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**Research Articles: Behavioral/Cognitive**

**Contributions of the ventral striatum to conscious perception: An intracranial EEG study of the attentional blink**

Heleen A Slagter<sup>1,2</sup>, Ali Mazaheri<sup>3</sup>, Leon C Reteig<sup>1</sup>, Ruud Smolders<sup>4</sup>, Martijn Figee<sup>4</sup>, Mariska Mantione<sup>4</sup>, P. Richard Schuurman<sup>5</sup> and Damiaan Denys<sup>4,6</sup>

<sup>1</sup>Department of Psychology, University of Amsterdam, The Netherlands

<sup>2</sup>Amsterdam Brain and Cognition, University of Amsterdam, The Netherlands

<sup>3</sup>School of Psychology, University of Birmingham, United Kingdom

<sup>4</sup>Department of Psychiatry, Academic Medical Center, Amsterdam, The Netherlands

<sup>5</sup>Department of Neurosurgery, Academic Medical Center, Amsterdam, The Netherlands

<sup>6</sup>The Netherlands Institute for Neuroscience, Royal Netherlands Academy of Arts and Science, Amsterdam, The Netherlands.

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Corresponding author: [h.a.slagter@uva.nl](mailto:h.a.slagter@uva.nl)

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5 Heleen A Slagter<sup>1,2\*</sup>, Ali Mazaheri<sup>3</sup>, Leon C Reteig<sup>1</sup>, Ruud Smolders<sup>4</sup>, Martijn  
6 Figeer<sup>4</sup>, Mariska Mantione<sup>4</sup>, P. Richard Schuurman<sup>5</sup> & Damiaan Denys<sup>4,6</sup>

7  
8 <sup>1</sup> Department of Psychology, University of Amsterdam, The Netherlands

9 <sup>2</sup> Amsterdam Brain and Cognition, University of Amsterdam, The Netherlands

10 <sup>3</sup> School of Psychology, University of Birmingham, United Kingdom

11 <sup>4</sup> Department of Psychiatry, Academic Medical Center, Amsterdam, The Netherlands

12 <sup>5</sup> Department of Neurosurgery, Academic Medical Center, Amsterdam, The Netherlands

13 <sup>6</sup> The Netherlands Institute for Neuroscience, Royal Netherlands Academy of Arts and  
14 Science, Amsterdam, The Netherlands.

15 \* Corresponding author: h.a.slagter@uva.nl

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22 **Abstract**

23 The brain is limited in its capacity to consciously process information, necessitating  
24 gating of information. While conscious perception is robustly associated with  
25 sustained, recurrent interactions between widespread cortical regions, subcortical  
26 regions, including the striatum, influence cortical activity. Here, we examined if the  
27 ventral striatum, given its ability to modulate cortical information flow, contributes to  
28 conscious perception. Using intracranial EEG, we recorded ventral striatum activity  
29 while 7 patients performed an attentional blink task in which they had to detect two  
30 targets (T1 and T2) in a stream of distractors. Typically, when T2 follows T1 within  
31 100-500ms, it is often not perceived (i.e., the attentional blink). We found that  
32 conscious T2 perception was influenced *and* signaled by ventral striatal activity.  
33 Specifically, the failure to perceive T2 was foreshadowed by a T1-induced increase in  
34 alpha and low beta oscillatory activity as early as 80ms post-T1, indicating that the  
35 attentional blink to T2 may be due to very early T1-driven attentional capture.  
36 Moreover, only consciously perceived targets were associated with an increase in  
37 theta activity between 200-400ms. These unique findings shed new light on the  
38 mechanisms that give rise to the attentional blink by revealing that conscious target  
39 perception may be determined by T1 processing at a much earlier processing stage  
40 than traditionally believed. More generally, they indicate that ventral striatum activity  
41 may contribute to conscious perception, presumably by gating cortical information  
42 flow.

43 **Significance statement**

44

45 What determines whether we become aware of a piece of information or not?

46 Conscious access has been robustly associated with activity within a distributed

47 network of cortical regions. Using intracranial electrophysiological recordings during

48 an attentional blink task, we tested the idea that the ventral striatum, because of its

49 ability to modulate cortical information flow, may contribute to conscious perception.

50 We find that conscious perception is influenced, and signaled by ventral striatal

51 activity; Short-latency (80-140ms) striatal responses to a first target determined

52 conscious perception of a second target. Moreover, conscious perception of the

53 second target was signaled by longer-latency (200-400ms) striatal activity. These

54 results suggest that the ventral striatum may be part of a subcortical network that

55 influences conscious experience.

56 **Introduction**

57 A fundamental question in the study of conscious perception relates to the neural  
58 basis of conscious access: what brain mechanisms determine whether a stimulus  
59 becomes available for explicit report? Research shows that only consciously  
60 perceived stimuli are associated with sustained, synchronized activity within a  
61 distributed network of cortical regions (Rees et al., 2002; Haynes, 2009; Dehaene and  
62 Changeux, 2011; Lau and Rosenthal, 2011; Aru et al., 2012; van Gaal and Lamme,  
63 2012). Based on this work, influential theories propose that conscious access stems  
64 from a cognitive architecture with an evolved function: the flexible sharing of  
65 information throughout the cortex (Baars, 1993; Dehaene and Naccache, 2001). Yet,  
66 the cortex operates in close interaction with subcortical regions, specifically the  
67 thalamus and the basal ganglia. The thalamus has rich and widespread reciprocal  
68 connections to the cortex and thus can synchronize activity across distant cortical  
69 regions. Several theories propose that this vast thalamocortical architecture may shape  
70 the boundary conditions for both the level of wakefulness (Vijayan and Kopell, 2012;  
71 MacDonald et al., 2015) as well conscious perception to occur (Newman and Baars,  
72 1993; Crick, 1995; Tononi and Edelman, 1998; Baars, 2005; Dehaene and Changeux,  
73 2005). Notably, thalamocortical interactions are modulated by the basal ganglia,  
74 which tonically inhibit the thalamus and thereby cortical activation. This raises the  
75 possibility that the basal ganglia may also contribute to conscious perception, through  
76 their influence on activity within the network of fronto-parietal regions that gives rise  
77 to conscious experience.

