On the Role of Dopamine in Cognitive Vision

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Abstract. Although dopamine is one of the most studied neurotransmitter in the brain, its exact function is still unclear. This short review focuses on its role in different levels of cognitive vision: visual processing, visual attention and working memory. Dopamine can influence cognitive vision either through direct modulation of visual cells or through gating of basal ganglia functioning. Even if its classically assigned role is to signal reward prediction error, we review evidence that dopamine is also involved in novelty detection and attention shifting and discuss the possible implications for computational modeling.

1 Introduction

Visual perception is well known to build upon multiple components. It starts with the extraction of simple basic features, such as motion, depth or oriented edges, which get incerasingly complex and end in very specific multi-sensorimotor patterns. Most vision systems compute a set of features at different levels of the hierarchy but largely ignore how vision and other sensors are linked to the task at hand. In part, this is due to the fact that little is known about this high-level cognitive processing. For example, it is well accepted that the attentive binding of different features requires mechanisms of selection and top-down modulation. The machinery that determines *what* is selected and *when* has been rarely the focus of modeling studies. In this paper we focus on the role of dopamine (DA) in cognitive vision, and present biological findings that suggests that DA is at a central place to favorize not only the guidance of attention towards relevant locations but also in more cognitive processes like visual working memory.

DA is a key neurotransmitter in the brain. It is mainly produced by two small groups of neurons in the mesencephalon: ventral tegmental area (VTA) and substancia nigra pars compacta (SNc). These areas send diffuse although segregated connections to different areas of the brain. DA has been involved in many aspects of brain functioning: control of movements, attention, memory, reward anticipation, pleasure, addiction to drugs (cocaine, amphetamines), motivation, arousal, etc. Dysfunctioning of the dopamine system leads to severe deficits as Parkinson's disease, schizophrenia, attention-deficit hyperactivity syndrome (AD-HD) or autism (see [1] for a review).

DA fibers reach a lot of brain regions: basal ganglia (striatum and globus pallidus), most parts of cerebral cortex (particularly cingulate, rhinal and medial prefrontal cortex), amygdala, hippocampus, thalamus and diverse midbrain

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structures like the superior colliculi [2]. Its effects are not precisely known at the moment. DA does not act like like a fast ionotropic neurotransmitter like acetylcholine, AMPA, NMDA or GABA but rather seems to modulate other receptor channels: activation of DA receptors alone does not induce large postsynaptic currents [3] but modifies the cell's excitability or the synaptic transmission of other neurotransmitters (see [4] for a review). Moreover, DA also has different effects depending on the type of dopamine receptors, which can be classified into two categories: the D1-like receptors (D1 and D5), that have principally excitatory effects and D2-like receptors (D2, D3, D4) that are mainly inhibitory.

The aim of this paper is not to review all these roles and effects of DA, but rather to illustrate its influence on vision in a very broad sense, i.e. on recognition, categorization and attention but also on visual search, reward prediction and working memory. We will try to pinpoint the need for cognitive vision systems to take into account the information carried by the dopaminergic signal.

2 Influence of Dopamine on Visual Processing

We will focus here on the influence of dopamine on cortical areas in the ventral pathway, that are thought to deal only with visual features. We will review evidences that this visual processing is influenced by the amount of reward associated to visual stimuli, what is thought to be mediated by the dopaminergic signal. On a more anecdotic level, dopamine is also produced by amacrine cells in the retina and is involved in light adaptation [5].

2.1 Dopamine Effects in the Cortical Visual Areas

Dopamine effects in the cerebral cortex are often considered to involve exclusively cingulate, rhinal and prefrontal cortices, whereas similar densities of receptors are found in several other areas, including parietal cortex, temporal lobe and occipital cortex [6,2]. What can be the role of DA modulation in visual areas? Müller and Huston [7] have observed an increase in the extracellular level of DA in the occipital cortex of freely moving rats, but not in their temporal cortex. Interestingly, they have observed a dose-dependent increase in both areas after cocaine injection, suggesting that DA levels in these visual areas signal the hedonistic value of visual information. When the rat moves freely (without a goal), visual stimuli are not associated to pleasure or food on a behavioral level - what would explain the lack of DA release in temporal cortex in this condition - but the richness of this stimulation may be interpreted by the dopaminergic cells innervating the occipital cortex as the possibility that something may be interesting. Dopamine could therefore act similarly to the vigilance parameter in the *adaptive resonance theory*, in a different way according to the considered cortical area [8].

Higher in the ventral pathway, Mogami and Tanaka [9] showed that inferotemporal (area TE) and perirhinal (PRh, part of the medial temporal lobe) cells showed significant reward dependence in their response to visual cues. This suggests that TE and PRh are involved in associating visual stimuli with reward

outcomes. However, by studying the time distribution of the reward-modulated part of these responses, they observed that TE reward-dependent responses occur before perirhinal reward-dependent responses, which can not be explained by a simple feedback mechanism from reward-related areas (amygdala, cingulate or orbitofrontal cortex) to perirhinal cortex and then TE. They propose that reward-association occurs very early in the visual pathway (already in the occipital cortex) but in increasing proportions culminating in PRh. This reward association is nevertheless not very adaptative: Rolls et al. [10] have shown that the stimulus selectivity of TE cells does not follow the reversal of the contingency between visual stimuli and the associated reward, contrary to orbitofrontal cortex cells [11]. This tends to show that reward-related activities in orbitofrontal cortex - and possibly cingulate cortex and amygdala - are context-dependent, whereas stimulus-reward associations in visual areas (at least until TE) represent more stable relationships that could participate to the salience of these stimuli.

