and losses with their respective probabilities, sum the weighted outcomes within an option, and choose the option with the higher EV. In a noncompensatory strategy of attribute-wise comparisons, decision-makers compare the two options on each attribute (gain amount, loss amount, loss probability) separately. If no dominance emerges, choice is based on the most important attribute (Tversky et al., 1988). If attributes are indeed compared, choices in which attributes present conflicting information may result in experienced decision conflict. Here, we will focus particularly on individuals using a noncompensatory strategy that minimizes loss amounts.

The GMT consists of two main types of choice conditions: In the simple condition, the two choice options differ only on one attribute, resulting in one option dominating the other. In the conflict condition, attributes provide conflicting information, meaning that in these choices one option is better on at least one attribute and the alternative is superior on at least one other attribute. Decision-makers may adapt their strategies depending on the requirements and choices at hand, for example, use a simple noncompensatory strategy if this suffices in a certain context (Tversky & Kahneman, 1974). Therefore, we expect the participants to use their predominant strategies in the conflict but not necessarily in the simple condition.

In summary, we expect no group differences in behavior or neural activation in the simple condition, given that such decision contexts elicit simple choice strategies in all participants. In contrast, in the conflict condition, we

expect individual differences in the use of decision strategies to emerge. More specifically, we expect that the compensatory, but not the noncompensatory group, shows EV coding in a network involving the VStr, vmPFC, PCC, and parietal cortex. In contrast, the noncompensatory, compared with the compensatory group, is expected to show increased dmPFC activation.

## METHODS

### Prestudy

#### Participants

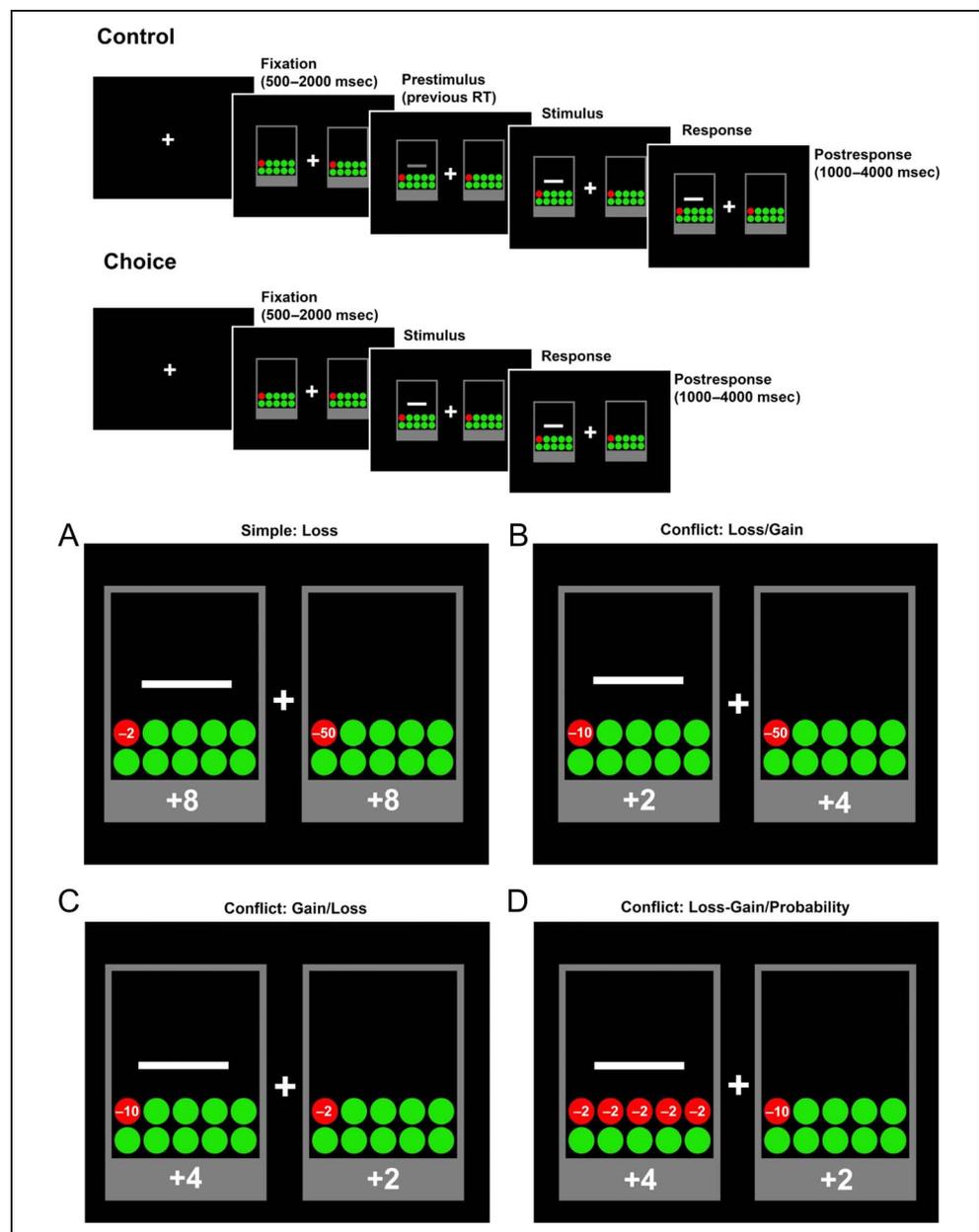
To recruit participants for the fMRI study in which we aimed to compare distinct strategy groups, we first ran a prestudy with a total of 77 participants (ages 18–30 years,

23 men) who were recruited through a university recruitment system and received payment or student credit for their participation. Our goal was to select a subset of participants that used either compensatory or noncompensatory decision strategies. Participants gave written informed consent, and all procedures for the behavioral prestudy were approved by the local ethics committee.

#### Gambling Machine Task

We used a risky choice task (the GMT) in which participants chose between two gambling machines, each characterized by a gain amount, a loss probability, and a loss amount. The current format of the task has been used in a set of studies to investigate distinct decision strategies (Bexkens, Jansen, Van der Molen, & Huizenga, 2016;

**Figure 1.** Task design of the fMRI-GMT (top): Each trial started with a fixation cross (1000–2500 msec; jittered in steps of 500 msec), followed by the presentation of a choice item, which was response terminated (max 16 sec). By means of a button press, participants indicated their choice after which a gray bar appeared below the chosen option. This display stayed on screen for 1–4 sec (jittered in steps of 1 sec), after which the next trial started. The control condition had a similar trial structure, except that the duration of the stimulus was based on participants' RT in preceding items. In the control condition, a gray bar appeared in one of the options signaling the participants to press the corresponding button. Examples of items in the simple (A) and conflict (B, C, D) condition (bottom).



Jansen et al., 2012; Van Duijvenvoorde et al., 2010) and uses a fixed gain combined with a probabilistic loss, as used in other risky choice paradigms such as the Iowa Gambling Task (Bechara, Damasio, Damasio, & Anderson 1994). Each machine contained 10 balls, some red and some green (see Figure 1A–D for an illustration). The proportion of red balls represented the loss probability. The number written on the red balls represented the loss amount; within a machine, all loss amounts were the same. The gain amount was depicted on the front of the machine and was not probabilistic, that is, that gain was received irrespective of whether a green or red ball would be drawn. It was explained to the participants that, upon choosing a machine, balls toss and tumble, one ball is drawn, and they should choose the best machine to play with. Drawing a

green ball would result in the stated gain, whereas drawing a red ball would result in the stated gain and loss. On each item, the participant could choose Machine A, Machine B, or indicate that the machines were equally profitable. In the GMT, no direct outcomes were presented and all outcomes differed in EV (see Table 1 for an overview of all of the items in the conflict condition of the fMRI-GMT).