78 The basal ganglia are connected to many frontal regions in parallel loops through  
79 the thalamus, and hence capable of modulating a wide range of associated processes.  
80 Loops connecting the basal ganglia to motor cortex have long been implicated in  
81 action selection. Notably, converging evidence indicates that these parallel loops  
82 connecting the basal ganglia and frontal cortex may serve a more generic “selection”  
83 function and may resolve conflicts not only in the motor domain, but also among  
84 cognitive resources (Redgrave et al., 1999a); Evidence from animal studies suggests  
85 that the striatum can act as an early gating system that enables the prioritization of  
86 salient stimuli by triggering frontal systems to orient attention (Redgrave and Gurney,  
87 2006; Overton et al., 2014). Moreover, the striatum receives longer-latency input from  
88 the hippocampus, which may provide longer-duration, context-dependent gating of

89 prefrontal activity (Newman and Grace, 1999). By gating cortical information flow  
90 the striatum could hence be part of a subcortical network that provides a “gateway” to  
91 conscious experience, for example by biasing attention (van Schouwenburg et al.,  
92 2015), and guide which information is selected for global broadcasting and conscious  
93 access.

94 Although the striatum is perfectly situated to influence conscious information  
95 processing, little is currently known about its exact contribution to conscious  
96 perception. This gap in knowledge is related to our inability to record subcortical  
97 activity with high temporal precision in healthy humans and difficulties in studying  
98 consciousness in animals. Human neuroimaging studies have provided some support  
99 for a role for the striatum and its irrigation by dopamine in conscious perception  
100 (Slagter et al., 2010, 2012; Van Opstal et al., 2014; Bisenius et al., 2015) For  
101 example, a recent positron emission tomography (PET) study (Slagter et al., 2012)  
102 revealed an association between striatal dopamine D2 receptor binding and the  
103 attentional blink (AB): a deficit in consciously perceiving the second target of two  
104 targets (T1 and T2) whenever it follows T1 within 100-500 ms in a rapid stream of  
105 distractors. Yet, these neuroimaging methods lack temporal precision and provide  
106 indirect measures of neural activity, leaving the specific contribution of the striatum  
107 to conscious perception unclear.

108 To shed light on the potential role of the striatum in conscious perception, we  
109 used the unique opportunity to directly record electrophysiological activity from the  
110 ventral striatum in patients, while they performed an attentional blink task. This task  
111 has two unique features. First, it has proven useful for studying the neural correlates  
112 of conscious perception by comparing the neural processing of T2 between T2-seen  
113 vs. T2-unseen trials. Second, it allows investigation of the conditions that are  
114 necessary for a stimulus to reach awareness by revealing how neural processing of T1  
115 affects the ability to consciously perceive T2 (Dux and Marois, 2009; Martens and  
116 Wyble, 2010). By linking conscious perception to real-time basal-ganglia  
117 measurements, we aimed to elucidate the role of the ventral striatum in conscious  
118 perception.

119 **METHODS AND MATERIALS**

120 **Participants**

121 Eight therapy-resistant patients eligible for deep brain stimulation (DBS) participated  
122 in the study. One patient had difficulty seeing any target, and the experiment had to be  
123 aborted prematurely. Of the seven remaining patients (mean age: 39.5; age range: 22  
124 to 63 years), six were female. Five patients had a diagnosis of obsessive-compulsive  
125 disorder (OCD), one patient had a diagnosis of major depressive disorder, and another  
126 patient had a drug addiction. They participated in the experiment one day (1 patient)  
127 or three days (6 patients) after undergoing DBS lead placement surgery and before  
128 DBS stimulator implantation. All the patients underwent careful screening before  
129 being included for DBS as described in (Denys et al., 2010). Patients were implanted  
130 following standard procedures with a four contact electrode (model 3387 with contact  
131 points 1.5 mm long and separated from adjacent contacts by 1.5 mm; Medtronic,  
132 Minneapolis, Minnesota) in each hemisphere. The surgical technique has been  
133 described elsewhere (Denys et al., 2010; van den Munckhof et al., 2013). The  
134 electrodes were implanted following the anterior limb of the internal capsule (ALIC),  
135 with the deepest contact targeted at the core of the nucleus accumbens and the three  
136 upper contacts in the ventral ALIC (Fig. 1). DBS leads were externalized at the back  
137 of the head based on individual anatomy to facilitate the recordings. Correct  
138 stereotactic position of the DBS leads was verified with postoperative stereotactic  
139 computer tomography. During the subsequent recordings, patients received normal  
140 medical treatment in addition to antibiotics and analgesics when requested or deemed  
141 medically necessary. However, to reduce the risk of brain haemorrhage any  
142 administration of Selective Serotonin Response Inhibitors was suspended. This study  
143 was approved by the Institutional Review Board of the Academic Medical Center of

144 the University of Amsterdam.

145

146 **Task and stimuli**

147 Stimulus presentation was performed using Presentation (Version 14.5;  
148 Neurobehavioural Systems, Inc.) and a laptop (HP 6730b) with a 15.4 inch display at  
149 a resolution of 1024 by 768 pixels (refresh rate of 60 Hz). The distance from the  
150 screen to the participants was kept around 60 cm. Participants performed a standard  
151 AB task in which they had to identify two digits (T1 and T2) presented in a rapid  
152 stream of distractors (letters and symbols) (Figure 1a). T2 followed T1 either in the  
153 time window of the attentional blink, after 200 msec (short-interval trial), or outside  
154 the time window of the attentional blink, after 800 msec (long-interval trial). Each  
155 trial started with a fixation-plus sign (+; 1500 msec), after which, the stimulus stream  
156 began, consisting of 22 stimuli. Stimuli were presented on a gray background (red,  
157 green, blue (RGB): 50, 50, 50) at the center of the screen (28 point Arial; 0.94 degrees  
158 visual angle) for 50 ms, followed by a 50ms blank. T1 was either green (RGB: 30,  
159 120, 55) or red (RGB: 140, 80, 125). T2 and the distractors were always blue (RGB:  
160 90, 90, 190). Stimulus colors were matched in luminance. T1 had a different color to  
161 make the T2 task more difficult. Digits were drawn randomly (without replacement)  
162 from the set 2–9. Distractors were randomly drawn (without replacement) from the  
163 following set of 30 letters and symbols: W, E, R, T, Y, U, P, A, D, F, G, H, J, K, L, Z,  
164 X, C, V, B, N, M, @, #, \$, %, }, &, <, =. T1 position was varied randomly between 7  
165 and 10. The temporal distance between T1 and T2 could be short (200 ms; 67.5% of  
166 trials) or long (800 ms; 32.5% of trials). 1500ms after the stream ended, participants  
167 were asked to report the two digits by typing the numbers in order using an external  
168 number pad. If they did not see one or both numbers, they were instructed to guess.



