NeuroImage 49 (2010) 3276–3285

Contents lists available at ScienceDirect

NeuroImage

journal homepage: www.elsevier.com/locate/ynimg

Dissociation of neural networks for anticipation and consumption of monetary and social rewards

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ARTICLE INFO

Article history:
Received 6 August 2009
Revised 28 October 2009
Accepted 30 October 2009
Available online 12 November 2009

Keywords:
Anticipation
Outcome
Social reward
Monetary reward
Nucleus accumbens
Amygdala
Thalamus

ABSTRACT

Human behaviour is generally guided by the anticipation of potential outcomes that are considered to be rewarding. Reward processing can thus be dissected into a phase of reward anticipation and a phase of reward consumption. A number of brain structures have been suggested to be involved in reward processing. However, it is unclear whether anticipation and consumption are mediated by the same or different neural networks. We examined the neural basis of these processes using functional magnetic resonance imaging (fMRI) in an incentive delay task offering either money or social approval. In both conditions participants ($N = 28$) were given a cue indicating potential reward. In order to receive reward a target button had to be pushed within a certain time window (adapted for individual reaction time). Cues triggering either monetary or social reward anticipation were presented sessionwise. Imaging was performed on a 1.5-Tesla Philips scanner in an event-related design. Anticipation of both reward types activated brain structures constituting the brain reward system including the ventral striatum. In contrast to the task independent activity in the anticipation phase, reward consumption evoked different patterns of activation for money and social approval, respectively. While social stimuli were mainly associated with amygdala activation, the thalamus was more strongly activated by the presentation of monetary rewards. Our results identify dissociable neural networks for the anticipation and consumption of reward. The findings implicate that the neural mechanisms underlying reward consumption are more modality-specific than those for reward anticipation, and that they are mediated by subjective reward value.

Introduction

Reward processing can be divided into two temporally distinct phases: an appetitive phase and a consummatory phase (Berridge, 1999). A large number of studies have indicated that several different brain structures, such as the midbrain, ventral striatum (including the nucleus accumbens (NAcc)), amygdala, orbital frontal and mesial prefrontal cortex, function as the reward system of the brain, attributing a special role to dopamine as the innervating neurotransmitter (see (Arias-Carrion and Poppel, 2007; McClure et al., 2004; O'Doherty, 2004; Schultz, 2006) for reviews). However, disentangling the neural mechanisms underlying the different processes of reward-related behaviour, in terms of both timing and reward specificity has been a challenge. In the present study anticipatory and consummatory processes were examined using two different types of reward: money and social approval. The aims were to differentiate the neural correlates of anticipatory and consummatory processes during reward-related behaviour, and to examine whether the neural basis of reward processing can be distinguished for different types of reward.

Earlier studies have shown that the ventral striatum, particularly the NAcc, plays a key role in the appetitive phase of reward processing (Kalivas and Volkow, 2005; Knutson et al., 2005). Based on conditioning processes dopaminergic nerve cells release dopamine into the NAcc when encountering cues signalling potential reward, hence alerting the organism that reward is within reach (Schultz, 1998). Moreover, animal studies have shown that dopamine release in the midbrain encodes the value of a potential outcome (Bromberg-Martin and Hikosaka, 2009; Morris et al., 2006; Wilson and Bowman, 2006). In humans, NAcc activity during reward anticipation seems to be relatively independent of incentive type and has been shown for money (Dreher et al., 2007; Ernst et al., 2004; Kirsch et al., 2003; Knutson et al., 2001a; Liu et al., 2007), pleasant taste (O'Doherty et al., 2002, 2004), verbal praise (Kirsch et al., 2003) and smiling faces (Spreckelmeyer et al., 2009). This implicates the NAcc as a universal mediator of reward prediction (Knutson and Cooper, 2005).

In contrast, the NAcc's role in reward consumption, i.e. when reward delivery is being processed, is less certain. While some studies report NAcc activation during the consumption of reward (Aharon et al., 2001; Bjork et al., 2004; Breiter et al., 2001; Delgado et al., 2000; Elliott et al., 2000; Ernst et al., 2005; Izuma et al., 2008; Martin-Soelch et al., 2003; Nieuwenhuis et al., 2005), others do not (Knutson et al., 2001b;

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doi:10.1016/j.neuroimage.2009.10.089

Knutson et al., 2003; O'Doherty et al., 2002, 2003). Some of these disparate findings can be explained by differences in research methods, experimental designs or paradigms used. Block design fMRI and PET studies do not allow for the dissociation of anticipatory and consummatory processes. Thus, the reported NAcc activity in studies using these methods (Aharon et al., 2001; Martin-Soelch et al., 2003) might well be a consequence of insufficient temporal resolution. Moreover, most reward paradigms lack a distinguishable anticipation phase because they do not make use of a reward predicting cue. It may be that presentation of the rewarding stimulus, which is usually simply a picture of a potential reward, is processed by the brain as a cue that real reward is upcoming. In that case, what has been interpreted as reflecting reward consumption would very likely be related to reward anticipation. Finally, many studies investigating reward consumption made use of gambling task paradigms (Delgado et al., 2000; Elliott et al., 2000; Ernst et al., 2005; Izuma et al., 2008; Nieuwenhuis et al., 2005). However, gains are unpredictable in these tasks because participants win or lose by pure chance. Striatal activation in such tasks is likely related to the brain's effort to establish reward predictability—a mechanism that has also been associated with dopaminergic activity in the ventral striatum (Berns et al., 2001). To sum up, only a few studies methodically allowed for a direct comparison of reward anticipation and reward consumption, the majority of which did not report NAcc activation in response to consumption if reward processing was contrasted to a neutral baseline (Dillon et al., 2008; Knutson et al., 2001b, 2003; O'Doherty et al., 2002), but see (Bjork et al., 2004; Bjork et al., 2008), who contrasted hits vs. misses.

Instead, a number of other brain structures have been suggested to play a role in reward consumption, including the orbitofrontal and mesial prefrontal cortex (Dillon et al., 2008; Knutson et al., 2003; O'Doherty et al., 2003), the thalamus (Izuma et al., 2008; Thut et al., 1997), the amygdala (Dreher et al., 2007; Hamann et al., 2004; Stoeckel et al., 2008), and the cingulate cortex (Knutson et al., 2003; Nieuwenhuis et al., 2005). However, due to a lack of studies presenting different types of reward within the same experiment, it is not yet clear whether any of these structures is active during reward consumption independent of incentive type. To address this issue, in the present study either monetary or social reward was offered within a standard incentive delay paradigm. In this paradigm an explicit anticipation phase is created by the introduction of a cue informing about the upcoming potential reward. The delay anticipation period can be distinguished from an outcome period during which rewards are delivered. In this task, anticipation of monetary reward has consistently been found to activate the ventral striatum, including the NAcc (Bjork et al., 2004; Knutson et al., 2001a, 2003; Wrase et al., 2007). It has been recently shown, by our own group, that this is also the case for anticipation of social rewards (Spreckelmeyer et al., 2009). In this study, however, we focussed on the outcome period and provide evidence for dissociable neural networks involved in reward anticipation and reward consumption.

Experimental procedures

Subjects

Thirty-two right-handed, healthy volunteers with normal vision and no past neurological or psychiatric history participated in the study. Four participants were excluded from later analyses as they did not reach the minimum hit rate of $N = 22$ hits per task, which had been set up as criterion to guarantee that a sufficient number of reward trials had been perceived by all participants[1]. Thus, data from 28 subjects (13 men: mean age = 29.5 years, range = 20–48 years; 15 women: mean age = 27.2 years, range = 20–45 years) were included. None of the subjects was taking psychoactive medication at the time of study or within the two months prior to their participation. The study was approved by the Ethics Committee of the Medical Faculty of the RWTH Aachen University. Subjects gave written informed consent and were paid a fee for participation in the study.