On the three items of the simple condition, a dominated choice was presented in which respectively only gain amounts (Simple: Gain), only loss amounts (Simple: Loss), and only loss probability (Simple: Probability) differed between choice options. On the three items of the conflict condition, at least one attribute was superior in one option, but another attribute was superior in the other option. These items differed systematically in the attribute-conflict

**Table 1.** Item Characteristics of the fMRI-GMT in the Conflict Condition

| <i>Items per Condition</i>      | <i>Loss Probability</i> |          | <i>Loss Amount</i> |          | <i>Gain</i> |          | <i>Expected Value</i> |          |
|---------------------------------|-------------------------|----------|--------------------|----------|-------------|----------|-----------------------|----------|
|                                 | <i>A</i>                | <i>B</i> | <i>A</i>           | <i>B</i> | <i>A</i>    | <i>B</i> | <i>A</i>              | <i>B</i> |
| <i>fMRI-GMT</i>                 |                         |          |                    |          |             |          |                       |          |
| Conflict: Loss/Gain             | .5                      | .5       | −2                 | −10      | +2          | +4       | 1                     | −1       |
| Conflict: Loss/Gain             | .1                      | .1       | −50                | −2       | +8          | +4       | 3                     | 3.8      |
| Conflict: Loss/Gain             | .1                      | .1       | −2                 | −50      | +2          | +4       | 1.8                   | −1       |
| Conflict: Loss/Gain             | .3                      | .3       | −2                 | −10      | +2          | +4       | 1.4                   | 1        |
| Conflict: Loss/Gain             | .3                      | .3       | −50                | −2       | +8          | +2       | −7                    | 1.4      |
| Conflict: Loss/Gain             | .3                      | .3       | −50                | −10      | +8          | +4       | −7                    | 1        |
| Conflict: Loss/Gain             | .1                      | .1       | −50                | −10      | +4          | +2       | −1                    | 1        |
| Conflict: Loss/Gain             | .3                      | .3       | −2                 | −50      | +4          | +8       | 3.4                   | −7       |
| Conflict: Gain/Loss             | .3                      | .3       | −2                 | −10      | +2          | +8       | 1.4                   | 5        |
| Conflict: Gain/Loss             | .1                      | .1       | −2                 | −50      | +2          | +8       | 1.8                   | 3        |
| Conflict: Gain/Loss             | .1                      | .1       | −10                | −2       | +8          | +2       | 7                     | 1.8      |
| Conflict: Gain/Loss             | .3                      | .3       | −10                | −2       | +8          | +4       | 5                     | 3.4      |
| Conflict: Gain/Loss             | .1                      | .1       | −2                 | −10      | +4          | +8       | 3.8                   | 7        |
| Conflict: Gain/Loss             | .5                      | .5       | −10                | −2       | +8          | +2       | 3                     | 1        |
| Conflict: Gain/Loss             | .1                      | .1       | −2                 | −10      | +2          | +4       | 1.8                   | 3        |
| Conflict: Gain/Loss             | .1                      | .1       | −50                | −10      | +8          | +2       | 3                     | 1        |
| Conflict: Loss–Gain/Probability | .3                      | .1       | −2                 | −10      | +4          | +2       | 3.4                   | 1        |
| Conflict: Loss–Gain/Probability | .3                      | .1       | −10                | −50      | +4          | +2       | 1                     | −3       |
| Conflict: Loss–Gain/Probability | .1                      | .5       | −10                | −2       | +4          | +8       | 3                     | 7        |
| Conflict: Loss–Gain/Probability | .1                      | .5       | −10                | −2       | +2          | +4       | 1                     | 3        |
| Conflict: Loss–Gain/Probability | .5                      | .1       | −10                | −50      | +8          | +4       | 3                     | −1       |
| Conflict: Loss–Gain/Probability | .5                      | .1       | −2                 | −10      | +8          | +2       | 7                     | 1        |
| Conflict: Loss–Gain/Probability | .3                      | .5       | −10                | −2       | +2          | +4       | −1                    | 3        |
| Conflict: Loss–Gain/Probability | .1                      | .5       | −50                | −10      | +2          | +8       | −3                    | 3        |

See main text for further description.

**Table 2.** Group Size and Estimated Probabilities to Choose the EV-maximizing Option for Each Group (Conditional Probabilities)

|   | <i>n</i> | <i>Items per Condition</i> |                     |                     |                            |                            |  |
|---|----------|----------------------------|---------------------|---------------------|----------------------------|----------------------------|--|
|   |          | <i>Simple: Probability</i> | <i>Simple: Loss</i> | <i>Simple: Gain</i> | <i>Conflict: Loss/Gain</i> | <i>Conflict: Gain/Loss</i> | <i>Conflict: Loss-Gain/Probability</i> |
| 1. Compensatory<br><i>EV max</i>                  | 35       | 1                          | .98 (1)             | .96 (1)             | .93 (1)                    | .83 (1)                    | .76 (1)                                |
| 2. Noncompensatory<br><i>Loss amount min</i>      | 27       | 1                          | 1.00 (1)            | .98 (1)             | .97 (1)                    | .03 (0)                    | .69 (1)                                |
| 3. Noncompensatory<br><i>Loss probability min</i> | 15       | 1                          | .90 (1)             | .96 (1)             | .78 (1)                    | .41 (0)                    | .14 (0)                                |

In parentheses: the probabilities of the EV-maximizing choice that are expected given specific strategies. min = minimizing; max = maximizing.

they represented. On the first item, one option presented a lower loss but the other option presented a higher gain, whereas the loss probability was equal in both option. The optimal choice according to EV was the option with the lowest loss (Conflict: Loss/Gain). The second item presented a similar conflict, but now the optimal choice according to EV was the option with the highest gain (Conflict: Gain/Loss). On the final item, all three attributes differed between the two options, and the option with the lower loss, the higher gain, and the higher loss probability was the optimal choice according to EV (Conflict: Loss-Gain/Probability). In the behavioral session, participants played four repetitions of each of the items in the simple and conflict condition, resulting in 24 trials.

To reduce memory effects and keep participants engaged, the numeric values of gains, losses, and probabilities were varied somewhat across repetitions of the same item: Gain amounts were +2 and +4, loss amounts were -2, -10, and -50, and loss probabilities were .1 and .5. Location of the EV-maximizing option (left/right) was counterbalanced across repetitions.

### Mixture Analysis

We performed a binomial mixture analysis with the package *flexmix* (Leisch, 2004) in R (R Development Core Team, 2015) to classify participants in groups with homogeneous choice patterns from which decision strategies were inferred. Participants' choices were converted to binary "EV-maximizing" and "non-EV-maximizing" responses and summed over repetitions per item. This generated a multivariate choice pattern for each participant across all items, that is, across both the simple and the conflict items.

The mixture analysis involved three steps (Jansen et al., 2012). First, the analysis was performed on a range of models, varying in the number (1–10) of subgroups. For each model, the estimation algorithm was run 100 times with different starting values to reduce the risk of local minima. Second, the solutions for the 10 models were compared by means of the Bayesian Information Criterion (Leisch, 2004), and the best fitting model was selected.

Third, we calculated each participant's probability that he/she belonged to a particular group and assigned each participant to his or her most likely group.

A mixture analysis on the prestudy data indicated that a model with three groups described the behavioral data best (see Table 2 for estimated response patterns and expected response patterns for each group interpretation). One group's choice patterns were indicative of a compensatory strategy ( $n = 35$ ; 45.5% of participants): These participants chose the option with the highest EV across all items. The two other groups' choice patterns were most indicative of noncompensatory strategies: In the simple condition, these participants chose the option with the highest EV (i.e., the dominating option), but they did not do so consistently in the conflict condition. More specifically, the second group ( $n = 27$ ; 35%) chose the option with the smallest loss amount. The third group's choice patterns suggested that they chose the lowest loss probability if loss probability differed between options, and if the two options had the same loss probability, they chose the option with the lowest loss amount ( $n = 15$ ; 19.5%).

### fMRI Study

#### Participants

Selection for the fMRI study occurred semi at-random from the prestudy. The first 20 participants that were willing and eligible for fMRI research using an EV strategy were included in the fMRI study. Similarly, 25 participants were included from the other strategy groups, resulting in a total of 45 participants in the fMRI study from the 77 participants in the prestudy (28 women, mean = 22.5 years,  $SD = 3.2$  years, min = 18 years, max = 29 years). All included participants were right-handed and reported normal or corrected-to-normal vision, an absence of any metal implants or braces, and an absence of neurological or psychiatric conditions. Testing was performed on a different day than the prestudy. Participants gave written informed consent, and procedures were approved by the local ethics committee. Data of three participants had to be removed because of technical problems, and two participants did not complete the fMRI session, leaving a total of

40 participants (24 women) for the fMRI analyses. Twenty of those participants used in the prestudy a compensatory, EV-maximizing strategy, and 20 participants used in the prestudy a noncompensatory strategy. In the noncompensatory group, we included 14 participants that used a loss amount-minimizing strategy and six participants that used a loss probability minimizing strategy in the prestudy.

To confirm stability of choice strategies, we first compared behavioral choices between the prestudy and the fMRI study. That is a close inspection of the response patterns across items on an individual level showed that nearly all participants maintained their strategy. Only two participants switched from a compensatory EV-maximizing strategy in the prestudy to a noncompensatory loss-minimizing strategy in the fMRI study. Additionally, a comparison of the choice patterns for each of the groups defined in the behavioral pre-session, showed that both noncompensatory groups followed a predominantly loss-minimizing choice pattern in the fMRI session (see Tables 2 and 3 for the expected response pattern). For further analysis, we therefore pooled participants from the two noncompensatory groups. For the fMRI analyses, this means that we compared a compensatory group of  $n = 18$  (9 women) versus a noncompensatory group of  $n = 22$  (15 women).