169 Participants first practiced the task for 15 trials. In the first 8 practice trials,  
170 stimuli were presented at half speed (100msec duration followed by a 100-msec  
171 blank). They then performed either 4 or 6 blocks (depending on the condition of the  
172 patient) of 37 trials each, resulting in 100 or 150 short-interval trials and 48 or 72  
173 long-interval trials, respectively, all intermixed within blocks.

174 Participants were instructed to keep their eyes at the center of the screen (at  
175 the fixation cross or at the stream of stimuli) at all times except during the question  
176 period. Specifically, they were asked to wait with moving their eyes down to their  
177 response hand until the first question ('what was the first number?') popped up, to  
178 reduce eye movements from creating artifacts in the EEG signal during target  
179 processing. They were also asked to move their eyes back to the center of the screen  
180 while or right after entering their second answer, so that they looked at the fixation  
181 cross again at the beginning of the next trial. Responses were unsped.

182

183 **iEEG Recording.** While patients performed the AB task, we recorded intracranial  
184 EEG from the implanted DBS electrodes using custom extension wires for the 64-  
185 channel (Advanced Neuro Technology (ANT) B.V.) amplifier. We also recorded both  
186 the horizontal and vertical electro-oculogram (EOG), with bipolar electrodes placed at  
187 the outer canthi of both eyes and above and below the left eye respectively. Although  
188 scalp EEG was also recorded using 64 shielded Ag/AgCl electrodes following the  
189 international '10/10' system, we did not process this data further because of the poor  
190 signal quality due to the placement of post-operative bandages on the scalp. The EEG  
191 was recorded at 1024 Hz (except for one patient where data was recorded at 512 Hz).

192

193

194 **Behavioral data analysis**

195 To determine the presence of an AB, T2 accuracy in short and long interval trials was  
196 compared using a paired t test. T2 accuracy was based only on those trials in which  
197 T1 was correctly reported.

198

199 **iEEG data analysis**

200 Offline pre-processing of iEEG data was performed using the EEGLAB toolbox  
201 running in MATLAB (The MathWorks, Inc., Natick, Massachusetts, United States).  
202 The iEEG at a given contact point was referenced to the local average activity of all  
203 contacts in that hemisphere. Visual inspection revealed that no data was recorded (flat  
204 line) at left contact point 4 for one patient, and at right contact point 2 for another  
205 patient (with contact point 1 being the most ventral one). Extreme high-frequency  
206 noise was visible at right contact point 2 for a third patient. These contact points in  
207 these patients were thus excluded from data analysis, and were also not used for  
208 average referencing. Data were subsequently high-pass filtered at 0.5Hz to remove  
209 slow drifts. For each condition of interest, trials were then epoched in synchrony with  
210 T1 onset (-2000 to 2500ms), and trials containing artifacts (drifts etc.) or eye blinks  
211 on the vertical EOG channel during target presentation were rejected through visual  
212 inspection. This led to rejection of 6 trials for one subject. No trials were rejected for  
213 the other subjects. Following previous studies, we focused our analysis on oscillatory  
214 activity in the lower frequency range (< 30Hz), as well as on the ventral-most contact  
215 points at the core of the nucleus accumbens in each hemisphere (Cohen et al., 2012;  
216 Figeo et al., 2013; Smolders et al., 2013), which appear to lie very close to where we  
217 previously observed greater conscious perception-related activity to consciously  
218 perceived compared to missed T2s with fMRI (Slagter et al., 2010).

219            *Time Frequency analysis* To examine the role of the ventral striatum in the  
220 AB, our primary analysis focused on differences in target-induced oscillatory activity  
221 (or power) between T2-seen (no-blink) and T2-unseen (blink) trials. Time-frequency  
222 representations of power and phase were calculated for each trial using sliding  
223 Hanning tapers with an adaptive time window of two cycles for each frequency (1-30  
224 Hz in logarithmically spaced steps). This approach has been used often in previous  
225 studies (e.g., (Mazaheri et al., 2009)). Time-frequency representations were computed  
226 separately for each condition (blink, no-blink), interval (short, long), contact point  
227 (left, right), and participant, and baseline corrected using the time window -500 to -  
228 200ms pre-T1.

229

### 230 **Statistics**

231 Given the relatively small number of patients, we utilized a nonparametric  
232 permutation procedure used in previous studies to assess statistical significance of the  
233 time-frequency data (Cohen et al., 2009). For the time-frequency data, we first  
234 randomly relabelled T2-unseen and T2-seen trials and created 1000 permuted  
235 difference maps of power. Next, at each time-frequency point, the true observed  
236 difference between T2-unseen and T2-seen trials was subtracted from the mean  
237 average permuted difference and divided by the standard deviation of the permuted  
238 differences to obtain a *Z* value. This *z*-value reflects the standardized distance away  
239 from the distribution of time-frequency power expected by chance. We chose a *Z*-  
240 value corresponding to a *p*-value of 0.05 as the threshold for significance, and a  
241 cluster size of 25 time-frequency pixels. In case of a significant effect, post hoc  
242 analyses were conducted to determine if effects were driven by changes in T2-seen or  
243 T2-unseen trials by comparing mean activity in the significant time-frequency cluster

244 in T2-seen (or T2-unseen) trials to mean baseline activity in a corresponding  
245 frequency range in T2-seen (or T2-unseen) trials.

246

## 247 **RESULTS**

248 Patients displayed a robust AB: they more often failed to perceive T2 when it  
249 followed T1 after 200 than after 800 ms (Fig. 2;  $t(6)=4.0$ ,  $p=.007$ ). Comparable to  
250 previous studies with healthy adults, T1 accuracy was high — on average 85% —  
251 regardless of the interval between the two targets ( $t(6)=0.04$ ,  $p=.972$ ).