Stimuli and task

The experiment consisted of two different tasks: the classic "monetary incentive delay" (MID) task as introduced by Knutson et al. (2000), and an adaptation of the MID, termed "social incentive delay" (SID), which aims to examine participants' striving to gain positive social feedback (see Fig. 1) (Spreckelmeyer et al., 2009).

Each task consisted of 88 trials, yielding a total of 176 trials. In all trials potential gain depended on the participants' ability to hit a button in time when a cued target symbol (white square) appeared on the screen (see Fig. 1). Task difficulty was standardized to a hit rate of ~66% for all participants by adjusting target duration to individual mean reaction times, which were calculated prior to the experiment based on a simple reaction time task.

In the main experiment, cues preceding the target symbol either signaled potential reward (n per task = 66; denoted by circles; i.e. ⊖<⊜<⊜) or "no outcome" (n per task = 22; denoted by a triangle; i.e. △). The degree of potential reward was varied on three levels as indicated by the number of horizontal lines in a cue. The three levels of monetary reward were 0.20 Euros ($n = 22$, circle with one horizontal line), 1.00 Euro ($n = 22$, circle with two horizontal lines), and 3.00 Euros ($n = 22$, circle with three horizontal lines). Success was acknowledged by presenting the picture of a wallet which either contained the respective amount of money in coins or–in the case of "no outcome"–was empty (see Fig. 1). Participants were encouraged to respond as fast as possible to all cue types. For the three levels of social reward magnitude (similarly indicated by one, two or three horizontal lines inside the circle cues; see above), three types of happy face expressions with increasing intensity levels (happy closed mouth; happy open mouth; happy open mouth exuberant) were used (see Fig. 1). For the face stimuli, 66 color photographs displaying 3 different expressions of 22 people (11 female, 11 male) were taken from a standard database of professional actors expressing various emotions (NimStim set of Facial Expressions, available at http://www.macbrain.org; (Tottenham et al., 2009)). To generate "no outcome" stimuli, 22 portraits were graphically dysmorphed (Adobe PhotoDeluxe Home Edition 3.0, Adobe Systems Incorporated) to eliminate all facial features while keeping size and luminance stable. This procedure resulted in a pool of 22 different "no outcome"-stimuli which–like the different persons' faces–differed from each other in colour and lightness.

In both tasks each trial started with the presentation of one of the four cues for 240 ms, followed by a delay period for a variable length of time (jittered between 2250 and 2750 ms) and the target (individually adjusted presentation time; between 160 and 260 ms). Feedback informing the participant about the outcome of the latest trial was presented for 1650 ms starting 300 ms after target onset. Trial types were pseudo-randomly ordered within two MID and two SID sessions of 44 trials each with inter-trial-intervals jittered between 2500 and 5000 ms. MID and SID sessions were presented interleaved with the order of tasks counterbalanced across participants. At the beginning of each session, participants were informed which task (MID or SID) would follow next.

Prior to entering the scanner participants performed a practice session composed of 44 trials per task to familiarize themselves with the experiment.

fMRI setting

For both tasks, stimuli presentations and recording of reaction times were performed using the software Presentation (Neurobehavioral

[1] The number of 22 was chosen to guarantee a number of hit trials that was equivalent to the number of trials in the baseline condition which was also 22.

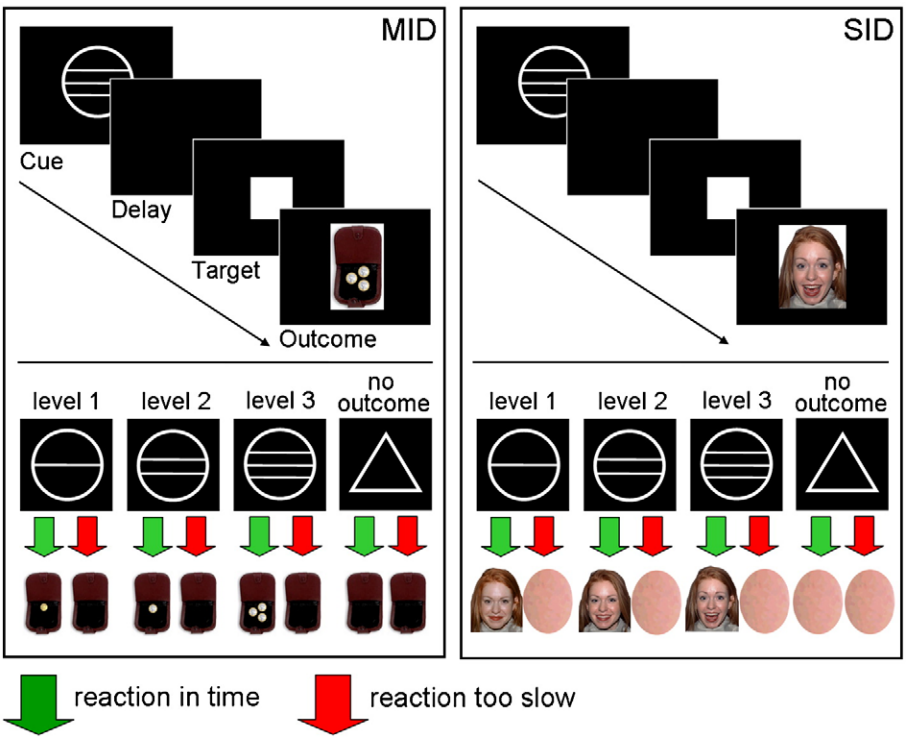

Fig. 1. Experimental paradigm for the Monetary (MID) and the Social (SID) Incentive Delay Task. Participants were asked to hit a button as fast as possible when a white target square appeared on the screen. To generate reward anticipation, target presentation was preceded by a cue signalling the reward that would be presented if the button was hit fast enough. Circle cues presented different levels of potential reward, while the triangle announced "no outcome" independent of reaction time.

Systems, Inc., San Francisco, CA). Participants indicated their response by pressing the button of a fibreoptic, custom-made, response box with the index finger of their right hand.

Image acquisition

Scanning was performed on a 1.5 T whole body scanner (Philips Medical Systems, Achieva, Best, Netherlands) using standard gradients and a standard quadrature head coil. Participants laid in a supine position, and their head movement was limited by foam padding within the head coil. In order to ensure optimal visual acuity, participants were offered fMRI-compatible glasses that could be fixed to the video goggles. For each participant, a series of 840 EPI-scans, lasting approximately 28 min, was acquired. Stimuli were presented in an event-related fashion.

Functional scans covered the region constituting the reward system of the brain (Bjork et al., 2004; Knutson et al., 2001b) and were aligned parallel to the AC/PC line (for a more detailed description see Spreckelmeyer et al., 2009). The fMRI recording, including five initial dummy scans, had the following parameters: number of slices (NS): 22; slice thickness (ST): 3.8 mm; interslice gap (IG): 0 mm; matrix size (MS): 64×64; field of view (FOV): 240 mm×240 mm; repetition time (TR): 2 s; echo time (TE): 50 ms; flip angle (FA): 90°. For anatomical localization, we acquired high resolution images with a T1-weighted 3D FFE sequence (TR = 25 ms; TE = 4.59 ms; NS = 170 (sagittal); ST = 2 mm; IG = 1 mm; FOV = 256×256 mm; voxel size = 1×1×2 mm).

Image analysis

The data were preprocessed and analyzed using the Statistical Parametric Mapping software package (SPM5) (http://www.fil.ion.ucl.ac.uk), implemented in MATLAB 7.0 (Mathworks Inc., Sherborn, MA, USA). The first five volumes from each participant were discarded from data analysis because of the non-equilibrium state of magnetization.

