Aversive disinhibition of behavior and striatal signaling in social avoidance

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Social avoidance is a major factor contributing to the development and maintenance of anxiety and depressive symptoms. Converging evidence suggests that social avoidance is associated with abnormal aversive processing and hyperactive amygdala signaling. However, what are the consequences of such abnormal aversive processing for action and for the neural mechanisms implementing action is unclear. Existing literature is conflicting, pointing at either enhanced or reduced action inhibition. We investigated the interaction between aversion and action in social avoidance by comparing the effects of aversive vs appetitive faces on a go/no-go task and associated striatal signals in 42 high and low socially avoidant individuals. We combined fMRI with a novel probabilistic learning task, in which emotional valence (angry and happy faces) and optimal response (go- and no-go-responses) were manipulated independently. High compared with low socially avoidant individuals showed reduced behavioral inhibition (proportion no-go-responses) for angry relative to happy faces. This behavioral disinhibition correlated with greater striatal signal during no-go-responses for angry relative to happy faces. The results suggest that social avoidant coping style is accompanied by disinhibition of action and striatal signal in the context of social threat. The findings concur with recent theorizing about aversive disinhibition and affective disorders.

Keywords: social avoidance; aversive disinhibition; behavioral inhibition; striatum; emotional faces; go/no-go probabilistic learning task

INTRODUCTION

Social avoidance is a major risk factor for the development and maintenance of anxiety and depression (Mazer and Cloninger, 1990; Barlow, 2002; Mineka and Zinbarg, 2006). These psychiatric disorders have often been associated with enhanced aversive processing and hyperactive amygdala signaling (Schneider et al., 1999; Sheline et al., 2001; Veit et al., 2002; Siegle et al., 2006; Staugaard, 2010). Moreover, amygdala abnormalities often accompany personality traits related to social avoidance (Schwartz et al., 2003; Idaka et al., 2006). Although there is converging evidence that social avoidance is associated with enhanced aversive processing, it remains unclear what are the consequences of abnormal aversive processing and aberrant amygdala signaling for action selection and for neural systems that implement action selection, such as the striatum. Insight in the emotional influence on action selection is critical for advancing our understanding of the (neurocognitive) mechanisms underlying the complex and impairing symptomatology of social avoidance.

Two competing hypotheses can be formulated on the basis of early theories and recent insights. The first and most intuitive hypothesis is that social avoidance, which is accompanied by enhanced aversive processing, is associated with an increase in the inhibition of actions. According to early theories on individual differences in avoidance motivation, avoidant traits and related affective disorders are associated with an enhanced tendency to respond intensely to signals of aversive stimuli, which facilitates behavioral inhibition in order to avoid punishment (Cloninger, 1987; Clark and Watson, 1991; Gray, 1994). Indeed, aversion seems to be intrinsically coupled with action inhibition: greater aversion elicits greater inhibition of actions (Bourreau and Dayan, 2011). Accordingly, social avoidance, which is accompanied by enhanced aversive processing, might be associated with increased inhibition of action in an aversive context.

In contrast, based on recent neurochemical theories related to affective disorders (Dayan and Huys, 2008, 2009), one might pose the alternative, more radical hypothesis that social avoidance is accompanied by a paradoxical decrease in inhibition in an aversive context (i.e. aversive disinhibition). According to these latter theories, affective disorders reflect a failure to inhibit aversive thoughts and actions (Dayan and Huys, 2008, 2009). Individuals suffering from these affective disorders may perceive situations as more threatening and may assume more negative attributions and outcomes. In turn, this may lead to more escape and avoidance behavior as a coping strategy to enhance personal safety (Kearney, 2004). By analogy, a failure to inhibit aversive thoughts and actions might be the underlying mechanism of social avoidance. The persistent tendencies to avoid social situations in daily life might then reflect a secondary strategy to cope with this aversive disinhibition, a notion compatible with the vigilance-avoidance theory (e.g. Mogg and Bradley, 1998; Bögels and Mansell, 2004; Mogg et al., 2004), and more recent interpretations of Gray’s theory of the Behavioral Inhibition System (BIS; Gray and McNaughton, 2000; McNaughton and Corr, 2004; p. 286). Here we tested these two opposing hypotheses by comparing high and low socially avoidant individuals on a task that quantifies aversive inhibition.