252 To examine the role of the ventral striatum in the AB, our primary analysis  
253 focused on the neural differences in target-induced oscillatory activity (or power)  
254 between trials in which T1 and T2 were both consciously perceived (T2-seen trials)  
255 and trials in which T1 was seen, but T2 was missed (T2-unseen trials). In line with a  
256 contribution of the ventral striatum to the AB, this analysis revealed differences in  
257 both T1 and T2 processing between trials in which T2 was seen vs. missed (Fig. 3 and  
258 4). First, the AB was foreshadowed by short-latency differences in T1 processing  
259 between T2-unseen and T2-seen trials. Specifically, the imminent failure to perceive  
260 T2 was signaled by a T1-induced transient burst of activity in the alpha and low beta  
261 range (8-16 Hz) occurring as early as 80ms after the first target (80-140ms) in the left  
262 ventral striatum (Fig. 3). Only in trials in which T2 was missed, did T1 induce this  
263 short-latency increase in alpha/low beta activity (Fig. 3b and 4). Post hoc analyses  
264 confirmed that this early response was only induced by T1 in T2-unseen trials ( $t(6)=$   
265  $2.75$ ,  $p=.055$ ), not in T2-seen trials ( $t(6)= 0.55$ ,  $p=.59$ ). This effect was not observed  
266 for the right ventral striatum.

267 Second, conscious T2 perception was associated with an increase in theta (4-  
268 8Hz) oscillatory activity between around 150 and 400ms in both the left and right

269 ventral striatum (Fig. 3). For the left ventral striatum, it can clearly be seen that when  
270 both targets were consciously perceived (T2-seen trials), this increase in theta activity  
271 was observed between 200 and 400ms after each target onset (Fig. 4b). However,  
272 when only T1 was seen (T2-unseen trials), only the first increase in theta activity was  
273 present (Fig. 4a). Of further note, when T2 followed T1 after a long interval, the  
274 second increase in theta activity was correspondingly shifted in time (Fig. 4c),  
275 confirming that the second increase in theta power observed in short-interval T2-seen  
276 trials is related to (conscious) T2 processing. The difference in T2-induced theta  
277 power between T2-seen and T2-unseen trials was significant between 215 and 400 ms  
278 post-T2 in the left ventral striatum and between 150 and 280 ms in the right ventral  
279 striatum (Fig. 3). Post hoc analyses confirmed that the theta response to T2 was only  
280 significantly increased from baseline in the left ventral striatum in trials in which T2  
281 was seen ( $t(6)=2.64$ ,  $p=.05$ ), and not in trials in which T2 was not seen ( $t(6)=1.05$ ,  
282  $p=.33$ ) (see Fig. 3b). However, in the right ventral striatum, the increase in theta  
283 activity to T2 was not significantly different from baseline in T2-seen trials  
284 ( $t(6)=1.87$ ,  $p=.1$ ), indicating that the effect was less robust in the right hemisphere.  
285 Thus, only consciously perceived T2's were signaled by theta oscillatory activity in  
286 the ventral striatum. This effect was also visible at the two most dorsal contact points  
287 (3 and 4) in the left ventral ALIC (data not shown).

288         Lastly, conscious T2 perception was associated with a greater increase in beta  
289 band (15-30Hz) activity in T2-seen vs. T2-unseen trials between 210 and 260ms post-  
290 T2 in the right ventral striatum (Fig. 3). This beta increase was not significantly  
291 increased from baseline in either condition (T2-seen trials:  $t(6)=1.84$ ,  $p=.2$ ; T2-unseen  
292 trials:  $t(6)=0.18$ ,  $p=.86$ ) (Fig. 3b and 4).

293            Thus, the attentional blink was associated with differences in both T1 and T2  
294 processing; T1 processing influenced the ability to consciously perceive T2, as  
295 indicated both by stronger alpha/beta activity between 80-140ms post-T1 in the left  
296 ventral striatum. Moreover, only perceived T2's elicited a theta response between 150  
297 and 280ms in the right ventral striatum and 200-400ms in the left ventral striatum, and  
298 a transient increase in beta activity between 210-260ms in the right ventral striatum.

299            As T1 could have one of two colors (red or green), we ran a control analysis to  
300 ensure that observed differences in T1 processing between T2-seen and T2-unseen  
301 trials could not be accounted for by differences in the relative contribution of red T1  
302 vs. green T1 trials between trial types. Specifically, we ran an additional repeated  
303 measures ANOVA with T1 color and Interval (short vs. long) as within-subject  
304 variables. This analysis importantly showed that T2 detection was not affected by T1  
305 color (no main effect of T1 color:  $F(1,6)=0.012$ ;  $p=.915$ ), nor was AB size (no  
306 interaction between T1 color and Interval:  $F(1,6)=0.336$ ;  $p=.583$ ), excluding this  
307 possibility.

308

### 309 **DISCUSSION**

310 This study aimed to shed more light on the role of the ventral striatum in conscious  
311 perception. We found that conscious T2 perception was not only reflected in, but also  
312 influenced by ventral striatal activity in that responses to T1 foreshadowed the AB to  
313 T2. Specifically, only in T2-unseen trials, T1 elicited a short-latency (80-140ms)  
314 increase in alpha and lower beta activity (8-16Hz) in the left ventral striatum. This  
315 novel finding suggests that the AB to T2 is determined by T1 processing at a much  
316 earlier processing stage than commonly assumed (Dux and Marois, 2009; Martens  
317 and Wyble, 2010). Our second main finding was that only consciously perceived T2s

318 were associated with an increase in theta activity between 215-400ms and 150-280ms  
319 in the left and right ventral striatum, respectively, as well as with transient beta-band  
320 activity between 210-260ms in the right ventral striatum. Thus, we also observed  
321 signals related to conscious experience in the ventral striatum prior to, or in the time  
322 range in which the global network of fronto-parietal regions implicated in  
323 consciousness is activated (Sergent et al., 2005; Dehaene and Changeux, 2011).  
324 Together, these findings suggest that the ventral striatum may contribute to conscious  
325 perception, and provide first insight into the time course of its contributions. They  
326 may also shed novel light on the mechanisms that give rise to one of the most studied  
327 phenomena in the consciousness literature: the attentional blink.

328         Strikingly, the AB was associated with differences in T1 processing as early as  
329 80ms post-T1, i.e., much earlier than previously shown with scalp-EEG (Sergent et  
330 al., 2005; Slagter et al., 2007). Specifically, this work has shown that in trials in which  
331 T2 goes undetected, the T1-elicited P3b, a brain potential with a latency of about 300-  
332 400ms, is delayed or larger in amplitude (Sergent et al., 2005; Slagter et al., 2007). As  
333 the P3b has been linked to several, albeit related, cognitive functions, including  
334 working memory updating (Donchin, 1981), event categorization (Kok, 2001), and  
335 decision making (Twomey et al., 2015), these findings have been taken as support for  
336 major AB theories that propose that T1 encoding renders some mechanism  
337 unavailable for T2 processing until T1 encoding is completed, and thus, that the AB is  
338 related to some later-stage information processing bottleneck (Duncan et al., 1994;  
339 Chun and Potter, 1995; Bowman and Wyble, 2007; Marti et al., 2015; for reviews, see  
340 Dux and Marois, 2009; Martens and Wyble, 2010). Yet, our findings relate the AB to  
341 differences in T1 processing starting at 80ms. This could suggest that T1  
342 consolidation starts much earlier, as soon as sufficient evidence has been collected.