All remaining images were realigned to the first image to correct for head movement, coregistered with the anatomical 3D image, and spatially normalized to the standard template of the Montreal Neurological Institute (MNI, Canada) using the unified segmentation approach (Ashburner and Friston, 2005). Subsequently, the normalized data were smoothed with a resliced voxel size of 4×4×4 mm with a 6 mm FWHM isotropic Gaussian kernel to allow for statistical inference using the Gaussian Random Field theory. Finally, a high-pass filter was applied to remove baseline drifts due to cardio-respiratory and other cyclical influences. A random-effects, event-related statistical analysis (Josephs et al., 1997) was performed with SPM5 in a two-level procedure. At the first level, a separate General Linear Model (GLM) was specified for each participant. To that end, the 22 "no outcome" trials (triangle) of each session (MID and SID) were contrasted with 22 "hit outcome" trials in which the target button had been hit in time and thus reward was received. The 22 "hit outcome" trials were recruited from the highest reward level. If participants had failed to get 22 hits in the highest reward category, the pool was complemented by "hit outcome" trials from the next lower level (for details see Table 1 in the supplements). Task-related changes in Blood Oxygen Level Dependent (BOLD) signal were estimated at each voxel by modelling the onsets of the 22 "no outcome" and the 22 "hit outcome" trials as delta functions convolved with a Hemodynamic Response Function (HRF) separately for the anticipation phase (onset = presentation of the cue) and the consumption phase (onset = presentation of the feedback). Eight separate regressors were modelled, four for each task (MID, SID): (1) anticipated "hit outcomes" (anticipation phase), (2) anticipated "no outcome" trials (anticipation phase), (3) gained "hit outcomes" (consumption phase) and (4) "no outcome" trials (consumption phase). Regression coefficients for all regressors were estimated using least squares within SPM5 (Friston et al., 1995). To assess the group effect (second level), a full factorial analysis (task × outcome × phase) implemented in SPM5 was performed to test for differences in brain activations between tasks (MID, SID) and phases (anticipation, consumption).

Other than during the anticipation phase (see Spreckelmeyer et al., 2009), there were no gender specific differences in activation patterns during the consumption phase. Thus, we collapsed the data sets of men and women to increase power for further analyses.

Following the approach by Dillon et al. (2008), a series of progressively more stringent analyses was used to identify brain regions specifically involved in anticipation versus consumption and vice versa. In the first step, contrasts were computed to identify brain regions involved during anticipation of monetary and social rewards, respectively, in comparison to the "no outcome" condition (anticipation $_{\text{hit outcome}}$>anticipation $_{\text{no outcome}}$). Only the 22 trials eventually resulting in a "hit outcome" were included (see above). The same contrasts were calculated for the consumption phase to identify brain areas activated during reward presentation compared to the "no outcome" condition (consumption $_{\text{hit outcome}}$>consumption $_{\text{no outcome}}$). In the second step, the results of the contrast (anticipation $_{\text{hit outcome}}$> anticipation $_{\text{no outcome}}$) were inclusively masked with a (anticipation $_{\text{hit outcome}}$>consumption $_{\text{hit outcome}}$) contrast. This step allows identifying brain regions more active during anticipation than consumption of rewards. The same process was applied to the consumption data to identify brain regions more active during consumption than anticipation of rewards: after computing the contrast (consumption $_{\text{hit outcome}}$>consumption $_{\text{no outcome}}$), the results were inclusively masked with a (consumption $_{\text{hit outcome}}$> anticipation $_{\text{hit outcome}}$) contrast. Furthermore, the contrasts (consumption $_{\text{MID hit outcome}}$>consumption $_{\text{SID hit outcome}}$) and (consumption $_{\text{SID hit outcome}}$>consumption $_{\text{MID hit outcome}}$) were calculated to allow a direct comparison of the brain regions specifically activated during monetary and social reward consumption.

To illustrate respective temporal activation patterns in brain areas involved during the anticipation and the consumption phase, BOLD contrast time courses were extracted from contrast-specific regions of interest (ROI). The activation foci (peak values) of each individual's ROI were used to construct spherical volumes of interests (VOIs) with 4 mm diameters. Using a Finite Impulse Response (FIR) model, BOLD contrast time courses were extracted from these VOIs for each trial type and for each individual calculated as percent signal change from overall intensity mean. Based on the result of the "anticipation $_{\text{hit outcome}}$> anticipation $_{\text{no outcome}}$" contrast a ROI encompassing the ventral striatum was chosen to demonstrate the time course of the anticipation specific BOLD response. Based on the "consumption $_{\text{hit outcome}}$> consumption $_{\text{no outcome}}$" contrasts, two ROI (amygdala and thalamus) were specified to illustrate individual BOLD contrast time courses during the outcome phase of the SID and the MID task.

To specifically test for signal changes correlated with increasing values of gained reward, a second random-effects, event-related statistical analysis was performed with SPM5 in a two-level procedure. Again a separate General Linear Model (GLM) was specified for each participant at the first level. However, in contrast to the analysis described above, not only the 22 "no outcome" and 22 "hit outcome" trials but all 176 trials were included (2 tasks × 4 reward levels × 22 trials). Task-related changes in Blood Oxygen Level Dependent (BOLD) signal were estimated at each voxel by modelling the onsets of the feedback phase of the trials (onset = presentation of the feedback) as delta functions convolved with a Hemodynamic Response Function (HRF). Both tasks (MID, SID) with their five potential feedback categories (no potential outcome, low reward, medium reward, high reward, missed reward) were modelled as separate regressors. For group inferences (second level) a full factorial analysis (task × reward magnitude), implemented in SPM5, was performed to test for the potential increase of activation with levels of gained reward. Parameter estimates were extracted from three regions of interest (ROI) defined *post-hoc* based on the results of the masked "consumption $_{\text{hit outcome}}$>consumption $_{\text{no outcome}}$" contrast reported above. Following this approach, spheres of 4 mm radius were created at the coordinates $x = 0$, $y = -8$, $z = 9$ for the thalamus, $x = -18$, $y = -4$, $z = -12$ for the left amygdala, and $x = 18$, $y = -6$, $z = -12$ for the right amygdala. The averaged parameter estimates of the amygdalae and the parameter estimates of the thalamus were entered into two separate analyses of variance (ANOVA) on repeated measures with within factors "task" (monetary, social) and "incentive magnitude" (no potential outcome, low, medium, high). Effect sizes were calculated using partial eta square (η^2_p). Note that the results of this analysis can only be regarded exploratory, given its *post-hoc*-nature and the heterogeneous number of hit trials in each participant and each incentive level category.

The reported voxel coordinates of activation peaks were transformed from MNI space to Talairach & Tournoux atlas space (Talairach and Tournoux, 1988) by non-linear transformations (www.mrc-cbu.cam.ac.uk).

Results

Behavioral results

An ANOVA on repeated measures with within factors "task" (monetary, social) and "incentive magnitude" (no outcome, low, medium, high) was performed on reaction times. The results are depicted in Fig. 2. A significant main effect of task ($F(1,27) = 19.22$, $p < 0.001$, $\eta^2_p = 0.42$) was found with faster mean reaction times in the MID than the SID task (219 ms (SE = 5.98) vs. 232 ms (SE = 5.38)). In addition, reaction times linearly decreased with increasing levels of potential reward in both tasks (main effect of incentive magnitude; $F(3,25) = 6.29$, $p = 0.002$, $\eta^2_p = 0.43$). *Post-hoc* comparisons (Bonferroni-corrected) revealed significantly faster reaction times for all levels of reward compared to the "no outcome" condition as well as faster reaction times to the highest level of potential reward compared to the lowest level of potential reward (all $p < 0.05$). The factors "task" and "incentive magnitude" did not interact ($p > 0.05$).

Neuroimaging results

Reward anticipation

For reward anticipation, the standard contrast (anticipation$_{\text{hit outcome}}$> anticipation$_{\text{no outcome}}$) revealed significant bilateral activation of the putamen, caudatus and NAcc. This finding was evident for both MID and SID (see Table 2 in the supplements). See also Spreckelmeyer et al. (2009) for a more detailed analysis of the anticipation phase.

