Consciousness and Cognition xxx (2015) xxx–xxx

Contents lists available at ScienceDirect

Consciousness and Cognition

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

Open and closed cortico-subcortical loops: A neuro-computational account of access to consciousness in the distractor-induced blindness paradigm ☆

Christian Ebner [a,b,1], Henning Schroll [a,c,d,e,1], Gesche Winther [f], Michael Niedeggen [f], Fred H. Hamker [a,c,*]

[a] Computer Science, Chemnitz University of Technology, 09111 Chemnitz, Germany
[b] Goethe University Frankfurt am Main, Frankfurt am Main, Germany
[c] Bernstein Center for Computational Neuroscience, Charité – Universitätsmedizin Berlin, Berlin, Germany
[d] Neurology, Charité – Universitätsmedizin Berlin, Berlin, Germany
[e] Psychology, Humboldt Universität zu Berlin, Berlin, Germany
[f] Experimental Psychology and Neuropsychology, Freie Universität Berlin, Berlin, Germany

ARTICLE INFO

Article history:
Received 28 October 2014
Revised 15 February 2015
Accepted 16 February 2015
Available online xxxx

Keywords:
Conscious access
Computational model
Basal ganglia
Global workspace theory
Cognitive control

ABSTRACT

How the brain decides which information to process 'consciously' has been debated over for decades without a simple explanation at hand. While most experiments manipulate the perceptual energy of presented stimuli, the distractor-induced blindness task is a prototypical paradigm to investigate gating of information into consciousness without or with only minor visual manipulation. In this paradigm, subjects are asked to report intervals of coherent dot motion in a rapid serial visual presentation (RSVP) stream, whenever these are preceded by a particular color stimulus in a different RSVP stream. If distractors (i.e., intervals of coherent dot motion prior to the color stimulus) are shown, subjects' abilities to perceive and report intervals of target dot motion decrease, particularly with short delays between intervals of target color and target motion.

We propose a biologically plausible neuro-computational model of how the brain controls access to consciousness to explain how distractor-induced blindness originates from information processing in the cortex and basal ganglia. The model suggests that conscious perception requires reverberation of activity in cortico-subcortical loops and that basal-ganglia pathways can either allow or inhibit this reverberation. In the distractor-induced blindness paradigm, inadequate distractor-induced response tendencies are suppressed by the inhibitory 'hyperdirect' pathway of the basal ganglia. If a target follows such a distractor closely, temporal aftereffects of distractor suppression prevent target identification. The model reproduces experimental data on how delays between target color and target motion affect the probability of target detection.

☆ This article is part of a special issue of this journal on Exploring the Visual (Un)conscious.

* Corresponding author at: Department of Computer Science, Chemnitz University of Technology, Straße der Nationen 62, 09111 Chemnitz, Germany. Fax: +49 371 53125739.
E-mail address: fred.hamker@informatik.tu-chemnitz.de (F.H. Hamker).

[1] C.E. and H.S. contributed equally.

http://dx.doi.org/10.1016/j.concog.2015.02.007
1053-8100/

1. Introduction

The global workspace theory proposes that only a subset of stimuli available in the outside world enter consciousness to become globally available for task-control processes (Baars, 1988). In this view, the 'global workspace' refers to a high-level processing and storage system that allows for the interaction of different specialized brain areas (cf. Baars, 2005). But how are visual stimuli gated into this processing and storage system? In a model proposed by Dehaene, Sergent, and Changeux (2003), the amount of stimulus activation is the critical factor. Their model explains why salient, well visible or attended stimuli are particularly amenable to becoming consciously available. Cognitive influences on conscious access, in contrast, cannot be explained by this model.

The neuronal determinants of stimulus-driven and cognitive influences on access to consciousness can be measured through distinct neuro-psychological paradigms. Stimulus-driven influences, for instance, are prominent in the attentional blink paradigm, where subjects are asked to detect target stimuli in a rapid serial visual presentation (RSVP) stream. In this paradigm, targets that follow previous targets by about 180–450 ms cannot be reliably detected by subjects (Raymond, Shapiro, & Arnell, 1992). Cognitive influences on access to consciousness, in contrast, have been investigated using the distractor-induced blindness paradigm (Niedeggen, Michael, & Hesselmann, 2012; Sahraie, Milders, & Niedeggen, 2001). In this paradigm, two separate RSVP streams are presented. In a global RSVP stream containing target two (T2) visual stimuli are presented in an annulus centered on a fixation point. In a local stream, presented in the center of the global stream, target one (T1) is included. By instruction, T2-like stimuli that occur after T1 presentation are to be detected as targets, while T2-like stimuli presented before T1 serve as distractors. Effects of distractor-induced blindness (i.e., non-detection of targets because of previous distractors) can be observed if the time interval between T1 and T2 (stimulus onset asynchrony, SOA) is below approximately 300 ms (cf. Sahraie et al., 2001). This blindness effect does not rely on an interaction of the two targets, in contrast to the attentional blink paradigm (Shapiro, 1994), but on cognitive effects of target-like episodes (i.e., distractors) preceding the onset of T1 (Hesselmann, Niedeggen, Sahraie, & Milders, 2006; Sahraie et al., 2001): these target-like episodes have been shown to result in a frontal negativity, cumulatively inhibiting target perception, as shown in a recent event-related brain potential (ERP) study (Niedeggen et al., 2012). This top-down *inhibitory* effect of distractors, i.e., their power to decrease the probability of getting conscious access to T2, is a unique characteristic of distractor-induced blindness. The specific neuronal mechanisms of this inhibition, however, remain speculative.

Empirical studies have shown an involvement of the prefrontal cortex (PFC; Demerzti, Soddu, & Laureys, 2013), sensory cortical areas (for a review, see Rees, Kreiman, & Koch, 2002), cortico-thalamo-cortical loops (Demerzti et al., 2013; Llinás, Ribary, Contreras, & Pedroarena, 1998) and the basal ganglia (BG) in consciousness (e.g., Balkin et al., 2002; Christensen, Ramsøy, Lund, Madsen, & Rowe, 2006; Gray, 1995; Kjaer et al., 2002; Mhuircheartaigh et al., 2010; Palmiter, 2011). Overall, therefore, it appears likely that there exists a widespread cerebral substrate of consciousness, in which different structures may fulfill different functions. The BG in particular are likely to exert modulatory control over consciousness with their direct, indirect and hyperdirect pathways. This should not be understood as implying that the BG are necessary for phenomenal awareness. Rather, they can enhance, but also suppress activity within the thalamus and cortex via these pathways, potentially contributing to the distractor-induced *inhibition* of access to consciousness in the distractor-induced blindness paradigm.

We here focus on the involvement of cortico-BG-thalamic loops in conscious perception, extending on our previous proposal that the BG are fundamentally involved in making contents globally available by their control over thalamo-cortical loops (Trapp, Schroll, & Hamker, 2012). We implemented a novel neuro-computational model that explains how conscious perception may evolve from reverberation of activity in closed cortico-subcortical loops. The model further predicts that BG pathways can control access to these closed loops based on top-down context information, determining which pieces of information will be allowed to reverberate. In contrast to the model by Dehaene et al. (2003) outlined above, thereby, our model explains cognitive inhibitory influences on access to consciousness. Via interconnected open cortico-BG-thalamic loops, BG pathways are additionally proposed to determine how conscious perception influences response selection in the motor cortex.

2. Methods

2.1. Model architecture

Based on anatomical observations (Haber, 2003), the model consists of two interconnected cortico-BG-thalamic loops (Fig. 1): A closed 'frontal' loop determines which pieces of information become consciously available, while an open 'motor' loop determines response selection. Each modeled nucleus and cortical area contains a population of artificial neurons. These neurons are interconnected in accordance with anatomical evidence (cf. Bolam, Hanley, Booth, & Bevan, 2000) as presented in Fig. 2.

