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Path dependence in risky choice: Affective and deliberative processes in brain and behavior☆

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ABSTRACT

Decision-makers show an increased risk appetite when they gamble with previously won money, the *house money effect*, and when they have a chance to make up for a prior loss, the *break even effect*. To explore the origins of these effects, we use functional magnetic resonance imaging to record the brain activities of subjects while they make sequential risky choices. The behavioral data from our experiment confirm the path dependence of choices, despite the short trial duration and the many task repetitions required for neuroimaging. The brain data yield evidence that the increased risk appetite after gains and losses is related to an increased activity of affective brain processes and a decreased activity of deliberative brain processes.

1. Introduction

The question of how individuals make decisions under risk is fundamental to economics and has captivated researchers for centuries. Over the past 25 years or so, behavioral studies have convincingly demonstrated that risk attitudes are path dependent, a notion that is at odds with rational choice theory. Most notably, Thaler and Johnson's (1990) experiments show that people tend to take more risk if they have a chance to make up for a prior loss, a phenomenon known as the break even

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effect (BEE). Conversely, people also display a greater risk appetite after a prior gain that is large enough to cover potential losses. Thaler and Johnson label this effect the house money effect (HME), referring to gamblers' feelings that they are not playing with their own money when they are ahead. In the present study, we employ functional neuroimaging techniques to examine the neural mechanisms that underlie this path dependence in risky choice.

Path dependence in risky choice has been observed in various settings. For example, McGlothlin (1956) reports that gamblers at racetracks display an increased propensity to bet on long shots at the end of the racing day, presumably in an attempt to recover earlier losses. Post et al. (2008) observe an increased risk appetite after gains and losses in the large-stake TV game show "Deal or No Deal", where contestants make a series of choices between cashing out with a certain lump sum or taking a risk for a larger reward by continuing to play. Studies by Smith et al. (2009) and Coval and Shumway (2005) point out that path dependence even extends to situations where decision-makers are experts in the domain: experienced online poker players take more risk after big losses, and Chicago Board of Trade proprietary traders display a greater risk appetite in afternoon trading sessions after morning losses. Barberis et al. (2001) show that path-dependent risk attitudes can have a substantial effect on asset returns.

Behaviorally, path dependence in risky choice can be explained within the framework of Kahneman and Tversky (1979). A distinguishing feature of their descriptive Prospect Theory relative to the more normative Expected Utility Theory (von Neumann and Morgenstern, 1947) is the reference-dependent valuation of outcomes: preferences are defined over gains and losses (relative to a reference point) rather than final wealth states. In general, people tend to show moderate risk-averse behavior in the gain domain and risk-seeking behavior in the loss domain, as well as a relatively strong risk aversion for mixed gambles due to loss aversion, that is, a greater sensitivity to losses than to gains. The value function in Prospect Theory captures these tendencies through diminishing sensitivity to increments in gains and losses and a steeper slope for losses than for gains of a similar size.[1] In a dynamic context, these properties entail a relatively high risk tolerance after both gains and losses if people do not update their reference point. If the reference point is sufficiently high after prior losses or low after gains, decision-makers display the risk-seeking behavior that is predicted by the convex shape of the value function for losses, or the moderate risk-averse behavior that is predicted by the concave value function for gains, respectively. In both cases, the impact of loss aversion is mitigated or absent.

Psychological evidence suggests that decisions are the result of both an affective (or "intuitive") and a deliberative (or "reflective") system of thinking (Chaiken and Trope, 1999; Kahneman, 2003). The former system is assumed to be fast, effortless, automatic and associative, while the latter is characterized by slower and more effortful processing. In this light, preferences are proposed to reflect a combined result of these two systems, with the affective system driving nonlinearities in valuation and the deliberative system valuing outcomes more linearly (Hsee and Rottenstreich, 2004; Mukherjee, 2010). As a consequence, choices can vary and depend on how strongly the two systems are involved in solving the decision problem.

Findings from neuroimaging research support the idea that two different brain systems drive choice behavior. De Martino et al. (2006) show that people's sensitivity to the manner in which choice options are presented is driven by affective neural processes, and that cognitive control mechanisms are more active when the behavior of the decision-maker is less sensitive to framing. A study by Roiser et al. (2009) finds that a subject group that exhibits only weak behavioral framing effects has increased connectivity between control and affective brain regions, suggesting the presence of dynamic regulatory control over emotional reactions, whereas a subject group exhibiting strong behavioral effects has weaker connectivity within this brain network. Similarly, loss aversion has been related to affective mechanisms in the brain. Knutson et al. (2008b) suggest that affective reactions in the brain, specifically in the insula, strengthen the endowment effect (Kahneman et al., 1991; Thaler, 1980) and thus increase loss aversion in selling situations. De Martino et al. (2010) report that patients with brain damage in another affect-related region, the amygdala, show a dramatically lower level of loss aversion than healthy people do.

Previous neuroscience and behavioral studies have also investigated how positive and negative experiences can influence subsequent choice behavior, with mixed findings reported to date. Kuhnen and Knutson (2005, 2011) show that negative (positive) affective states precede future tendencies to avoid (accept) uncertain prospects. In contrast, the findings of Andrade and Iyer (2009) and Demaree et al. (2012) suggest that a negative affective state entails an increased risk appetite.

The role of deliberative processes also remains unclear. Xue et al. (2011) report that the tendency to take more risk after a loss than after a gain is associated with higher activity of cognitive control processes, while Campbell-Meiklejohn et al. (2008) find increased cognitive control-related and anxiety-related activity when people decide to stop chasing previous losses.

In the light of this literature and given the key roles of framing and nonlinear preferences for path dependence, we conjecture that the BEE and HME are driven by affective processes and suppressed by deliberative processes.[2] A number of recent behavioral studies have already found some evidence in this direction (Andrade and Iyer, 2009; Demaree et al., 2012; Monga and Rao, 2006). To examine whether people's brain responses to gains and losses are also in accordance with this

[1] We ignore the effect of probability weighting here. Prospect Theory actually describes a fourfold pattern of risk aversion, as probability weighting can lead to risk aversion for low-probability losses and risk seeking for low-probability gains. The relevant choice problems in our experiment always use 50/50 gambles.

[2] To some degree it is still an open question as to whether "affective processes" form a unitary system that is activated after both gains and losses, or whether there is a complex network of affect-related mechanisms that are different for gain and loss situations. Similarly, we consider it an open question whether the "deliberative process" is truly a single mechanism, or rather an aggregate description of a network of different processes involved in controlling behavior.

hypothesis, we record the brain activities of decision-makers with functional magnetic resonance imaging (fMRI) while they make a series of sequential choices. We map the activity of affective and deliberative processes of our subjects during two phases: when they are informed of the prior outcome, and when they make their subsequent decision. To explore whether brain activity is associated with subsequent choice behavior, we also investigate whether safe and risky choices are preceded by differential patterns of activity in the affective and deliberative brain networks.

In our experimental design, subjects repeatedly make two choices, where the second choice problem depends on the outcome of the first choice. As a novel feature, our design includes not only gain and loss outcomes, but also a third "neutral" outcome. Another uniqueness is that we have designed our experiment in such a way that it allows us to study the effects of prior outcomes without potential confounds. We also use relatively large monetary incentives.

Behaviorally, our participants display both the BEE and the HME while undergoing fMRI. More importantly, our analyses of the brain data reveal that the increased risk appetite after both gains and losses is indeed related to an increased activity of affective processes and a decreased activity of deliberative processes. Moreover, brain activity in the outcome phase is associated with choice in the subsequent decision stage. These findings are important and novel, because they show that path dependence in risky choice is driven by the dynamics of the balance between affective and deliberative systems. Transient external events (such as a volatile, arousing market situation) or changes in internal states (such as mood) can affect this balance. Our results have the potential to inform policy, implying that interventions to either system can be considered to reduce a decision-maker's path dependence.

The remainder of the paper is organized as follows. In Section 2, we provide background on affective and deliberative brain mechanisms. Section 3 presents our neuroimaging experiment. Section 4 reports the behavioral patterns of our subjects, explains the methods that we use to analyze the fMRI data, and presents the fMRI results. In Section 5 we summarize and discuss our results, put forward some general limitations of neuroimaging, and conclude.