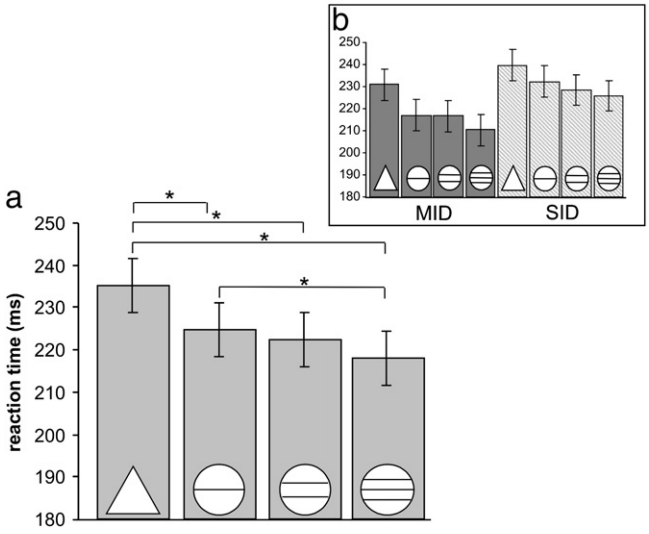

Fig. 2. Mean reaction times to the reward signalling cues in the monetary (MID) and the social incentive delay task (SID). Circles with one, two, and three bars announce increasing levels of potential reward, triangles represent "no outcome." Error bars indicate the standard error of the mean (s.e.m.). *$p < 0.05$ (Bonferroni corrected).

The ventral striatum activation remained significant for both tasks when the (anticipation $_{\text{hit outcome}}$ > consumption $_{\text{hit outcome}}$) mask was applied (see Table 1, Fig. 3), indicating that this region was more activated during anticipation of rewards than during reward consumption.

Reward consumption

The standard contrast (consumption $_{\text{hit outcome}}$ > consumption$_{\text{no outcome}}$) yielded task specific activation patterns. Monetary reward consumption strongly activated the thalamus, while social reward consumption was associated with pronounced bilateral amygdalae activation (see Table 3 in the supplements). These regions were also detected in a direct comparison of monetary and social reward consumption: the contrast (consumption $_{\text{MID hit outcome}}$ > consumption $_{\text{SID hit outcome}}$) yielded thalamus activation, while amygdala activity was found for the reversed contrast (consumption $_{\text{SID hit outcome}}$ > consumption $_{\text{MID hit outcome}}$) (see Table 4 in the supplements).

Masking the results of the standard contrasts with the respective (consumption $_{\text{hit outcome}}$ > anticipation $_{\text{hit outcome}}$) contrast also revealed thalamus activation for the MID and amygdalae recruitment for the SID task (see Figs. 4 and 5, Table 2), indicating that these regions were more activated during reward consumption than during the anticipation phase.

The ANOVA performed on the parameter estimates extracted at the coordinates $x=0$, $y=-8$, $z=9$ for the thalamus revealed a main effect of task ($F(1,27)=6.40$, $p=0.018$, $\eta_p^2=0.19$) with higher parameter estimates in the MID (mean: 2.00, SE: 0.30) than in the SID (mean: 1.47, SE: 0.23) trials. Furthermore, a significant main effect of incentive magnitude ($F(3,81)=19.56$, $p<0.000$, $\eta_p^2=0.42$) was found, reflecting a linear increase of activations with higher levels of reward independent of incentive type. In addition, task and incentive magnitude significantly interacted ($F(3,81)=3.63$, $p=0.016$, $\eta_p^2=0.12$), reflecting a linear increase of thalamic activation with increasing levels of monetary but not social reward (Fig. 6, boxplots; see supplementary data (Tables 5, 6 and 8) for complete results of the ANOVA and *post-hoc* comparisons).

The ANOVA performed on the averaged parameter estimates extracted at the coordinates $x=-18$, $y=-4$, $z=-12$ for the left amygdala and $x=18$, $y=-6$, $z=-12$ for the right amygdala revealed a significant main effect of incentive magnitude ($F(3,81)=26.04$, $p<0.000$, $\eta_p^2=0.49$), reflecting stronger activation in response to reward compared to "no outcome," independent of incentive type. Moreover, a significant interaction of task and incentive magnitude was found ($F(3,81)=3.93$, $p=0.011$, $\eta_p^2=0.13$). This was explained by the fact, that activation was lower to the lowest level of monetary reward than to the lowest level of social reward (Fig. 6; boxplots; see supplementary data (Tables 5, 7 and 9) for complete results of the ANOVA and *post-hoc* comparison).

Discussion

The aim of the present study was to disentangle the neural networks mediating anticipatory versus consummatory aspects of reward processing. To test for reward specificity of the respective networks, we offered two different types of incentives, money and social approval. Anticipation of both reward types activated brain structures constituting the brain reward system including the ventral striatum. This result is consistent with prior studies examining the anticipatory phase of reward processing (Ernst et al., 2004; Kirsch et al., 2003; Knutson et al., 2000) and confirms the role of the NAcc as the universal mediator of reward prediction.

In contrast to the task independent activity in the anticipation phase, reward consumption evoked different patterns of activation for money and social approval, respectively. While social stimuli were associated with amygdala activation, the thalamus was strongly activated by the presentation of monetary rewards. However, ventral striatal structures were not found activated during the consumption

Table 1
Brain regions activated for anticipated reward in comparison to the "no outcome" condition inclusively masked with the (anticipation $_{\text{hit outcome}}$ > consumption $_{\text{hit outcome}}$) contrast (uncorrected, $p<0.001$).

		BA	Talairach coordinates			Peak *z*-score	Cluster size
			x	*y*	*z*		
MID ($N=28$)							
R	Caudate, NAcc		12	12	−1	6.24	218
L	Putamen, NAcc		−18	12	−1	5.36	104
L	Precuneus	7	−12	−65	34	5.12	160
R	Precuneus	19	30	−68	31	4.97	118
	Culmen, substantia nigra		0	−29	−1	4.97	191
L	Inferior frontal gyrus	9	−59	10	24	4.47	80
R	Inferior frontal gyrus	9	53	5	33	4.28	14
L	Inferior frontal gyrus	46	−33	30	12	4.24	65
L	Cingulate gyrus	32	−6	25	29	4.15	33
R	Precentral gyrus	44	59	6	8	4.07	9
R	Inferior parietal lobule	40	39	−36	43	4.05	10
L	Superior frontal gyrus	9	−36	36	26	4.04	26
R	Middle frontal gyrus	10	33	39	17	3.95	7
L	Precentral gyrus	6	−50	2	30	3.92	14
L	Postcentral gyrus	2	−36	−24	40	3.86	7
L	Inferior parietal lobule	40	−50	−34	24	3.81	12
L	Insula	13	−50	−19	20	3.77	17
R	Caudate		24	−37	21	3.60	5
L	Insula		−45	9	0	3.53	5
SID ($N=28$)							
R	Inferior frontal gyrus	47	33	32	1	4.40	6
L	Cerebellum, culmen		0	−50	−13	4.11	27
R	Putamen		21	9	11	4.03	17
R	Insula		30	−34	21	3.96	9
L	Putamen, NAcc		−18	9	−3	3.77	9
R	Precuneus	7	18	−62	34	3.67	5
R	Anterior cingulate	32	12	25	26	3.32	6