Inputs presented to the model activate input cortical neurons that represent visual or categorical features, while responses are recorded in the motor cortex. BG pathways connect input cortices to the closed frontal loop, where stimulus-induced activity may reverberate, i.e., cause recurrent self-excitation. The closed frontal loop comprises the PFC which also is the origin of an interconnected open motor loop, where stimulus-induced activity is processed towards response

Please cite this article in press as: Ebner, C., et al. Open and closed cortico-subcortical loops: A neuro-computational account of access to consciousness in the distractor-induced blindness paradigm. *Consciousness and Cognition* (2015), http://dx.doi.org/10.1016/j.concog.2015.02.007

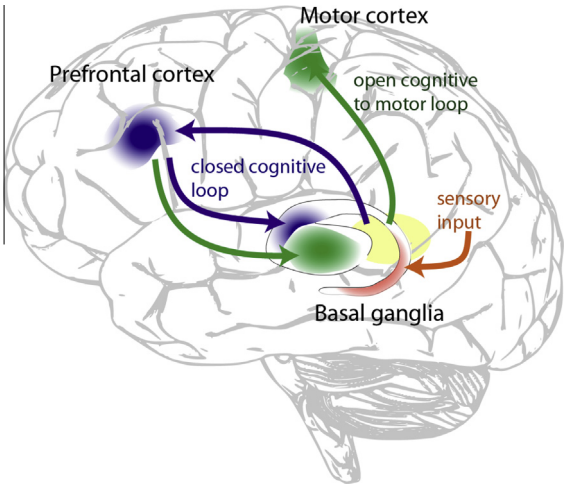

Fig. 1. Schematic overview of implemented cortico-BG-thalamic loops in the anatomical context of a human brain. Orange arrows depict sensory input to the BG from visual cortical areas. In the BG, these signals enter the closed frontal cortico-BG-thalamic loop (blue arrows) that contains the PFC. From the PFC, signals enter the open motor loop (green arrows) that proceeds via a different part of the BG to the motor cortex. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

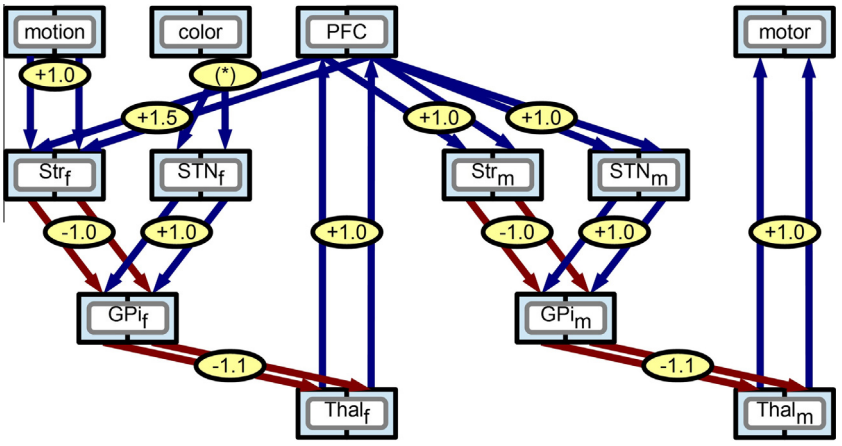

Fig. 2. Model architecture. Boxes signify anatomical structures, light blue squares within these boxes represent single neurons. Connections between these neurons are depicted by arrows, where excitatory connections are shown in blue, while inhibitory connections are shown in red. The strengths of connections are given in ellipses on top of arrows. Connections marked with asterisks are systematically varied in strength, as explained in the main text. Frontal-loop nuclei are marked with a subscript *f*, motor-loop nuclei with a subscript *m*. Abbreviations: GPi: internal segment of the globus pallidus; motor: motor cortex; PFC: prefrontal cortex; STN: subthalamic nucleus; Str: striatum; Thal: thalamus. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

selection and may eventually reach the motor cortex. Whenever accumulated activity of the population of motor-cortical neurons crosses a static response threshold within a given time interval, we assume a response to be performed.

Our model contains direct and hyperdirect pathways of the BG (Nambu, Tokuno, & Takada, 2002; cf. Schroll & Hamker, 2013). The direct pathway proceeds from the cortex to the internal segment of the globus pallidus (GPi) via the striatum. It inhibits tonically active GPi cells which thereby remove inhibition from the thalamus and cortex. In line with general agreement (cf. Schroll & Hamker, 2013), we assume the direct pathway to facilitate specific (cognitive or motor-related) cortical representations. The hyperdirect pathway connects the cortex to the GPi via the subthalamic nucleus (STN) and consists of excitatory connections only, thereby increasing the inhibition of the thalamus and cortex (Nambu et al., 2002). In agreement with previous modeling studies, we assume the hyperdirect pathway to globally inhibit responses or cognitive representations (cf. Schroll & Hamker, 2013). Two additional indirect pathways were left out from implementation in our model. A 'short' indirect pathway connects the cortex to the GPi via the striatum and the external segment of the globus pallidus (GPe; Smith, Bevan, Shink, & Bolam, 1998), while a longer indirect pathway additionally traverses the STN (Smith et al., 1998). Short and long indirect pathways have been suggested to inhibit cortical representations specifically and globally,

Please cite this article in press as: Ebner, C., et al. Open and closed cortico-subcortical loops: A neuro-computational account of access to consciousness in the distractor-induced blindness paradigm. *Consciousness and Cognition* (2015), http://dx.doi.org/10.1016/j.concog.2015.02.007

respectively (e.g., Frank, 2006; O'Reilly & Frank, 2006; Schroll & Hamker, 2013; Schroll, Vitay, & Hamker, 2014); their inhibition involves a relatively long latency due to a higher number of successive synaptic contacts than there exist in the hyperdirect BG pathway. In the distractor-induced blindness paradigm, such long latencies are maladaptive due to rapid serial visual presentation of stimuli. We therefore implemented the hyperdirect pathway instead of the indirect pathways for all inhibitory effects in our model.

To recapitulate, our model features a closed frontal loop that determines which information becomes consciously available and an open motor loop that determines which motor responses become selected given the present context. Both loops contain direct and hyperdirect BG pathways, where the direct pathway activates specific cortical representations (motor or cognitive), while the hyperdirect pathway globally suppresses cortical representations.

2.2. Paradigm

The distractor-induced blindness paradigm was first conducted by Sahraie et al. (2001). These authors simultaneously presented two separate RSVP streams to subjects. A local stream, a continuously changing color of the fixation point (10 Hz), was shown in the center of a stimulus display, while a global dot motion stream was shown in its periphery. Subjects were asked to detect an episode of coherent motion (100 ms) in the motion stream that occurred after a red stimulus was shown in the color stream. The delay between the red color target (T1) and the coherent motion target (T2), i.e., the stimulus onset asynchrony (SOA), was varied in this experiment. Short intervals of coherent dot motion before T1 (each of length 100 ms) served as distractors and had to be ignored. Sahraie et al. (2001) reported that subjects often missed T2 occurrences in trials with SOAs below 300 ms. In their paradigm, T2 occurrences had to be indicated at the ends of the respective trials.

When implementing this paradigm (Fig. 3), we did not feed the original color and motion stimuli into the model but used abstract cognitive representations of these stimuli as inputs. The color stream of the original paradigm was implemented as a target context stream, representing *distractor context* during distractor presentation and switching to *target context* with the onset of T1. The motion stream of the original paradigm was implemented as a target probing input, showing relevant T2 probes at different SOAs, but omitting continuous irrelevant stimuli (i.e., incoherent motion) at other times for simplification. Overall, thereby, we used cognitively pre-processed representations of color and motion stimuli as inputs to the model to better focus on the mechanisms of distractor-induced blindness. We further adapted the original paradigm in that each trial lasted for 2000 ms (Fig. 3) and in that motor responses (indicating T2 detection) could be performed directly after T2 presentation rather than at the end of each trial. The latter choice was made to avoid implementation of a temporal delay mechanism that prevents activity to spread into the motor loop before the trial is over. In line with the original paradigm, we simulated 60 trials per SOA condition (comprising SOAs of 0 to 700 ms in steps of 100 ms), resulting in a total of 480 trials.

2.3. Model details

In contrast to previous models by our group (e.g., Schroll, Vitay, & Hamker, 2012; Schroll et al., 2014; Vitay & Hamker, 2010), the model implemented here relies on hardwired connections between neurons that do not change via learning. This can be compared to a human subject familiar with the instructions of an experiment or after completed training.