Before entering the scanner, task explanations were briefly repeated. To increase motivation, all participants were paid a flat rate for their participation plus an additional variable amount between €0 and €8 purportedly related to their decisions: The participants were instructed that a set of their choices would be played out after the task, of which one randomly selected choice would be paid out. Eventually, all participants were presented with the same set of played out choices, and one of these played out gambles was added to the participant's flat rate (€2, 4, or 8). Given that participants were not presented with an endowment, losses were treated as a €0 outcome in the final payment, which was mentioned in the instructions.

### fMRI-GMT

In the scanner, an adjusted version of the GMT was used that included three changes. First, the fMRI-GMT ex-

cluded the answer category that the machines were equally profitable. This third category has been used to better differentiate between strategies (Bexkens et al., 2016; Jansen et al., 2012; Van Duijvenvoorde et al., 2010) that, however, were not of interest in the current fMRI study.

Second, the fMRI-GMT consisted of three runs that each presented eight repetitions of all three items in the conflict and simple condition (see Table 1 for a complete overview of all items in the conflict condition). Because of the increased repetition of items, variations in numeric values were extended: Gain amounts were +2, +4, and +8, loss amounts were -2, -10, and -50, and loss probabilities were .1, .3, and .5. The two options within a choice pair never had the same EV.

Finally, to control for activations triggered by processes unrelated to decision-making, for example, perceptual or motor processes, the fMRI-GMT included a "control" condition that had a similar structure as the choice condition, except that participants were not presented with an actual choice. In the control condition, two identical options were shown. After an interval, a gray bar appeared in one of the options, and participants were instructed to choose the option indexed by the gray bar. Attributes used in the control trials were based on the attribute values used in the choice trials, leading to 27 unique combinations of gains (2, 4, 8), probabilities (.1, .3, .5), and losses (-2, -10, -50). These control trials were randomly and evenly divided per participant into simple and conflict control trials. That is, the interval between the onset of the stimulus and the appearance of the gray bar was modeled based on each individual's RT on a preceding trial. Half of the control trials were matched to the participant's response duration in the simple condition (simple-control), and half of the control trials were matched to the participant's response duration in the conflict condition (conflict-control). Matching of RTs was applied to account for neural differences that might be due to a Condition (simple, conflict) × Strategy Group (Compensatory/Noncompensatory) interaction in decision duration.

Trials in the fMRI-GMT had the following structure (see Figure 1): First, a fixation cross was displayed on the

**Table 3.** Average Proportion of EV-maximizing Choices in the fMRI Session for Each Item and the Strategy Groups as Found in the Behavioral Session, with the Two Compensatory Groups Split Out

|                    | <i>n</i> | <i>Items per Condition</i> |                     |                     |                            |                            |  |
|--------------------|----------|----------------------------|---------------------|---------------------|----------------------------|----------------------------|--|
|                    |          | <i>Simple: Probability</i> | <i>Simple: Loss</i> | <i>Simple: Gain</i> | <i>Conflict: Loss/Gain</i> | <i>Conflict: Gain/Loss</i> | <i>Conflict: Loss-Gain/Probability</i> |
| 1. Compensatory    | 18       | 1                          | .99                 | .99                 | .96                        | .93                        | .98                                    |
| 2. Noncompensatory | 16       | 1                          | .99                 | .98                 | .98                        | .07                        | .86                                    |
| 3. Noncompensatory | 6        | .97                        | .99                 | .97                 | .94                        | .14                        | .81                                    |

The two participants that switched from a compensatory (behavioral) to a noncompensatory loss-minimizing decision strategy (fMRI) are included in the second noncompensatory group.

screen for 1–2.5 sec (jittered in steps of 500 msec; uniform distribution). Then, the two choice options were presented, and participants could indicate their choice by pressing one of two buttons with their left hand: A button press with the middle finger indicated choice of the left option and a button press with the index finger indicated choice of the right option. After a decision was made, a gray bar appeared under the chosen option for 1–4 sec (jittered in steps of 1 sec with a uniform distribution), after which a new trial started. Participants had a maximum time allowance of 16 sec to make their decision, which was sufficient in all cases. In approximately 25% of the trials, intertrial intervals were included to the fixation screen that presented a fixation cross and were jittered exponentially to improve signal detection (2, 4, 6, and 8 sec, with 2 sec as the most frequent and 8 sec as the least frequent interval).

In the fMRI-GMT, participants thus played three runs, with 48 choice trials and 27 control trials included per run. Control and choice trials were presented in pseudorandom order. The control trials were not included for possible payout.

### *Imaging*

fMRI data were acquired with a standard whole-head coil using a 3-T Philips (Amsterdam, The Netherlands) Achieva scanner. T2\*-weighted echoplanar images were obtained during three functional runs of which the first two volumes were discarded to allow for equilibration of T1 saturation effects. Volumes covered the whole brain (34 slices; 3 mm slice thickness; 0.3 mm slice spacing; 220<sup>2</sup> mm field of view; 962<sup>2</sup> in plane resolution; ascending orientation) and were acquired every 2000 msec (echo time = 28 msec). A high-resolution T1-weighted anatomical scan was obtained from each participant after the functional runs.

fMRI analysis was performed using FEAT (fMRI Expert Analysis Tool) Version 5.98, part of FSL 4.1 (FMRIB's Software Library, [www.fmrib.ox.ac.uk/fsl](http://www.fmrib.ox.ac.uk/fsl)). The data were high-pass filtered with a cutoff frequency of 90 sec to remove baseline drift in the signal. Functional volumes were spatially smoothed with a 5-mm FWHM isotropic Gaussian kernel motion-corrected and slice time-corrected. Finally, the functional data were prewhitened using FSL. All functional data sets were individually registered into 3-D space using the participant's individual high-resolution anatomical images. The individual 3-D image was then used to normalize the functional data into the Montreal Neurological Institute (MNI) template. Registration to high-resolution structural and/or standard space images was carried out using FLIRT. Registration from high-resolution structural to standard space was then further refined using FNIRT nonlinear registration. The statistical analysis was performed using the general linear model (GLM). The design matrix of the GLM was convolved with a double gamma hemodynamic response function and its first derivative.

## **Imaging Analysis**

### *Conflict Analysis*

In a GLM, we included two discrete regressors that coded choices for the (1) simple and (2) conflict condition respectively and two discrete regressors that coded (3) the control items based on RT in the simple condition and (4) the control items based on RT in the conflict condition. Accordingly, the first of the two control regressors was used in whole-brain contrasts with the simple condition and the second in whole-brain contrasts with the conflict condition. The duration of each event in all regressors was modeled by the respective RT.

### *EV Analysis*

In a subsequent GLM, we included additional parametric regressors of absolute EV differences between options, separately for the simple and conflict condition. For these parametric regressors, the height of each event was modeled by the absolute difference in EV.

In both GLMs, motion regressors, and inconsistent responses (see below) were included as regressors of no interest. For all analyses, only trials were included in which participants chose in accordance to their predominant strategies (e.g., EV-maximizing or loss amount-minimizing choices; see Table 3). Trials with deviating responses were included in a regressor of no interest. This happened relatively rarely. In the simple condition, a nondominating choice occurred on 0.7% of trials in the compensatory group and on 1.5% of trials in the noncompensatory group. In the conflict condition, a non-EV-based choice occurred in 4.6% of trials for the compensatory group. In the noncompensatory group, a non-loss amount-minimizing choice occurred in 9.5% of trials.