343 Alternatively, the AB may be affected by T1 processing at a much earlier processing  
344 stage. Notably, animal work has shown that the ventral striatum responds to salient  
345 stimuli within 100ms. This response is thought to reflect a signal to frontal regions to  
346 orient attention to potentially relevant information for further processing (Redgrave et  
347 al., 1999b; Horvitz, 2000; Overton et al., 2014). Although this warrants further  
348 research, the short-latency increase in alpha/beta activity may thus reflect an early  
349 alerting signal triggered by a salient stimulus, the strength of which determines the  
350 extent of higher-order stimulus processing, and ability of the brain to pick up on a  
351 subsequently presented target stimulus (Slagter et al., 2009). This possibility receives  
352 initial support from a study in which early evoked activity (80-180ms) in the nucleus  
353 accumbens related to deviancy detection predicted the amplitude of the scalp P3b  
354 (Dürschmid et al., 2015). Yet, future studies that manipulate e.g., T1 saliency, are  
355 necessary to draw firm conclusions regarding the functional significance of our early  
356 alpha/beta effect.

357 Intriguingly, the short-latency difference in T1 processing was observed in the  
358 ventral striatum. This raises the question how the ventral striatum, which does not  
359 directly receive visual input, can in some trials “know” so quickly after T1, which  
360 differed from the distractors in color and shape/identity, that a salient stimulus has  
361 been presented. Animal research shows that short-latency visual information is  
362 provided by the superior colliculus to dopaminergic neurons, which in turn project to  
363 the striatum (Overton et al., 2014). Yet, the superior colliculus is relatively primitive,  
364 and allegedly not capable of distinguishing between colors (McPeck and Keller,  
365 2002) or complex visual stimuli (Schiller and Koerner, 1971; Goldberg and Wurtz,  
366 1972), making it unlikely that the here observed short-latency response reflects input  
367 from this region. At a slightly longer latency (after ~150ms), the ventral striatum can



368 receive the results of complex stimulus processing via a recently discovered route by  
369 which cortical areas project to dopaminergic neurons via the superior colliculus  
370 (Overton et al., 2014). Yet, the alpha/beta effect occurred earlier. While the ventral  
371 striatum also receives visual information via the thalamus, this is primarily from  
372 higher-order thalamic nuclei, which receive little direct sensory input. Therefore, our  
373 findings - which indicate very short-latency information processing in the ventral  
374 striatum dependent on non-spatial visual features – cannot easily be explained by  
375 existing ideas about subcortical visual processing and warrant further research.  
376 Moreover, the fact that the early alpha/beta effect was only triggered by T1 in T2-  
377 unseen trials further suggests that it does not simply reflect sensory T1 processing.  
378 This observation also raises the question as to why a physically identical T1 only  
379 elicited this response in T2-unseen, but not T2-seen trials. Several studies have shown  
380 that differences in attentional state can influence T1 processing and AB magnitude  
381 (Olivers and Nieuwenhuis, 2005; Slagter et al., 2007; van Vugt and Slagter, 2014),  
382 raising the possibility that trial-to-trial fluctuations in attentional state may have  
383 contributed to observed effects. Notably, alpha and beta oscillations have been  
384 specifically related to top-down, feedback-related processing (Bastos et al., 2015).  
385 Thus, although speculative, the extent to which T1 captured attention or placed  
386 demands on consolidation processes may have depended on pre-target attentional  
387 state. Alternatively, differences in bottom-up T1 strength across trials, caused by  
388 variability across trials in the specific target-distracter sequence and hence distractor  
389 masking, may have also influenced T1 processing (Bowman and Wyble, 2007).  
390 Future studies that manipulate attentional state and T1 saliency are necessary to  
391 determine the functional significance of our early alpha/beta effect. Nevertheless, our  
392 findings indicate that the AB is related to T1 processing at a much earlier processing

393 stage than is commonly assumed, at the subcortical level. They may also extend  
394 recent findings that indicate that while visual consciousness and endogenous (top-  
395 down) attention may be neurally dissociated (Koch and Tsuchiya, 2007; Wyart and  
396 Tallon-Baudry, 2008), exogenous spatial attention (Chica and Bartolomeo, 2012) and  
397 phasic alertness (Kusnir et al., 2011) are important antecedents of conscious  
398 experience and facilitate conscious access by calling upon fronto-parietal networks to  
399 orient attention.

400           Importantly, striatal responses to T1 not only prevented conscious access to  
401 T2, conscious T2 perception was also signaled by striatal activity in the theta band.  
402 This is notable as most theories of consciousness focus exclusively on the cortex  
403 and/or thalamo-cortical interactions (Rees et al., 2002; Haynes, 2009; Dehaene and  
404 Changeux, 2011; Lau and Rosenthal, 2011; Aru et al., 2012; van Gaal and Lamme,  
405 2012), and are agnostic on striatal contributions. Theta oscillations are associated with  
406 the active intake of sensory stimuli (Bastos et al., 2015), but are also observed in  
407 structures further down the processing stream, including the hippocampus (Colgin,  
408 2013). Hippocampal-generated theta oscillations are key in setting the dynamics for  
409 memory encoding and retrieval within cortical circuits (Hasselmo and Stern, 2014).  
410 As the ventral striatum allegedly does not generate theta oscillations itself (van der  
411 Meer and Redish, 2011) and intracranial EEG measures local field potentials (i.e.,  
412 neuronal input), the observed T2 detection-related theta response may thus reflect  
413 hippocampal-dependent processes. The nucleus accumbens, which also receives  
414 inputs from the amygdala, prefrontal cortex and VTA, projects to both the  
415 mediodorsal nucleus of the thalamus (MDT) and the thalamic nucleus reticularis  
416 (TNR), which in turn project to the prefrontal cortex. The nucleus accumbens is thus  
417 hence well positioned to integrate information from different sources and select which

418 information is broadcasted through the brain (via the TNR (Scheibel, 1980)) or  
419 selected for sustained cortical representation (via the MDT) (Goto and Grace, 2008).  
420 This is significant as several influential theories propose that consciousness is related  
421 to the ‘broadcasting’ of sensory information to the whole brain and that  
422 thalamocortical circuits serve as an important mediator of such broadcasting  
423 (Newman and Baars, 1993; Crick, 1995; Tononi and Edelman, 1998; Baars, 2005;  
424 Dehaene and Changeux, 2005, 2011). Future studies are necessary to determine how  
425 the striatum may precisely influence thalamocortical interactions and conscious  
426 perception.