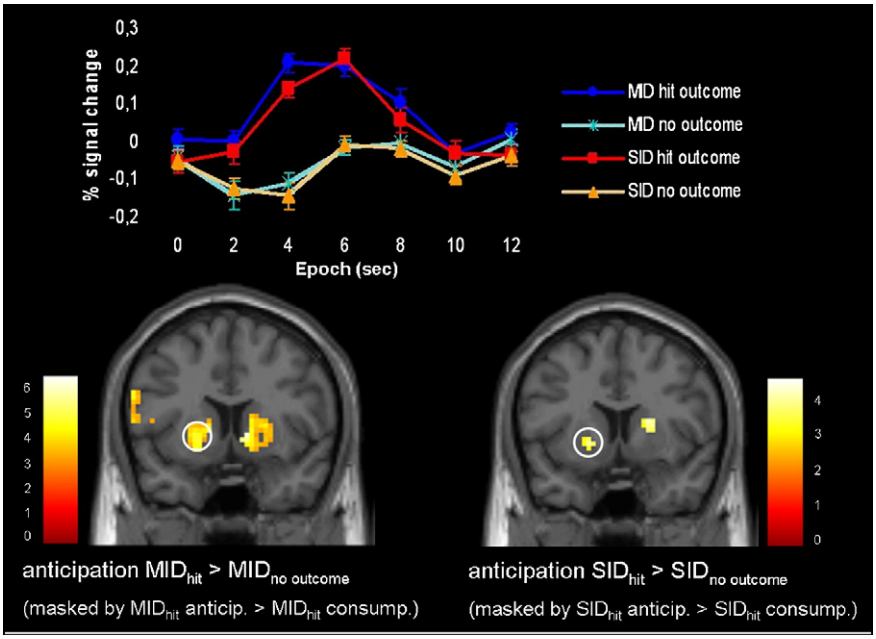

Fig. 3. Activation foci during MID (left) and SID (right) "hit outcome" anticipation compared to "no outcome" anticipation masked by the respective contrast ("hit outcome" anticipation > "hit outcome" consumption) ($p<0.001$, uncorrected; $y=9$). A conjunction analysis between the two tasks (MID, SID) was performed and the resulting peak activation appearing in the left ventral striatum (NAcc) was used to define a spherical VOI of 4 mm radius. Using a Finite Impulse Response (FIR) model, BOLD contrast time courses were extracted from this VOI calculated as percent signal change from overall intensity (starting point (Epoch = 0) of the diagram is the onset of cue presentation).

phase. This is in line with findings linking dopaminergic processes in the midbrain to reward prediction (Schultz, 1998). It is known from single cell studies that dopaminergic cells in the ventral tegmental area, which project into the NAcc, respond to reward if it occurs unpredicted (Schultz et al., 1993). However, as soon as the reward becomes predictable by a conditioned stimulus (CS), the dopaminergic cells respond more vigorously to the CS than to the reward itself (Schultz et al., 1997). If one assumes that the circle cues in our study function as CS for upcoming reward, it seems plausible that the striatal activation during the anticipation phase results from such a shift in dopaminergic cell activity.

The finding of pronounced thalamic activation for the monetary task adds to the results of prior imaging studies, which show recruitment of this structure during the processing of monetary reward (Bjork et al., 2004; Izuma et al., 2008; Martin-Soelch et al., 2003; Thut et al., 1997). It is known from animal studies that the thalamus receives projections from the nucleus accumbens, via the ventral pallidum (Mogenson et al., 1983; O'Donnell et al., 1997; Young et al., 1984), and in turn projects to prefrontal areas (Baev et al., 2002; Block et al., 2007). By linking reward signals with higher cognition processes, and preparing behavioural output, this basal ganglia-thalamo-cortical system may play a crucial role in reward-related processing (Elliott et al., 2000; McAlonan et al., 1993). Specifically, single-cell recordings in rats have demonstrated that neurons in the posterior thalamus are sensitive to the rewarding value of expected rewards (Komura et al., 2001). In an incentive delay paradigm very much like the one used in our study, thalamic neurons increasingly fired during the delay period of the experiment, peaking right before reward was delivered. Moreover, responses increased more vigorously with increasing reward value (water<mild sucrose solution<strong sucrose solution<low intracranial stimulation<high intracranial stimulation). It is thus speculated that the thalamic activation observed in the current experiment may reflect such a reward value prediction process. The linear increase of thalamus activation with increasing levels of monetary reward supports this notion. The fact, that facial stimuli also evoked thalamus activation but to a lower extent (only if the correction level was relaxed) might indicate that social feedback is perceived as less valuable than money. Shorter reaction times in response to cues predicting money compared to social reward corroborated the findings (see also (Kohls et al., 2009).

In our study, contrasting monetary "hit outcomes" with a neutral "no outcome"-baseline did not yield any activation in prefrontal

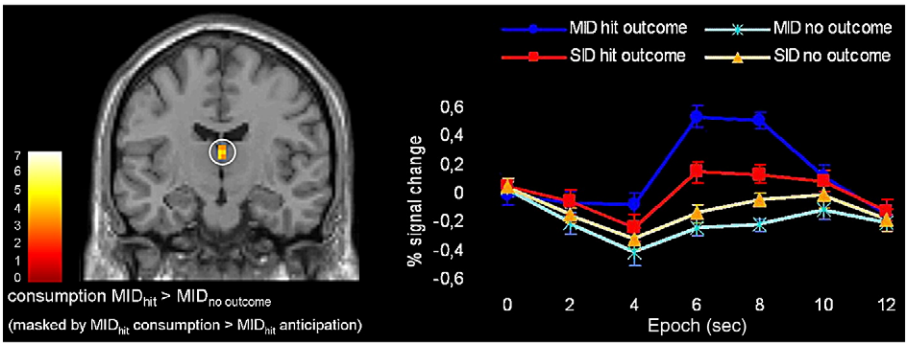

Fig. 4. Activation focus during MID consumption compared to "no outcome" masked by the contrast (MID "hit outcome" consumption > MID "hit outcome" anticipation) ($p<0.001$, uncorrected; $y=-12$). Using the peak activation appearing in the thalamus, a spherical volume of interest (VOI) of 4 mm diameter was constructed to extract time courses calculated as percent signal change from overall intensity. To allow for comparison with the NAcc time course the diagram starts (Epoch = 0) with the onset of cue presentation.

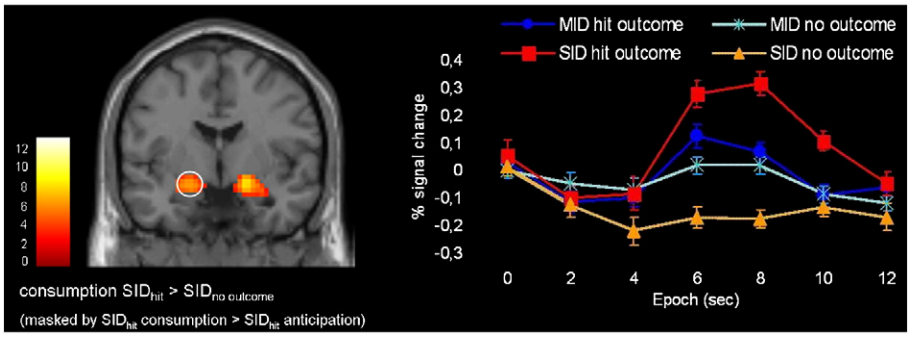

Fig. 5. Activation foci during SID consumption compared to "no outcome" masked by the contrast (SID "hit outcome" consumption>SID "hit outcome" anticipation) ($p<0.001$, uncorrected; $y=-3$). Using the peak activation appearing in the left amygdala, a spherical volume of interest (VOI) of 4 mm diameter was constructed to extract time courses calculated as percent signal change from overall intensity. To allow for comparison with the NAcc time course the diagram starts (Epoch = 0) with the onset of cue presentation.

cortex areas. In contrast, Knutson et al. (2001b, 2003) who included miss trials in their analysis, reported ventral medial frontal cortex deactivation in response to hit plus miss trials versus "no outcome" trials (Knutson et al., 2001b), and activation of regions of the mesial prefrontal cortex when directly contrasting hits vs. misses (Knutson et al., 2003). The findings implicate that prefrontal activation is specifically related to monitoring of unwanted outcomes (see also Dillon et al., 2008). Indeed, computing the respective contrast (consumption $_{\text{hit outcome}}$>consumption $_{\text{missed hit outcome}}$) in our data, also yielded activation of a region of the mesial prefrontal cortex close to the one reported by Knutson et al. (2003) (see supplements). Surprisingly, contrasting hits vs. the "no outcome"-baseline in the SID task also yielded prefrontal activation. Following the assumption that social feedback was considered less valuable than money, it can be speculated that winning a smile instead of money yielded feelings of disappointment or anger. More specific research is needed to elucidate the role of the prefrontal cortex in outcome processing.