There is no evidence yet that these reward-related activities in visual areas are due to dopamine modulation, except in PRh. Liu et al. [12] suppressed the expression of D2 receptors in PRh of monkeys, leading to deficits in processing reward-related information (in that case, the number of successful trials required to obtain reward). According to the hypothesis of Mogami and Tanaka, reward association occurs in a feed-forward way along the ventral pathway, what supports the idea that the reward information is carried to visual areas by DA and is incorporated progressively in visual processing.

2.2 Selective Categorization in Perirhinal Cortex

To investigate the computational interest of using DA-mediated reward information in the visual areas, we focused on the role of PRh in categorization, multimodal integration and memory retrieval [13]. PRh is primarily involved in the learning and representation of novel objects [14], but also in the generalization of visual object discrimination [15]. Its lower stimulus selectivity compared to TE and its connections with multimodal areas tend to show that PRh represents an integrated view of the different characteristics of an object, perhaps with some categorization. Similarly to what is observed in TE, some PRh cells exhibit sustained activities in delayed match-to-sample tasks, even if it does not seem robust to distractors [16].

In the computational model we propose (Fig. 1), objects are represented by different aspects that may not always occur at the same time. For example, it could be different visual parts of an object (the back, the seat and the feet of a chair), different modalities (vision, audition, touch) or parts of several exemplars of a category (cups having a similar shape but different details). All these aspects may not be perceived at the same time, but through repetitive presentation of the same object we assume that on average all the possible combinations will occur. The role of the model is to integrate these different parts into a global representation, through the activation of a *cluster* of cells. Each individual cell is selective for only one part, but the interaction of all cells of a cluster through their lateral connections creates a population code of the object. The model

consists of excitatory and inhibitory cells, reciprocally connected, that follow an ordinary differential equation. The connections between excitatory cells are modifiable with experience, according to a homeostatic learning rule that ensures that the learning of the weights has long-term stability [17]. Every connection in the model is modulated by a global DA level, supposed to be constant in a short time scale, either in an excitatory - the efficiency of the connection is enhanced - or an inhibitory way - the efficiency is reduced.

After repeated and alternated presentations of two objects to the model, with only a random number of active aspects at each time, we have observed that the learned connections between the excitatory cells provoked several interesting phenomena. First of all, the activation of a majority of aspects of an object induce activity in the corresponding PRh cells, but also, under optimal dopamine levels, to the cells that usually represent aspects that do not receive external stimulation. DA favorizes the propagation of activity within a cluster, even in cells that do not receive external inputs. This property raises the possibility that cells in PRh can virtually enlarge their selectivity: without DA, they are selective for restricted aspects of an object, whereas with an optimal level of DA they are selective for the object as a whole. DA could therefore control the level of abstraction needed in PRh representations according to the task.

We also observe sustained activities in a cluster under optimal dopamine levels, without any feedback connections from prefrontal cortex. This implies that the working memory of the visual details of an object can be processed in PRh and that prefrontal cortex is preferentially involved in manipulating this representation, not memorizing it (see section 4 for a discussion). Interestingly, activity can propagate within a cluster under optimal DA levels with only 40% of its cells receiving external simulation. This raises the possibility that an external system - either prefrontal cortex or basal ganglia through thalamus - can retrieve the visual details of an object even if its representation in PRh has disappeared. An abstract and compressed representation of this object (similar to a pointer in the computer language C) that would be memorized elsewhere is sufficient to retrieve the global visual information about this object so that it can be used for example in visual search through top-down connections [18]. In this framework, DA acts as a gating signal allowing propagation of activity and memory retrieval.

3 Attention and Dopamine

In the preceding section, we stated that DA carries reward-related information, without any further details. We will now focus on the firing of dopaminergic cells and see what kind of reward-related information causes the cells to fire and what are the computational implications.

3.1 Classical Conditioning View of Dopamine

Midbrain dopaminergic neurons exhibit highly stereotyped phasic excitatory responses of high amplitude, short duration (<200ms) and short latency (70-100 ms) after several types of events: delivery of primary rewards like food or liquids,