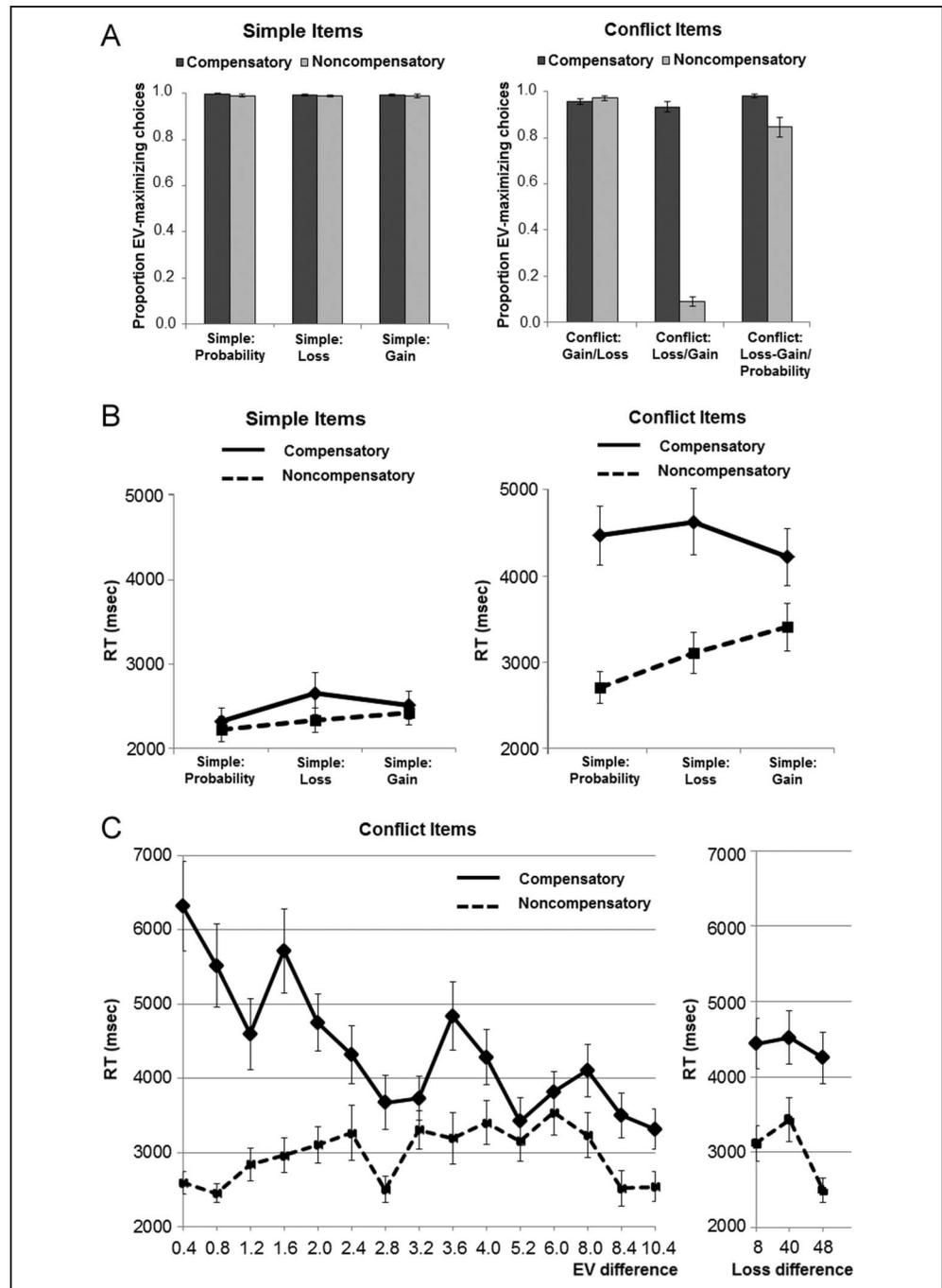
Higher-level analysis was performed using FLAME (FMRIB's Local Analysis of Mixed Effects) Stage 1 with automatic outlier detection. For the whole-brain analysis, Z-statistic images were thresholded with Gaussian Random Field Theory cluster-wise correction,  $Z > 2.3$  and family-wise error (FWE)-corrected with  $p < .05$  (unless indicated otherwise).

## **RESULTS**

### **Behavioral Results**

Figure 2A depicts the proportion of EV-maximizing choices as a function of condition (three simple, three conflict items) and strategy group. We performed a generalized linear mixed model using the lme4 package (Bates, Maechler, Bolker, & Walker, 2015) in R (R Development Core Team, 2015). The unit of analysis was the binary decision level (choose EV-maximizing option/non-EV-maximizing option), including Condition (simple vs. conflict), Group (noncompensatory vs. compensatory), and Condition  $\times$  Group as fixed effects. To

**Figure 2.** Behavioral task performance: (A) The proportion of EV-maximizing choices for each simple and conflict item for compensatory and noncompensatory groups. (B) RTs for each simple and conflict item for compensatory and noncompensatory groups. (C) RTs in the conflict condition, plotted for each strategy group, as a function of differences in EV and differences in loss amounts between choice items. Error bars indicate  $\pm SE$  around the mean.



account for the nested structure of the data, a random intercept and a random slope of Condition (simple vs. conflict) per participant was included. Self-evidently (as we grouped participants according to their behavior), log-likelihood ratio tests (Singmann, Bolker, & Westfall, 2015) indicated a significant interaction between Condition and Group,  $B = -.45$ ,  $p = .001$ . There were no significant group differences in the simple condition. Generally, both groups chose the option that dominated the other (and thus had the largest EV). In the conflict condition, however, the compensatory group showed a larger proportion of

EV-maximizing choices than the noncompensatory group,  $B = 1.26$ ,  $p < .001$ . Note that on two items in the conflict condition, EV-maximizing and loss-minimizing strategies led to the same choice (see Figure 2A). These items are of particular interest, because potential behavioral RT differences or neural differences cannot be attributed to choosing a different option but should be attributed to a difference in decision strategy.

To test whether RTs would support our hypothesis that choice strategies are adaptively applied and that groups would particularly differ in their decision process

on conflict trials, we performed a similar linear mixed model analyses, but now with RT as the dependent variable (Figure 2B). This analysis indicated a significant interaction between Condition and Group,  $B = -302.69$ ,  $p < .001$ . There were no group differences in the simple condition ( $p = .46$ ). In the conflict condition, the compensatory group was generally slower than the noncompensatory group,  $B = 678.3$ ,  $p < .001$ . Note that RTs were longer in the compensatory group for all items in the conflict condition (see Figure 2B), even those that yielded similar choices as the noncompensatory group, providing further evidence that the groups used different decision strategies.

An additional check on the nature of decision strategies was obtained from an analysis in which we tested whether the continuous differences in EV were related to the RT differences in choice behavior. To this end, we calculated the absolute difference in Expected Value between options and performed a similar mixed-model analysis with RT on each trial predicted by the fixed effects of Expected Value difference, Condition (simple vs. conflict), Group (noncompensatory vs. compensatory), and all possible interactions. Additionally, a random intercept per participant and random slopes of Expected Value difference and Condition were included. As expected, results showed a three-way interaction; Expected Value difference  $\times$  Group  $\times$  Condition,  $B = 54.8$ ,  $p < .001$ . Follow-up tests showed that greater differences in Expected Value led to faster RTs, but this was only the case in the conflict condition, and only present in the compensatory group (Expected Value difference  $\times$  Group  $p < .001$  in the conflict condition, but  $p = .21$  in the simple condition; see Figure 2C). Moreover, a similar mixed-model analysis with the absolute value of loss differences (instead of Expected Value differences) showed no significant main effect of loss difference ( $p = .13$ ), nor significant interactions of Group or Condition with loss difference (all

$ps > .1$ ). Together these results indicate that, as expected, the compensatory group uses EV in their decision strategy, but only in the conflict condition. On the other hand, these results show that the difference in amount of loss is not continuously coded. This seems to strengthen the idea that the noncompensatory (loss-minimizing) strategy group particularly focuses on the presence or absence of a loss difference, without considering *continuous* loss differences.

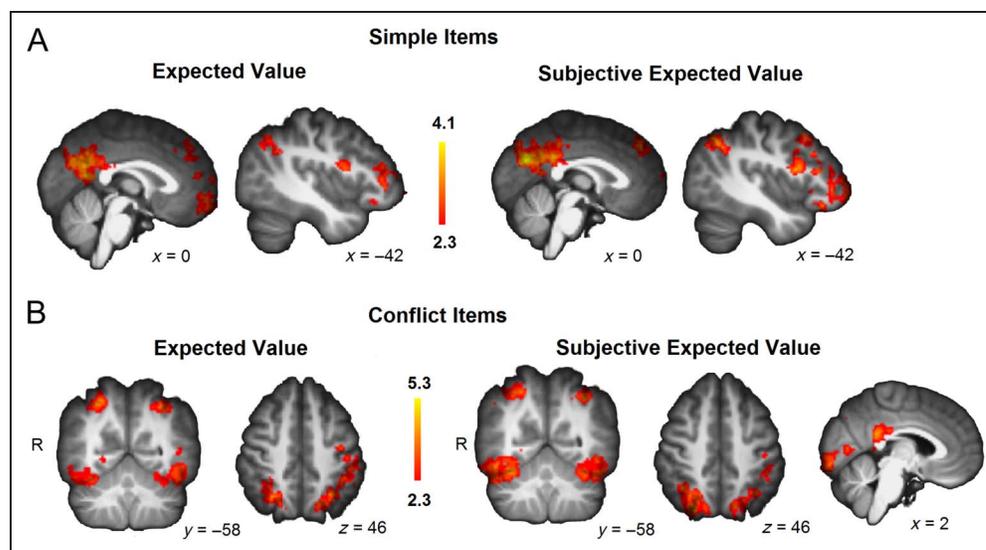
Finally, to test whether choice behavior was stable across the task, we additionally tested the mixed model on choice behavior with Run included as an additional fixed and random effect. There was no significant main effect of Run ( $p = .47$ ), nor a Group  $\times$  Run interaction ( $p = .67$ ) or a significant Group  $\times$  Run  $\times$  Condition interaction ( $p = .055$ ). Taken together, these effects show that choice behavior was stable for both strategy groups.