Specifically, we used functional magnetic resonance imaging (fMRI) combined with a novel paradigm to investigate effects of aversive (relative to appetitive) processing on behavioral inhibition vs activation in social avoidance. Participants were presented with aversive (angry) faces and appetitive (happy) faces and had to learn by trial and error whether to make a go- or a no-go-response in order to obtain reward or avoid punishment. We manipulated emotional valence (angry/happy), the optimal response (go/no-go) and instrumental valence (reward/punishment) independently in a probabilistic learning task, in which subjects were unlikely to detect and apply an explicit rule.
The rationale for using a probabilistic learning paradigm was to ensure that subjects recruit a (habit-based) behavioral control system that is thought to be particularly sensitive to emotional influences (Dickinson et al., 1995; Holland et al. 2004; Dayan and Huys, 2008).

In our analyses we focused on two regions of interest. Our first region of interest was the amygdala, which is well known to be involved in aversive processing (e.g. Staugaard, 2010) and emotional accounts in motivated behavior through its interaction with other brain areas (e.g. Cardinal et al., 2002). Our second region of interest was the striatum, which is known to implement action selection and to interact with the amygdala (Cardinal et al., 2002). Critically, the striatum has long been shown to be associated with behavioral activation (vs inhibition) when facing appetitive stimuli (Schultz et al., 1997; Berridge and Robinson, 1998; Salamone et al., 2005; Niv et al., 2007), and has recently been demonstrated to represent predominantly (go) action independent of valence (Guitart-masip et al., 2011). The current paradigm was used to disentangle two sets of alternative hypotheses pointing at either aversive inhibition or aversive disinhibition underlying social avoidance. Revealing these distinct mechanisms is important, not only for understanding the neural process underlying social avoidance, but also for advancing (preventative) therapies targeted either at decreasing behavioral inhibition, or contrarily, gaining control over aversive disinhibition.

**METHODS AND MATERIALS**

**Participants**
Forty-five female students from the Radboud University Nijmegen participated in this study after giving written informed consent. We selected only women, because of the higher prevalence of affective symptoms and disorders (Kessler et al., 1993; Nolen-Hoeksema, 2001) and the higher levels of emotional reactivity reported for women than men (Koch et al., 2007; Domes et al., 2010). They received payment or course credits as a reimbursement for participation. All participants were healthy, right-handed and had normal or corrected-to-normal visual acuity. Exclusion criteria were claustrophobia, neurological or cardiovascular diseases, psychiatric disorders, regular use of medication or marijuana, use of psychotropic drugs, heavy smoking and metal parts in the body. For one participant, the fMRI session was aborted, due to headache. Two other participants were excluded from data analyses, because their performance pattern did not meet our predefined criteria of adequate performance, which may indicate poor task compliance or motivation; in all but these two participants, simple regression analyses revealed a significant linear effect of outcome probability (i.e. the experimentally manipulated action-outcome contingencies, which determined the optimal response, significantly predicted the actual response given by the participants). Thus, data of 42 participants were analyzed. To investigate the effects of social avoidance on the interaction between emotional valence and behavioral inhibition, we divided participants in a low-(low-avoidant) and a high socially avoidant (high-avoidant) group using a median-split procedure based on the avoidance subscale of the Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987). The low-avoidant group (M = 3.33, SD = 2.8) differed significantly from the high-avoidant group (M = 19.81, SD = 7.2) in score on the avoidance subscale of the LSAS [t(25,827) = −9.838, P < 0.001].

**Learning paradigm**
The goal of our design was to investigate the effects of social avoidance on the influence of aversive (relative to appetitive) processing on behavioral inhibition vs activation, and on associated striatal BOLD signal. We manipulated emotional valence (angry/happy), optimal response (go/no-go) and instrumental valence (reward/punishment) independently in a novel probabilistic learning paradigm. Participants were presented with aversive (angry) faces and appetitive (happy) faces. They had to learn by trial and error whether to make a go- or a no-go-response (i.e. press a button, or withhold a button press, respectively) in order to obtain monetary reward or avoid monetary punishment (Figure 1). Our primary research question was focused on the effects of social avoidance on the influence of angry (relative to happy) faces on behavioral inhibition (relative to activation). We included an additional factor of instrumental outcome valence to explore the effects of social avoidance on the influence of reward and punishment anticipation on behavioral inhibition, and whether social avoidance would be associated with the extent to which reward or punishment anticipation would add to or potentiate the effects of a compatible emotional valence on behavioral inhibition.