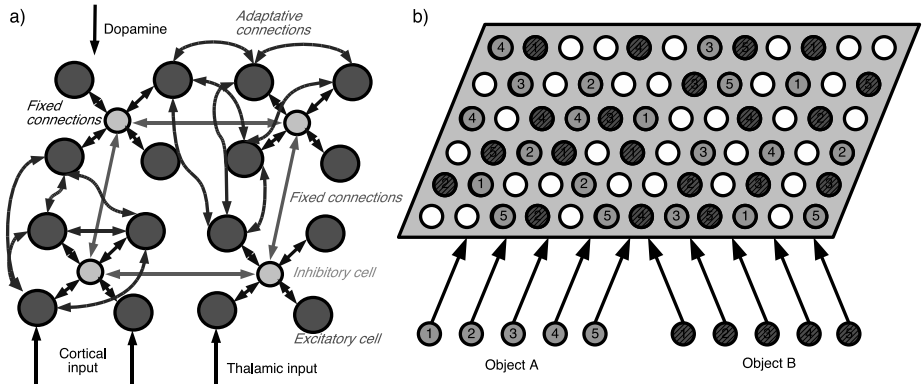

Fig. 1. a) Architecture of our model of PRh. It is composed of $N \times N$ excitatory cells and $\frac{N}{2} \times \frac{N}{2}$ inhibitory cells. Each inhibitory cell is reciprocally connected with four excitatory cells and to neighbouring inhibitory cells. Excitatory cells are reciprocally connected with each other, but the strength of the connections is learned. Each excitatory cell receives a cortical input from other areas like TE. Additionnally, some excitatory cells receive a thalamic input. All connections are modulated by dopamine. b) Feed-forward connectivity. Two different objects have to be learned by the model: object A (light grey) and B (dark grey, hatched) are each represented by five aspects, corresponding to different views or modalities. Each part is connected to four excitatory cells, what makes each object being represented by a cluster of 20 cells.

arbitrary stimuli classically conditioned by association with primary rewards and sudden appearance of novel, intense or salient stimuli [19]. The first type of events corresponds to the hedonic value of a stimulus, what can be signaled by the lateral hypothalamic area which responds to primary rewards without habituation [20]. The lateral hypothalamic area has excitatory connections to the midbrain dopaminergic neurons through the pedonculopontine tegmental nucleus [21].

The second type of events that produce phasic activation of dopaminergic neurons is the appearance of a visual stimulus that reliably predicts reward delivery after a certain amount of time. This activation appears during learning and progressively replaces the activation at the time of the actual reward. However, if the reward is omitted, the dopamine activity falls beyond its baseline level. In the Pavlovian conditioning paradigm, such a behaviour suggests that dopamine reflects a reward-prediction error: after learning, when the reward-predicting stimulus appears, DA signals that nothing was expected but that reward will be delivered within a certain delay: this is a positive reward-prediction error. At the time of reward delivery, the expected reward is equal to what is obtained, and DA does not show any activation. If reward is omitted, actual reward is lower than what was expected, and DA activity decreases: this is a negative reward-prediction error.

This behaviour has been frequently compared to the temporal difference (TD) algorithm [22] and lead to various computational models of the dopaminergic neurons acting as a reward-prediction error system, especially in actor-critic

architectures [23,24,25]. However, this algorithm does not fully cover all aspects of DA firing. First of all, it needs a constant time interval between the predicting stimulus and the reward delivery during learning, whereas animals are robust to some variability [26]. Brown et al. [27] and O'Reilly et al. [28] have proposed computational models avoiding this problem by computing separately, in different areas, the reward-prediction error associated to the predicting stimulus and the one associated to the reward. The link between the two events is ensured by a working memory of the predicting stimulus.

The TD analogy of DA firing also does not take into account the uncertainty of reward: if the cue predicts that reward will be delivered with a probability of 0.5, DA cells will first show a phasic response to the cue and then progressively increase their activity until the expected instant of reward delivery. This is not captured by TD models. Finally, contrary to TD reward-prediction error, DA cells also respond to the sudden appearance of novel, intense or salient stimuli, even if they are not associated to reward [29]. However, this response decreases when the subject becomes habituated to such an unrewarding stimulus. As a conclusion, TD analogy is a computationally efficient and formally elegant method, but it does not cover all aspects of DA firing.

3.2 DA Signals Novelty

Despite the different sizes, shapes or complexity of the reward-predicting stimuli, the phasic dopamine signal is highly stereotyped. It responds with a latency comprised between 70 and 100 ms, which is shorter than the latency of the saccades bringing the stimulus onto the fovea for a more detailed analysis (150 to 200 ms). Signals regarding the identity of visual objects, even if they are not foveated, are detected in the inferotemporal cortex with a latency of 80 to 100 ms after stimulus onset, therefore at least at the same time as dopamine firing, rising the problem of how this information can reach the dopamine areas almost immediately. According to the current state of the art in neuroanatomy, such a projection from inferotemporal cortex to dopamine cells would at least need a relay in the medial basal nucleus of the amygdala or the ventral striatum [30]. Redgrave and Gurney [31] consequently conclude that the rich and detailed representations in the cerebral cortex are a bad candidate to provide the dopamine system with accurate reward prediction. They propose that dopamine responses are triggered as a consequence of limited pre-attentive, pre-saccadic sensory processing.

Where does this information come from? Redgrave and Gurney also note that, in almost all studies showing that phasic dopamine responses look like a reward prediction error signal, the reward predicted by the cue is correlated with its spatial location. Would spatial position - and not stimulus identity - be the core parameter of DA activity? The dopaminergic midbrain areas receive strong connections from the deep layers of the superior colliculus (SC) [32,33]. SC superficial layers show a very early visual response 40 ms after the stimulus onset. SC deep layers (involved in saccade execution) also quickly habituate to the repetition of the same stimuli without any reward contingency [34] but not

when reward is associated [35]. This suggests that SC can quickly provide the dopaminergic areas with information about the novelty or the reward association of a stimulus at a fixed position, without having to process its visual details.