427         As to the T2 perception-related increase in beta activity, a recent iEEG study  
428 reported reduced beta-band activity in the ventral striatum at a similar latency to rare  
429 versus frequent scene images (Zaehle et al., 2013), suggesting that beta-band activity  
430 may signal contextual deviancy. Yet, we observed an increase in beta activity to  
431 perceived T2s. This discrepancy in findings might relate to differences in the specific  
432 task or reference procedure used.

433         Our results extend findings from neuroimaging studies (Slagter et al., 2012;  
434 Van Opstal et al., 2014; Christensen et al., 2006; Slagter et al., 2010; Bisenius et al.,  
435 2015; Chica et al., 2016) by revealing the time course of conscious perception-related  
436 striatal activity. Importantly, some of these studies used simple backward masking  
437 tasks with only one stimulus (Christensen et al., 2006; Van Opstal et al., 2014;  
438 Bisenius et al., 2015), in which conscious access is not dependent on attentional  
439 selection (Dehaene and Changeux, 2011) as in the AB task. Yet, many fMRI studies  
440 did not report conscious-related activity in the striatum. Possibly, fMRI does not  
441 provide a sensitive measure of consciousness-related activity in the basal ganglia due  
442 to the nature of the blood-oxygen-level dependent (BOLD) response; BOLD activity

443 mostly reflects local field potential activity between 20-60Hz (Goense and Logothetis,  
444 2008) and correlates negatively with alpha power (Laufs et al., 2003; Scheeringa et  
445 al., 2011). Low-frequency oscillatory activity, as observed here, may hence not be  
446 reflected in increased BOLD activity or even result in decreased BOLD activity.  
447 Combined with the fact that basal ganglia activity cannot be measured with scalp-  
448 EEG (Cohen et al., 2011), this might explain the exclusion of a striatal contribution in  
449 many theories of consciousness. Yet, the striatum is well positioned to gate cortical  
450 information flow and integrate the massively parallel and distributed information  
451 capacity of the cerebral cortex into the limited-capacity, sequential mode of operation  
452 required to form a coherent percept of our environment. Albeit speculative, our  
453 observations may provide initial support this idea.

454         A role for striatum-dependent gating mechanisms in the AB fits with  
455 influential theories that attribute the AB to dysfunctional gating of information (Di  
456 Lollo et al., 2005; Bowman and Wyble, 2007; Olivers and Meeter, 2008). For  
457 example, in the episodic Simultaneous Type Serial Token model (Bowman & Wyble,  
458 2007), target perception critically relies on the capacity to rapidly detect that a  
459 stimulus is salient when a fleeting representation of it arises amongst (temporally)  
460 competing stimuli, and the ability to sustain this representation through transient  
461 attentional enhancement for memory encoding. In this model, ongoing consolidation  
462 of T1 has an inhibitory effect on T2 attentional selection, resulting in impaired T2  
463 detection. According to another influential theory (Olivers and Meeter, 2008), the AB  
464 reflects the workings of a rapidly responding gating system. Specifically, T1 elicits  
465 transient excitatory feedback activity within 100ms meant to provide access to  
466 working memory. However, accidentally, the subsequent post-T1 distracter is  
467 'boosted', resulting in a strong inhibitory feedback response, which closes the gate to

468 working memory for T2. This theory actually postulated a possible role for the basal  
469 ganglia in the AB.

470         Although behaviorally, our mixed-patient data are very similar to typical  
471 findings in healthy adults, the question remains how our findings translate to the  
472 healthy human brain. Answering this question requires the development of methods  
473 that can noninvasively measure subcortical activity with high temporal precision in  
474 healthy humans.

475         To conclude, our results shed new light on the mechanisms that give rise to the  
476 attentional blink, by revealing that the AB may be due to very early T1-driven  
477 attentional capture. More generally, by showing that conscious perception is  
478 modulated by ventral striatal activity, they suggest that the neural mechanisms  
479 underlying conscious access may not be confined to the thalamo-cortical complex  
480 alone.

481

482

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- 691
- 692

693 **FIGURE LEGENDS**

694

695 **Figure 1** (a) Schematic illustration of deep-brain electrodes in the ventral striatum. The core  
696 of the nucleus accumbens (NAc) is indicated in red. Adapted from (Figuee et al., 2013). (b)  
697 The attentional blink (AB) task. Subjects had to detect two targets (T1 and T2; two numbers)  
698 in a rapid stream of distractor stimuli. Shown is an example of a short T1-T2 interval trial.

699

700 **Figure 2** Attentional blink task performance. Percentage (%) T1 accuracy (left panel) and  
701 percentage T2 accurate given T1 correct (T2/T1) (right panel) are shown separately for Short  
702 (200ms) and Long (800ms) T1-T2 interval trials, for each patient (lines) and at the group  
703 level (bars). As can be seen, a robust AB was observed as reflected by lower T2/T1 accuracy  
704 in short interval compared to long interval trials. Error bars show the standard error of the  
705 mean.

706

707 **Figure 3** The AB is predicted by, and reflected in ventral striatal activity. Shown are  
708 intracranial EEG data from the left and right ventral striatum. **A:** Time-frequency  
709 representations show z-values reflecting the strength of differences in striatal activity between  
710 T2-seen and T2-unseen short-interval trials. Time-frequency windows where the difference  
711 reached significance are highlighted in black. Average power (in dB) within these significant  
712 windows is shown in **B** separately for T2-unseen and T2-seen trials. The AB was associated  
713 with an early difference in T1-induced alpha and low beta activity (8-16) between 80-140ms  
714 post-T1 in the left ventral striatum. Moreover, conscious T2 perception was associated with  
715 an increase in theta activity (4-8Hz) between 215-400ms post-T2 in the left ventral striatum  
716 and between 150-280ms post-T2 in the right ventral striatum. Finally, in the right ventral  
717 striatum, perceived T2's elicited greater activity in the beta band (15-30Hz) activity between  
718 210 and 260ms post-T2 than T2's that went undetected. Thus, the AB to T2 was