The task consisted of four cue-types: angry-reward, angry-punishment, happy-reward and happy-punishment. We used different colors to distinguish between reward and punishment cues of the same emotional category; yellow and grayscale were randomly assigned to signal either a reward or punishment condition for each participant (which leads to either one of the two options for a participant: (i) Faces in yellow indicative of reward, and faces in grayscale indicative of punishment; or (ii) the other way around, faces in yellow indicative of punishment, and faces in grayscale indicative of reward). Participants were instructed that the combination of emotional category and color (signaling reward/punishment conditions) distinguished the four cue-types and that they had to learn the optimal response for each of the four cue-types separately. The optimal response (go- or no-go-response) was manipulated for each cue-type independently across time, by changing the action-outcome contingencies or the probability of a positive outcome given a go-response, \(p_{\text{Pos_Out}(\text{Go})}\), for each cue-type over time. Specifically, for each cue-type separately, the probability of a positive outcome given a go-response could be low \(\{p_{\text{Pos_Out}(\text{Go})} = 0.20\}\) or high \(\{p_{\text{Pos_Out}(\text{Go})} = 0.80\}\) and was changed pseudorandomly over time. Thus, at different times during the experiment, a go-response for a certain cue-type would be rewarded or not punished on a certain percentage of trials (20% or 80%). Participants were instructed to learn the optimal response (to maximize reward, or minimize punishment) for each cue-type separately by trial and error. They were informed that the action-outcome contingencies were probabilistic and would change unpredictably over time. They were not informed about the nature of the probabilistic associations or about the time intervals across which they changed.

Each participant completed three sessions, with a 1-min break in between the sessions. Each session consisted of 160 trials, with 40 trials per cue-type. For an example of a time series, see Figure 1B. For each cue-type within a session, the probability of a positive outcome given a go-response could take one of the following combinations in two consecutive blocks: (i) 0.20, 0.20; (ii) 0.20, 0.80; (iii) 0.80, 0.80. The block lengths varied between 12 and 18 trials per cue-type, so that participants could not predict exactly when a change in contingency would occur. Moreover, to avoid instantaneous and complete reversals of the contingencies during the task, there were always short blocks (2–8 trials) of nonpredictive trials \(\{p_{\text{Pos_Out}(\text{Go})} = 0.50\}\) at the beginning of a session and in between the blocks. We used 24 different sets of pseudorandom sequences across participants.

**Timing and visual stimuli**
Visual stimuli were adult Caucasian faces [trimmed to exclude influence from hair and nonfacial contours (van Peer et al., 2007; Roelofs et al., 2009)] from 36 models (18 men) taken from several databases (Ekman and Friesen, 1976; Matsumoto and Ekman, 1988; Martinez and Benavente, 1998; Lundqvist et al., 1998). Model identity...
was counterbalanced, such that the model occurred equally often for each cue-type. For each model there were two emotions (angry and happy), which occurred in both yellow and grayscale (randomly assigned to signal the possibility of reward or punishment), matched for brightness and contrast values, displayed against a black background. The stimuli were projected onto a mirror above the subjects’ head, subtending a visual angle of 21° by 14.6°. On each trial, one of the face cues was presented centrally for 100 ms. After cue onset, participants were required to make either a go- or a no-go-response as fast as possible within 1000 ms. If no response was made within 1000 ms, then a no-go-response was recorded. After a response–outcome delay of maximally 2000 ms (depending on the response time), the outcome was presented for 1000 ms (+10 cents for reward, −10 cents for punishment, and +0 cents for omitted reward or avoided punishment). The intertrial interval was jittered (3500 ± 1000 ms). Stimulus presentation and response acquisition were controlled by a PC running Psychophysics Toolbox Version 3 with Matlab version 7.9.0 R2009a.

**Procedure**

Upon arrival, the participants were reminded of the experimental procedure. They completed the LSAS. Subsequently, they were familiarized with the learning task by means of instructions (see below) and a short training session before being positioned in the MR scanner for the fMRI session to start. To increase ecological validity and participants’ motivation during the learning task, we told participants that the sum of the amount of money gained and lost from the learning task would be calculated at the end of the experiment. They would receive the actual amount of monetary gain as a payment.

**Behavioral data analysis**

The behavioral data were analyzed using the statistics software SPSS 16.0. The proportion of no-go-responses that was made by the participants and reaction times (RTs) were analyzed using a mixed design ANOVA with group (high-avoidant/low-avoidant) as between-subject factor, and emotion (angry/happy), and outcome (instrumental valence: punishment/reward) as within-subject factors. Significant interactions were broken down by using simple interaction effects analyses. Finally, we conducted an additional analysis to test the success of the action–outcome contingency manipulation (i.e., probability of a positive outcome given a go-response). This analysis confirmed that participants were able to track the manipulation by showing the ‘optimal response’.