Membrane potentials $\left(m_{j,t}^{post}\right)$ of single neurons are computed using

$$\tau_m^{post} \cdot \frac{dm_{j,t}^{post}}{dt} = \sum_{i \in pre} \left(w_{i,j}^{pre \to post} \cdot r_{i,t}^{pre} \right) + B_j^{post} + \varepsilon_{j,t}^{post} - m_{j,t}^{post}, \quad (1)$$

where τ_m^{post} is a time constant, $r_{i,t}^{pre}$ is the firing rate of the 'presynaptic' transmitting neuron i, $w_{i,j}^{pre \to post}$ the weight from the transmitting neuron i to the receiving 'postsynaptic' neuron j, B_j^{post} is the baseline membrane potential of postsynaptic layer

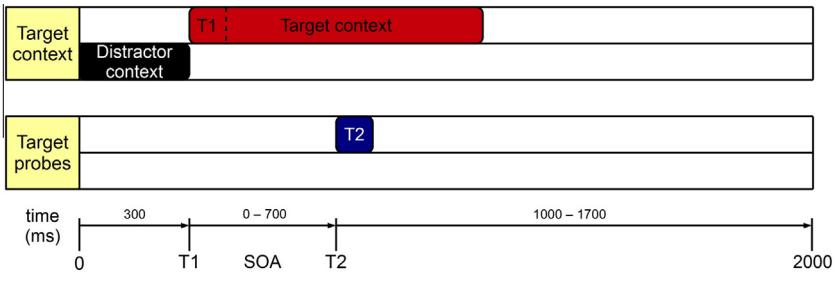

Fig. 3. Trial setup. Two inputs are simultaneously presented to the model. One is the target context as defined by the color stream, which switches from *distractor context* to *target context* as a consequence of the presentation of T1 (i.e., red). The other input includes target probes (T2s) as defined by the motion stream, where T2 (i.e., coherent motion) is presented between 0 and 700 ms after T1 onset (400 ms in this example), with a duration of 100 ms. Once the model's response threshold is crossed, it remains in an idle state until the trial is over, which is the case 2000 ms after trial onset. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Please cite this article in press as: Ebner, C., et al. Open and closed cortico-subcortical loops: A neuro-computational account of access to consciousness in the distractor-induced blindness paradigm. *Consciousness and Cognition* (2015), http://dx.doi.org/10.1016/j.concog.2015.02.007

post and $\varepsilon_{j,t}^{post}$ is a random noise term. In brief, Eq. (1) determines that a neuron's membrane potential converges to the sum of the neuron's inputs. For striatal neurons, membrane potentials are equipped with an additional normalization term to avoid excesses in striatal activity:

$$\tau_m^{post} \cdot \frac{dm_{j,t}^{post}}{dt} = \sum_{i \in pre} \left(w_{i,j}^{pre \to post} \cdot r_{i,t}^{pre} \right) + B_j^{post} + \varepsilon_{j,t}^{post} - m_{j,t}^{post} - m_{j,t}^{post} \cdot k \cdot \sum_{i \in pre} (w_{i,j}^{pre \to post} \cdot r_{i,t}^{pre}), \quad (2)$$

with $k = 0.8$.

Membrane potentials are converted into firing rates using

$$r_{j,t} = (m_{j,t})^{+}, \quad (3)$$

where $()^{+}$ sets negative values to zero.

All connection strengths $w_{i,j}^{pre \to post}$ of the model are displayed in Fig. 2. Connectivity between nuclei is implemented as based on anatomical evidence (cf. Bolam et al., 2000; Braak & Del Tredici, 2008), but focuses on direct and hyperdirect BG pathways as motivated in Section 2.1. In the interest of simplicity, most connection strengths were set to 1.0 for excitatory connections (blue arrows in Fig. 2) and to −1.0 for inhibitory connections (red arrows in Fig. 2). Three exceptions from this rule have to be noted. First, the connections from the cortical context layer to the frontal-loop STN were systematically varied in strength across simulations as motivated in Section 2.4. Second, the strength of the connections from PFC neurons to those of the frontal-loop striatum was set to 1.5, such that PFC activity may safely establish reverberating activity in the closed frontal loop independent of stimulus inputs. Third, the strength of the connections from GPi to thalamic neurons was set to −1.1 such that the GPi could safely inhibit the thalamus when both nuclei fired around their baseline rates, even in the presence of noise. The following paragraph describes how this specific wiring pattern affects signaling in the model.

The model's input signals are varied in strength (uniform distribution between 0.5 and 1.0) to account for noise in the system. Whenever target probe stimuli (T2s) are presented to the model, they are directly transmitted to the striatal parts of the frontal loop (direct pathway), where the first neuron encodes potential T2s (i.e., coherent motion) and the second neuron encodes non-T2s (i.e., incoherent motion). This pattern of representations is used for all simulated areas and nuclei of the model. For target context inputs as defined by T1, we assumed that the cortical neuron encoding *distractor context* provides input to both frontal-loop STN neurons (hyperdirect pathway) which may inhibit any activation of the GPi and subsequent nuclei. Potential influences of *target context* on the STN were left out for simplicity. The direct pathway, modulated by the hyperdirect pathway, forms a closed loop with the PFC.

For response selection, PFC signals are forwarded to the motor loop. While both T2s and non-T2s are transmitted to the motor-loop striatum which may subsequently activate the motor cortex, non-T2s additionally connect to the STN which will inhibit motor-cortex activity. Whenever motor-cortex activity, as accumulated over a particular time interval, reaches a threshold value r_{thresh}, we record a (physical) response of the model, which is true if

$$\sum_{t=1}^{300} \left(\sum_{j=1}^{n} \left(r_{j,t}^{motor} \right) \right) \geqslant r_{thresh}, \quad (4)$$

where n denotes the total number of motor neurons and r_{thresh} lies between 85 and 115 for different simulations, as specified in Section 3. When a response is detected, its identity is determined by the neuron that contributed most to the accumulated activity of the population. The accumulation process starts as soon as T2 is presented and lasts for 300 ms. After these 300 ms, a global 'reset pulse' is evoked in the frontal-loop STN for 300 ms which deletes any reverberating activity from the closed frontal loop.

2.4. Parameters that determine task performance

Three model parameters relevantly influence the model's performance on the paradigm outlined above. The first of these is the strength of the connection between the distractor context neuron and the frontal-loop STN ($w_{context \to STN}$). This connection belongs to the hyperdirect pathway of the frontal loop which is capable of globally suppressing PFC activity. The higher this parameter value, the stronger the hyperdirect pathway's suppression. The second parameter is the decay time constant of activity in input cortices ($\tau_{act,dec}$ represented by τ_m in Eq. (1)). This time constant determines that input cortical activity decreases only slowly after stimuli have disappeared in the outside world, thereby mimicking iconic memory (Dick, 1974; Sperling, 1960), which in turn affects how fast the rule for distractor suppression disappears. The third parameter is the accumulation threshold of the motor cortex, controlling how much motor-cortical activity is required for an overt response (r_{thresh} in Eq. (4)). On average, lower thresholds result in more trials in which a response is performed. r_{thresh} interacts with the other two factors: higher weights of the hyperdirect-pathway $w_{context \to STN}$ (stronger inhibition) and lower values of the decay constant $\tau_{act,dec}$ (faster decay of activity) require lower accumulation thresholds, if the same percentage of trials is supposed to result in physical responses. In order to illustrate network performance under different sets of parameter values, multiple simulations were run using deterministic variables, meaning that random variables (as used for neural noise and varying stimulus input strengths) took on the same values across parameter sets.

Please cite this article in press as: Ebner, C., et al. Open and closed cortico-subcortical loops: A neuro-computational account of access to consciousness in the distractor-induced blindness paradigm. *Consciousness and Cognition* (2015), http://dx.doi.org/10.1016/j.concog.2015.02.007

3. Results

3.1. Conscious perception and response selection in the model

We propose that BG pathways can control access to consciousness. Via these pathways, cortical distractor and target stimuli bias information processing in closed cortico-subcortical loops. Before we address distractor-induced blindness in particular, Fig. 4 shows our model's computations from cognitive to motor signals ($w_{context \rightarrow STN} = 0.50$, $\tau_{act,dec} = 50$ ms, SOA = 0 ms) when faced with the distractor-induced blindness paradigm.

Presentation of a non-target fixation color activates model inputs that encode the distractor context. These inputs increase the activities of both neurons of the frontal-loop STN (hyperdirect pathway) which suppresses activity in the closed frontal loop (Fig. 4). As T1 is presented, this suppression decays. Presentation of T2 (first trial depicted in Fig. 4) then excites the neuron encoding T2 in the frontal-loop striatum. The frontal-loop GPi integrates excitatory inputs from the STN and inhibitory inputs from the striatum. Whenever the amount of inhibition (direct pathway) exceeds the amount of excitation (hyperdirect pathway), GPi activity decreases below baseline and the GPi's inhibition of the PFC is (partially) removed. As a consequence, activity can reverberate in the closed frontal loop – which we assume to go along with conscious awareness of the corresponding information. The strength of activity in the PFC therefore provides a marker of access to consciousness, where weak activations prohibit further processing in the "global workspace". The open motor loop originates from the PFC and terminates in the motor cortex. Via this loop, the PFC transmits T2 information to the motor striatum which, via the GPi and thalamus, removes inhibition from the motor cortex and thereby elicits a physical response implying the model's decision "I saw T2".