2. Affective and deliberative processes in the brain

Even though the human brain consists of anatomically separable brain areas, the functional specificity of each region is not clear-cut. Different brain regions are heavily interconnected, and a single cognitive process is often performed by a network of interacting areas. The following discussion is limited to the central brain networks that relate to affective processing and deliberation.

2.1. Affective mechanisms

Damasio and Carvalho (2013) define emotions as a set of physiological reactions, such as alteration in heart rate or attention, that are triggered by an external stimulus and aim to restore or maintain homeostatic balance, "a status-quo of the body". Emotional feelings arise when interoception of bodily state changes reaches consciousness. Brain regions on all levels of processing, from the midbrain to the celebral cortex, have been linked to the generation of emotions and affective arousal. Converging evidence from neuroscience suggests that each specific basic emotional state, such as happiness or sadness, is associated with a distinct network of brain regions (Vytal and Hamann, 2010). In this discussion we focus on the general affective arousal mechanisms.

The neuroscience literature often distinguishes between the processing of positive and negative events. The processing of positive events and rewards is closely related to the functioning of the neurotransmitter dopamine. While dopamine itself cannot be directly measured by the use of noninvasive and radiation-free technologies such as fMRI, we can image areas known to be targets of dopaminergic neurons in the midbrain, such as the striatum and the ventromedial prefrontal cortex (VMPFC). These regions are activated during the receipt of both primary and secondary rewards (Bartra et al., 2013), such as drinks (Berns et al., 2001; Plassmann et al., 2008) and monetary amounts (Delgado et al., 2003; Knutson et al., 2000; O'Doherty et al., 2001; Thut et al., 1997), and they also reflect the hedonic value of rewards (Kringelbach, 2005; Plassmann et al., 2008). In line with the behavioral reference dependence of valuation, the reward circuits—especially the striatum—process stimuli largely in a reference-dependent manner. A variety of contextual aspects have been shown to influence the evaluation of received rewards (Hytönen and Sanfey, 2011), such as other possible outcomes (Breiter et al., 2001) and the outcomes of others (Fliessbach et al., 2007). In fact, the striatum is often argued to reflect the receipt of a better-than-expected outcome of a past choice (Delgado et al., 2000; Hare et al., 2008).

Dopamine also has an important role in guiding behavior (Schultz and Dickinson, 2000). In general, increased dopamine activity at the time of reward receipt—as reflected by activation in the midbrain and striatum—reinforces behaviors that have led to the rewarded outcomes. There is evidence that such reinforcement signals can increase the preference for risky assets (Cohen, 2008; Kuhnen and Knutson, 2005). Even exogenous modulation of striatum activity, for example by presenting visual stimuli with a strong positive valence, increases subjects' risk appetite (Knutson et al., 2008a). Thus, based on the role of dopamine both in the receipt of rewards and in guiding future choice behavior, the dopamine system and the associated brain regions such as the striatum are likely candidates for driving the increase in risk appetite after prior gains.

Negative experiences often evoke activity in multiple brain structures. The amygdala and the anterior insula, for example, are broadly associated with negative emotions such as fear and disgust (Dalgleish, 2004). Both of these brain areas have also been implicated in the contexts of negative monetary outcomes in uncertain situations (Kuhnen and Knutson, 2005; Yacubian et al., 2006) and receiving worse outcomes than expected (Seymour et al., 2004). Negative affective reactions

can also influence subsequent risk-taking behavior. For instance, increased preference for certainty has been shown to be preceded by a negatively valenced insula reaction (Kuhnen and Knutson, 2005). In addition to the amygdala and the insula, previous studies have also indicated the role of the lateral orbitofrontal cortex (OFC) when receiving punishments (Kringelbach, 2005). Some studies also report that increasing negative monetary outcomes decreases activity in the reward structures, such as the striatum (Delgado et al., 2000; Tom et al., 2007).

Although the insula is often related to negative experiences, converging evidence indicates that the anterior insula can also reflect positive affective arousal. The reaction in the anterior insula when experiencing subjective emotions frequently co-occurs with activations of sub-regions of the anterior cingulate cortex (ACC). As summarized by Craig (2009), the "network" of anterior insula and ACC is consistently activated in studies of emotional arousal, including a vast range of emotional states ranging from love and happiness to anger and disgust. Multiple studies have linked the anterior insula and the ACC to risky decision making. A recent comprehensive meta-analysis across various experimental paradigms shows that risk consistently activates the insular cortex (Mohr et al., 2010). This suggests that insular activity serves as a fast affective estimate of a risky situation, and supports the risk-as-feeling hypothesis that emphasizes the role of emotions in decision making under risk (Loewenstein et al., 2001). There is, however, less convergence about the relation between insula activity and behavior. Some studies report a negative correlation between anterior insula activity and the propensity to take risk (Campbell-Meiklejohn et al., 2008; Knutson and Bossaerts, 2007; Liu et al., 2007), whereas others find a positive correlation (Clark et al., 2009; Paulus et al., 2003; Platt and Huettel, 2008; Xue et al., 2010). The results for the ACC seem to be more consistent, and point at a positive correlation between activity level and the propensity to take risk (Christopoulos et al., 2009; Cohen et al., 2005). We also note, however, that the ACC is a relatively large structure. It performs a wide range of cognitive functions, and is also associated, for example, with conflict, error detection, and attention (Carter and van Veen, 2007; Ericson and Fuster, 2011).

Based on these neuroscience insights, we may expect to find higher involvement of the emotion-related, interconnected salience network (Seeley et al., 2007)—consisting of the midbrain, the striatum, the insula, the ACC, and the amygdala—when people experience gain or loss outcomes, compared to relatively neutral outcomes. This network may have dissociable areas that respond differently to gains and losses. Last, we may also expect that activity in these affective brain areas correlates with the propensity to take risk in the second decision stage of the two-stage choice problems that we use.

2.2. Executive control mechanisms

Executive control mechanisms are top-down mental processes that are used when task performance requires operation and coordination of multiple basic cognitive tasks. The executive functions (e.g., error detection, behavioral control, and task-switching) are considered to be "cool" mental processes that can regulate "hot" processes such as undesired emotional reactions (Diamond, 2013; Hofmann et al., 2012). These control mechanisms are associated with an interconnected network of brain regions that includes the dorsolateral prefrontal cortex (DLPFC), the ventrolateral prefrontal cortex (VLPFC), parts of the cingulate cortex, and the lateral parietal cortices (including the intraparietal lobule; Seeley et al., 2007). Related controlling actions range from inhibiting the execution of planned motor responses (Liddle et al., 2001) to exerting self-control over dietary choices (Hare et al., 2009). Previous research in neuroeconomics has also demonstrated the relevance of this network for controlling risky choice. For instance, Campbell-Meiklejohn et al. (2008) report increased activity in the parietal cortices when people decide to stop chasing previous losses, and experiments by Knoch et al. (2006) and Fecteau et al. (2007) indicate a causal relationship between activity in the right DLPFC and risk appetite.

Taken together, these studies suggest that we will see less activity in the deliberation-related, interconnected executive control network after gains and losses than after neutral outcomes. Also, we may expect that the activity in these executive control mechanisms correlates negatively with the subsequent propensity to take risk.

3. The experiment

To examine the processes that underlie path dependence in risky choice behavior, we utilize fMRI to record the brain activity of subjects while they engage in dynamic risky choice problems. fMRI measures brain activation indirectly, by registering modulations in the local magnetic properties which relate to changes in the blood flow, as it is widely assumed that the blood flow dynamics in a particular brain region reflect the level of neural activity in that area. We refer to Huettel et al. (2009) for extensive background on fMRI.

3.1. Design

The use of fMRI imposes a number of constraints on our experimental design. Due to the slow nature of the hemodynamic responses (blood flow changes) in the brain, fMRI signal changes that are caused by a neuronal activity are delayed (start ~2 s after stimulus onset) and blurred (one single short event can cause a response of ~10–25 s). Other typical features of fMRI data are that the signals are noisy and that they represent a relative rather than an absolute measure of brain activity. These features require experimenters to repeat the events of interest many times, to separate them in time, and to include one or more reference conditions in the experimental design that serve as a comparison base for the degree of brain activity.