According to Redgrave and Gurney, such a signal would only be used to reinforce the learning of the correlation between the appearance of the stimulus, the action that possibly preceded it and the context. Through repetition of interactions between the agent and his environment, DA would help to distinguish between the consequences of the agent's own actions and what is caused by external events. This hypothesis highlights the role of DA in operant conditioning, contrary to the classical Pavlovian conditioning interpretation. This last paradigm, requiring a fine analysis of the details of the stimulus, would be treated by other cortical structures like orbitofrontal cortex.

3.3 DA Modulation of the Basal Ganglia Can Direct Attention Through Superior Colliculus

DA is also a key element of attentional processes: lesioning the midbrain DA cells reduce the attentive component of behavior [36], either by decreasing the frequency of saccades or by neglecting interesting stimuli. One major site of dopaminergic innervation is the basal ganglia (BG), which is known to control the preparation of saccades in the superior colliculus by means of sustained inhibition [37]. Two major pathways are often considered between the input structure of the BG - the striatum - and the output structures - substancia nigra pars reticulata (SNr) and the internal part of the globus pallidus (GPi). The direct pathway disinhibits the deep layers of SC at the location of a planned saccade whereas the indirect pathway through the external part of globus pallidus (GPe) increases this inhibition on SC to prevent the saccade from being executed. Both pathways are modulated by the dopaminergic fibers originating in SNc or VTA, controlling the competition between the two pathways as well as modulating the learning of the connections between the cerebral cortex and the striatum.

Thanks to this modulation by dopamine, BG can orient the eye towards rewarded locations and is therefore implicated in overt attention [38]. The question that arises is whether basal ganglia can also control covert attention. First, it is now known that SC sends feedback information about the planned saccade to the frontal eye fields (FEF) through the mediodorsal part of the thalamus [39]. BG can use this pathway to send information to FEF, but can also directly disinhibit the mediodorsal thalamus and therefore favorize FEF activation [40]. Even if FEF is principally considered as a premotor area, it is known to play a key role in visual attention, especially in modulating the gain of V4 and IT cells at the location of a prepared saccade [41,18]. In such a premotor theory of attention [42], covert and overt attention are supposed to use the same structures, the only difference being the actual execution of a saccade, which is under control of BG through dopamine modulation.

This hypothesis considers that BG only controls the spatial reentry from premotor to visual areas, but Silkis [43] goes further by hypothesizing that visual perception is based on neuronal pattern selection in parallel but interdependent

loops between different cortical areas (visual, limbic, premotor or cognitive), basal ganglia and thalamus. In this framework, visual processing is not only due to the feedforward activation of visual areas in the ventral pathway, but also to the disinhibition of the corresponding cortico-basal-thalamo-cortical loops according to dopamine activation. The proposed mechanism for DA activation is the following: DA is directly activated by the deep layers of SC when a stimulus appears, but as SC is under inhibition of SNr, DA activation first requires the inhibition of SNr. This can be achieved by the stimulation of striatal cells by visual nuclei of the thalamus (median-parafascicular complex), which has been discovered by Matsumoto et al. [44]. Since only strong and salient stimuli can depolarize striatal cells and therefore evoke dopamine firing, the modulation of segregated cortico-basal-thalamo-cortical is a good candidate for bottom-up attention. The same mechanism can be used for top-down attention, where prefrontal cortex or hippocampus can directly or indirectly - through a relay in the striosomes of the nucleus accumbens - elicit DA activation, therefore influencing visual processing. Silkis' theory is still for the moment very hypothetical, but proposes an interesting explanation to several biological data (like the earlier reward-dependent activation of TE cells compared to PRh cells, see section 2.1) and makes several testable predictions.

4 Visual Working Memory Is Mediated by Dopamine

Attention is not the only cognitive process that is influenced by - or under control of - dopamine. Working memory (WM), the ability to hold a few items in mind for later use, is known to heavily depend on the integrity of dopaminergic cells, either in human parkinsonian subjects [45] or in lesioned animals [46]. The two major structures involved in WM are the prefrontal cortex and the basal ganglia, which are both subject to dopaminergic modulation [47]. The key mechanism for WM is sustained activity, which have been discovered in prefrontal cortex [48] and striatum [49] but also in diverse cortical areas like inferotemporal [16] or parietal cortex [50]. What is the role of dopamine in these sustained activities and more generally in WM processes?

4.1 Dopamine Modulates Sustained Activity in Recurrent Circuits

There are many evidences for a direct involvement of dopamine receptors on prefrontal sustained activities (see [4] for a review). Several mechanisms are involved: modification of the cell's excitability, increase of the efficacity of NMDA- or GABA-mediated synapses, decrease of AMPA-mediated ones, dependence on the cell's activity, differential modulation of excitatory pyramidal neurons and inhibitory fast-spiking interneurons, D1 vs. D2 receptors densities, etc. Several computational models have tried to capture some of these mechanisms at different levels of detail [51,52,53,54,55]. The common point between all these models is that they consider that sustained activities are not an intrinsic property of isolated single cells, but rather an emergent property of an assembly of recurrently

connected cells. These assemblies form fixed-point attractors of the network and tonic levels of DA act principally as a stabilizer of these states, either against internal noise or against intervening distractors. The model of PRh we proposed in section 2.2 use the same kind of mechanisms at the visual level.