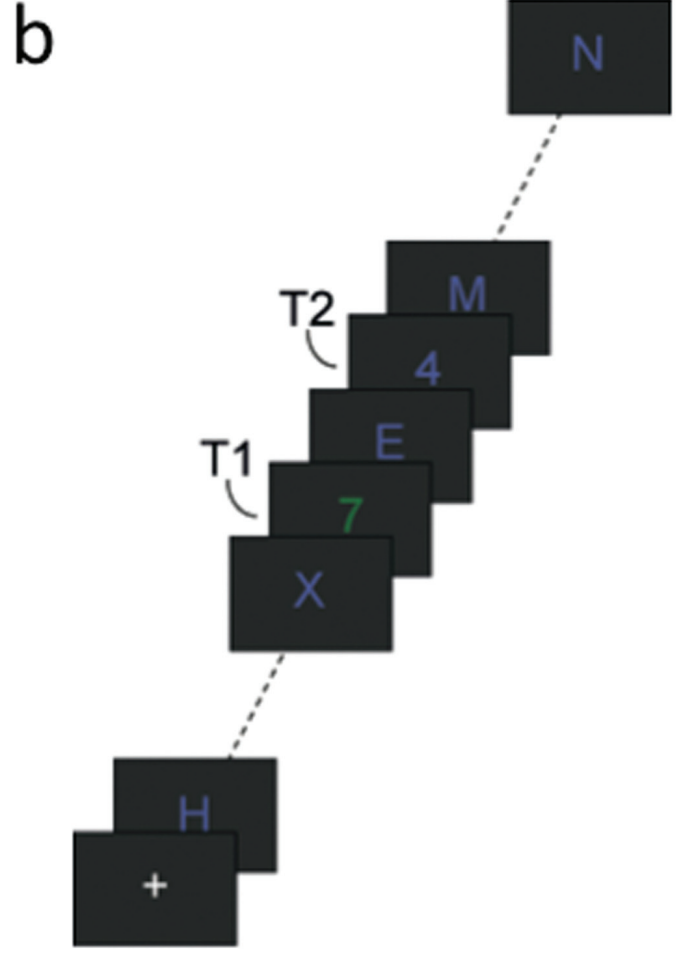
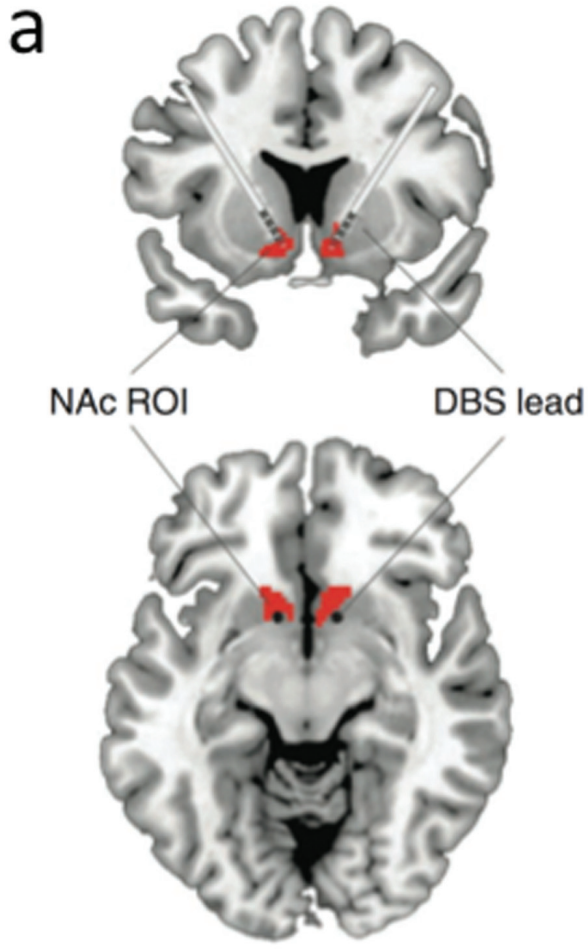
719 foreshadowed by a short-latency response to T1 in the left ventral striatum, and conscious T2  
720 perception was signaled by longer-latency ventral striatal activity in the theta and beta band.

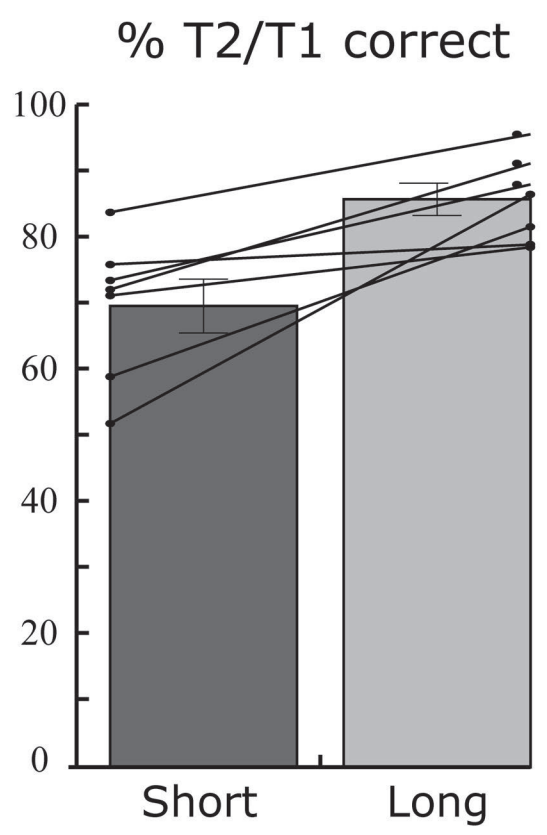
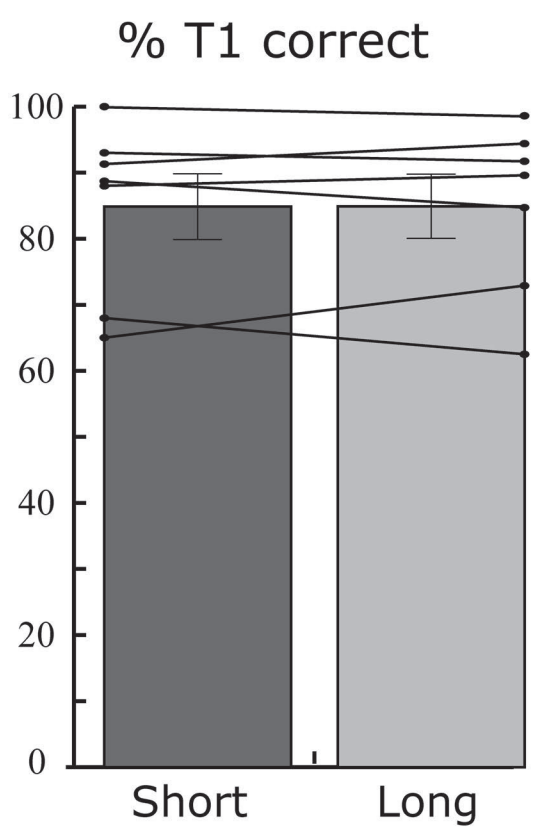
721