Based on previous behavioral research by Post et al. (2008), we developed an fMRI-compatible sequential choice paradigm. In our design, subjects experience changes in the expected value of a lottery that they own in the first decision stage of a

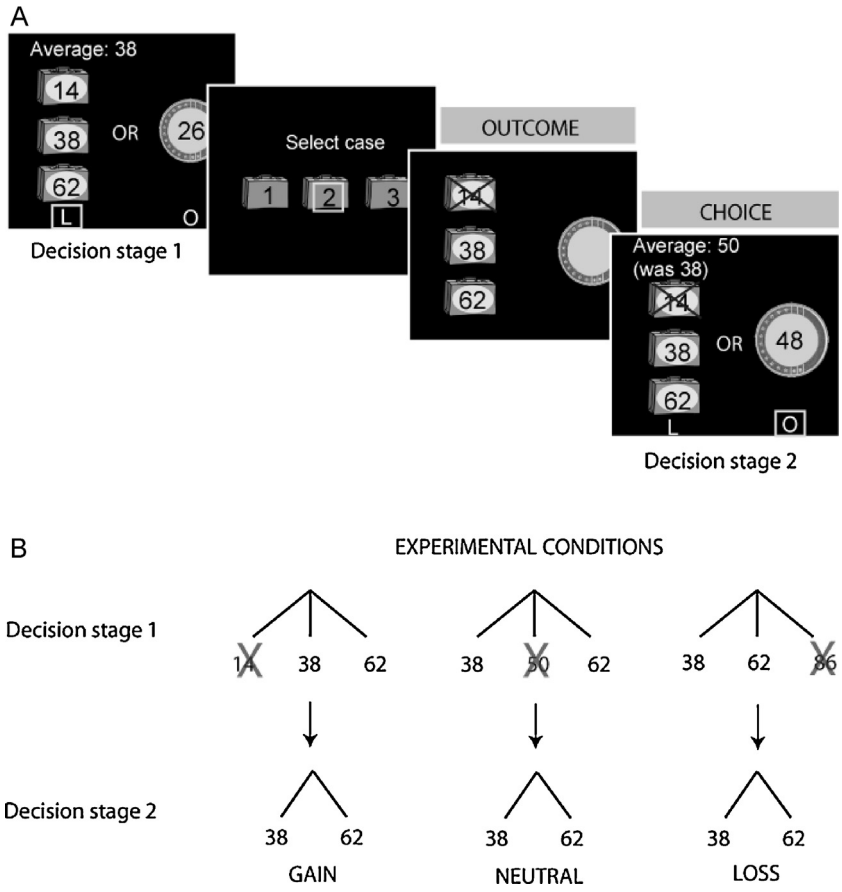

Fig. 1. Sequential choice paradigm. (A) After choosing the three-prize lottery (L) in the first decision stage, subjects select one briefcase containing a prize to be removed from the lottery. In the outcome phase, subjects find out which prize has been removed. Next, they proceed to the second decision stage. Our analysis concentrates on the brain activities that occur during the outcome phase and during the subsequent choice phase. (B) The design allows us to compare numerically identical choices in the second decision stage that differ only in how they were reached. Each two-prize lottery-offer pair from a set of 24 is presented under three conditions: once after experiencing a gain (smallest prize removed after the first decision stage), once after a neutral outcome (middle prize removed), and once after a loss (largest prize removed).

two-stage dynamic choice problem. This lottery initially consists of three equally likely prizes, and one of these prizes is randomly eliminated after the first decision stage. In line with a growing literature that suggests that reference points for gains and losses are expectation-based (Abeler et al., 2011; Ericson and Fuster, 2011; Kőszegi and Rabin, 2006, 2007), we assume that the resulting change in the expected value of the lottery generates the experience of a gain or a loss.

More specifically, the subjects' task was to choose between a risky lottery (lottery choice: L) and a sure amount of money (offer choice: O). Each dynamic choice problem (henceforth: trial) consisted of a maximum of two decision stages, depending on the subjects' choice in the first decision stage. See Fig. 1A for an example. In the first stage, subjects chose either a sure money amount or a lottery with three money prizes. Choosing the offer ended the trial. After a lottery choice, subjects had to choose one of three visually identical briefcases, knowing that the randomly assigned and hidden prize inside this briefcase would be removed from the lottery. With this procedure, we hoped to stimulate subjects' awareness of the change in their endowment. During the outcome phase, subjects found out which prize they had selected and which two prizes still remained. Next, subjects entered the second decision stage, where they had to choose either the two-prize lottery or a new sure amount.

To avoid possible carry-over effects between the many trials, subjects who chose the lottery in the second decision stage were not informed which of the two prizes they had ultimately won. At the end of the experiment, one of the trials was randomly selected and subjects were paid according to the outcome of this trial. If the selected trial had ended with a lottery choice in the second decision stage, the outcome was determined by the roll of a die. See Baltussen et al. (2012) for a discussion and test of this "random incentive system". On average, subjects earned €46.

The choice problems were designed such that subjects faced each second-stage lottery-offer combination from a pre-determined set of 24 combinations under three experimental conditions: once after experiencing a gain (smallest prize removed after the first decision stage), once after a neutral outcome (middle prize removed), and once after a loss (largest prize removed). We refer to the 72 trials that ended with one of these 24 special combinations as trials of interest. This

approach allows us to compare choices across decision problems that are numerically identical but different in the way they were reached. See Fig. 1B for an example. The monetary amounts used as prizes varied from €1 to 116, with the smallest and the largest in each starting set of three prizes differing by €12–56. The middle prize was always equal to the expected value of the lottery. In addition to the 72 trials of interest, we conducted 24 filler trials to ensure that subjects would also experience losses (gains) in tasks that started with the very lowest (highest) prizes.

The riskless money offers in the first decision stage were set considerably lower than the expected value of the lottery, in order to encourage subjects to select the lottery and thus to proceed to the second decision stage. To ascertain that the first-stage choices were not seen as trivial, the offers were adjusted for each individual subject on the basis of her choices during the experiment, in such a way that they would be high enough to be considered but low enough to motivate the subject to select the lottery. If a subject ended a trial of interest in the first decision stage by selecting the riskless offer, that specific trial was repeated once at the end of the experiment with a lower riskless offer in the first decision stage. The offers for the second-stage choices were set at a level where an average subject would be roughly indifferent between the lottery and the offer. These presupposed certainty equivalents were based on results from independent pretests with 20 subjects in a behavioral lab.

In 12 additional "catch trials", we intentionally set extremely low or extremely high offers to ensure that subjects continued to pay attention throughout the entire experiment. In total, each subject performed at least 108 different trials (mean = 115; stdev = 6). The trials were presented in a semi-random order to make sure that similar first and/or second decision stage lotteries did not occur in close proximity to each other. The semi-random ordering was individually predetermined for each subject.

Before the brain scan, subjects were given written instructions about the task and briefly trained outside and inside the scanner. The actual fMRI session was divided into two ∼25-min scanning parts (mean = 25.4; stdev = 0.6) that were separated by a ∼5-min break outside the fMRI scanner. The stimuli were presented using Presentation® software (Neurobehavioral Systems). The trials were separated by a jittered 4–5 s delay interval. In the decision stages, subjects had 5.5 s to respond (after a forced delay of 1.5 s), using one of two response buttons (left for a lottery choice and right for an offer choice). After the first decision stage, subjects had 2.5 s to use one of three buttons to indicate which hidden prize was to be removed from the lottery (see Fig. 1A). The outcome screen was shown for 4 s, and separated from the other screens by a jittered 1–2 s delay interval. These random delays are used to better separate brain activities that are related to different phases of the task.[3] We ended each session with an anatomical brain scan.