To show how the model reacts to non-T2-like stimuli under comparable conditions, we presented such a non-T2-like stimulus under the same protocol (second trial in Fig. 4). This causes analogous reverberating activity in the frontal loop. In the motor loop, however, it results in high STN activity which cancels any below-baseline activity of the motor-loop

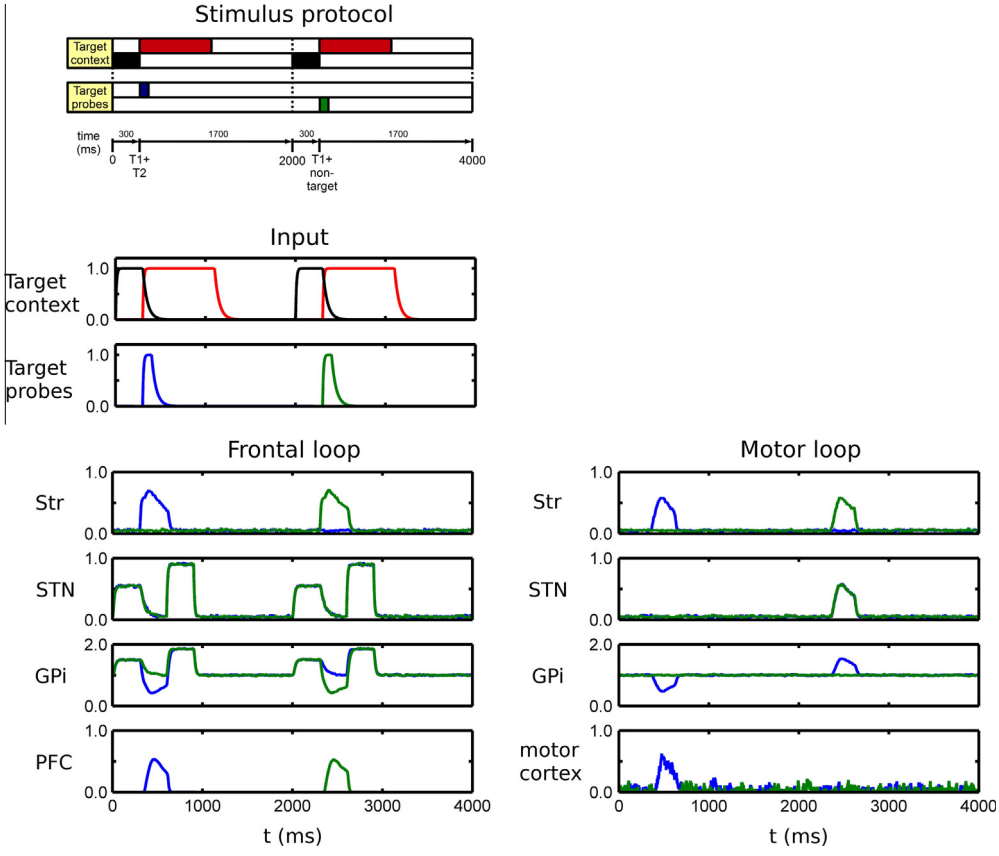

Fig. 4. Frontal and motor activity in the distractor-induced blindness task. Graphs depict the activities of frontal- and motor-loop nuclei for two separate trials, each lasting for 2000 ms with SOAs of 0 ms. The left column contains all nuclei of the frontal loop, the right column all nuclei of the motor loop. Blue lines depict the activities of neurons encoding potential T2 target stimuli (i.e., coherent motion), green lines the activities of neurons encoding non-target stimuli (i.e., incoherent motion). Black and red lines correspond to the activities of neurons encoding distractor and target contexts, respectively. Parameter settings for this example are $w_{context \rightarrow STN} = 0.5$ and $\tau_{act,dec} = 50$ ms. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Please cite this article in press as: Ebner, C., et al. Open and closed cortico-subcortical loops: A neuro-computational account of access to consciousness in the distractor-induced blindness paradigm. *Consciousness and Cognition* (2015), http://dx.doi.org/10.1016/j.concog.2015.02.007

GPi. As a result, non-T2-like stimuli do not elicit any motor response in the model which implies the decision "I did not see T2". Please note the memory reset pulse in the frontal-loop STN at about 600 ms after trial onset, resetting the closed loop and thereby clearing any memorized information.

3.2. Distractor-induced blindness in the model

Fig. 5 illustrates how distractor-induced blindness originates in the model. It occurs only for relatively high time constants of cortical activity decay $\tau_{act,dec}$ (right column of subplots in Fig. 5). For those time constants, the cortical neuron encoding the distractor context is still active for some time after T1 has appeared (removing the distractor context in favor of the target context). For short SOAs (i.e., short time intervals between T1 and T2 onset), then, cortical activity encoding the distractor context reaches into the time interval where T2 is presented. With it, the hyperdirect pathway remains active such that the activity of the frontal-loop GPi is still enhanced during T2 presentation. This increased GPi activity suppresses any reverberation of activity in the closed frontal loop which would otherwise be induced by T2 onset. By definition, any suppression of frontal-loop reverberation of T2-related activity results in reduced conscious awareness of this T2, i.e., in blindness of the T2.

3.3. Fitted networks reproduce human performance curves

In our model, three parameters relevantly influence task performance: the decay of activity in the input cortices ($\tau_{act,dec}$), the strength of the inhibitory hyperdirect pathway ($w_{context \rightarrow STN}$) and the response threshold of the motor cortex (r_{thresh}). We systematically varied the first two parameters to investigate how each of them influenced task performance for different SOA conditions; the third parameter was adjusted for each combination of the first two parameters such that all performance curves remained at the same height level, i.e. it shifts the overall performance curves equally for all SOAs. In order to

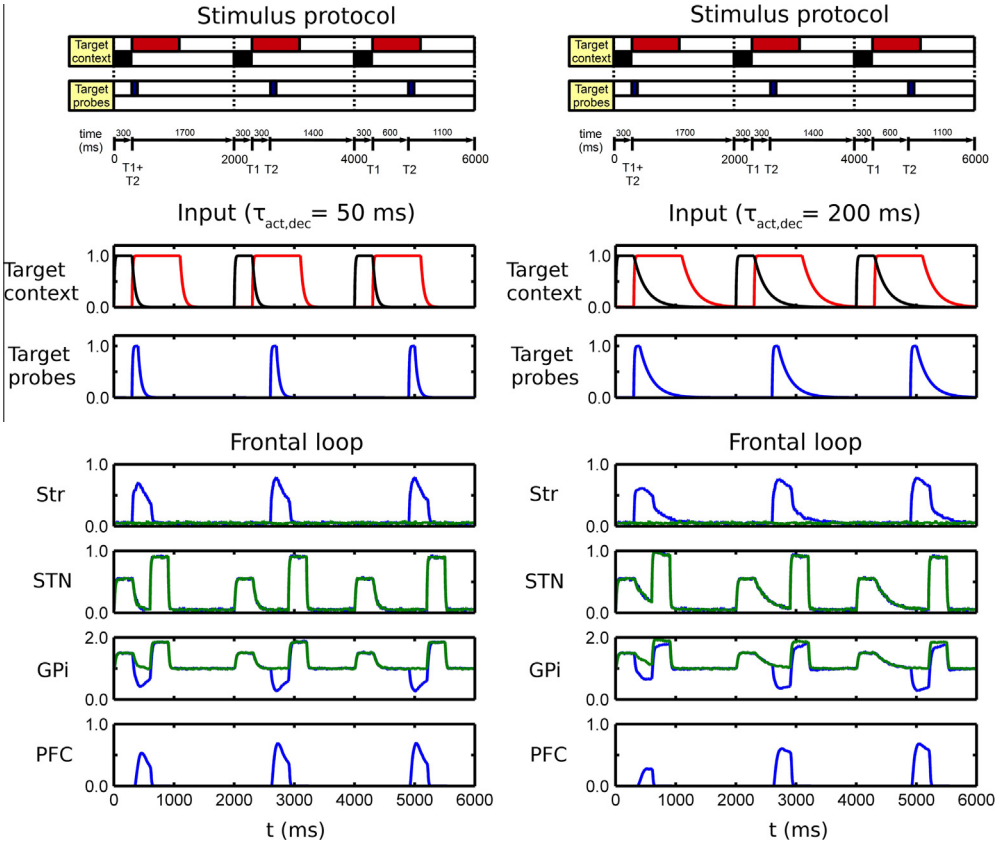

Fig. 5. Stimulus-induced activity in the model in the presence of distractor-induced blindness effects. Activities of frontal-loop nuclei are shown for time constants controlling cortical activity decay ($\tau_{act,dec}$) of 50 ms (left subplots) and 200 ms (right subplots) and for SOAs of 0, 300 and 600 ms (left, center and right trials within each subplot). Blue lines depict the activities of neurons encoding potential T2s (i.e., coherent motion), green lines the activities of neurons encoding non-T2s (i.e., incoherent motion). Black and red lines correspond to the activities of neurons encoding distractor and target contexts, respectively. $w_{context \rightarrow STN}$ in these simulations was set to 0.5. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Please cite this article in press as: Ebner, C., et al. Open and closed cortico-subcortical loops: A neuro-computational account of access to consciousness in the distractor-induced blindness paradigm. *Consciousness and Cognition* (2015), http://dx.doi.org/10.1016/j.concog.2015.02.007