The amygdala is considered a central component of the neural network commonly referred to as the reward system of the brain. Previous functional imaging studies have demonstrated amygdala response to reward consumption processes for sexually arousing visual stimuli (Hamann et al., 2004), food images (Beaver et al., 2006; Stoeckel et al., 2008), monetary reward (Bjork et al., 2004; Breiter et al., 2001; Dreher et al., 2007; Elliott et al., 2003; Ernst et al., 2005), and positive verbal stimuli (Hamann et al., 2004). The present study confirmed these results, but showed differences in the activation level for distinct incentive types. While the amygdala was strongly activated in response to socially rewarding pictures, the activation evoked by monetary stimuli was much weaker (again, only present if the correction level was lowered). An explanation for these disparate activation intensities might be that the amygdala is also associated with the processing of facial expressions (Adolphs et al., 2002, 1994; Tovee, 1995; Young et al., 1996). Traditionally, amygdala recruitment was believed to be restricted to the processing of fearful expressions (Adolphs et al., 1995; Calder, 1996; Morris et al., 1996), but there is increasing evidence supporting an involvement in facial emotion processing in general (Breiter et al., 1996; Canli et al., 2002; Yang et al., 2002). Thus, the present finding of pronounced amygdala activation during the consumption of social reward compared to monetary reward might be attributable to its sensitivity to facial cues.

Table 2
Brain regions activation during reward consumption in comparison to the "no outcome" condition inclusively masked with the (consumption $_{\text{hit outcome}}$>anticipation $_{\text{hit outcome}}$) contrast (uncorrected, $p<0.001$).

		BA	Talairach coordinates			Peak *z*-score	Cluster size
			x	*y*	*z*		
MID ($N=28$)							
L/R	Middle occipital gyrus	18	30	−85	−3	6.81	1010
L	Parahippocampal gyrus	27	−24	−27	−6	5.56	41
L/R	Thalamus[a]		3	−35	5	5.31	187
R	Parahippocampal gyrus		24	−24	−9	3.91	24
L	Hippocampus		−33	−12	−15	3.84	7
	Anterior cingulate	24	0	38	9	3.81	8
SID ($N=28$)							
L/R	Middle occipital gyrus, fusiform gyrus	18, 37	−27	−87	−1	Inf[b]	1678
R	Amygdala		18	−6	−12	Inf[b]	299
R	Medial frontal gyrus	11	3	58	−13	6.42	50
L	Amygdala		−18	−4	−12	6.13	215
R	Superior temporal gyrus	38	39	19	−24	5.28	20
L	Middle temporal gyrus	39	−48	−63	25	4.85	59
L	Inferior frontal gyrus	47	−39	31	−12	4.74	16
R	Inferior frontal gyrus	45/46	45	30	10	4.43	29
L	Medial frontal gyrus	10	−6	59	19	4.33	21
L	Superior temporal gyrus	38	−36	5	−20	4.30	9
R	Cingulate gyrus, posterior cingulate	31	3	−51	25	4.25	102
L	Middle temporal gyrus	21	−56	−6	−12	3.89	12
R	Middle frontal gyrus	11	36	34	−12	3.86	8
L	Anterior cingulate	24	0	38	6	3.84	7
R	Superior temporal gyrus	22	53	−9	−10	3.77	5

[a] Please note that the listed peak activation coordinates do not belong to the thalamus. However, the cluster does include two thalamic peak activations (coordinates 0, −8, 9 (*z*-score = 4.9) and 15, −34, 10 (*z*-score = 4.23).
[b] Inf = *z* is infinite (*z* score>8).

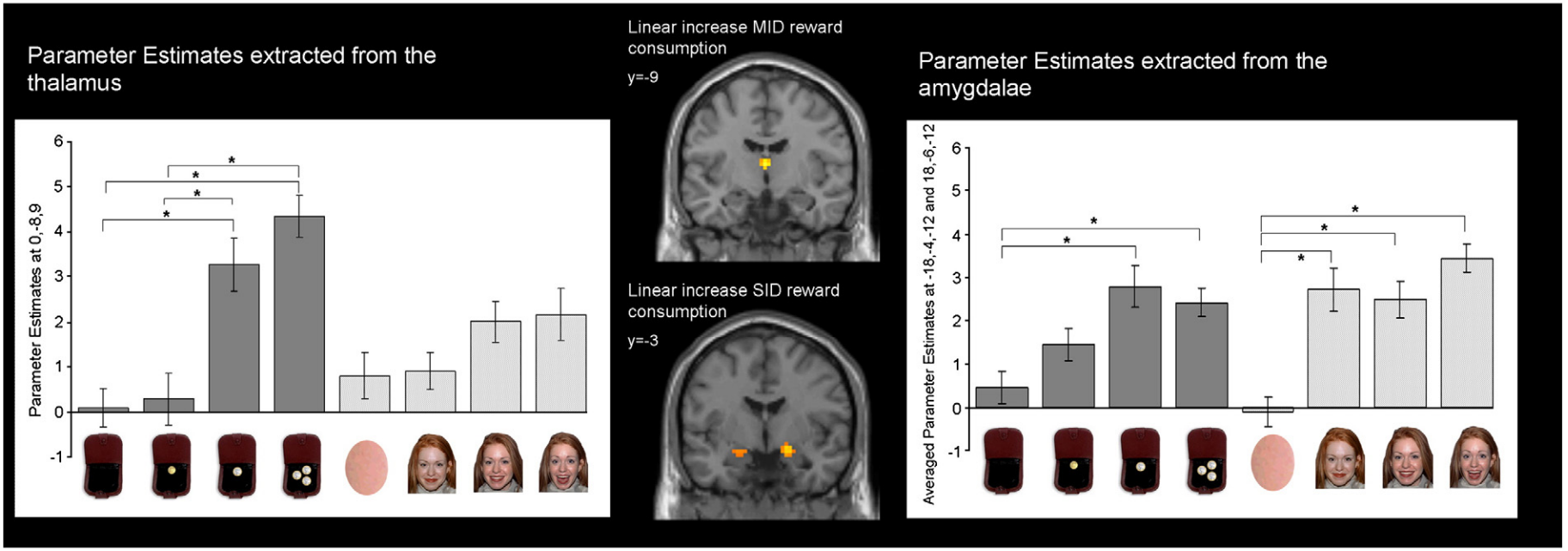

Fig. 6. Activation foci during MID and SID linear reward consumption ($p<0.001$, uncorrected). Bars represent parameter estimates for the MID (dark gray) and SID task (light gray) in regions of interest for the thalamus (left boxplots) and the amygdalae (right boxplots) which were defined based on the results of the masked "consumption $_{\text{hit outcome}}$>consumption $_{\text{no outcome}}$ " contrast. Error bars indicate the standard error of the mean. *$p<0.05$ (Bonferroni corrected for 28 comparisons). Please note, that for reasons of clarity, only significant differences between conditions within one task were reported in the figure. Please refer to the supplements for between-task effects.