3.2. MRI data acquisition

We collected the brain data on an Avanto 1.5 Tesla (Siemens, Munich) scanner. During the fMRI measurements, subjects were placed on a movable examination table inside the magnet bore. The subject's head was placed tightly in position with foam padding to minimize artificial signal intensity changes due to head motion. While lying inside the MRI scanner, subjects could view the visual stimulation from a screen at the end of the magnet bore with the help of a mirror system.

We measured the blood oxygenation level-dependent (BOLD) signals with ascending slice acquisition, and used a T2* weighted echo-planar imaging (EPI) sequence with the following standard imaging parameters: 32 axial slices; 2.34 s volume repetition time (TR); 35 ms echo time (TE); 90° flip angle; 64 × 64 slice matrix; 3.5 mm slice thickness; 0.35 mm slice gap; and 212 mm field of view. We acquired anatomical brain images with a T1-weighted GRAPPA sequence: 176 sagittal slices; 2.25 s TR; 2.95 ms TE; 15° flip angle; 256 × 256 slice matrix; 1.0 mm slice thickness; 0.5 mm slice gap; and 256 mm field of view.

3.3. Subjects

Twenty-nine healthy students from Radboud University Nijmegen and HAN University of Applied Sciences participated in the study. Ten were not suited for inclusion in our final sample. For some, the certainty equivalents that we used in the second decision stage did not work well. To avoid ceiling and floor effects in the second-stage choice data, we excluded subjects who chose either the lottery or the offer more than 90 percent of the time (across all trials of interest for the three experimental conditions combined). Five subjects did not pass this criterion. We also excluded subjects who were not successful in detecting at least six of the 12 catch trials. Four subjects did not pass this criterion (one of which also did not satisfy the 10-percent condition). One subject completed insufficient trials for our fMRI analysis by frequently selecting the sure outcome in the first decision stage (we applied a minimum requirement of 12 successful repetitions per experimental condition). We also had to exclude one subject as a result of technical problems during scanning. Our final sample comprised 19 subjects (nine male), with a mean age of 22.1 years (stdev = 2.2).

[3] Typically, interstimulus intervals are longer than the intervals that we employed here, although similarly short intervals have been used previously in event-related fMRI experiments (e.g., Brown et al., 2006; Rubia et al., 2005). In our design, the relatively short jittered intervals provided sufficient separation between outcome phase and second decision stage to enable modeling of both time windows with a general linear model.

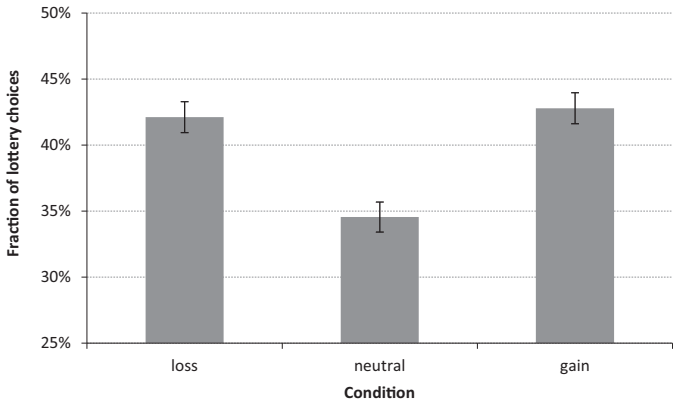

Fig. 2. Break even and house money effects in behavior. For each condition, the figure displays the observed percentages of lottery choices in the second decision stage for the trials of interest of all subjects combined. The sets of choice problems are normatively identical across the three conditions, but different in how they were reached. In the loss (gain) condition, subjects experienced a drop (rise) in the expected value in the preceding outcome phase. In the neutral condition, the expected value remained the same.

4. Methods and results

4.1. Path dependence in behavior

We start with an analysis of subjects' choices in the second stage of the trials of interest. That is, we compare the decision situations that have similar lottery-offer pairs, but differ in how they were reached (after either a gain, neutral, or loss outcome). This way we can examine the influence of prior outcomes without any confounding effects.

Fig. 2 displays the pattern of path dependence in the choice data. Subjects selected the lottery option 42.1 percent of the time after a loss and 42.8 percent of the time after a gain, but only 34.6 percent of the time in the neutral condition. In line with the BEE and HME, they have a statistically significantly higher propensity to take risk after losses and after gains than after neutral outcomes (BEE: $z = 2.308$, $p = 0.021$; HME: $z = 2.510$, $p = 0.012$; paired proportion tests).

Although the set of second-stage choice problems is identical across the three conditions, the 24 choice problems within this set are different. To control for variations in the expected value and risk of the lottery option and in the attractiveness of the offer option, we also run the following linear probit regression model:

$$l_{i,j} = \Phi(\beta X_{i,j} + \alpha_i) + u_{i,j} \quad (1)$$

where the dependent variable $l_{i,j}$ is the decision of subject i in trial of interest j (with a value of 1 for a lottery choice and 0 for an offer choice), $\Phi(\cdot)$ is the standard normal cumulative distribution function, β is the parameter vector, $X_{i,j}$ is the matrix of explanatory variables, α_i is the subject-specific fixed effect included to control for unobserved heterogeneity at the subject level, and $u_{i,j}$ represents an unobserved stochastic component. The explanatory variables of interest are two dummy variables that take the value of 1 for observations that are from the loss (BEE) or gain (HME) condition, respectively. Positive coefficients for these variables imply a higher propensity to take risk after a loss or gain, respectively, than after a relatively neutral outcome. To control for the stakes, for the expected return from taking the lottery option, and for the riskiness of the lottery option, we also include the variables EV (the expected value of the two remaining prizes of the lottery option), EV/O (the average remaining prize divided by the riskless offer), and Stdev/EV (the standard deviation of the remaining prizes divided by the average remaining prize), respectively.

Table 1 shows the regression results. The results confirm the above evidence for both a BEE ($\beta = 0.229$; $p = 0.013$) and a HME ($\beta = 0.248$; $p = 0.007$) in the data. Translated into marginal effects at the means, the sizes of the BEE and HME are 8.8 and 9.6 percentage points, respectively. Note that the insignificant coefficients for the control variables indicate that the offers for the second-stage choice problems were properly set.

All in all, these analyses of the behavioral data point out that our experimental design evoked clear prior outcome effects in our subjects' behavior, despite the short duration of each task and the many repetitions. The next step is to analyze the interaction of prior outcome experiences, choices, and brain activations.

4.2. Methods of fMRI analysis

We performed the data analyses using SPM5 (Wellcome Department of Imaging Neuroscience, London). As a standard procedure, the first four EPI scans were discarded to allow for the stabilization of magnetic properties (T1 equilibration). No actual data was lost, because the task began only after the measurement of these discarded scans. The remaining images were analyzed in two steps. First, we performed the standard preprocessing steps necessary for fMRI data analysis, and then we modeled the design-specific brain activations and conducted statistical analyses.

Table 1
Binary probit regression results. The table displays the results from the probit regression analysis of subjects' decisions to choose the lottery (1) or offer option (0) in the second decision stage of the trials of interest. BEE and HME are dummy variables taking the value of 1 for observations after a loss or gain, respectively. EV, EV/O and Stdev/EV are control variables that represent the stakes (the average value of the two remaining prizes for the lottery option), the expected return from taking the lottery option (the average remaining prize divided by the riskless offer option), and the riskiness of the lottery option (the standard deviation of the remaining prizes divided by the average remaining prize). In addition to the maximum likelihood estimates for the regression coefficients, the table reports the log-likelihood, McFadden's R^2, and the number of observations. The regression model includes fixed effects to control for unobserved heterogeneity at the subject level.