The main problem of most of these models is that they only explain memory maintenance and not memory access (gating) of the relevant items. Dopamine modulation has a very slow timecourse in cortical substrates (several seconds) and long-lasting effects (up to several hours): phasic DA activation can not update the content of WM. Similarly, the diffuse innervation of dopaminergic axons does not allow the selective updating of WM: in most tasks involving WM (like the 1-2 AX task used in [56]), some items must be maintained over long periods of time whereas others are only useful for immediate decisions. A common gating signal can not be used to update these different categories of items.

4.2 Basal Ganglia Gates Working Memory

Whereas direct dopamine modulation of prefrontal cells plays certainly a role in working memory, especially its robustness to distractors, other systems have to deal with the selective updating of the content of WM. The basal ganglia is known to play an important role in WM processes, as revealed by imaging studies [57,58]. Striatal medium spiny neurons have been shown to exhibit a region of bistability (up/down states) under elevated dopamine levels [59]. This intrinsic mechanism of sustained activities is therefore under direct control of DA, whose effects have very quick latencies in the striatum, allowing phasic DA discharges to control the updating of working memory at this level. This information can then be sent back to prefrontal cortex through direct connections between the striatum and the mediodorsal part of the thalamus [60] and/or indirectly through GPi.

Several computational models have already tried to address the interplay between the prefrontal cortex and the basal ganglia. The PBWM model of Frank and O'Reilly [56] is designed to learn different cognitive tasks involving WM on the same substrate, thanks to a robust reward-prediction error algorithm. However, time is not explicitely considered, making no distinction possible between phasic and tonic modes of DA firing. The FROST model of Ashby et al. [61] uses BG to maintain information in prefrontal cortex, but does not deal with updating. Finally, the model of Gruber et al. [62] is the first that distinguishes between the tonic long-lasting effect of DA on prefrontal cells (increasing the cortical gain) and the phasic effect of DA on striatal cells (intrinsic bistability). Prefrontal cortex and BG in this model cooperate to provide a robust and dynamic WM depending on dopamine modulation. However, the model would need to be integrated into a realistic task, with an efficient learning algorithm for DA activation.

4.3 Role of Working Memory in Cognitive Vision

Previous models about the role of DA in working memory either address spatial WM - the ability to remember the location of a previously flashed stimulus [63] - or abstract representations of objects that are entirely copied into WM

(as in [56]). In most delayed match-to-sample tasks (DMS), the subject has to remember the identity (whatever this means) of a stimulus in order to find it after a certain delay in an array of distractors. Which kind of representation of this stimulus is stored in WM during such a task? Most visual search theories suggest that selective attention is achieved through feedback modulation of visual areas to enhance the processing of the desired features [64,65,66,67,18]. In DMS tasks, the representation of the cue in WM should therefore be able to retrieve the visual features needed to bias the visual search of the target. However, visual WM seems to store integrated representations of objects rather than individual features for complex stimuli [68,69].

To solve this apparent contradiction, Ranganath [70] suggests a model of visual WM that relies on the the interaction of three main structures. First, the visual aspects of an object are maintained through sustained activity in visual cortical areas, starting from low-level features in V1 [71] to object representations in IT [14]. Then, additional feedback from the medial temporal lobe (PRH, entorhinal cortex, hippocampus) helps to reconstruct the different aspects of a novel object [72]. Finally, top-down inputs from dorsolateral prefrontal cortex (DLPFC) helps to maintain visual representations in the face of distractors [73] and to manipulate the content of WM [74].

In this theoretical model, there is a clear distinction between the content of WM - which is maintained in visual areas - and its manipulation (robustness to distractors, selective updating) that is thought to occur in prefrontal cortex and, according to what was reviewed before, in basal ganglia. Our model of PRh can be easily integrated in such a framework as its sustained activities are not the core system of its short-term memorizing capacities, but rather its ability to retrieve through external stimulation - either prefrontal cortex or basal ganglia through thalamus - the detailed information that needs to be reactivated. What is actually memorized in these external systems is therefore a representation of which group of neurons should be reactivated when needed. The key mechanism here is supposed to be dopaminergic modulation of the relevant visual areas.

5 Conclusion

Parent and Cicchetti said that *"Models in science tend to reassure and appease researchers who do not like to wander alone in the universe of knowledge. However, models may have a perverse effect, such as the selective neglect of data that do not fit into the model (modellus deformans disease). It would be unwise to rush into the formulation of a new basal ganglia model until the real significance of the enormous amount of new data on basal ganglia becomes clear"* [75]. Without being so pessismistic about the usefulness of modeling, one can wonder if the convenient TD analogy of DA firing did not lead to a misunderstanding of the roles of dopamine and basal ganglia in cognitive functions. New developments in dopamine research has shed a new light on the role of this neurotransmitter in various aspects of cognition, such as quickly directing attention to potentially - and not predicted as - rewarding events.