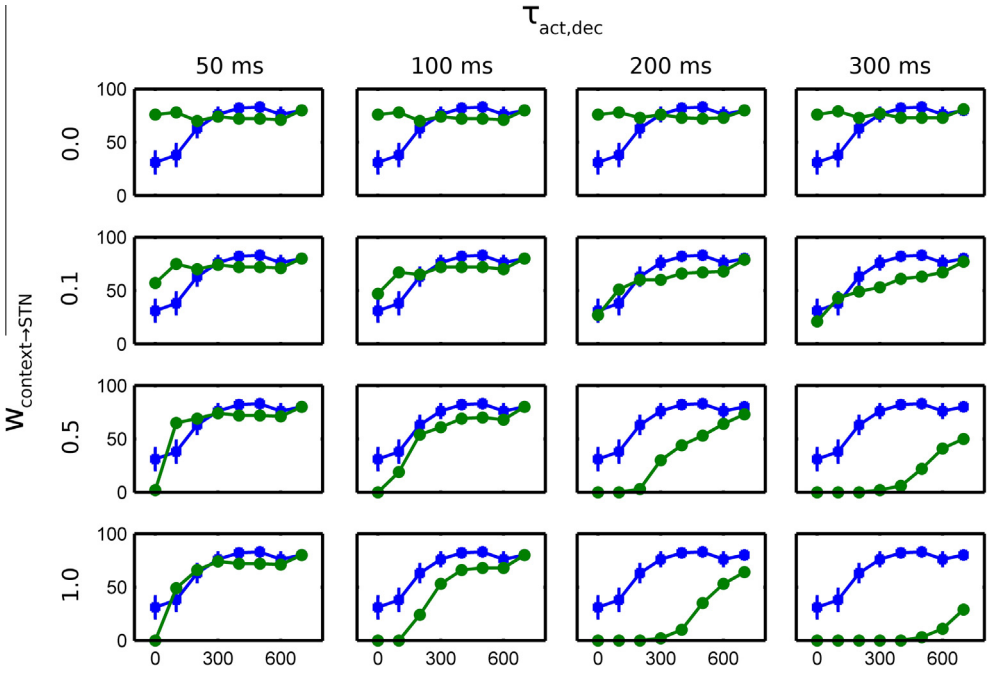

Fig. 6. Effects of relevant model parameters on distractor-induced blindness. Subplots depict the dependence of correct-response rates on SOAs for different values of $w_{context \rightarrow STN}$ (inhibitory strength of the hyperdirect pathway) and $\tau_{act,dec}$ (cortical activity decay). The blue line is a copy of the results by Sahraie et al. (2001; see Fig. 2). Values for r_{thresh} were 87, 95, 100 and 102 from the first to the last column. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

illustrate network performance under different sets of these values, multiple simulations were run using deterministic variables, meaning that random variables (i.e., neuronal noise and stimulus input strengths) were set to the same values across parameter sets.

Fig. 6 shows the results of these simulations and compares them to the results reported by Sahraie et al. (2001). The time constant of cortical activity decay ($\tau_{act,dec}$; columns of Fig. 6) controls if performance is affected only for small or also for larger SOAs, while the strength of the hyperdirect pathway ($w_{context \rightarrow STN}$; rows of Fig. 6) controls how strongly performance is impaired for affected SOA conditions. This implies that the strength of the hyperdirect pathway has to be greater than zero for a drop in performance.

A parameter set that reproduced the performance curve reported by Sahraie et al. (2001) was determined by a systematic search in the parameter space $\tau_{act,dec}$: [100 350] ms, $w_{context \rightarrow STN}$: [0.12 0.17] and r_{thresh}: [85 115], using a least squares criterion (Fig. 7). In accordance with the data reported by Sahraie et al. (2001), five independent subjects were simulated by running five independent, randomly initialized networks. The best parameter set was the following: $\tau_{act,dec} = 250$ ms, $w_{context \rightarrow STN} = 0.15$ and $r_{thresh} = 94.0$ (Fig. 7). The fitted performance curve for this parameter set is depicted in Fig. 8.

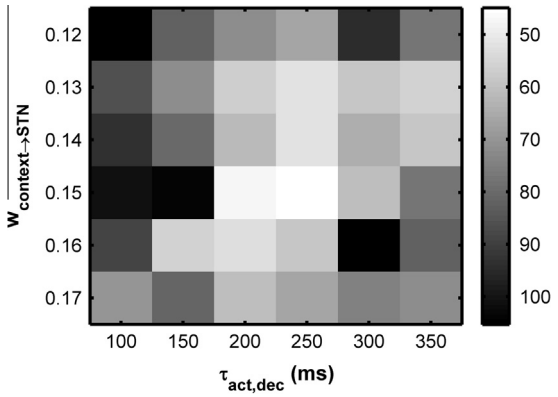

Fig. 7. Error matrix for model fits of the results reported by Sahraie et al. (2001). Grayscale fields depict sum-of-squares deviations between our fitted results and the results reported by Sahraie et al. (2001; Fig. 2) for systematically varied values of cortical activity decay ($\tau_{act,dec}$) and the inhibitory strength of the hyperdirect pathway ($w_{context \rightarrow STN}$) in our model. The smallest deviation was found for a combination of $\tau_{act,dec} = 250$ ms and $w_{context \rightarrow STN} = 0.15$.

Please cite this article in press as: Ebner, C., et al. Open and closed cortico-subcortical loops: A neuro-computational account of access to consciousness in the distractor-induced blindness paradigm. *Consciousness and Cognition* (2015), http://dx.doi.org/10.1016/j.concog.2015.02.007

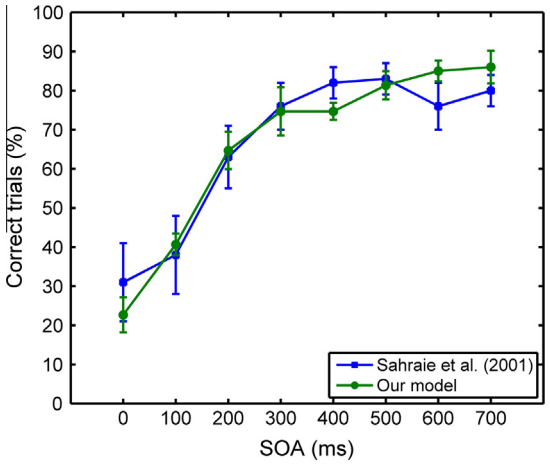

Fig. 8. Behavioral results for fitted model parameters. The plot shows the effects of SOAs on correct-response rates for both our fitted model parameters (green line) and the results reported by Sahraie et al. (2001; blue line). Error bars show standard errors of the mean. Parameter settings for this best reproduction of Sahraie et al.'s (2001) data are $w_{context \rightarrow STN} = 0.15$, $\tau_{act,dec} = 250$ ms and $r_{thresh} = 94.0$. Consistent with Sahraie et al.'s (2001) report, we simulated five independent 'subjects' in non-deterministic mode, i.e., with randomized variables for noise functions. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

While $\tau_{act,dec}$ determines that cortical activity after stimulus offset has a half-value time of about 175 ms, $w_{context \rightarrow STN}$ implies a rather weak hyperdirect pathway compared to the direct pathway's connections (cf. Fig. 2).