**Image acquisition**

Whole-brain imaging was performed on a 3 T MR scanner (Magnetom Trio Tim; Siemens Medical Systems) equipped with an 32-channel head coil using a multi-echo GRAPPA sequence (Poser et al., 2006) [repetition time (TR): 2.32 ms, echo times (TEs, 4): 9.0/19.3/30/40 ms, 38 axial oblique slices, ascending acquisition, distance factor: 17%, voxel size 3.3 × 3.3 × 2.5 mm, field of view (FoV): 211 mm; flip angle, 90°]. At the end of the experimental session, high-resolution anatomical images were acquired using a magnetization prepared rapid gradient echo sequence (TR: 2300 ms, TE: 3.03 ms, 192 sagittal slices, voxel size 1.0 × 1.0 × 1.0 mm, FoV: 256 mm).

**fMRI data analysis**

Images were preprocessed with SPM5, while statistical analyses were conducted with SPM8 (Statistical Parametric Mapping; http://www.fil.ion.ucl.ac.uk/spm). Given the multiecho GRAPPA MR sequence (Poser et al., 2006), the head motion parameters were estimated on the MR images with the shortest TE (9.0 ms), because these images are the least affected by possible artifacts. These motion-correction parameters, estimated using a least-squares approach with six rigid body transformation parameters (translations, rotations), were then applied to the four echo images collected for each excitation. After spatial realignment, the four echo images were combined into a single MR volume using an optimized echo weighting method (Poser et al., 2006). The T1-weighted image was spatially coregistered to the mean of the functional images. The fMRI time series were transformed and resampled at an isotropic voxel size of 2 mm into the standard Montreal Neurological Institute (MNI) space using both linear and non-linear transformation parameters as determined in a probabilistic generative model that combines image registration, tissue classification, and bias correction (i.e. unified segmentation and normalization) of the coregistered T1-weighted image (Ashburner and Friston, 2005). The normalized functional images were spatially smoothed using an isotropic 8 mm full-width at half-maximum Gaussian kernel. The fMRI time series of each subject were analyzed using an event-related approach in the context of the general linear model (GLM). For each session, eight conditions of interest were modeled as separate regressors in a GLM as a function of emotion, the response that was made by the participant, and outcome: angry-punishment-go, angry-punishment-no-go, happy-punishment-go, happy-punishment-no-go, angry-reward-go, angry-reward-no-go, happy-reward-go and happy-reward-no-go. The six realignment parameters were added to capture residual head movement-related artifacts. An additional three regressors were included, describing the time course of signal intensities averaged across different image compartments of no interest (i.e. white
RESULTS

Behavioral results

Proportion no-go

ANOVA of the proportion of no-go-responses revealed a Group × Emotion interaction \([F(1,40) = 6.4, P = 0.016]\), which was due to a lower proportion of no-go-responses for angry vs happy faces in high-avoidant participants relative to low-avoidant participants. In fact, the difference in no-go-responses between angry and happy faces was absent in high-avoidant participants \([P = 0.657; \text{Figure 2}\), but not in the low-avoidant participants \([F(1,20) = 18.9, P < 0.001]\). A main effect of outcome \([F(1,40) = 17.1, P < 0.001]\) indicated that the proportion of no-go-responses was greater when participants avoided punishment than when they maximized reward. Finally, there was also a significant Emotion × Outcome interaction \([F(1,40) = 9.4, P = 0.004]\) due to greater proportion of no-go-responses for angry (\(M = 49.8, \text{SEM} = 1.6\)) vs happy faces (\(M = 45.2, \text{SEM} = 1.6\)) in the reward condition \([F(1,40) = 9.3, P = 0.004]\), but not in the punishment condition \([F(1,40) = 0.722, P = 0.4]\); angry (\(M = 53.1, \text{SEM} = 1.3\)), happy (\(M = 54.2, \text{SEM} = 1.5\)). No other significant interaction effects were found (all \(P < 0.160\)). Raw data are presented in Table 1.