Together, the data indicate that consumption of either reward type triggered neural processes in the thalamus and the amygdala. This suggests both structures play an important role in reward consumption. Furthermore, the activation levels demonstrate a specific interaction between structure and incentive type. While the thalamus was more strongly activated by monetary rewards, the amygdala was more sensitive to social rewards. We suggest that the opposing patterns of activation can be attributed to different underlying mechanisms. Stronger thalamus activation to money than to friendly faces is likely reflecting the thalamus' sensitivity to subjective reward value (Komura et al., 2001). The data obtained here attributes to the thalamus an important function in goal-directed behaviour through ranking of incentive value. In this case, the monetary reward can be understood to have a greater value to the participants than a smiling face. With regard to the amygdala, we speculate that increased activation in the social condition reflects facial processing rather than reward related function. However, more specific experiments are needed to consolidate these thoughts.

To conclude, our findings demonstrate that brain responses during anticipation and consumption of rewards are dissociable. The data further indicate that in the time course of (cued) reward processing, the underlying neural mechanisms become more and more reward specific as a consequence of reward value and/or reward characteristics. This specialization is likely to serve as preparation of reward-specific consume-behaviour.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.neuroimage.2009.10.089.

References

Adolphs, R., Baron-Cohen, S., Tranel, D., 2002. Impaired recognition of social emotions following amygdala damage. J. Cogn. Neurosci. 14, 1264–1274.

Adolphs, R., Tranel, D., Damasio, H., Damasio, A., 1994. Impaired recognition of emotion in facial expressions following bilateral damage to the human amygdala. Nature 372, 669–672.

Adolphs, R., Tranel, D., Damasio, H., Damasio, A.R., 1995. Fear and the human amygdala. J. Neurosci. 15, 5879–5891.

Aharon, I., Etcoff, N., Ariely, D., Chabris, C.F., O'Connor, E., Breiter, H.C., 2001. Beautiful faces have variable reward value: fMRI and behavioral evidence. Neuron 32, 537–551.

Arias-Carrion, O., Poppel, E., 2007. Dopamine, learning, and reward-seeking behavior. Acta Neurobiol. Exp. (Wars). 67, 481–488.

Ashburner, J., Friston, K.J., 2005. Unified segmentation. Neuroimage 26, 839–851.

Baev, K.V., Greene, K.A., Marciano, F.F., Samanta, J.E., Shetter, A.G., Smith, K.A., Stacy, M.A., Spetzler, R.F., 2002. Physiology and pathophysiology of cortico-basal ganglia-thalamocortical loops: theoretical and practical aspects. Prog. Neuro-psychopharmacol. Biol. Psychiatry 26, 771–804.

Beaver, J.D., Lawrence, A.D., van Ditzhuijzen, J., Davis, M.H., Woods, A., Calder, A.J., 2006. Individual differences in reward drive predict neural responses to images of food. J. Neurosci. 26, 5160–5166.

Berns, G.S., McClure, S.M., Pagnoni, G., Montague, P.R., 2001. Predictability modulates human brain response to reward. J. Neurosci. 21, 2793–2798.

Berridge, K.C., 1999. Pleasure, pain, desire, and dread: hidden core processes of emotion. In: Kahneman, D., Diener, E., Schwarz, N. (Eds.), Well-being: The foundations of hedonic psychology. Russell Sage Foundation, New York, pp. 525–557.

Bjork, J.M., Knutson, B., Fong, G.W., Caggiano, D.M., Bennett, S.M., Hommer, D.W., 2004. Incentive-elicited brain activation in adolescents: similarities and differences from young adults. J. Neurosci. 24, 1793–1802.

Bjork, J.M., Smith, A.R., Hommer, D.W., 2008. Striatal sensitivity to reward deliveries and omissions in substance dependent patients. Neuroimage 42, 1609–1621.

Block, A.E., Dhanji, H., Thompson-Tardif, S.F., Floresco, S.B., 2007. Thalamic-prefrontal cortical–ventral striatal circuitry mediates dissociable components of strategy set shifting. Cereb. Cortex 17, 1625–1636.

Breiter, H.C., Etcoff, N.L., Whalen, P.J., Kennedy, W.A., Rauch, S.L., Buckner, R.L., Strauss, M.M., Hyman, S.E., Rosen, B.R., 1996. Response and habituation of the human amygdala during visual processing of facial expression. Neuron 17, 875–887.

Breiter, H.C., Aharon, I., Kahneman, D., Dale, A., Shizgal, P., 2001. Functional imaging of neural responses to expectancy and experience of monetary gains and losses. Neuron 30, 619–639.

Bromberg-Martin, E.S., Hikosaka, O., 2009. Midbrain dopamine neurons signal preference for advance information about upcoming rewards. Neuron 63, 119–126.

Calder, A.J., 1996. Facial emotion recognition after bilateral amygdala damage: differentially severe impairment of fear cognitive neurospsychology 13, 699-745.

Canli, T., Sivers, H., Whitfield, S.L., Gotlib, I.H., Gabrieli, J.D., 2002. Amygdala response to happy faces as a function of extraversion. Science 296, 2191.

Delgado, M.R., Nystrom, L.E., Fissell, C., Noll, D.C., Fiez, J.A., 2000. Tracking the hemodynamic responses to reward and punishment in the striatum. J. Neurophysiol. 84, 3072–3077.

Dillon, D.G., Holmes, A.J., Jahn, A.L., Bogdan, R., Wald, L.L., Pizzagalli, D.A., 2008. Dissociation of neural regions associated with anticipatory versus consummatory phases of incentive processing. Psychophysiology 45, 36–49.

Dreher, J.C., Schmidt, P.J., Kohn, P., Furman, D., Rubinow, D., Berman, K.F., 2007. Menstrual cycle phase modulates reward-related neural function in women. Proc. Natl. Acad. Sci U. S. A. 104, 2465–2470.

Elliott, R., Friston, K.J., Dolan, R.J., 2000. Dissociable neural responses in human reward systems. J. Neurosci. 20, 6159–6165.

Elliott, R., Newman, J.L., Longe, O.A., Deakin, J.F., 2003. Differential response patterns in the striatum and orbitofrontal cortex to financial reward in humans: a parametric functional magnetic resonance imaging study. J. Neurosci. 23, 303–307.

Ernst, M., Nelson, E.E., McClure, E.B., Monk, C.S., Munson, S., Eshel, N., Zarahn, E., Leibenluft, E., Zametkin, A., Towbin, K., Blair, J., Charney, D., Pine, D.S., 2004. Choice selection and reward anticipation: an fMRI study. Neuropsychologia 42, 1585–1597.

Ernst, M., Nelson, E.E., Jazbec, S., McClure, E.B., Monk, C.S., Leibenluft, E., Blair, J., Pine, D.S., 2005. Amygdala and nucleus accumbens in responses to receipt and omission of gains in adults and adolescents. Neuroimage 25, 1279–1291.

Friston, K.J., Holmes, A.P., Worsley, K.J., Poline, J.-P., Frith, C.D., Frackowiak, R.S.J., 1995. Statistical parametric maps in functional imaging: a general linear approach. Hum. Brain Mapp. 2, 189–210.

Hamann, S., Herman, R.A., Nolan, C.L., Wallen, K., 2004. Men and women differ in amygdala response to visual sexual stimuli. Nat. Neurosci. 7, 411–416.

Izuma, K., Saito, D.N., Sadato, N., 2008. Processing of social and monetary rewards in the human striatum. Neuron 58, 284–294.

Josephs, O., Turner, R., Friston, K.J., 1997. Event-related fMRI. Hum. Brain Mapp. 5, 243–248.

Kalivas, P.W., Volkow, N.D., 2005. The neural basis of addiction: a pathology of motivation and choice. Am. J. Psychiatry 162, 1403–1413.

Kirsch, P., Schienle, A., Stark, R., Sammer, G., Blecker, C., Walter, B., Ott, U., Burkart, J., Vaitl, D., 2003. Anticipation of reward in a nonaversive differential conditioning paradigm and the brain reward system: an event-related fMRI study. Neuroimage 20, 1086–1095.