4. Discussion

We propose a computational model that predicts conscious perception to arise from reverberating activity within closed cortico-subcortical loops. The model predicts that BG pathways control access to consciousness by either enhancing or suppressing gating of information into these closed loops. The model moreover predicts that the characteristics of information processing in BG pathways explain the phenomenon of distractor-induced blindness (cf. Sahraie et al., 2001): we show that the excitatory direct BG pathway is capable of gating information into consciousness, while the inhibitory hyperdirect pathway may suppress gating of irrelevant information into consciousness. When a relevant stimulus closely follows an irrelevant stimulus, the hyperdirect pathway's suppression is not reduced fast enough for the relevant stimulus to receive access, resulting in distractor-induced blindness. Our model is in line with existing empirical evidence and provides specific predictions for future experiments that will be outlined below.

4.1. Existing empirical data corroborate our model

Niedeggen, Hesselmann, Sahraie, Milders, and Blakemore (2004) recorded ERPs during a distractor-induced blindness paradigm and concluded that the inhibition of target perception occurs at a post-sensory level. In a later study, moreover, they showed that the inhibition causes an ERP EEG negativity over frontal cortical areas (Niedeggen et al., 2012). These results fit well with our proposal that access to consciousness is controlled by those BG pathways that modulate activity in the prefrontal cortex. Michael, Hesselmann, Kiefer, and Niedeggen (2011) recently showed that distractor-induced blindness works not only for motion stimuli but also for changes in the orientation of static stimuli. Our model readily embraces these results by assuming that different input stimuli stimulate the same BG pathways.

Two model parameters were systematically varied to investigate their effects on performance: the strength of the hyperdirect pathway ($w_{context \rightarrow STN}$) and the decay time of activity in cortical input layers ($\tau_{act,dec}$). The latter parameter determines how fast the inhibition of via the hyperdirect pathway is removed and may relate to iconic memory, i.e., a brief maintenance of information that has already disappeared in the outside world (see Dick, 1974; Sperling, 1960). For those parameter values that best reproduced subjects' performance, the time constant $\tau_{act,dec}$ that controls this decay time took on the value of 250 ms. Empirically, Loftus, Duncan, and Gehrig (1992) reported time intervals of 200–300 ms concerning the decay of iconic memory, which fits well with the fact that distractor-induced blindness occurs strongly only for SOAs up to 300 ms (Sahraie et al., 2001). Moreover, these 200–300 ms correspond well to our fitted $\tau_{act,dec}$ of 250 ms, defining that 300 ms after stimulus disappearance activity has decreased to about 30% of its original value. The strength of the hyperdirect pathway (fit value 0.15) was found relatively weak compared to the direct pathway's strength (1.0 for striatal afferents from the input cortex and 1.5 for afferents from the PFC; cf. Fig. 2). We did not find any meaningful empirical data to compare these value against. Electrophysiological studies in which the activities of various BG nuclei are recorded after cortical stimulation usually show that the hyperdirect pathway causes strong increases in GPi activity (e.g., Kita & Kita, 2011; Nambu et al., 2000). However,

Please cite this article in press as: Ebner, C., et al. Open and closed cortico-subcortical loops: A neuro-computational account of access to consciousness in the distractor-induced blindness paradigm. *Consciousness and Cognition* (2015), http://dx.doi.org/10.1016/j.concog.2015.02.007

these recordings were performed after large-scale external stimulation of the cortex rather than under natural conditions. Because of the hyperdirect pathway's profuse and divergent connectivity on the GPi, such large-scale stimulation may have exaggerated the hyperdirect pathway's effects on the GPi.

4.2. Comparison with previous models

The present model is based on the concept of open and closed loops as suggested by Trapp et al. (2012). These authors proposed that consciousness arises out of closed loops, involving the cortex, thalamus and BG. In such closed loops, activity can reverberate, allowing for persistent integration of information and for its maintenance independent of sensory stimulation; unconscious processing, in contrast, is assumed to result from cessation of stimulation before closed-loop activity has started to reverberate. Here, we present a computational implementation of these ideas and apply it to a specific experimental paradigm. Thereby, we show that Trapp et al.'s (2012) ideas can be used to derive specific predictions that can be tested empirically.

How information processing in closed loops influences response selection in interconnected open loops has been previously modeled by Schroll et al. (2012). Closed loops in this model contribute to the maintenance of information in working memory. And indeed, working memory and consciousness have been proposed closely related within the global workspace theory (Baars & Franklin, 2007): both involve enhanced processing of limited amounts of information for limited periods of time. The present model differs from Schroll et al.'s (2012) in that it does not include synaptic plasticity, but runs on fixed synaptic connections. This allowed us to focus on the mechanisms behind distractor-induced blindness, a phenomenon that presumably does not depend on synaptic learning.

From our perspective, the BG are of general functional importance for the modulation of cortical and thalamic activities (i.e., for selecting which cortical representations become active). While previous computational models of the BG (e.g., Ashby, Ennis, & Spiering, 2007; Brown, Bullock, & Grossberg, 2004; Frank, 2006; Stocco, Lebiere, & Anderson, 2010) have largely focused on cognitive and motor impacts of these nuclei, we here showed that their modulation of cortical activities can also explain top-down influences on access to consciousness.

An influential previous neuro-computational model of access to consciousness, not including the BG, was presented by Dehaene et al. (2003; cf. also Dehaene, Kerszberg, & Changeux, 1998, and Dehaene & Changeux, 2005, for earlier and later version of this model, respectively). Their neural network explains stimulus-driven influences on access to consciousness. It consists of several specialized processing systems and a central high-level system that interconnects these. Conscious perception is assumed to arise whenever information crosses from a specialized into the high-level system. Such access to consciousness occurs if self-amplifying activity is strong enough to support global activation. Attentional blink, in contrast, occurs when a relevant stimulus is prohibited from access to the central system (due to limited capacity), because it occurs simultaneously with or follows closely after another relevant stimulus. Dehaene et al.'s (2003) model well reproduces data from the attentional blink paradigm, where a T2 that follows a T1 with a lag of 180–450 ms cannot be reported by subjects that are instructed to report both (see Raymond et al., 1992). Dehaene et al. (2003) proposed that their high-level system prominently includes the PFC, which is in line with our proposal. In contrast to our approach, however, their model does not explain how the contents of consciousness influence motor responding. Moreover, neither the specialized nor the central processing system of their model includes the BG. These differences in model assumptions go along with differences in the experimental paradigms implemented. In the attentional blink paradigm modeled by Dehaene et al. (2003), a conflict is evoked by two competing stimuli both of which are supposed to be gated into consciousness. "Blindness" (i.e., lack of access to the second target) in this paradigm plausibly results from competition among these stimuli in line with Dehaene et al.'s (2003) suggestion. In the distractor-induced blindness paradigm, in contrast, response conflict arises, because a stimulus that is supposed to be gated into consciousness is presented after distractors that are to be suppressed. Blindness in this paradigm likely results from aftereffects of distractor suppression in line with our suggestions. Therefore, our model and the model by Dehaene et al. (2003) may capture different mechanisms of the control of access to consciousness which co-exist in the brain.

A more fundamental difference between our model and the one by Dehaene et al. (2003) is the specification of how rules and constraints influence the gate to consciousness. In the model by Dehaene et al. (2003), stimulus activation has to be strong enough to support itself. Any cognitive influence on access to consciousness is outside the description of this model unless it is conceived that it acts on the strength of stimulus representations. Such an enhancement of stimulus representations is typically referred to as attention, but attention as a gate-keeper to consciousness has been recently challenged (Rahnev et al., 2011; van Boxtel, Tsuchiya, & Koch, 2010). Our approach, in contrast, provides an explicit description of how cognitive factors directly influence access to consciousness. From a neuro-anatomical point of view it is now well known that the BG are interconnected with the cortex and thalamus in multiple parallel cortico-subcortical loops (Haber, 2003) which may be an ideal substrate not only for cognitive control but also for access to consciousness. A potentially interesting experimental paradigm in the context of cognitive control is the inattentional blindness task in which salient, but task-irrelevant stimuli fail to enter consciousness (Mack & Rock, 1998; the term attention should here be considered as referring to a cognitive set and not to sensory enhancement; Trapp et al., 2012). In conclusion, Dehaene et al. (2003) proposed a sensory model of access consciousness while we suggest a cognitive model of access consciousness. In line with our cognitive focus, Niedeggen et al. (2012) observed a distractor-related EEG negativity over frontal cortical areas in their distractor-induced blindness paradigm, but no modulation of EEG activity over posterior, sensory cortices.