	Coefficient	*p*-Value
BEE	0.229	0.013
HME	0.248	0.007
EV	−0.002	0.705
EV/O	−13.932	0.390
Stdev/EV	2.368	0.539
LL	−765.6	
McFadden R^2	0.141	
No. obs.	1325	

The fMRI images were preprocessed to correct for known inaccuracies in the data and to prepare the data for group-level analysis. We first corrected the data for the slight head movements that occurred despite the supporting foam padding around the subject's head. Next, we corrected for differences in slice acquisition time. This step is necessary because the fMRI data is collected one "brain slice" at a time, and thus the data from different brain locations are collected at different time points. Then, because the size and shape of the brain varies from person to person, we transformed the data of individual subjects to a common brain coordinate system (the Montreal Neurological Institute (MNI) coordinate system) by first matching the functional data with each subject's own anatomical scan (coregistration), and then spatially squashing, or normalizing, the anatomical scan into the shape and size of a "standard brain" (MNI T1 template). After the transformation, we resampled the data into 2 mm × 2 mm × 2 mm voxels, or "three-dimensional pixels", and spatially smoothed the data (Gaussian kernel of 8 mm full-width at half-maximum). The spatial smoothing is essential for later statistical inferences where we used random field theory.

To make inferences from our data to the broader population, we used mixed-effects analysis with a two-level summary-statistics approach (Friston et al., 2005). In the first-level (within-subject) analysis, the design-specific brain activations were modeled separately for each subject. More specifically, we modeled the data in an event-related manner using the general linear model approach (Friston et al., 1995), whereby a linear model of expected time-series patterns (regressors) was fitted to all brain locations (voxels). In the final step on the single-subject level, we calculated summary statistics ("statistical maps") for the influence of the regressors of interest. In the second-level, random-effects (between-subject) analysis, we tested whether the combined individual statistics from the subjects show significant effects across experimental conditions. The approach is similar to standard statistical tests, the only difference being that the input for the statistical tests originates from the first-level statistics and not directly from the measured data.

We were particularly interested in two phases of the sequential choice paradigm: the outcome phase and the subsequent choice phase (marked with gray bars in Fig. 1A). The first-level model included three explanatory variables ("regressors") of interest for each of the two phases, one for each experimental condition (gain, neutral and loss). To allow for clear comparisons without any differences in the expected value and risk of the remaining two-prize lotteries, these six explanatory variables (three per phase) capture trials of interest only (trials where the second-stage lottery was numerically identical to that of trials in the other experimental conditions). In addition to these regressors of interest, we included regressors for trials that were not of interest, for the expected value and risk of the lotteries, for activation during the first-stage choice problems, and for residual movement artifacts (movement correction parameters).[4]

When modeling the fMRI signal time course it is necessary to take into account the dynamics of blood flow (hemodynamic response). We constructed the time course of each explanatory variable to reflect the expected hemodynamic response through a two-step procedure. First, we modeled the outcome phase with an impulse response function, and the choice phase with a boxcar function (starting at the time of the stimuli appearance and lasting 1.5 s, corresponding to the forced delay before any button could be pressed). To create the parametric regressors, we modulated the impulse response and boxcar functions by the expected value and risk. Second, we convolved the resulting time courses with a canonical hemodynamic response function to model the delayed reaction in the blood flow.

In the second-level analysis, we used analysis of covariance with three treatment levels (gain, neutral, loss) for the outcome phase and for the choice phase, while controlling for differences in the strength of the HME and BEE (as measured

[4] Expected value (EV) and risk (Stdev/EV) were calculated on the basis of the second-stage lottery. However, for the outcome phase, we used the first-stage EV in order to account for possible carry-over effects of differential reactions to the initial three-prize lotteries. By construction, trials with identical second-stage two-prize lotteries started with first-stage three-prize lotteries that differed systematically in expected value across gain (low EV), neutral (average EV) and loss (high EV) trials.

Table 2
Brain activity in the outcome phase. The table provides statistical information on the activated brain areas during the outcome phase, both at a cluster level (extent and strength of activation) and at a single-voxel level (voxel size: 2 mm × 2 mm × 2 mm). All clusters reported in the table are significant at the cluster level. The p-values are corrected for multiple comparisons. The peak coordinates indicate the location of the most strongly activated voxel in MNI coordinates. Unless stated otherwise, the neutral condition is used as a reference. L = left; R = right.

Anatomical region	Cluster-level		Voxel-level		Peak coordinates			
	Cluster size	*p*-Value	*Z*-Score	*p*-Value	*x*	*y*	*z*	
Activation during gains when compared to losses								
R ventral striatum	468	0.000	6.29	0.000	14	6	−10	
L ventral striatum	295	0.002	6.05	0.000	−14	4	−10	
R dorsal striatum	257	0.005	4.57	0.120	24	6	14	
VMPFC	672	0.000	4.67	0.084	6	42	−10	
MPFC	312	0.002	4.16	0.457	−22	62	8	
Activation during gains								
ACC	3715	0.000	5.85	0.000	−4	40	4	
L anterior insula	397	0.000	5.31	0.005	−32	20	−16	
R anterior insula	802	0.000	5.22	0.008	32	18	−16	
Midbrain, basal ganglia	564	0.000	5.93	0.013	8	−12	−12	
Activation during losses								
ACC	254	0.005	4.03	0.612	−2	38	4	
L anterior insula	189	0.021	4.05	0.586	−34	26	−12	
R anterior insula	155	0.046	4.14	0.478	34	18	−16	
L middle insula	56	0.020	4.24	0.009	−36	8	0	+
Activation during gains and losses								
ACC	220	0.010	4.03	0.612	−2	38	4	
L anterior insula	156	0.044	4.05	0.586	−34	26	−12	
R anterior insula	70	0.013	4.14	0.013	34	18	−16	+
Deactivation during gains								
L IPL	130	0.013	4.30	0.049	−32	−48	40	+
R IPL	82	0.0496	4.28	0.052	44	−40	54	+
Deactivation during losses								
L IPL	1570	0.000	5.79	0.000	−16	−56	50	
R IPL	827	0.000	5.03	0.018	44	−40	54	
Dorsomedial striatum	172	0.030	4.60	0.109	−14	22	8	
R DLPFC	242	0.006	4.21	0.392	58	14	38	
Occipital gyrus	348	0.001	4.26	0.349	30	−92	0	
Paracentral lobule	742	0.000	5.19	0.009	16	−32	62	
Precuneus	224	0.009	3.96	0.703	28	−68	38	
Deactivation during gains and losses								
L IPL	130	0.003	4.30	0.009	−32	−48	40	+
R IPL	82	0.011	4.28	0.009	44	−40	54	+
Dorsomedial striatum	64	0.019	4.24	0.011	−14	22	6	+

+ Significant in region-of-interest analysis only (statistics from the ROI analysis; see Section 4.2).

by the difference in the fraction of lottery choices between experimental conditions) across subjects. We also computed a "reduced model" without these covariates, and found very similar results.

We then compared a minimum of two regressors to localize those brain regions that were different across experimental conditions. For example, we tested which brain areas showed a stronger activation in the gain condition than in the neutral condition (gain > neutral). We used a conservative conjunction analysis with a conjunction null (gain > neutral and loss > neutral; Nichols et al., 2005) to identify the brain areas that were activated by both the gain and the loss events in contrast to the neutral condition, and used family-wise error correction on the basis of random field theory ($p < 0.05$) to account for multiple comparisons (due to the large number of voxels). We report activation that reached significance either at the voxel-level (with a 10-voxel extent threshold) or at the cluster-level. In the cluster-level inferences, the statistical maps were thresholded at a standard level of $Z > 3.1$ (corresponding to $p < 0.001$ uncorrected). This same threshold was used when creating the illustrations of brain activations. In addition to a whole-brain analysis, we also ran region of interest (ROI) analyses where we limited the search volume to those predefined brain regions which are associated with the affective and executive control processes, such as the insula, ACC, parietal cortex, and DLPFC (search volumes were defined anatomically with WFU PickAtlas). Tables 2 and 3 report the significant results of the various statistical analyses. Whole-brain corrected findings are also significant in ROI analyses, but the reverse is not always true. Results that are based on ROI analyses only are explicitly marked in the tables.

Table 3
Brain activity in the decision-making phase. The table provides statistical information on the activated brain areas during the second-stage choice phase. The results are presented in the same format as those in Table2.