Knutson, B., Adams, C.M., Fong, G.W., Hommer, D., 2001a. Anticipation of increasing monetary reward selectively recruits nucleus accumbens. J. Neurosci. 21, RC159.

Knutson, B., Fong, G.W., Adams, C.M., Varner, J.L., Hommer, D., 2001b. Dissociation of reward anticipation and outcome with event-related fMRI. Neuroreport 12, 3683–3687.

Knutson, B., Fong, G.W., Bennett, S.M., Adams, C.M., Hommer, D., 2003. A region of mesial prefrontal cortex tracks monetarily rewarding outcomes: characterization with rapid event-related fMRI. Neuroimage 18, 263–272.

Knutson, B., Cooper, J.C., 2005. Functional magnetic resonance imaging of reward prediction. Curr. Opin. Neurol. 18, 411–417.

Knutson, B., Westdorp, A., Kaiser, E., Hommer, D., 2000. FMRI visualization of brain activity during a monetary incentive delay task. Neuroimage 12, 20–27.

Knutson, B., Taylor, J., Kaufman, M., Peterson, R., Glover, G., 2005. Distributed neural representation of expected value. J. Neurosci. 25, 4806–4812.

Kohls, G., Peltzer, J., Herpertz-Dahlmann, B., Konrad, K., 2009. Differential effects of social and non-social reward on response inhibition in children and adolescents. Dev. Sci. 12, 614–625.

Komura, Y., Tamura, R., Uwano, T., Nishijo, H., Kaga, K., Ono, T., 2001. Retrospective and prospective coding for predicted reward in the sensory thalamus. Nature 412, 546–549.

Liu, X., Powell, D.K., Wang, H., Gold, B.T., Corbly, C.R., Joseph, J.E., 2007. Functional dissociation in frontal and striatal areas for processing of positive and negative reward information. J. Neurosci. 27, 4587–4597.

Martin-Soelch, C., Missimer, J., Leenders, K.L., Schultz, W., 2003. Neural activity related to the processing of increasing monetary reward in smokers and nonsmokers. Eur. J. Neurosci. 18, 680–688.

McAlonan, G.M., Robbins, T.W., Everitt, B.J., 1993. Effects of medial dorsal thalamic and ventral pallidal lesions on the acquisition of a conditioned place preference: further evidence for the involvement of the ventral striatopallidal system in reward-related processes. Neuroscience 52, 605–620.

McClure, S.M., York, M.K., Montague, P.R., 2004. The neural substrates of reward processing in humans: the modern role of FMRI. Neuroscientist 10, 260–268.

Mogenson, G.J., Swanson, L.W., Wu, M., 1983. Neural projections from nucleus accumbens to globus pallidus, substantia innominata, and lateral preoptic-lateral hypothalamic area: an anatomical and electrophysiological investigation in the rat. J. Neurosci. 3, 189–202.

Morris, J.S., Frith, C.D., Perrett, D.I., Rowland, D., Young, A.W., Calder, A.J., Dolan, R.J., 1996. A differential neural response in the human amygdala to fearful and happy facial expressions. Nature 383, 812–815.

Morris, G., Nevet, A., Arkadir, D., Vaadia, E., Bergman, H., 2006. Midbrain dopamine neurons encode decisions for future action. Nat. Neurosci. 9, 1057–1063.

Nieuwenhuis, S., Slagter, H.A., von Geusau, N.J., Heslenfeld, D.J., Holroyd, C.B., 2005. Knowing good from bad: differential activation of human cortical areas by positive and negative outcomes. Eur. J. Neurosci. 21, 3161–3168.

O'Doherty, J.P., 2004. Reward representations and reward-related learning in the human brain: insights from neuroimaging. Curr. Opin. Neurobiol. 14, 769–776.
O'Donnell, P., Lavin, A., Enquist, L.W., Grace, A.A., Card, J.P., 1997. Interconnected parallel circuits between rat nucleus accumbens and thalamus revealed by retrograde transynaptic transport of pseudorabies virus. J. Neurosci. 17, 2143–2167.
O'Doherty, J.P., Deichmann, R., Critchley, H.D., Dolan, R.J., 2002. Neural responses during anticipation of a primary taste reward. Neuron 33, 815–826.
O'Doherty, J., Winston, J., Critchley, H., Perrett, D., Burt, D.M., Dolan, R.J., 2003. Beauty in a smile: the role of medial orbitofrontal cortex in facial attractiveness. Neuropsychologia 41, 147–155.
O'Doherty, J., Dayan, P., Schultz, J., Deichmann, R., Friston, K., Dolan, R.J., 2004. Dissociable roles of ventral and dorsal striatum in instrumental conditioning. Science 304, 452–454.
Schultz, W., 1998. Predictive reward signal of dopamine neurons. J. Neurophysiol. 80, 1–27.
Schultz, W., 2006. Behavioral theories and the neurophysiology of reward. Annu. Rev. Psychol. 57, 87–115.
Schultz, W., Dayan, P., Montague, P.R., 1997. A neural substrate of prediction and reward. Science 275, 1593–1599.
Schultz, W., Apicella, P., Ljungberg, T., 1993. Responses of monkey dopamine neurons to reward and conditioned stimuli during successive steps of learning a delayed response task. J. Neurosci. 13, 900–913.
Spreckelmeyer, K.N., Krach, S., Kohls, G., Rademacher, L., Irmak, A., Konrad, K., Kircher, T., Grunder, G., 2009. Anticipation of monetary and social reward differently activates mesolimbic brain structures in men and women. Soc. Cogn. Affect. Neurosci. 4, 158–165.
Stoeckel, L.E., Weller, R.E., Cook 3rd, E.W., Twieg, D.B., Knowlton, R.C., Cox, J.E., 2008. Widespread reward-system activation in obese women in response to pictures of high-calorie foods. Neuroimage 41, 636–647.
Talairach, J., Tournoux, P., 1988. Co-planar Stereotaxic Atlas of the Human Brain: 3-Dimensional Proportional System—an Approach to Cerebral Imaging. Thieme Medical Publishers, New York.
Thut, G., Schultz, W., Roelcke, U., Nienhusmeier, M., Missimer, J., Maguire, R.P., Leenders, K.L., 1997. Activation of the human brain by monetary reward. Neuroreport 8, 1225–1228.
Tottenham, N., Tanaka, J.W., Leon, A.C., McCarry, T., Nurse, M., Hare, T.A., Marcus, D.J., Westerlund, A., Casey, B., Nelson, C., 2009. The NimStim set of facial expressions: Judgments from untrained research participants. Psychiatry Res. 168 (3), 242–249.
Tovee, M.J., 1995. Face recognition. What are faces for? Curr. Biol. 5, 480–482.
Wilson, D.I., Bowman, E.M., 2006. Neurons in dopamine-rich areas of the rat medial midbrain predominantly encode the outcome-related rather than behavioural switching properties of conditioned stimuli. Eur. J. Neurosci. 23, 205–218.
Wrase, J., Schlagenhauf, F., Kienast, T., Wustenberg, T., Bermpohl, F., Kahnt, T., Beck, A., Strohle, A., Juckel, G., Knutson, B., Heinz, A., 2007. Dysfunction of reward processing correlates with alcohol craving in detoxified alcoholics. Neuroimage 35, 787–794.
Yang, T.T., Menon, V., Eliez, S., Blasey, C., White, C.D., Reid, A.J., Gotlib, I.H., Reiss, A.L., 2002. Amygdalar activation associated with positive and negative facial expressions. Neuroreport 13, 1737–1741.
Young III, W.S., Alheid, G.F., Heimer, L., 1984. The ventral pallidal projection to the mediodorsal thalamus: a study with fluorescent retrograde tracers and immunohistofluorescence. J. Neurosci. 4, 1626–1638.
Young, A.W., Hellawell, D.J., Van De Wal, C., Johnson, M., 1996. Facial expression processing after amygdalotomy. Neuropsychologia 34, 31–39.