## Imaging Results

### EV Coding

As expected, in the conflict condition, the Group  $\times$  EV effect showed significantly stronger EV difference coding in the compensatory than in the noncompensatory group; these differences were found in the visual and the parietal cortex (see Figure 3B and Table 4). The reversed group contrast (noncompensatory  $>$  compensatory) showed no significant clusters, even at more lenient thresholds (e.g., uncorrected  $p < .001$ ; minimally 15 contiguous voxels). Follow-up tests for each group separately showed a positive effect of EV in the compensatory group in similar regions as those identified in the Group  $\times$  EV effect (see Table 4), whereas the main effect of the noncompensatory group showed a cluster of activation in the visual cortex only.

**Figure 3.** EV coding (left) and EU coding (right) by the compensatory group in the simple (A) and conflict (B) conditions. The noncompensatory group did not show any EV- or EU-related effects. The figure displays cluster-corrected results ( $Z > 2.3$ ,  $p < .05$  FWE-corrected); coordinates of EV activation are reported in Table 4.



**Table 4.** Coordinates for the Brain Regions Showing Parametric EV Difference Coding

| <i>Anatomical Area</i>  | <i>Cluster Size</i> | <i>MNI Coordinates (mm)</i> |          |          | <i>Z-max Value</i> |
|---|---------------------|-----------------------------|----------|----------|--------------------|
|   |                     | <i>x</i>                    | <i>y</i> | <i>z</i> |                    |
| <i>Conflict Condition</i>   |                     |                             |          |          |                    |
| Compensatory > noncompensatory  |                     |                             |          |          |                    |
| R lateral occipital cortex  | 11,414              | 30                          | -78      | 16       | 4.21               |
| L lateral occipital cortex  |                     | -30                         | -78      | 22       | 4.07               |
| L superior parietal cortex, including intraparietal sulcus and inferior parietal cortex |                     | -30                         | -56      | 44       | 3.99               |
| Mean activation compensatory group  |                     |                             |          |          |                    |
| R occipital pole  | 13,070              | 22                          | -98      | 2        | 5.17               |
| L inferior parietal lobe, including intraparietal sulcus and superior parietal cortex   | 408                 | -52                         | -28      | 32       | 3.29               |
| Noncompensatory > compensatory  |                     |                             |          |          |                    |
| None  |                     |                             |          |          |                    |
| Mean activation noncompensatory group   |                     |                             |          |          |                    |
| R occipital pole  | 755                 | 20                          | -94      | -12      | 3.96               |
| <i>Simple Condition</i>   |                     |                             |          |          |                    |
| Compensatory > noncompensatory  |                     |                             |          |          |                    |
| R frontal pole  | 2882                | 32                          | 48       | -8       | 3.74               |
| Posterior cingulate gyrus   | 2050                | 6                           | -54      | 14       | 4.18               |
| L angular gyrus   | 472                 | -48                         | -54      | 40       | 3.32               |
| Medial frontal gyrus  | 408                 | 8                           | 56       | 16       | 3.03               |
| R middle frontal gyrus  | 401                 | 44                          | 30       | 22       | 3.02               |
| Mean activation compensatory group  |                     |                             |          |          |                    |
| Posterior cingulate gyrus   | 1670                | 6                           | -54      | 14       | 4.09               |
| L inferior frontal gyrus  | 854                 | -52                         | 24       | -4       | 3.99               |
| L angular gyrus   | 388                 | -48                         | -54      | 40       | 3.27               |
| L frontal pole  | 357                 | -14                         | 68       | -4       | 3.43               |
| Frontal medial cortex   |                     | 4                           | 54       | -18      | 2.75               |
| Noncompensatory > compensatory  |                     |                             |          |          |                    |
| None  |                     |                             |          |          |                    |
| Mean activation noncompensatory group   |                     |                             |          |          |                    |
| None  |                     |                             |          |          |                    |

Main effects per strategy group and group differences are reported for cluster-corrected  $Z > 2.3$ ,  $p < .05$  thresholds. MNI coordinates are given for the (local) peaks of activation.

Surprisingly, compensatory decision-makers also showed evidence of EV coding in the simple condition. In this condition, the compensatory, compared with the noncompensatory group, showed significantly stronger EV coding in parietal cortex, vmPFC, and PCC (see

Figure 3A and Table 4). The reversed group contrast (non-compensatory > compensatory) showed no significant clusters, even not at more lenient thresholds (e.g., uncorrected  $p < .001$ ; minimally 15 contiguous voxels). Follow-up tests for each group separately showed EV coding in the

compensatory group in similar regions as those identified in the Group  $\times$  EV effect, but not in the noncompensatory group (see Table 4).

### Control Analyses

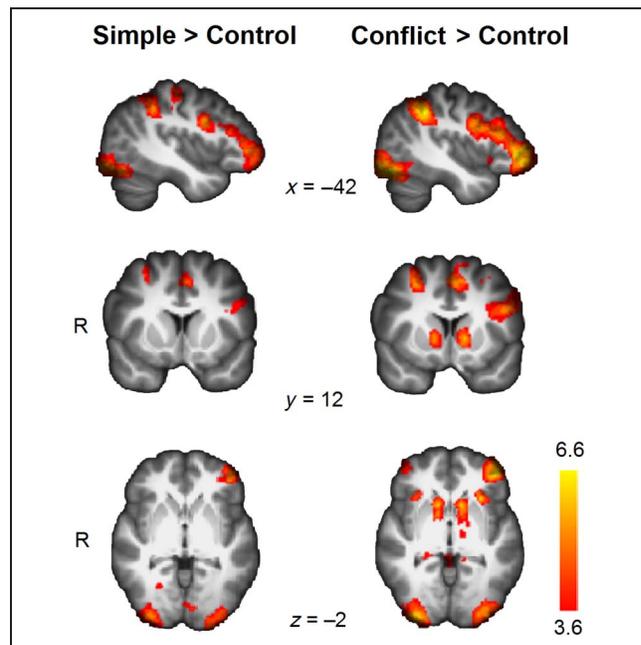
We tested three alternative explanations for this differential between group EV coding. First, it may be argued that the noncompensatory group does use a compensatory strategy, but instead of objective EV, codes some form of EU. That is, the noncompensatory group may integrate subjective attributes and base their decisions on a subjective integrated value signal. Particularly, the noncompensatory loss-minimizing strategy may actually be a compensatory, but highly loss-averse, strategy. Therefore, we estimated per participant a loss aversion parameter from the behavioral data in the fMRI session. To do so, we fitted a cumulative prospect theory model (Tversky & Kahneman, 1974, 1992) that included a loss aversion parameter and a weight parameter, the latter indicating to what extent choices are guided by differences in subjective utility. The cumulative prospect theory model was fitted to all items simultaneously, we assumed a logistic choice rule, and estimates were obtained by iteratively minimizing the negative log-likelihood function. The algorithm was restarted 100 times to avoid local minima. Note that the estimated loss aversion parameters were significantly different between the strategy groups,  $t(21.01) = -3.5, p = .002$ , with lower loss aversion in the compensatory than the noncompensatory group (compensatory:  $M = 1.09, SD = 0.21$ ; noncompensatory:  $M = 12.34, SD = 15.09$ ). Subsequently, we used this loss aversion parameter of each participant to calculate the subjective utility difference between options on each trial. This EU parametric regressor was used in a whole-brain fMRI analysis (Trepel et al., 2005). That is, in this analysis we included, instead of two parametric EV regressors, two parametric EU regressors (coding subjective utility) separately for the simple and conflict condition.

Results of brain regions coding EU were highly similar to the EV results, showing greater activation in the conflict condition for the compensatory compared with the noncompensatory group, in similar regions as with the EV regressor ( $Z > 2.3, p < .05$ , FWE cluster-corrected; see Figure 3B), including a cluster in the visual cortex ( $x = 24, y = -98, z = 2$ ; voxels = 14,395), parietal cortex ( $x = -42, y = -24, z = 52$ ; voxels = 526), and additionally in the PCC ( $x = -4, y = -40, z = 22$ ; voxels = 499). The reversed group contrast (noncompensatory  $>$  compensatory) showed no significant clusters in the conflict condition, even at more lenient thresholds (e.g., uncorrected  $p < .001$ ; minimally 15 contiguous voxels). Follow-up tests for each group separately showed a positive effect of EU in the compensatory group in similar regions as those identified in the Group  $\times$  EU effect, whereas the noncompensatory group showed clusters of activation in the visual cortex only ( $x = 34, y = -94, z = 6$ , voxels =

1490;  $x = -22, y = -98, z = 14$ , voxels = 571). Also in the simple condition, EU results were highly similar to the EV results, showing greater EU activation for the compensatory compared with the noncompensatory group (see Figure 3A). The reversed group contrast (noncompensatory  $>$  compensatory) and the positive main effect in the noncompensatory group showed no significant clusters of activation, even at more lenient thresholds (e.g., uncorrected  $p < .001$ ; minimally 15 contiguous voxels).