In this brief review, we emphasized the role of DA in cognitive vision at several levels. Dopamine is crucial for a goal-directed behavior of visual agents. One potential role is the direct modulatory influence of DA on visual areas in order to favorize learning of the features that are associated to potential rewards. This role of DA in low-level feature learning remains to be established but, according to a more integrated view of learning, should propose a different explanation on which visual features are learned. Secondly, the TD analogy of DA firing does not cover all aspects of DA neurons' behaviour and emphasis has been put on its role in directing attention. Finally, its role in working memory process through its influence on basal ganglia has been decribed in an integrated framework. Concluding, when the role of DA will be more fully understood, there will be a potential for developing flexible, more brain-like visual agents that acquire task-related knowledge and learn to integrate context information to make appropriate decisions.

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References

1. Nieoullon, A.: Dopamine and the regulation of cognition and attention. Prog Neurobiol. 67(1), 53–83 (2002)
2. Hurd, Y.L., Suzuki, M., Sedvall, G.C.: D1 and D2 dopamine receptor mRNA expression in whole hemisphere sections of the human brain. J. Chem. Neuroanat. 22(1-2), 127–137 (2001)
3. Yang, C.R., Seamans, J.K.: Dopamine D1 receptor actions in layers V-VI rat prefrontal cortex neurons in vitro: modulation of dendritic-somatic signal integration. J. Neurosci. 16(5), 1922–1935 (1996)
4. Seamans, J.K, Yang, C.R: The principal features and mechanisms of dopamine modulation in the prefrontal cortex. Prog. Neurobiol. 74(1), 1–58 (2004)
5. Witkovsky, P.: Dopamine and retinal function. Doc. Ophthalmol. 108(1), 17–40 (2004)
6. Reader, T.A., Quesney, L.F.: Dopamine in the visual cortex of the cat. Experientia 42(11-12), 1242–1244 (1986)
7. Müller, C.P., Huston, J.P.: Dopamine activity in the occipital and temporal cortices of rats: dissociating effects of sensory but not pharmacological stimulation. Synapse 61(4), 254–258 (2007)
8. Carpenter, G.A., Grossberg, S.: A massively parallel architecture for a self-organizing neural pattern recognition machine. Comput. Vis. Graphs Image Proc. 37, 54–115 (1987)
9. Mogami, T., Tanaka, K.: Reward association affects neuronal responses to visual stimuli in macaque te and perirhinal cortices. J. Neurosci. 26(25), 6761–6770 (2006)
10. Rolls, E.T., Judge, S.J., Sanghera, M.K.: Activity of neurones in the inferotemporal cortex of the alert monkey. Brain Res. 130(2), 229–238 (1977)

11. Thorpe, S.J., Rolls, E.T., Maddison, S.: The orbitofrontal cortex: neuronal activity in the behaving monkey. Exp. Brain Res. 49(1), 93–115 (1983)
12. Liu, Z., Richmond, B.J, Murray, E.A, Saunders, R.C, Steenrod, S., Stubblefield, B.K, Montague, D.M, Ginns, E.I: DNA targeting of rhinal cortex D2 receptor protein reversibly blocks learning of cues that predict reward. Proc. Natl. Acad. Sci. 101(33), 12336–12341 (2004)
13. Vitay, J., Hamker, F.H.: Sustained activities and retrieval in a computational model of perirhinal cortex. Submitted to J. Cog. Neurosci. (June 2007)
14. Ranganath, C., D'Esposito, M.: Directing the mind's eye: prefrontal, inferior and medial temporal mechanisms for visual working memory. Curr. Opin. Neurobiol. 15(2), 175–182 (2005)
15. Buckley, M.J., Gaffan, D.: Perirhinal cortex ablation impairs visual object identification. J. Neurosci. 18(6), 2268–2275 (1998)
16. Miller, E.K., Gochin, P.M., Gross, C.G.: Suppression of visual responses of neurons in inferior temporal cortex of the awake macaque monkey by addition of a second stimulus. Brain Res. 616, 25–29 (1993)
17. Hamker, F.H., Wiltschut, J.: Homeostatic scaling and hebbian learning in dynamic rate-coded neurons (in preparation, 2007)
18. Hamker, F.H: The reentry hypothesis: the putative interaction of the frontal eye field, ventrolateral prefrontal cortex, and areas V4, IT for attention and eye movement. Cereb Cortex 15(4), 431–447 (2005)
19. Schultz, W., Dayan, P., Montague, P.R.: A neural substrate of prediction and reward. Science 275(5306), 1593–1599 (1997)
20. Nakamura, K., Ono, T.: Lateral hypothalamus neuron involvement in integration of natural and artificial rewards and cue signals. J. Neurophysiol. 55(1), 163–181 (1986)
21. Semba, K., Fibiger, H.C.: Afferent connections of the laterodorsal and the pedunculopontine tegmental nuclei in the rat: a retro- and antero-grade transport and immunohistochemical study. J. Comp. Neurol. 323(3), 387–410 (1992)
22. Sutton, R.S., Barto, A.G.: Reinforcement Learning: An Introduction. MIT Press, Cambridge, MA (1998)
23. Houk, J.C., Adams, J.L., Barto, A.G.: A model of how the basal ganglia generate and use neural signal that predict reinforcement. In: Houk, J.C., Davis, J.L., Beiser, D.G. (eds.) Models of information processing in the basal ganglia, The MIT Press, Cambridge, MA (1995)
24. Suri, R.E., Schultz, W.: Temporal difference model reproduces anticipatory neural activity. Neural Comput. 13(4), 841–862 (2001)
25. Daw, N.D, Touretzky, D.S: Long-term reward prediction in td models of the dopamine system. Neural Comput. 14(11), 2567–2583 (2002)
26. Kirkpatrick, K., Church, R.M.: Stimulus and temporal cues in classical conditioning. J. Exp. Psychol. Anim. Behav. Process 26(2), 206–219 (2000)
27. Brown, J., Bullock, D., Grossberg, S.: How the basal ganglia use parallel excitatory and inhibitory learning pathways to selectively respond to unexpected rewarding cues. J. Neurosci. 19(23), 10502–10511 (1999)
28. O'Reilly, R.C., Frank, M.J.: Making working memory work: A computational model of learning in the frontal cortex and basal ganglia. Neur. Comput. 18, 283–328 (2006)
29. Horvitz, J.C.: Mesolimbocortical and nigrostriatal dopamine responses to salient non-reward events. Neuroscience 96(4), 651–656 (2000)