Reaction time on go-trials

ANOVA of RT data revealed a main effect of outcome \([F(1,40) = 10.1, P = 0.003]\), as well as an Emotion × Outcome interaction \([F(1,40) = 5.7, P = 0.022]\). In addition, a main effect of emotion \([F(1,40) = 11.7, P = 0.001]\) indicated overall faster RTs for happy vs angry faces. No other (interaction) effects were found (all \(P > 0.434\)). A supplementary Emotion × Probability (of a positive outcome given a go-response) × Outcome ANOVA on the proportion of no-go-responses revealed a main effect of Probability \([F(2,39) = 135.7, P < 0.001]\), which was due to significant differences between all probabilities in the expected direction [i.e. 20% (\(M = 69.3, \text{SEM} = 1.6\)) > 50% (\(M = 51.5, \text{SEM} = 1.4\)) > 80% (\(M = 31.0, \text{SEM} = 1.6\))]; all \(P < 0.001\) suggesting that participants were able to track the action–outcome contingencies.

fMRI results

Effects of emotion in the amygdala

ROI analysis of data from the anatomically defined amygdala replicated prior work (Staugaard, 2010) by showing a main effect of emotion \([F(1,40) = 7.4, P = 0.010]\): Amygdala signal was significantly greater for angry vs happy faces. Consistent with our hypothesis, we also found a Group × Emotion interaction effect \([F(1,40) = 10.9, P = 0.002]\) due to greater amygdala response to angry vs happy faces in high-avoidant participants \([F(1,20) = 12.4, P = 0.002]\) relative to low-avoidant participants \([F(1,20) = 0.316, P = 0.581; \text{Figure 3}\). In addition, a main effect of response \([F(1,40) = 7.9, P = 0.007]\) indicated that amygdala signal was greater for go- than for no-go-responses. No other significant main or interaction effects were found (all \(P > 0.153\)).

Effects of emotion on striatal signals associated with behavioral activation

ROI analysis of data from the functionally defined striatum confirmed a main effect of response, due to greater signals during go than
within high-avoidant participants \[
\text{F(1,20) = 7.8, } P = 0.011 \]
and not within low-avoidant participants \[
\text{F(1,20) = 0.101, } P = 0.754 \].
Further analyses within the high-avoidant participants showed that the Emotion \times Response interaction was due to greater striatal signal for angry vs happy faces in the no-go-condition \[
\text{F(1,20) = 6.07, } P = 0.445 \].
As shown in Figure 4, this enhanced striatal ‘activation’ signal for angry vs happy faces on no-go-trials was only apparent in high-avoidant participants and not in low-avoidant participants \[
\text{Group \times Emotion interaction; } F(1,40) = 5.5, P = 0.024 \].
Raw data are presented in Table 2. Finally, the effect of emotion on striatal ‘activation’ signal during no-go-trials correlated with the effect of emotion on behavioral inhibition, as measured in terms of the proportion of no-go-responses: greater striatal ‘activation’ signal on angry vs happy no-go-trials was associated with reduced behavioral inhibition for angry vs happy faces \[
(r = -0.358, P = 0.020; \text{Figure 5}) \].
No other interaction effects were found (ROI analyses: all \(P > 0.116 \)).

We refer to the Supplementary Materials for the results of our whole-brain voxel-wise analyses, which revealed main effects of emotion, response and outcome. No significant interaction effects were found using our stringent statistical threshold of \(P_{\text{FWE}} < 0.05 \).

**DISCUSSION**

We used fMRI combined with our novel paradigm to investigate the effects of social avoidance on the influence of aversive (relative to appetitive) processing on behavioral inhibition vs activation, and on associated striatal signaling. We aimed to test the two opposing hypotheses of either increased aversive inhibition or decreased aversive inhibition (aversive disinhibition) in social avoidance. Consistent with previous literature regarding emotional processing in psychiatric disorders characterized by social avoidance (e.g. Schneider et al., 1999; Veit et al., 2002; Staugaard, 2010), our fMRI data showed that amygdala signaling was greater for angry vs happy faces in high-avoidant compared with low-avoidant participants. This finding suggests enhanced aversive processing in high-avoidant compared with low-avoidant participants. However, the crucial finding of this study is that high-avoidant participants compared with low-avoidant participants showed aversive disinhibition of behavior, i.e. they showed reduced behavioral inhibition for angry vs happy faces. Furthermore, this behavioral effect correlated significantly with greater striatal signaling, associated with behavioral activation, during no-go-trials for angry vs happy faces. Thus, our results support the hypothesis of aversive disinhibition rather than increased aversive inhibition in social avoidance.