Anatomical region	Cluster-level		Voxel-level		Peak coordinates			
	Cluster size	*p*-Value	*Z*-Score	*p*-Value	*x*	*y*	*z*	
Activation after losses								
ACC	243	0.008	4.06	0.557	6	34	18	
Deactivation after gains								
Occipital	505	0.000	4.59	0.105	−6	−88	20	
L IPL	130	0.015	4.56	0.017	−36	−56	42	+
R IPL	119	0.020	4.38	0.034	34	−54	34	+
Activation after losses when compared to gains								
Occipital	415	0.000	4.53	0.135	−6	−94	12	
ACC	186	0.027	4.51	0.141	8	34	18	
R thalamus	255	0.006	4.49	0.155	14	−18	6	
L thalamus	202	0.019	4.37	0.236	−26	−22	4	
Claustrum	228	0.011	4.25	0.334	−38	−4	0	
Insula	334	0.001	4.16	0.440	−48	4	10	
VLPFC, lateral OFC	415	0.000	3.95	0.696	30	58	10	
Superior temporal gyrus	191	0.024	3.91	0.736	58	−42	16	

+ Significant in region-of-interest analysis only (statistics from the ROI analysis; see Section 4.2).

For both the outcome phase and the choice phase we also tested whether the brain regions that showed differential brain activity across the experimental conditions were related to choice behavior. Ideally, we would have analyzed each experimental condition separately, but this was not possible due to a lack of observations.[5] Because of this limitation, we pooled the data from gain, neutral and loss trials, and tested whether the brain regions displayed a different degree of activity during the outcome or choice phase for offer choices than for lottery choices. We extracted the average signal strengths for the lottery and offer choices from the brain regions with the MarsBaR toolbox and compared these with a paired *t*-test.

4.3. *fMRI results*

4.3.1. *Outcome phase*

To examine whether gain, neutral, and loss experiences have different effects on the affective and executive control networks, we compare the brain activities at the time of the gain and loss experiences with those that occur when the neutral outcomes are revealed. Recall that the set of second-stage decision problems is identical across the three experimental conditions, implying that reported brain activity differentials are independent of the expected value and risk of the remaining two-prize lottery and the attractiveness of the offer option. Table 2 shows the results and the statistical details of all the contrasts for the outcome phase.

Our experimental paradigm relies on the assumption that subjects will evaluate the second-stage prospect relative to the (first-stage) prospect that they were originally endowed with. Before concentrating on the neural correlates of the HME and BEE, we first check the validity of this assumption by testing whether the brain's reward network—consisting of the striatum and the VMPFC—is more strongly activated during the outcome phase by a second-stage lottery that consists of the highest prizes from the first-round lottery (gain condition) than by an identical lottery that consists of the lowest prizes (loss condition). When we compare the gain condition to the loss condition, we indeed find stronger striatal, VMPFC and medial prefrontal cortex (MPFC) activity. We do not observe any other significant differences between the two conditions for the outcome phase. This indicates that prospects are indeed evaluated in a path-dependent manner in the brain's valuation network.

4.3.1.1. Affective network. We hypothesized that both the HME and the BEE are driven by affective processes, and thus expect that affective brain areas are more strongly activated by gains and losses than by neutral outcomes. To test this for the HME, we compare the brain responses during the outcome phase in gain trials with those in neutral trials. As predicted, the gain outcomes activate the left and right anterior insula, the ACC, and a cluster consisting of the midbrain and striatum (a part of the basal ganglia), all areas known to be engaged in affective processing. For the BEE, we compare the neural activity in loss and neutral trials and find more left and right anterior insula, left middle insula and (rostral) ACC activity for loss outcomes than for neutral outcomes. Strikingly, these regions overlap with those of the gain versus neutral comparison. Indeed, a strict conjunction analysis indicates that identical brain regions in the left and right anterior insula and the ACC are more activated

[5] The subjects included in the analysis all chose each option (lottery and offer) at least 10 percent of the time across the three experimental conditions combined, but in one single condition (consisting of 24 trials) the fraction of lottery or offer choices could be less than 10 percent (implying as few as 0-2 successful lottery or offer trials).

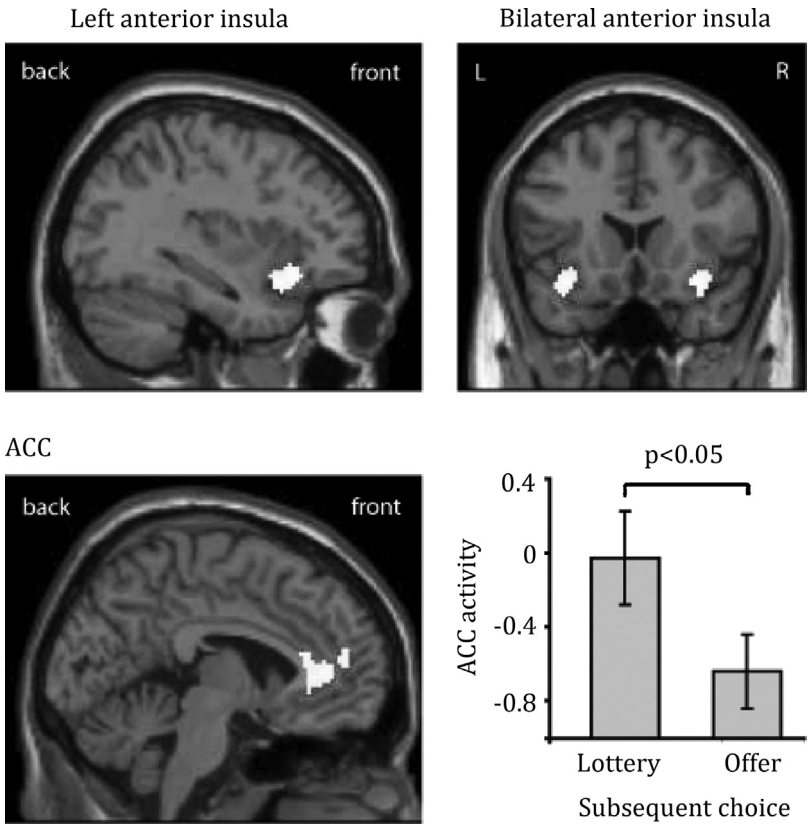

Fig. 3. Outcome phase: increased brain activity for gains and losses. Affective brain areas, including the left and right (bilateral) anterior insula and the ACC, were more activated by both gains and losses than by neutral outcomes. The bar graph shows that the average activity of the ACC cluster in the outcome phase was significantly higher in trials where subjects subsequently chose the lottery option than in trials where they chose the offer option. Note that subjects are unaware of the precise riskless alternative of this second-stage choice problem in the outcome phase.

by both gains and losses than by neutral outcomes, as shown in Fig. 3. We thus find that gains and losses evoke activity in the very same parts of the affective brain network (bilateral anterior insula and ACC) while some other parts of the affective network (the striatum and midbrain) are activated by gain outcomes only.

Given that gain and loss outcomes activate overlapping parts of the affective network, we test whether the activation of those brain regions is stronger in trials where subjects subsequently chose the lottery option than in those where they subsequently chose the offer option. As shown in Fig. 3, the analysis indicates that the ACC is more strongly activated in the outcome phase when subjects next chose the lottery option than when they next chose the sure offer ($p = 0.03$). Thus, the ACC shows increased activity levels during gain and loss experiences, with this increased activity preceding subsequent lottery choices. These results indicate that the ACC may be driving the behaviorally observed HME and BEE. In contrast, the activations of the cluster consisting of the midbrain and basal ganglia ($p = 0.18$) and the anterior insula (left side: $p = 0.69$; right side: $p = 0.84$) are not significantly different for lottery and offer choices.

4.3.1.2. Executive control network. We also hypothesized that experiences of gains and losses are associated with a decreased degree of activity in the executive control network relative to neutral outcomes. For gains, both the left and right intraparietal lobule (IPL) show decreased activity levels. These areas are part of the executive control network in the parietal cortices. For losses, we also observe decreased activity in the executive control network (see Fig. 4). More specifically, there is deactivation in the parietal cortices, including the left and right IPL, and in the right DLPFC. In addition, there is decreased activity in the occipital gyrus and in the dorsomedial striatum. The latter is a sub-part of the striatum, suggested to be involved in behavioral flexibility and cognitive control of behavior (mainly based on animal studies; Devan et al., 2011). The activity in the occipital gyrus (typically associated with visual perception) may reflect the visual differences between the screens. Taken together, these findings support our hypothesis.