30. Cheng, K., Saleem, K.S., Tanaka, K.: Organization of corticostriatal and corticoamygdalar projections arising from the anterior inferotemporal area te of the macaque monkey: a phaseolus vulgaris leucoagglutinin study. J. Neurosci. 17(20), 7902–7925 (1997)
31. Redgrave, P., Gurney, K.: The short-latency dopamine signal: a role in discovering novel actions? Nat. Rev. Neurosci. 7(12), 967–975 (2006)
32. Coizet, V., Comoli, E., Westby, G.W.M., Redgrave, P.: Phasic activation of substantia nigra and the ventral tegmental area by chemical stimulation of the superior colliculus: an electrophysiological investigation in the rat. Eur. J. Neurosci. 17(1), 28–40 (2003)
33. Dommett, E., Coizet, V., Blaha, C.D., Martindale, J., Lefebvre, V., Walton, N., Mayhew, J.E.W., Overton, P.G., Redgrave, P.: How visual stimuli activate dopaminergic neurons at short latency. Science 307(5714), 1476–1479 (2005)
34. Oyster, C.W., Takahashi, E.S.: Responses of rabbit superior colliculus neurons to repeated visual stimuli. J. Neurophysiol. 38(2), 301–312 (1975)
35. Wurtz, R.H., Albano, J.E.: Visual-motor function of the primate superior colliculus. Annu. Rev. Neurosci. 3, 189–226 (1980)
36. Ljungberg, T., Ungerstedt, U.: Sensory inattention produced by 6-hydroxydopamine-induced degeneration of ascending dopamine neurons in the brain. Exp. Neurol. 53(3), 585–600 (1976)
37. Hikosaka, O., Takikawa, Y., Kawagoe, R.: Role of the basal ganglia in the control of purposive saccadic eye movements. Physiol. Rev. 80(3), 953–978 (2000)
38. Hikosaka, O., Nakamura, K., Nakahara, H.: Basal ganglia orient eyes to reward. J. Neurophysiol. 95(2), 567–584 (2006)
39. Sommer, M.A, Wurtz, R.H: Influence of the thalamus on spatial visual processing in frontal cortex. Nature 444(7117), 374–377 (2006)
40. Alexander, G.E., Crutcher, M.D., DeLong, M.R.: Basal ganglia-thalamocortical circuits: parallel substrates for motor, oculomotor, "prefrontal" and "limbic" functions. Prog. Brain Res. 85, 119–146 (1990)
41. Moore, T., Fallah, M.: Control of eye movements and spatial attention. Proc. Natl. Acad. Sci. 98(3), 1273–1276 (2001)
42. Rizzolatti, G., Riggio, L., Dascola, I., Ulmita, C.: Reorienting attention across the horizontal and vertical meridians: Evidence in favor of a premotor theory of attention. Neuropsychol. 25, 31–40 (1987)
43. Silkis, I.: A hypothetical role of cortico-basal ganglia-thalamocortical loops in visual processing. Biosystems 89(1-3), 227–235 (2007)
44. Matsumoto, N., Minamimoto, T., Graybiel, A.M., Kimura, M.: Neurons in the thalamic CM-Pf complex supply striatal neurons with information about behaviorally significant sensory events. J. Neurophysiol. 85(2), 960–976 (2001)
45. Lange, K.W., Robbins, T.W., Marsden, C.D., James, M., Owen, A.M., Paul, G.M.: L-dopa withdrawal in parkinson's disease selectively impairs cognitive performance in tests sensitive to frontal lobe dysfunction. Psychopharmacology (Berl) 107(2-3), 394–404 (1992)
46. Kori, A., Miyashita, N., Kato, M., Hikosaka, O., Usui, S., Matsumura, M.: Eye movements in monkeys with local dopamine depletion in the caudate nucleus. ii. deficits in voluntary saccades. J. Neurosci. 15(1 Pt 2), 928–941 (1995)
47. Goldman-Rakic, P.S.: Cellular basis of working memory. Neuron. 14(3), 477–485 (1995)
48. Fuster, J.M., Alexander, G.E.: Neuron activity related to short-term memory. Science 173, 652–654 (1971)