Our findings are in line with recent neurochemical theories on behavioral inhibition in affective disorders. These theories suggest that aversive disinhibition, might be the underlying mechanism of negative thoughts and/or a lack of positive bias in psychopathology.
no-go-responses for angry vs disinhibition underlying socially avoidant coping and we recommend this is the first human investigation in this intriguing field of aversive (Dayan and Huys, 2008, 2009). While the natural tendency to inhibit actions and thoughts leading to aversive states protects us from psychopathology, a failure to show this type of inhibition may lead to enhanced negative thoughts. Escape and avoidance behavior might then represent a coping strategy to enhance personal safety (Kearney, 2004). Accordingly, social avoidance may be a secondary consequence of aversive disinhibition. This idea is compatible with the vigilance–avoidance hypothesis, which describes the phenomenon of an enhanced initial automatic orienting to threat, followed by avoidance as a strategic attempt to alleviate the negative affective state elicited by the aversive stimuli (e.g. Mogg and Bradley, 1998; Bogels and Mansell, 2004; Mogg et al., 2004). Moreover, our findings are consistent with more recent interpretations of Gray’s theory of the BIS (Gray and McNaughton, 2000; McNaughton and Corr, 2004; p. 286). The BIS is proposed to play a role in resolving conflicts among competing goals by actively engaging in risk assessment behaviors. Enhanced BIS promotes scanning for threat-relevant information, and as such may act in favor for avoidance behavior. It might be noted that the absolute behavioral data suggest less inhibition for happy faces in the low-avoidant group (Table 1). However, in the absence of a neutral control condition, we think our results cannot easily be interpreted in terms of absolute scores, but rather must be interpreted in terms of difference scores between angry relative to happy faces. Moreover, appetitive disinhibition in the low-avoidant group would be difficult to reconcile with the group difference in the fMRI data, which was driven by enhanced striatal signaling for angry relative to happy faces (during no-go-trials) in the high-avoidant participants. One could argue that this aberrant disinhibition in response to angry relative to happy faces in the high-avoidant participants could be driven by excessive inhibition to happy relative to angry faces. However, given the empirical evidence for abnormal processing of aversive faces in social avoidance in the current and previous studies (e.g. Schneider et al., 1999; Veit et al., 2002; Staugaard, 2010), as well as contemporary theories regarding the coupling of inhibition with aversion (Dayan and Huys, 2008, 2009), these preliminary results are best interpreted as aversive disinhibition in the high-avoidant participants. This is the first human investigation in this intriguing field of aversive disinhibition underlying socially avoidant coping and we recommend replication including neutral faces in future research to avoid interpretational limitations.

Our hypothesis that social avoidance is accompanied by abnormal influences of aversive processing, mediated by the amygdala, on action selection, mediated by the striatum, would have been strengthened by an effect of group on the functional connectivity between the amygdala and the striatum. Unfortunately, we did not detect such an effect, presumably due to low statistical power. We suggest that the open question whether aversive disinhibition in social avoidance is accompanied by abnormal amygdala–striatal connectivity should be investigated in future research.

Note that the majority of previous investigations on affective (anxiety and depressive) disorders, which show high comorbidity (Kaufman and Charney, 2000), have adopted a categorical approach, largely ignoring potentially shared mechanisms that may underlie these disorders. In line with current transdiagnostic approaches, we did not focus on social anxiety or depression per se, but we focused on a factor that has previously been described as the major maintaining factor underlying affective symptoms: social avoidance.

We developed a novel paradigm to investigate the effects of social avoidance on the emotional influence on behavioral activation. We successfully found differences between high and low socially avoidant individuals on the emotional influences on behavioral activation, as indicated by both behavioral and neural measures. Future research should examine the specificity of the findings for social avoidance vs its correlates (e.g. negative affectivity, mood and anxiety disorders). Finally, it remains unknown whether our behavioral and neural effects reflect the reflexive (Pavlovian) system or the more goal-driven (instrumental) system. Further research is needed, using a design which allows disentangling the Pavlovian response from the instrumental response, to investigate whether our results, suggestive of aversive disinhibition in social avoidance, involve Pavlovian control of instrumental action selection (Dayan and Huys, 2008).

In sum, we found that self-reported social avoidance tendencies are associated with aversive disinhibition of behavior and striatal signaling. These findings concur with recent behavioral neurochemical theorizing about aversion, behavioral inhibition and affective disorders and suggest that aversive disinhibition of behavior and striatal signaling might represent a core phenomenon of social avoidance behavior.

**SUPPLEMENTARY DATA**

Supplementary data are available at SCAN online.

**Conflict of Interest**

None declared.

**REFERENCES**