Second, it might be argued that the noncompensatory group does not code the difference in EV between options, but instead continuously codes differences in loss amounts, consistent with the notion that this is the main choice criterion in this group. Therefore, we calculated the absolute loss difference between choice options on each trial. This loss difference parametric regressor was used in a whole-brain fMRI analysis. Thus, in this second control analysis, we included, instead of two (objective) EV regressors, two parametric loss difference regressors separately for the simple and conflict condition. For both conditions, these analyses indicated no greater coding of loss differences in the noncompensatory group, even at more lenient thresholds (uncorrected  $p < .001$ , 15 contiguous voxels).

Third, it might be argued that the noncompensatory group coded the chosen loss amount instead of the difference in loss, focusing not on the comparison between choice options but on the potential loss that may be



**Figure 4.** Whole-brain results across groups showing brain regions that were activated for the contrasts simple  $>$  control condition (left) and conflict  $>$  control condition (right). Figure displays cluster-corrected results ( $Z > 3.6, p < .05$  FWE-corrected); coordinates are reported in Table 5.

incurred. This chosen loss parametric regressor was similarly included in a whole-brain fMRI analysis. In the conflict condition, the Group  $\times$  Chosen loss effect showed that there was significantly stronger loss coding in the noncompensatory than in the compensatory group.

These differences were found in, among other regions, the visual cortex ( $x = -46, y = -74, z = 6$ , voxels = 29,577), extending into the parietal cortex, and the putamen ( $x = 28, y = 8, z = -8$ , voxels = 536), with greater activations for higher chosen loss amounts. The reversed

**Table 5.** Coordinates for the Brain Regions Showing Activations for the Simple > Control and Conflict > Control Contrast at  $Z > 3.6, p < .05$  FWE Cluster-corrected

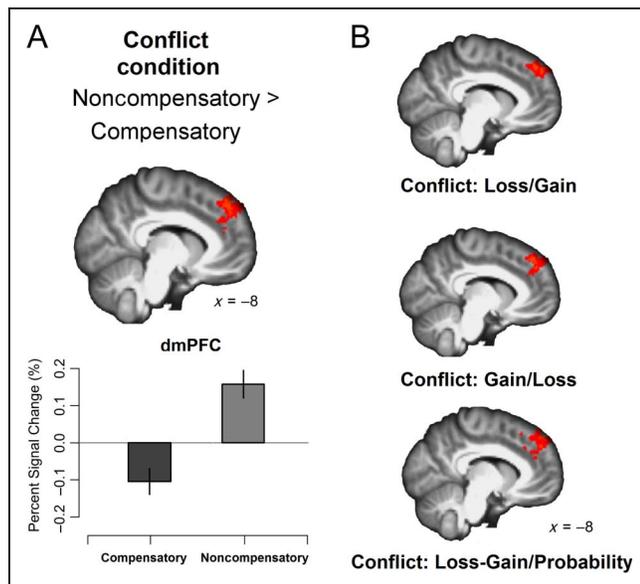
| <i>Anatomical Area</i>                | <i>Cluster Size</i> | <i>MNI Coordinates (mm)</i> |          |          | <i>Z-max Value</i> |
|---------------------------------------|---------------------|-----------------------------|----------|----------|--------------------|
|                                       |                     | <i>x</i>                    | <i>y</i> | <i>z</i> |                    |
| <i>Simple &gt; Control</i>            |                     |                             |          |          |                    |
| Mean activity $Z > 3.6$               |                     |                             |          |          |                    |
| R lateral occipital cortex            | 5656                | 36                          | -90      | -10      | 6.07               |
| R precuneus                           | 2177                | 26                          | -64      | 46       | 6.14               |
| L lateral occipital cortex            | 1768                | -30                         | -68      | 30       | 5.68               |
| L middle frontal gyrus                | 1455                | -46                         | 48       | 0        | 5.34               |
| Paracingulate gyrus                   | 961                 | 4                           | 28       | 36       | 5.36               |
| R middle frontal gyrus                | 767                 | 48                          | 38       | 16       | 5.62               |
| L precentral gyrus                    | 158                 | -42                         | -22      | 58       | 4.58               |
| Cerebellum                            | 142                 | 0                           | -78      | -24      | 4.71               |
| R superior frontal gyrus              | 112                 | 30                          | 10       | 50       | 4.54               |
| <i>Conflict &gt; Control</i>          |                     |                             |          |          |                    |
| Mean activity $Z > 3.6$               |                     |                             |          |          |                    |
| L lateral occipital cortex            | 6679                | -28                         | -66      | 38       | 6.65               |
| L lateral occipital cortex (inferior) | 3904                | -40                         | -78      | -18      | 6.16               |
| L frontal pole                        | 3551                | -46                         | 50       | -4       | 6.41               |
| R lateral occipital cortex (inferior) | 3230                | 36                          | -88      | -12      | 6.45               |
| Cerebellum                            | 2206                | 2                           | -54      | -28      | 5.78               |
| Paracingulate gyrus                   | 1788                | -2                          | 18       | 44       | 6.14               |
| R middle frontal gyrus                | 1490                | 46                          | 36       | 20       | 5.62               |
| R superior frontal gyrus              | 519                 | 28                          | 10       | 46       | 5.53               |
| R striatum (caudate)                  | 454                 | 12                          | 14       | -2       | 5.63               |
| L insula                              | 295                 | -30                         | 20       | -2       | 5.76               |
| L superior frontal gyrus              | 234                 | -26                         | 6        | 52       | 4.95               |
| R insula                              | 140                 | 34                          | 24       | -6       | 5.0                |
| R frontal pole                        | 133                 | 20                          | 46       | -18      | 4.9                |
| R hippocampus                         | 104                 | 22                          | -32      | -8       | 4.34               |
| L hippocampus                         | 94                  | -24                         | -28      | -10      | 4.83               |
| L orbitofrontal cortex                | 53                  | -16                         | 28       | -20      | 3.76               |
| L frontal pole                        | 52                  | -16                         | 52       | -24      | 4.1                |

MNI coordinates are given for the peaks of activation.

group contrast (compensatory > noncompensatory) showed no significant clusters of activation, even at more lenient thresholds (uncorrected  $p < .001$ , 15 contiguous voxels). Follow-up tests for each group separately showed a positive effect of chosen loss amount in the noncompensatory group in similar regions as those identified in the Group  $\times$  Chosen loss effect, whereas the compensatory group did not show any neural activation tracking the chosen loss between choice options.

In the simple condition, the noncompensatory group also showed greater coding of the chosen loss amount compared with the compensatory group, resulting in activation in the PCC and lateral PFC. However, inspection of the main effects per group indicated that this effect was predominantly driven by a negative relation in the compensatory group between chosen loss amount and neural activation. This can be explained from the relatively high negative correlation in the simple condition between the chosen loss amount and the difference in EV (simple condition:  $r = -.65$ ; conflict condition:  $r = .06$ ). Therefore, it seems that the results in the simple condition have limited interpretability for the noncompensatory group.

Taken together, these control analyses showed limited evidence that the noncompensatory group coded subjective utility differences or coded continuous loss differences. However, these results do suggest that the magnitude of the chosen loss amount is tracked particularly by the noncompensatory group, albeit only in the conflict condition.



**Figure 5.** Group differences in the conflict condition. (A, left) Brain regions in which the noncompensatory group showed more activation than the compensatory group in conflict > control (left). For illustration purposes, the average extracted values are plotted per strategy group. (B, right) Brain regions in which the noncompensatory group showed more activation than the compensatory group in each of the items of the conflict condition. The figure displays cluster-corrected results ( $Z > 2.3$ ,  $p < .05$  FWE-corrected).

### Conflict Detection

Finally, we focused on group differences in neural activation in simple and conflict conditions. That is, we specifically tested for group differences in neural activation on the simple > control and conflict > control contrasts. Main effects of neural activation across groups are presented in Figure 4 and Table 5.