Please cite this article in press as: Ebner, C., et al. Open and closed cortico-subcortical loops: A neuro-computational account of access to consciousness in the distractor-induced blindness paradigm. *Consciousness and Cognition* (2015), http://dx.doi.org/10.1016/j.concog.2015.02.007

We have here primarily addressed the control of closed loops for the access to consciousness. With respect to the concept of open loops (Trapp et al., 2012), similar mechanisms can operate to control top-down attention. This concept relates to the attentional sensitization concept (Kiefer & Martens, 2010). As a core tenet of this idea, task sets cannot only influence the conscious, but also the unconscious processing of stimuli in a top-down way. While top-down effects on the processing of consciously available information are generally agreed upon, the extent of top-down effects on unconscious information processing is controversial. Our own model is rather focused on the closed loop concept and thus does not specifically address the issue whether top-down effects can modulate the ways in which unconscious information is processed or not. Rather, it states that top-down effects may determine whether or not information *becomes* conscious. A more refined model would be required to capture the details of how unconscious information is processed.

4.3. Model predictions

Our model predicts functional contributions of BG pathways to the control of access to consciousness, but should not be misinterpreted as stating that the BG are required for phenomenal awareness. Clinical impairments in access to consciousness are therefore predicted to result from alterations in BG functions. Hallucinatory conscious perceptions, for instance, as occurring in schizophrenia, are predicted to result either from overactivity of the excitatory direct BG pathway or from reduced activation of the inhibitory hyperdirect pathway. Both of these effects would allow irrelevant representations to access consciousness. While schizophrenia has indeed been shown to involve BG dysfunctions (e.g., Mamah et al., 2007, 2008; Menon, Anagnoson, Glover, & Pfefferbaum, 2001; Sjøholm, Bratlid, & Sundsfjord, 2004), the exact nature of these dysfunctions remains to be clarified. For patients with Parkinson's disease on dopamine replacement therapy, in contrast, who occasionally suffer from hallucinations as well (Factor, Molho, Podskalny, & Brown, 1994; Holroyd, Currie, & Wooten, 2001), excess activity of the excitatory direct BG pathway has indeed been shown (Schroll et al., 2014; Shen, Flajolet, Greengard, & Surmeier, 2008).

For the distractor-induced blindness paradigm, our model predicts increased PFC activity in trials with identified as compared to missed targets. Moreover, it predicts increased activity of the STN during distractor presentation and during intervals where subjects fail to detect T2. These predictions can for instance be tested in patients with intracranial deep-brain electrodes.

4.4. Limitations and future directions

Our model cannot yet explain all aspects of BG contributions to the control of consciousness. For instance, it is simplified in that it includes only two artificial neurons in each layer. Moreover, only direct and hyperdirect BG pathways are modeled, while indirect pathways were omitted because of assumed irrelevance for the distractor-induced blindness paradigm. Therefore, our model does not predict how indirect pathways may contribute to the control of conscious perception in different environments. Moreover, additional contributions of a cortico-thalamic pathway were left unmodeled which is capable of bypassing the BG for well-established processes in the brain (Schroll et al., 2014) and which likely contributes to consciousness as well (cf. Llinás et al., 1998).

Apart from our simplifications of BG anatomy, we modeled cognitive influences on access to consciousness only for the distractor-induced blindness paradigm, thereby leaving open to what extent cognitive influences in different experimental paradigms might differ from the proposed mechanisms. In principle, our model can explain any cognitive inhibitory effects on access to consciousness; with a few small changes, moreover (i.e., inclusion of a connection from the cortical context layer to the striatum; cf. Fig. 2), *excitatory* effects could be included as well. Then, our model could, for instance, also account for the facilitative versus inhibitory effects of congruent versus incongruent primes on access to consciousness (e.g., Draine & Greenwald, 1998; Naccache & Dehaene, 2001). In priming paradigms, prime stimuli activate high-level representations, task sets or schemes in a bottom-up way which then bias processing of subsequent stimuli in a top-down manner. In general, primes facilitate processing of subsequent stimuli if these are congruent, but inhibit processing of incongruent stimuli (e.g., Draine & Greenwald, 1998; Kiefer & Martens, 2010; Naccache & Dehaene, 2001). To judge for the ability of our model to *quantitatively* reproduce empirically known priming effects, however, computational simulations would be required which are outside the scope of this manuscript.

As to the implementation of the distractor-induced blindness paradigm, we abstracted the stimulus protocol by assuming that the model receives target versus distractor *contexts* as inputs, elicited by distractors and T1s, respectively, rather than being presented with these stimuli themselves. As a consequence, the model does not account for findings that the blindness effect increases in magnitude with increasing numbers of distractors (Hesselmann et al., 2006; Michael et al., 2011; Niedeggen et al., 2004, 2012). Moreover, the current implementation of the reset pulse that terminates reverberation of activity in the closed frontal loop is of a rather abstract nature as it is evoked from outside the model rather than originating from model dynamics.

4.5. Conclusion

We showed that a model of cortico-basalganglio-thalamic loops can account for the phenomenon of distractor-induced blindness, reproducing human performance curves. From a wider perspective, our model suggests that reverberating activity

Please cite this article in press as: Ebner, C., et al. Open and closed cortico-subcortical loops: A neuro-computational account of access to consciousness in the distractor-induced blindness paradigm. *Consciousness and Cognition* (2015), http://dx.doi.org/10.1016/j.concog.2015.02.007

in cortico-subcortical loops results in the emergence of conscious perceptions. BG pathways, by their both excitatory and inhibitory control over activity in these loops, are assumed to control access to consciousness. Distractor-induced blindness is predicted to result from aftereffects of distractor suppression, required to prevent inadequate distractor-induced response tendencies.

Acknowledgments

This research was supported by a grant from the German Research Foundation (DFG HA2630/6-1) to Fred Hamker, within the Research Network "Neuro-Cognitive Mechanisms of Conscious and Unconscious Visual Perception" (PAK 270/1 and 2). The authors would like to thank Frederik Beuth (Department of Computer Science, Chemnitz University of Technology) for his generous technical support.