To test whether there is a network of brain areas that shows decreased activation for both gain and loss outcomes, we perform a region of interest analysis by testing whether the gain outcomes deactivate the same brain regions as the loss

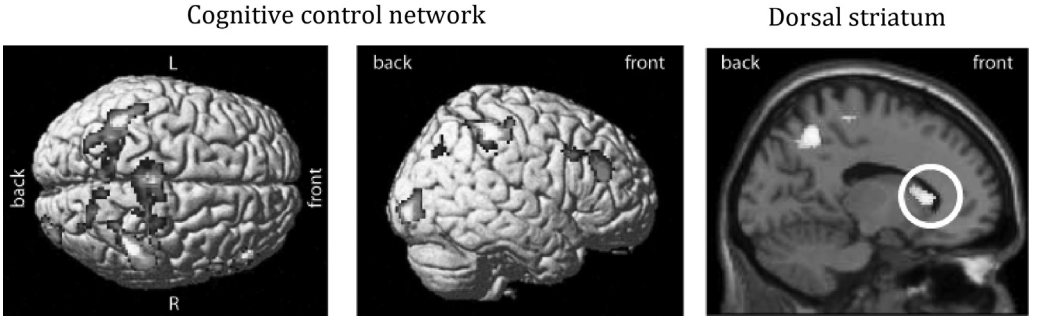

Fig. 4. Outcome phase: decreased brain activity for losses. Cognitive control network areas in the parietal cortices and in the right DLPFC, as well as in the dorsomedial striatum, deactivate for losses. For visualization purposes, the activation in the cognitive control network is projected on the cortical surface.

outcomes do.[6] Indeed, we find that the left and right IPL, and also the dorsomedial striatum, are significantly deactivated, indicating that overlapping regions are less strongly activated by both gain and loss experiences than by neutral outcomes.

Given that gain and loss outcomes deactivate overlapping areas of the executive control network, we test whether those areas have a higher level of activity during the outcome phase when subjects subsequently chose the offer compared to when they chose the lottery in the second decision stage. All but one of the parietal clusters are indeed (marginally significantly) more strongly activated in the outcome phase when subjects chose the offer than when they chose the lottery ($p < 0.10$; left IPL: $p = 0.18$). This suggests that the HME and BEE are related to the deactivation of cognitive control regions that occurs when gains and losses are being experienced.

4.3.2. Second-stage choice phase

To examine whether the history of a gain, neutral or loss outcome leads to particular affective or executive control processing during the subsequent second-stage choice phase, we also compare the brain activations for this phase. Recall that the choice problems that subjects face are numerically equivalent across the conditions, and that the only differences are in the prior outcomes. Table 3 summarizes the statistical information on the activated clusters for this time window.

4.3.2.1. Affective network. We first compare the brain activity for gain and neutral trials, but observe no significant difference. We then compare the loss trials with the neutral trials. In line with our hypothesis of more strongly activated affective brain areas, the ACC is more activated in the loss trials than in the neutral trials during the second-stage choice phase. Interestingly, the precise area is more dorsal than the ACC cluster found in the outcome phase. The two ACC clusters only slightly overlap (seven voxels based on a region of interest analysis), indicating that the activated brain areas are mostly different between the two phases. Also, and in contrast to the findings for the outcome phase, the ACC activity is not different for second-stage lottery and offer choices ($p = 0.16$).

4.3.2.2. Executive control network. We hypothesized that gain and loss experiences may lead to decreased activity levels in the executive control network during the subsequent choice phase. As expected, the right and left IPL are less strongly activated in this phase for gain trials than for neutral trials. In contrast, we do not find any significant deactivation of brain activity when we compare the loss trials with the neutral trials. Thus, we report decreased activity in the executive control network during the second-stage choice phase only for the gain trials but not for the loss trials. Finally, we find that activity in the executive control network is not statistically different for trials that ended up with offer choices than for those that ended up with lottery choices (right IPL: $p = 0.24$; left IPL: $p = 0.19$).

The findings for the second-stage choice phase suggest that there is a higher level of both affective and executive control processing in loss trials than in gain trials during this phase. To more formally test this, we directly compare the activity of the affective and executive control regions in loss trials with that in gain trials. The results are in Table 3, and some are illustrated in Fig. 5. As expected, we find stronger activation in parts of the affective brain network for loss trials: both the insula and the ACC are more strongly activated. Additionally, the loss trials show increased activity in a cluster that consists of the VLPFC and the lateral OFC. The lateral OFC has previously been related to experiencing negative outcomes (Kringelbach, 2005), while the VLPFC is part of the executive control network. The lower involvement of the VLPFC in gain trials reinforces earlier indications that decreased deliberation occurs in the gain trials only.

5. Discussion and conclusions

In this study, we find robust evidence of path dependence when subjects repeatedly make choices between lotteries and sure amounts in a two-stage lottery game. They take more risk both after a prior gain (HME) and after a prior loss (BEE).

[6] This region-of-interest approach is less conservative than a strict conjunction analysis. The latter did not yield significant results.

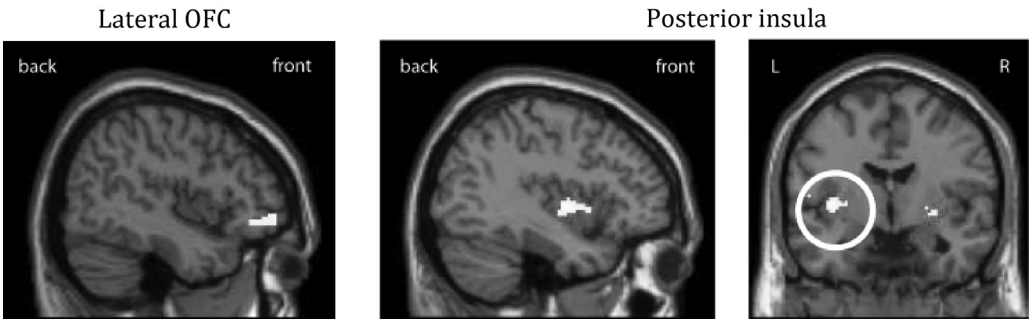

Fig. 5. Second-stage choice phase: higher brain activity for losses than for gains. The stronger activation of the orbitofrontal cortex (OFC) and the insula indicates more affective processing during the choice phase after prior losses than after prior gains.

As hypothesized, our analyses of the brain data reveal that these effects are related to increased activity of affective brain processes and decreased activity of deliberative brain processes. Moreover, brain activity in the outcome phase is associated with choice behavior in the subsequent second decision stage. The latter holds in particular for the affective network. For the deliberative network, the relations between brain activity and choice are only marginally significant. Taken together, these results indicate that the strength of path dependence in risky choice is driven by the balance between affective and deliberative systems. Below, we elaborate on our findings.

Theoretical accounts of the HME and the BEE suggest that the effects are driven by the integration of the current choice problem with prior outcomes. Most of the previous investigations into the relation between prior outcomes and subsequent choices have been conducted with experimental designs where consecutive choice problems were either not linked at all (Xue et al., 2011), or only weakly through cumulative earnings (Andrade and Iyer, 2009; Demaree et al., 2012; Gehring and Willoughby, 2002; Kuhnen and Knutson, 2005). In the present study, we encouraged integration with prior outcomes by having a structure where only two of the three prizes of a first-stage lottery were taken to the second decision stage. Our experimental setup indeed led to the path-dependence of second-stage choices, and—in line with previous neuroscience studies on reference-dependent valuation (Breiter et al., 2001; De Martino et al., 2009; Fliessbach et al., 2007)—to a path-dependent evaluation of the subsequent lotteries in the neural valuation network of striatum and VMPFC. Importantly, the choice problems were identical across the gain, neutral and loss conditions, which excludes the possibility that any variability in the choice problems other than prior outcomes could be driving the results. Our findings demonstrate the robustness of the HME and the BEE by showing that the two effects can be elicited within a reasonably short time frame in a within-subject experimental design with many repetitions.