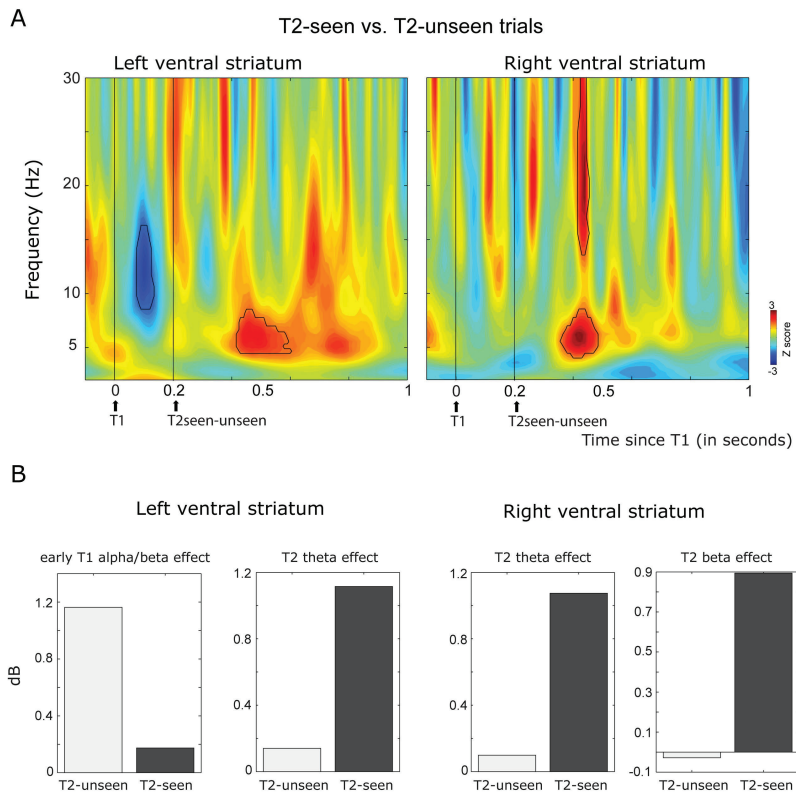
722 **Figure 4** The failure to perceive T2 is foreshadowed by early (80-140ms) T1 processing in  
723 the ventral striatum and conscious T2 target perception is associated with later (200-400ms)  
724 ventral striatal activity. Shown are intracranial EEG data from the left (left figures) and right  
725 (right figures) ventral striatum. Time-frequency representations show oscillatory activity  
726 induced by T1 and T2 in short-interval T2-unseen (top panel), short-interval T2-seen (middle  
727 panel), and long-interval T2-seen (bottom panel) trials. In trials in which T2 was not seen, T1  
728 induced a strong increase in alpha and low beta oscillatory activity (8-16) between 80-140ms  
729 post-T1 in the left ventral striatum. Moreover, in the left ventral striatum, when T2 was  
730 consciously perceived, an increase in theta oscillatory activity (4-8Hz) was observed 215-  
731 400ms after each target onset. However, when only T1 was seen, only the first increase in  
732 theta activity was present. Moreover, the second increase in theta activity is shifted in time  
733 with the presentation of T2 in long-interval trials, confirming that is related to conscious  
734 perception of T2. Thus, only consciously perceived targets were signaled by theta oscillatory  
735 activity between 200-400ms in particular in the left ventral striatum. Finally, conscious T2  
736 perception was associated with a transient increase in beta band activity (15-30Hz) in the  
737 right ventral striatum 210-260ms post-T2.

738

739





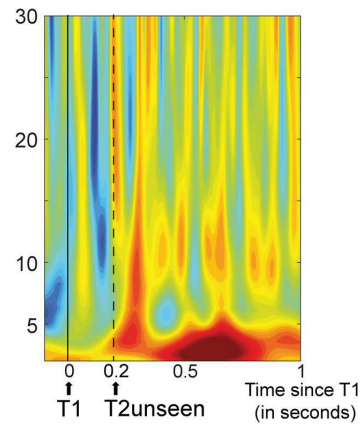
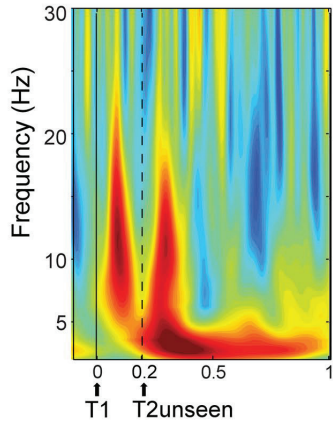




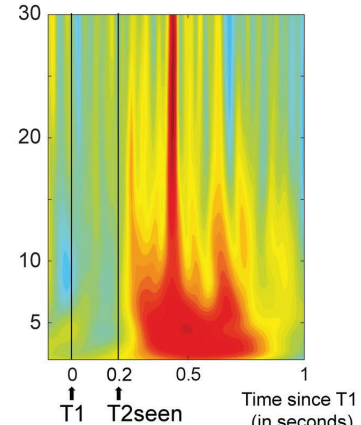
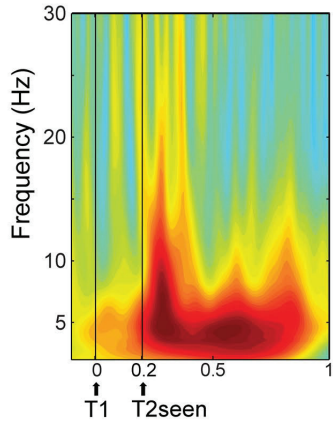
Left ventral striatum

Right ventral striatum

T2-unseen trials (short)



T2-seen trials (short)



T2-unseen trials (long)