49. Alexander, G.E.: Selective neuronal discharge in monkey putamen reflects intended direction of planned limb movements. Exp. Brain Res. 67(3), 623–634 (1987)
50. Courtney, S.M., Ungerleider, L.G., Keil, K., Haxby, J.V.: Transient and sustained activity in a distributed neural system for human working memory. Nature 386(6625), 608–611 (1997)
51. Braver, T.S., Barch, D.M., Cohen, J.D.: Cognition and control in schizophrenia: A computational model of dopamine and prefrontal function. Biol. Psychiatry 46(3), 312–328 (1999)
52. Durstewitz, D., Seamans, J.K., Sejnowski, T.J.: Neurocomputational models of working memory. Nat. Neurosci. Supp. 3, 1184–1191 (2000)
53. Compte, A., Brunel, N., Goldman-Rakic, P.S., Wang, X.J.: Synaptic mechanisms and network dynamics underlying spatial working memory in a cortical network model. Cereb. Cortex 10(9), 910–923 (2000)
54. Brunel, N., Wang, X.J.: Effects of neuromodulation in a cortical network model of object working memory dominated by recurrent inhibition. J. Comput. Neurosci. 11(1), 63–85 (2001)
55. Dreher, J.C., Guigon, E., Burnod, Y.: A model of prefrontal cortex dopaminergic modulation during the delayed alternation task. J. Cogn. Neurosci. 14(6), 853–865 (2002)
56. Frank, M.J., Loughry, B., O'Reilly, R.C.: Interactions between frontal cortex and basal ganglia in working memory: a computational model. Cogn. Affect Behav. Neurosci. 1(2), 137–160 (2001)
57. Postle, B.R., D'Esposito, M.: Dissociation of human caudate nucleus activity in spatial and nonspatial working memory: an event-related fmri study. Brain Res. Cogn. Brain Res. 8(2), 107–115 (1999)
58. Lewis, S.J G, Dove, A., Robbins, T.W, Barker, R.A, Owen, A.M: Striatal contributions to working memory: a functional magnetic resonance imaging study in humans. Eur. J. Neurosci. 19(3), 755–760 (2004)
59. Wilson, C.J., Kawaguchi, Y.: The origins of two-state spontaneous membrane potential fluctuations of neostriatal spiny neurons. J. Neurosci. 16(7), 2397–2410 (1996)
60. Middleton, F.A, Strick, P.L: Basal-ganglia 'projections' to the prefrontal cortex of the primate. Cereb Cortex 12(9), 926–935 (2002)
61. Ashby, F.G., Ell, S.W, Valentin, V.V, Casale, M.B: Frost: a distributed neurocomputational model of working memory maintenance. J. Cogn. Neurosci. 17(11), 1728–1743 (2005)
62. Gruber, A.J, Dayan, P., Gutkin, B.S, Solla, S.A: Dopamine modulation in the basal ganglia locks the gate to working memory. J. Comput. Neurosci. 20(2), 153–166 (2006)
63. Funahashi, S., Bruce, C.J., Goldman-Rakic, P.S.: Mnemonic coding of visual space in the monkey's dorsolateral prefrontal cortex. J. Neurophysiol. 61, 331–349 (1989)
64. Koch, C., Ullman, S.: Shifts in selective visual attention: towards the underlying neural circuitry. Hum. Neurobiol. 4, 219–227 (1985)
65. Desimone, R., Duncan, J.: Neural mechanisms of selective visual attention. Ann. Rev. Neurosci. 18, 193–222 (1995)
66. Itti, L., Koch, C.: Computational modelling of visual attention. Nat. Rev. Neurosci. 2, 1–10 (2001)
67. Deco, G., Rolls, E.T: A neurodynamical cortical model of visual attention and invariant object recognition. Vision Res. 44(6), 621–642 (2004)
68. Luck, S.J., Vogel, E.K.: The capacity of visual working memory for features and conjunctions. Nature 390(6657), 279–281 (1997)

69. Lee, D., Chun, M.M.: What are the units of visual short-term memory, objects or spatial locations? Percept Psychophys. 63(2), 253–257 (2001)
70. Ranganath, C.: Working memory for visual objects: complementary roles of inferior temporal, medial temporal, and prefrontal cortex. Neurosci. 139(1), 277–289 (2006)
71. Supèr, H., Spekreijse, H., Lamme, V.A.: A neural correlate of working memory in the monkey primary visual cortex. Science 293(5527), 120–124 (2001)
72. Rolls, E.T.: Hippocampo-cortical and cortico-cortical backprojections. Hippocampus 10(4), 380–388 (2000)
73. Sakai, K., Rowe, J.B., Passingham, R.E.: Active maintenance in prefrontal area 46 creates distractor-resistant memory. Nat. Neurosci. 5(5), 479–484 (2002)
74. D'Esposito, M., Postle, B.R., Ballard, D., Lease, J.: Maintenance versus manipulation of information held in working memory: an fMRI study. Brain and Cognition 41, 66–86 (1999)
75. Parent, A., Cicchetti, F.: The current model of basal ganglia organization under scrutiny. Mov. Disord. 13(2), 199–202 (1998)