We did not observe any group differences in neural activation in the simple condition. In the conflict condition, a Group  $\times$  (conflict > control) effect showed increased dmPFC activity in the noncompensatory as compared with the compensatory group ( $x = -24$ ,  $y = 36$ ,  $z = 44$ , number of voxels = 638; see Figure 5A). Follow-up tests on each conflict condition separately (conflict: Loss/Gain; conflict: Gain/Loss; conflict: Loss-Gain/Probability; see Figure 5B) indicated that increased dmPFC activation in the noncompensatory group was present on each of these conditions separately. No other clusters of activation were found.

## DISCUSSION

In this study, we investigated the neural mechanisms underlying compensatory (EV-maximizing) versus noncompensatory (loss-minimizing) decision strategies. Therefore, we compared individuals using these compensatory and noncompensatory strategies in a simple and in a conflict choice condition. Conflict choices were expected to trigger an individual's typical strategy, resulting in behavioral and neural differences, which were not expected on simple (in our study, dominating) choices.

In the conflict condition, we expected that particularly the compensatory, EV-maximizing, group would show neural activation in relation to a value signal that reflected the difference in EV between options; we expected this value signal in regions such as the VStr, vmPFC, PCC, lateral PFC, and parietal cortex (Mc Kell Carter et al., 2010; Kable & Glimcher, 2007; Tom et al., 2007; Blair et al., 2006). Indeed, we observed that primarily the compensatory group showed EV coding in the parietal cortex, whereas the noncompensatory group showed limited evidence for such a parametric EV signal. Parietal cortex activation was more prominent than neural activation in more typical value-based regions, which may suggest that EV coding in the current task is a more deliberative process. These findings are supported by the increased RTs of the compensatory group in the conflict condition and their scaling of RT with the difference in EV. In addition to parietal cortex activation that tracked the difference in EV in conflict items, we also observed activation in the visual cortex. Previous studies of value coding in the visual cortex (Krajbich, Armel, & Rangel, 2010; Serences, 2008) speculated that this activation is related to attentional processes. That is, visual cortex activation may be an inherent part of allocating attentional resources to value-based comparisons, in which a larger difference in EV indicates

that there is more at stake, leading to more attentional resources allocated.

Because we assumed that decision-makers would adopt a simple strategy if that sufficed, we expected no group differences in EV coding in the simple condition. Surprisingly, we did observe differences. In the simple condition, the compensatory, but not the noncompensatory, group showed evidence of EV coding in, among other regions, the vmPFC and PCC. It is tempting to interpret these findings as evidence that the compensatory group codes EV also in contexts where a dominating option is present (e.g., the simple condition). However, an alternative and more probable explanation is that this activation reflects the continuous coding of differences on choice attributes (gain amount, loss amount, loss probability), which are, in the simple condition, perfectly correlated with EV. This interpretation is also supported by RT analyses indicating that EV differences do not scale with RTs of the compensatory group in the simple condition.

Second, we expected that noncompensatory decision-makers would experience decision conflict from their attribute-wise comparisons, but only when a dominating choice option was unavailable. Alternatively, it may be expected that, in a strict noncompensatory decision strategy (only considering loss amounts), no decision conflict is experienced whatsoever. Here, we observed enhanced dmPFC activation particularly in the noncompensatory group and only in the conflict condition. This is consistent with an attribute-wise comparison leading to increased decision conflict as proposed by Tversky and colleagues (1988) and fits well with the interpretation of the dmPFC in detecting decision conflict and heuristic biases (Venkatraman & Huettel, 2012; De Neys et al., 2008). It should be noted, however, that dmPFC activation has been related to other interpretations such as detecting errors, choice uncertainty (Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004), and representing action outcome contingencies (Rushworth, Buckley, Behrens, Walton, & Bannerman, 2007).

The conflict monitoring function of the dmPFC may be associated with signaling the need to respecify the intensity of the executed control (Shenhav, Botvinick, & Cohen, 2013). In a previous study, it was indeed observed that a more anterior dmPFC region was related to strategy-related control (Venkatraman, Payne, Bettman, Luce, & Huettel, 2009; Venkatraman, Rosati, et al., 2009). That is, this region became active when participants chose counter to their preferred strategy, and it interacted with other brain regions to drive local choice behaviour. In the current study, we were limited in testing such strategy-related control given the stable response patterns in both groups.

Additional analyses were performed to check whether the noncompensatory group coded parametric subjective utility differences or loss differences instead of parametric EV differences. Generally, the imaging results show limited evidence that the participants adopted the current loss-minimizing strategy use continuous loss differ-

ences between options, which is supported by an absence of RT scaling with increasing loss differences. Note that a strategy such as loss minimization indeed does not require a parametric coding of loss differences, as a discrete detection of attribute differences between options is sufficient to arrive at a decision (Minati, Grisoli, Seth, & Critchley, 2012). Additional analyses indicated that the noncompensatory group, however, did code the chosen loss amount, more prominently than the compensatory group, and particularly in the conflict items. Although future studies are needed to allow further interpretation of neural activation related to the chosen versus differences in choice attributes, these control analyses do support the particular focus on losses for the noncompensatory group.

A limitation of the current study is that the noncompensatory group seems more heterogeneous than the compensatory group. Therefore, an absence of integrated value coding might be related to decreased power to detect this coding. Note, however, that even testing at lenient thresholds, as well as including a subjective value regressor, yielded limited evidence for value coding, making this explanation less likely. Second, we used a limited set of items for strategy detection, which may lead to the possibility that we misinterpreted participants' decision strategies. That is, theoretically, it may be possible that the noncompensatory group, for instance, would have used a strategy in which they calculated Probability  $\times$  Loss amount and only considered gains if a difference in such expected losses was not present. That this misspecification was present in the current study is, however, unlikely given that the distinction of compensatory and noncompensatory strategies is not only present in choice behavior but also in RTs. Furthermore, we already considered potential alternative strategies, which, however, were not supported by our data. To distinguish between a greater set of possible strategies, a more elaborate set of choice items would be necessary in future studies. Another limitation is that we only considered one type of noncompensatory and one type of compensatory strategy; the two investigated strategies commonly occur in risky choice, but many others are known to exist (Riedl, Brandstätter, & Roithmayr, 2008; Payne, Bettmann, & Johnson, 1993). To what extent the current results are particularly related to these types of decision strategies or generalize to other noncompensatory strategies is an important question for future studies. A final limitation is that, although the current task presented an additional payout based on a random selection of a set of played out gambles, this payout was only experienced at the end of the task and payout loss was treated as a €0 outcome. Therefore, eventually choices were indirectly related to payout and a true loss was not experienced. It could be argued that the fact that participants knew that they could not experience a true loss influenced their decision strategy. However, it would then be more likely that they would have neglected losses,

whereas the current results indicate that both the compensatory and noncompensatory group did take losses into account. Notwithstanding, future studies should include an incentive-compatible payout.

This study highlights the importance of studying individual differences in decision strategies by including paradigms and analyses that allow for strategy assessment. These individual differences in decision strategies may go unnoticed if data are averaged across participants and thus may obscure results. For example, value coding would have been less pronounced if we would have neglected individual differences in strategy use and pooled across all participants. Therefore, such experimental paradigms and analysis techniques allowing assessment of strategy differences may be highly beneficial to decision neuroscience.

To conclude, compensatory and noncompensatory decision-makers do not only differ in behavior but also in underlying neural mechanisms. That is, compensatory, EV-maximizing, decision-makers, but not noncompensatory, loss amount-minimizing decision-makers, coded the difference in EV between options. Noncompensatory decision-makers, on the other hand, showed increased dmPFC activation when choice attributes provided conflicting information, indicative of experienced decision-conflict. These results indicate the need for decision neuroscience and neuroeconomics to expand its scope by focusing not only on compensatory but also on noncompensatory decision strategies.

## Acknowledgments

This research was supported by a grant from the Amsterdam Brain Imaging Platform and by NWO VICI grant 016.130.070. The authors thank Jasper Wijnen for his help with programming the task.

Reprint requests should be sent to Anna C. K. van Duijvenvoorde, Wassenaarseweg 52, 2333 AK Leiden, The Netherlands, or via e-mail: a.c.k.van.duijvenvoorde@fsw.leidenuniv.nl.

## REFERENCES

Arsalidou, M., & Taylor, M. J. (2011). Is  $2 + 2 = 4$ ? Meta-analyses of brain areas needed for numbers and calculations. *Neuroimage*, *54*, 2382–2393.

Badre, D., & Wagner, A. D. (2004). Selection, integration, and conflict monitoring; assessing the nature and generality of prefrontal cognitive control mechanisms. *Neuron*, *41*, 473–487.

Bartra, O., McGuire, J. T., & Kable, J. W. (2013). The valuation system: A coordinate-based meta-analysis of BOLD fMRI experiments examining neural correlates of subjective value. *Neuroimage*, *76*, 412–427.