References

Ashby, F. G., Ennis, J. M., & Spiering, B. J. (2007). A neurobiological theory of automaticity in perceptual categorization. *Psychological Review, 114*, 632–656.
Baars, B. J. (1988). *A cognitive theory of consciousness*. Cambridge, MA: Cambridge University Press.
Baars, B. J. (2005). Global workspace theory of consciousness: Toward a cognitive neuroscience of human experience. *Progress in Brain Research, 150*, 45–53.
Baars, B. J., & Franklin, S. (2007). An architectural model of conscious and unconscious brain functions: Global Workspace Theory and IDA. *Neural Networks, 20*, 955–961.
Balkin, T. J., Braun, A. R., Wesensten, N. J., Jeffries, K., Varga, M., Baldwin, P., et al (2002). The process of awakening: A PET study of regional brain activity patterns mediating the reestablishment of alertness and consciousness. *Brain, 125*, 2308–2319.
Bolam, J. P., Hanley, J. J., Booth, P. A. C., & Bevan, M. D. (2000). Synaptic organisation of the basal ganglia. *Journal of Anatomy, 196*, 527–542.
Braak, H., & Del Tredici, K. (2008). Cortico-basal ganglia-cortical circuitry in Parkinson's disease reconsidered. *Experimental Neurology, 212*, 226–229.
Brown, J. W., Bullock, D., & Grossberg, S. (2004). How laminar frontal cortex and basal ganglia circuits interact to control planned and reactive saccades. *Neural Networks, 17*, 471–510.
Christensen, M. S., Ramsøy, T. Z., Lund, T. E., Madsen, K. H., & Rowe, J. B. (2006). An fMRI study of the neural correlates of graded visual perception. *Neuroimage, 31*, 1711–1725.
Dehaene, S., & Changeux, J. P. (2005). Ongoing spontaneous activity controls access to consciousness: A neuronal model for inattentional blindness. *Public Library of Science Biology, 3*, e141.
Dehaene, S., Kerszberg, M., & Changeux, J. P. (1998). A neuronal model of a global workspace in effortful cognitive tasks. *Proceedings of the National Academy of Sciences, 95*, 14529–14534.
Dehaene, S., Sergent, C., & Changeux, J. (2003). A neuronal network model linking subjective reports and objective physiological data during conscious perception. *Proceedings of the National Academy of Sciences, 100*, 8520–8525.
Demerzti, A., Soddu, A., & Laureys, S. (2013). Consciousness supporting networks. *Current Opinion in Neurobiology, 23*, 239–244.
Dick, A. O. (1974). Iconic memory and its relation to perceptual processing and other memory mechanisms. *Perception & Psychophysics, 16*, 575–596.
Draine, S. C., & Greenwald, A. G. (1998). Replicable unconscious semantic priming. *Journal of Experimental Psychology: General, 127*, 286–303.
Factor, S. A., Molho, E. S., Podskalny, G. D., & Brown, D. (1994). Parkinson's disease: Drug-induced psychiatric states. *Advances in Neurology, 65*, 115–138.
Frank, M. J. (2006). Hold your horses: A dynamic computational role for the subthalamic nucleus in decision making. *Neural Networks, 19*, 1120–1136.
Gray, J. A. (1995). Dopamine release in the nucleus accumbens: The perspective from aberrations of consciousness in schizophrenia. *Neuropsychologia, 33*, 1143–1153.
Haber, S. N. (2003). The primate basal ganglia: Parallel and integrative networks. *Journal of Chemical Neuroanatomy, 26*, 317–330.
Hesselmann, G., Niedeggen, M., Sahraie, A., & Milders, M. (2006). Specifying the distractor inhibition account of attention-induced motion blindness. *Vision Research, 46*, 1048–1056.
Holroyd, S., Currie, L., & Wooten, G. F. (2001). Prospective study of hallucinations and delusions in Parkinson's disease. *Journal of Neurology, Neurosurgery and Psychiatry, 70*, 734–738.
Kiefer, M., & Martens, U. (2010). Attentional sensitization of unconscious cognition: Task sets modulate subsequent masked semantic priming. *Journal of Experimental Psychology: General, 139*, 464–489.
Kita, H., & Kita, T. (2011). Cortical stimulation evokes abnormal responses in the dopamine-depleted rat basal ganglia. *Journal of Neuroscience, 31*, 10311–10322.
Kjaer, T. W., Bertelsen, C., Piccini, P., Brooks, D., Alving, J., & Lou, H. C. (2002). Increased dopamine tone during meditation-induced change of consciousness. *Cognitive Brain Research, 13*, 255–259.
Llinás, R., Ribary, U., Contreras, D., & Pedroarena, C. (1998). The neuronal basis for consciousness. *Philosophical Transactions of the Royal Society London B, 353*, 1841–1849.
Loftus, G. R., Duncan, J., & Gehrig, P. (1992). On the time course of perceptual information that results from a brief visual presentation. *Journal of Experimental Psychology: Human Perception and Performance, 18*, 530–549.
Mack, A., & Rock, I. (1998). *Inattentional blindness*. Cambridge, MA: MIT Press.
Mamah, D., Harms, M. P., Wang, L., Barch, D., Thompson, P., Kim, J., et al (2008). Basal ganglia shape abnormalities in the unaffected siblings of schizophrenia patients. *Biological Psychiatry, 64*, 111–120.
Mamah, D., Wang, L., Barch, D., de Erausquin, G. A., Gado, M., & Csernansky, J. G. (2007). Structural analysis of the basal ganglia in schizophrenia. *Schizophrenia Research, 89*, 59–71.
Menon, V., Anagnoson, R. T., Glover, G. H., & Pfefferbaum, A. (2001). Functional magnetic resonance imaging evidence for disrupted basal ganglia function in schizophrenia. *American Journal of Psychiatry, 158*, 646–649.
Mhuircheartaigh, R. N., Rosenorn-Lanng, D., Wise, R., Jbabdi, S., Rogers, R., & Tracey, I. (2010). Cortical and subcortical connectivity changes during decreasing levels of consciousness in humans: A functional magnetic resonance imaging study using Propofol. *Journal of Neuroscience, 30*, 9095–9102.
Michael, L., Hesselmann, G., Kiefer, M., & Niedeggen, M. (2011). Distractor-induced blindness for orientation changes and coherent motion. *Vision Research, 51*, 1781–1787.
Naccache, L., & Dehaene, S. (2001). Unconscious semantic priming extends to novel unseen stimuli. *Cognition, 80*, 215–229.
Nambu, A., Tokuno, H., Hamada, I., Kita, H., Imanishi, M., Akazawa, T., et al (2000). Excitatory cortical inputs to pallidal neurons via the subthalamic nucleus in the monkey. *Journal of Neurophysiology, 84*, 289–300.
Nambu, A., Tokuno, H., & Takada, M. (2002). Functional significance of the cortico-subthalamo-pallidal 'hyperdirect' pathway. *Neuroscience Research, 43*, 111–117.
Niedeggen, M., Hesselmann, G., Sahraie, A., Milders, M., & Blakemore, C. (2004). Probing the prerequisites for motion blindness. *Journal of Cognitive Neuroscience, 16*, 584–597.
Niedeggen, M., Michael, L., & Hesselmann, G. (2012). Closing the gates to consciousness: Distractors activate a central inhibition process. *Journal of Cognitive Neuroscience, 24*, 1294–1304.

Please cite this article in press as: Ebner, C., et al. Open and closed cortico-subcortical loops: A neuro-computational account of access to consciousness in the distractor-induced blindness paradigm. *Consciousness and Cognition* (2015), http://dx.doi.org/10.1016/j.concog.2015.02.007

O'Reilly, R., & Frank, M. (2006). Making working memory work: A computational model of learning in the prefrontal cortex and basal ganglia. *Neural Computation, 18*, 283–328.
Palmiter, R. D. (2011). Dopamine signaling as a neural correlate of consciousness. *Neuroscience, 198*, 213–220.
Rahnev, D., Maniscalco, B., Graves, T., Huang, E., de Lange, F. P., & Lau, H. (2011). Attention induces conservative subjective biases in visual perception. *Nature Neuroscience, 14*, 1513–1515.
Raymond, J. E., Shapiro, K. L., & Arnell, K. M. (1992). Temporary suppression of visual processing in an RSVP task: An attentional blink? *Journal of Experimental Psychology: Human Perception and Performance, 18*, 849–860.
Rees, G., Kreiman, G., & Koch, C. (2002). Neural correlates of consciousness in humans. *Nature Reviews Neuroscience, 3*, 261–270.
Sahraie, A., Milders, M., & Niedeggen, M. (2001). Attention induced motion blindness. *Vision Research, 41*, 1613–1617.
Schroll, H., & Hamker, F. H. (2013). Computational models of basal-ganglia pathway functions: Focus on functional neuroanatomy. *Frontiers in Systems Neuroscience, 7*, 122.
Schroll, H., Vitay, J., & Hamker, F. H. (2012). Working memory and response selection: A computational account of interactions among cortico-basalganglio-thalamic loops. *Neural Networks, 26*, 59–74.
Schroll, H., Vitay, J., & Hamker, F. H. (2014). Dysfunctional and compensatory synaptic plasticity in Parkinson's disease. *European Journal of Neuroscience, 39*, 688–702.
Shapiro, K. L. (1994). The attentional blink: The Brain's "eyeblink". *Current Directions in Psychological Science, 3*, 86–89.
Shen, W., Flajolet, M., Greengard, P., & Surmeier, D. J. (2008). Dichotomous dopaminergic control of striatal synaptic plasticity. *Science, 321*, 848–851.
Sjøholm, H., Bratlid, T., & Sundsfjord, J. (2004). 123I-β-CIT SPECT demonstrates increased presynaptic dopamine transporter binding sites in basal ganglia in vivo in schizophrenia. *Psychopharmacology, 173*, 27–31.
Smith, Y., Bevan, M. D., Shink, E., & Bolam, J. P. (1998). Microcircuitry of the direct and indirect pathways of the basal ganglia. *Neuroscience, 86*, 353–388.
Sperling, G. (1960). The information available in brief visual presentations. *Psychological Monographs: General and Applied, 74*, 1–29.
Stocco, A., Lebiere, C., & Anderson, J. R. (2010). Conditional routing of information to the cortex: A model of the basal ganglia's role in cognitive coordination. *Psychological Review, 117*, 541–574.
Trapp, S., Schroll, H., & Hamker, F. H. (2012). Open and closed loops: A computational approach to attention and consciousness. *Advances in Cognitive Psychology, 8*, 1–8.
van Boxtel, J. J. A., Tsuchiya, N., & Koch, C. (2010). Consciousness and attention: On sufficiency and necessity. *Frontiers in Psychology, 1*, 217.
Vitay, J., & Hamker, F. H. (2010). A computational model of basal ganglia and its role in memory retrieval in rewarded visual memory tasks. *Frontiers in Computational Neuroscience, 4*, 1–18.

Please cite this article in press as: Ebner, C., et al. Open and closed cortico-subcortical loops: A neuro-computational account of access to consciousness in the distractor-induced blindness paradigm. *Consciousness and Cognition* (2015), http://dx.doi.org/10.1016/j.concog.2015.02.007