In line with neuroeconomic findings on framing (De Martino et al., 2006; Roiser et al., 2009) and loss aversion (De Martino et al., 2010; Knutson et al., 2008b), we proposed that path dependence in risky choice is driven by increased affective processing and decreased deliberative processing from gain and loss experiences. In other words, we expected more affective processing for positive and negative prior outcomes than for neutral prior outcomes. When we compare the brain activities during the outcome phase of the task across our three experimental conditions, we indeed find increased activity in partially overlapping networks of brain areas for the gain and loss conditions compared to the neutral condition, namely in the bilateral anterior insula and in the ACC. These regions are known to be part of an emotion-related, interconnected salience network and they co-activate in virtually all types of experienced emotional feelings, regardless of the emotional valence (Craig, 2009; Seeley et al., 2007). This network has also been previously found to be activated by positive outcomes and negative near-miss (non-win) events in a simulated slot-machine task (Clark et al., 2009).

A neuroscience study by Kuhnen and Knutson (2005) suggests that positive anticipatory affect—as reflected in the ventral striatum (more precisely: the nucleus accumbens)—is followed by risk-seeking behavior, while negative anticipatory affect—reflected by insula activation—precedes a preference for certainty. Although we also find reward-specific activation in the ventral striatum, we do not find that striatum or insula activity precedes a preference for risk or certainty. A possible explanation for this difference is that Kuhnen and Knutson' task included probabilities that were unknown to their subjects, whereas our task was a pure risk task where both the magnitude and the probability of possible outcomes were always known. The ventral striatum and the insula have been associated with learning from experience, and are found to reflect reward prediction error and risk prediction error, respectively (d'Acremont et al., 2009; Hare et al., 2008; Preuschoff et al., 2008). Thus, in the case of Kuhnen and Knutson, striatal and insular activity may have predicted subsequent choice because subjects were learning from experience.

Instead of the ventral striatum and the insula, we find that the ACC has a higher level of activity when subjects subsequently chose the lottery rather than the sure amount. This suggests that the ACC operates as a mediator between affective arousal and subsequent choice, a role that has previously been suggested in emotion formation (Craig, 2002, 2009). In his work, Craig (2002, 2009) argues that the insula and the ACC constitute the "limbic sensory and motor cortices", respectively, with the insula engendering a feeling and the ACC relating to behavioral motivation and agency. The ACC has also been more broadly related to executive functions, even outside the emotional context. The superior parts of the dorsal ACC (extending

to the dorsomedial frontal cortex) in particular are related to executive functions and conflict detection (Carter et al., 1999; Carter and van Veen, 2007; Seeley et al., 2007). It is also noteworthy that the rostral part of the ACC—which is associated with subsequent choice behavior in our experiment—has been found to be activated by near-miss (non-win) events, and that this result was only observed in gambling situations where subjects had control over the task (Clark et al., 2009). The presence of agency in our task could thus be an important factor that distinguishes our work from prior studies that did not find a link between ACC activity and subsequent choice (e.g., Kuhnen and Knutson, 2005).

We also expected that gain and loss experiences would decrease the use of deliberation processes. In line with this, we find a decreased level of activity in the executive control network for gain and loss experiences. There is an overlap in the control areas that show decreased activation, indicating that similar types of control processes function at a decreased level for gains and losses. Furthermore, for multiple parts of the executive control network, we find some evidence of lower activity during the outcome phase when decision-makers subsequently chose the risky lottery option over the sure offer option. In sum, these findings indicate that both gain and loss outcomes induce decreased activity in the cognitive control network, and that a decreased use of these control mechanisms increases the propensity to take risk.

The findings discussed so far are related to the outcome phase of the task, that is, the brain activity that occurred prior to the second-stage choice phase. In contrast to the overlapping activations for gain and loss outcomes in the outcome phase, the patterns are different in the subsequent choice phase. More specifically, when we compare the gain condition to the neutral condition, we find deactivation in the executive control network but no increased activation of affective brain regions. However, when we compare the loss condition to the neutral condition, we find a higher level of activity in affective brain regions (in the ACC in particular) but no deactivation in the executive control network.[7] In contrast to the outcome phase, the activity levels of the affective or deliberative brain regions during the choice phase are not correlated with subsequent risk seeking or avoidant behavior, suggesting that the brain response to the outcome phase may already be influencing the subsequent choice in important ways.

5.1. Limitations

To properly evaluate the contribution of neuroscience to the study of economic behavior, some remarks have to be made about the limitations of fMRI. The interpretation of fMRI results is challenged by the difficulty in associating a brain region with one specific cognitive function, or the inverse inference problem (Poldrack, 2006). For instance, even though the amygdala and insula are often found to process experienced and anticipated negative emotions, it does not imply that amygdala or insula activity is specific for negative emotions per se, as both of these structures can also be active in positive events and emotions (Baxter and Murray, 2002; Craig, 2009). This limited functional specificity restricts the possibility of inferring specific mental states of decision-makers from their brain activities. In the current study, we attempt to circumvent, or at least minimize, the inverse inference problem by basing our interpretations on larger networks of brain regions that are known to be interconnected and to have at least some level of specificity to affective and executive control processes (Seeley et al., 2007).

There are also some limitations on the methodology itself. Typically, fMRI research is correlational in nature, indicating the brain regions that show fluctuations in line with experimental conditions, but of course this does not imply direct causality from the activity in the brain region to behavior. Also, fMRI provides a relative and indirect measure of brain activity rather than an absolute and direct measure, leading to the necessity for relative comparisons, and to difficulties in interpreting whether a difference between experimental conditions is caused by increased activity in one condition or decreased activity in the other.

5.2. Implications

The present work is an example of how neuroscientific tools can contribute to the process-level understanding of human decision making, in particular in the context of path dependence in risky choice. Learning more about underlying brain mechanisms and gaining better understanding of the causes of behavior helps the development of new behavioral hypotheses, and potentially reduces the amount of trial and error (Clithero et al., 2008). A good example of this is the series of studies by Kuhnen and Knutson (2005, 2011) and Knutson et al. (2008a), where the two neuroscience studies informed the later behavioral study.

Our study is also an example of how neuroscience methodology can be used in providing practical suggestions. It underlines the crucial role of emotions and reflective processes in financial decision making and risk taking behavior. Transient emotions, whether externally or internally activated, can easily induce and intensify break even and house money effects to the detriment of the decision-maker. Reducing path dependence can materially benefit the decision-maker, and insight into the underlying process can help inform both personal and policy interventions. Our brain imaging findings distinguish

[7] Interestingly, the ACC activation in the loss condition is located in more dorsal parts during the choice phase than during the outcome phase. In general, the ACC has an integrative role in behavioral control and especially the most dorsal parts of the ACC are involved in executive functions, while the ventral and rostral parts are more purely affect-related (Carter et al., 1999; Carter and van Veen, 2007; Craig, 2009; Seeley et al., 2007). The dorsal ACC activity in the loss trials might thus relate to behavioral control in the second-stage choice which is triggered by the negative emotional state.

two separate networks that promote path dependence: increased affective and decreased deliberative processing induced by gain and loss outcomes are generally related to an increased tendency to subsequently select risky prospects. Interestingly, the induced affective brain processes persist until the subsequent choice phase, in particular after loss outcomes. As for the gain outcomes, the present data suggest that decision-makers use insufficient deliberation in the subsequent decision stage. To achieve more rational choice behavior, decision-makers might benefit from neutralizing their emotional state before proceeding with the subsequent choice task (especially after losses), by, for instance, employing emotion regulation techniques such as cognitive reappraisal strategies which are known to decrease decision biases related to negative moods (Heilman et al., 2010) and loss aversion (Sokol-Hessner et al., 2009, 2013). Decision-makers could also reduce biases by intentionally increasing deliberation in their choice behavior (especially after gains), by, for instance, utilizing self-regulation strategies which increase control over behavior during emotional arousal (Leith and Baumeister, 1996).

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