Bates, D., Maechler, M., Bolker, B., & Walker, S. (2015). Fitting Linear Mixed-Effects Models Using lme4. *Journal of Statistical Software*, *67*, 1–48.

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

Bekkens, A., Jansen, B. R., Van der Molen, M. W., & Huizenga, H. M. (2016). Cool decision-making in adolescents with

behavior disorder and/or mild-to-borderline intellectual disability. *Journal of Abnormal Child Psychology*, *44*, 357–367.

Blair, K., Marsh, A. A., Morton, J., Vythilingam, M., Jones, M., Mondillo, K., et al. (2006). Choosing the lesser of two evils, the better of two goods: Specifying the roles of ventromedial prefrontal cortex and dorsal anterior cingulate in object choice. *Journal of Neuroscience*, *26*, 11379–11386.

Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., & Cohen, J. D. (2001). Conflict monitoring and cognitive control. *Psychological Review*, *108*, 624–652.

Carter, C. S., Braver, T. S., Barch, D. M., Botvinick, M. M., Noll, D., & Cohen, J. D. (1998). Anterior cingulate cortex, error detection, and the online monitoring of performance. *Science*, *280*, 747–749.

De Neys, W., Vartanian, O., & Goel, V. (2008). Smarter than we think: When our brains detect that we are biased. *Psychological Science*, *19*, 483–489.

Gigerenzer, G., & Goldstein, D. G. (1996). Reasoning the fast and frugal way: Models of bounded rationality. *Psychological Review*, *103*, 650–669.

Huizenga, H. M., Crone, E. A., & Jansen, B. J. (2007). Decision-making in healthy children, adolescents and adults explained by the use of increasingly complex proportional reasoning rules. *Developmental Science*, *10*, 814–825.

Hunt, L. T., Kolling, N., Soltani, A., Woolrich, M. W., Rushworth, M. F., & Behrens, T. E. (2012). Mechanisms underlying cortical activity during value-guided choice. *Nature Neuroscience*, *15*, 470–476, S471–S473.

Jansen, B. R., van Duijvenvoorde, A. C., & Huizenga, H. M. (2012). Development of decision making: Sequential versus integrative rules. *Journal of Experimental Child Psychology*, *111*, 87–100.

Kable, J. W., & Glimcher, P. W. (2007). The neural correlates of subjective value during intertemporal choice. *Nature Neuroscience*, *10*, 1625–1633.

Knutson, B., Taylor, J., Kaufman, M., Peterson, R., & Glover, G. (2005). Distributed neural representation of expected value. *Journal of Neuroscience*, *25*, 4806–4812.

Krajich, I., Armel, C., & Rangel, A. (2010). Visual fixations and the computation and comparison of value in simple choice. *Nature Neuroscience*, *13*, 1292–1298.

Leisch, F. (2004). FlexMix: A general framework for finite mixture models and latent class regression in R. *Journal of Statistical Software*, *11*, 1–18.

Liston, C., Matalon, S., Hare, T. A., Davidson, M. C., & Casey, B. J. (2006). Anterior cingulate and posterior parietal cortices are sensitive to dissociable forms of conflict in a task-switching paradigm. *Neuron*, *50*, 643–653.

Mc Kell Carter, R., Meyer, J. R., & Huettel, S. A. (2010). Functional neuroimaging of intertemporal choice models: A review. *Journal of Neuroscience, Psychology & Economics*, *3*, 27–45.

Minati, L., Grisoli, M., Seth, A. K., & Critchley, H. D. (2012). Decision-making under risk: A graph-based network analysis using functional MRI. *Neuroimage*, *60*, 2191–2205.

Payne, J. W., Bettmann, J. R., & Johnson, E. J. (1993). *The adaptive decision-maker*. Cambridge, UK: Cambridge University Press.

Platt, M. L., & Glimcher, P. W. (1999). Neural correlates of decision variables in parietal cortex. *Nature*, *400*, 233–238.

Pochon, J. B., Riis, J., Sanfey, A. G., Nystrom, L. E., & Cohen, J. D. (2008). Functional imaging of decision conflict. *Journal of Neuroscience*, *28*, 3468–3473.

Rao, L. L., Zhou, Y., Xu, L., Liang, Z. Y., Jiang, T., & Li, S. (2011). Are risky choices actually guided by a compensatory process? New insights from fMRI. *PLoS One*, *6*, e14756.

- R Development Core Team (2015). *R: A language and environment for statistical computing*. R Foundation for Statistical Computing, Vienna, Austria. URL <https://www.R-project.org/>
- Ridderinkhof, K. R., Ullsperger, M., Crone, E. A., & Nieuwenhuis, S. (2004). The role of the medial frontal cortex in cognitive control. *Science*, *306*, 443–447.
- Riedl, R., Brandstätter, E., & Roithmayr, F. (2008). Identifying decision strategies: A process- and outcome-based classification method. *Behavior Research Methods*, *40*, 795–807.
- Rolls, E. T., McCabe, C., & Redoute, J. (2008). Expected value, reward outcome, and temporal difference error representations in a probabilistic decision task. *Cerebral Cortex*, *18*, 652–663.
- Rushworth, M. F., Buckley, M. J., Behrens, T. E., Walton, M. E., & Bannerman, D. M. (2007). Functional organization of the medial frontal cortex. *Current Opinion in Neurobiology*, *17*, 220–227.
- Russo, J. E., & Doshier, B. A. (1983). Strategies for multiattribute binary choice. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *9*, 676–696.
- Sanfey, A. G. (2004). Neural computations of decision utility. *Trends in Cognitive Sciences*, *8*, 519–521.
- Sanfey, A. G. (2007). Social decision-making: Insights from game theory and neuroscience. *Science*, *318*, 598–602.
- Scheibehenne, B., Rieskamp, J., & Wagenmakers, E. J. (2013). Testing adaptive toolbox models: A Bayesian hierarchical approach. *Psychological Review*, *120*, 39–64.
- Serences, J. T. (2008). Value-based modulations in human visual cortex. *Neuron*, *60*, 1169–1181.
- Shenhav, A., Botvinick, M. M., & Cohen, J. D. (2013). The expected value of control: An integrative theory of anterior cingulate cortex function. *Neuron*, *79*, 217–240.
- Singmann, H., Bolker, B., & Westfall, J. (2015). *Afex: Analysis of factorial experiments*. R package version 0.15–12. <https://CRAN.R-project.org/package=afex>.
- Smith, D. V., & Huettel, S. A. (2010). Decision neuroscience: Neuroeconomics. *Wiley Interdisciplinary Reviews. Cognitive Science*, *1*, 854–871.
- Sugrue, L. P., Corrado, G. S., & Newsome, W. T. (2004). Matching behavior and the representation of value in the parietal cortex. *Science*, *304*, 1782–1787.
- Tom, S. M., Fox, C. R., Trepel, C., & Poldrack, R. A. (2007). The neural basis of loss aversion in decision-making under risk. *Science*, *315*, 515–518.
- Trepel, C., Fox, C. R., & Poldrack, R. A. (2005). Prospect theory on the brain? Toward a cognitive neuroscience of decision under risk. *Brain Research, Cognitive Brain Research*, *23*, 34–50.
- Tversky, A., & Kahneman, D. (1974). Judgment under uncertainty: Heuristics and biases. *Science*, *185*, 1124–1131.
- Tversky, A., & Kahneman, D. (1992). Advances in prospect theory: Cumulative representation of uncertainty. *Journal of Risk and Uncertainty*, *5*, 297–323.
- Tversky, A., Sattath, S., & Slovic, P. (1988). Contingent weighting in judgment and choice. *Psychological Review*, *95*, 371–384.
- Van Duijvenvoorde, A. C., Jansen, B. R., Visser, I., & Huizenga, H. M. (2010). Affective and cognitive decision-making in adolescents. *Developmental Neuropsychology*, *35*, 539–554.
- Venkatraman, V., & Huettel, S. A. (2012). Strategic control in decision-making under uncertainty. *European Journal of Neuroscience*, *35*, 1075–1082.
- Venkatraman, V., Payne, J. W., Bettman, J. R., Luce, M. F., & Huettel, S. A. (2009). Separate neural mechanisms underlie choices and strategic preferences in risky decision making. *Neuron*, *62*, 593–602.
- Venkatraman, V., Rosati, A. G., Taren, H. H., & Huettel, S. A. (2009). Resolving response, decision, and strategic control: Evidence for a functional topography in dorsomedial prefrontal cortex. *Journal of Neuroscience*, *29*, 13158–13164.
- Vlaev, I., Chater, N., Stewart, N., & Brown, G. D. (2011). Does the brain calculate value? *Trends in Cognitive Sciences*, *15*, 546–554.
- Von Neumann, J., & Morgenstern, O. (1944). *Theory of games and economic behavior*. Princeton, NJ : Princeton University Press.