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Attention as an effect not a cause

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Attention is commonly thought to be important for managing the limited resources available in sensory areas of the neocortex. Here we present an alternative view that attention arises as a byproduct of circuits centered on the basal ganglia involved in value-based decision making. The central idea is that decision making depends on properly estimating the current state of the animal and its environment and that the weighted inputs to the currently prevailing estimate give rise to the filter-like properties of attention. After outlining this new framework, we describe findings from physiological, anatomical, computational, and clinical work that support this point of view. We conclude that the brain mechanisms responsible for attention employ a conserved circuit motif that predates the emergence of the neocortex.

Frameworks for thinking about attention

Current thinking about attention is guided by several well-established metaphors: 'bottleneck', 'spotlight', 'zoom lens' [1–3]. These metaphors share the central idea that there is a fundamental resource limitation that constrains information processing by the brain [4]. This resource limitation enforces trade-offs – some objects are selected as the focus of perception and action but only at the expense of others, which are given lower priority. Such metaphors also imply that attention is responsible for determining how sensory data are represented in the brain: what is illuminated by the spotlight? In this opinion article, we present an alternate framework that does not treat attention as a cause but instead views it as an effect – in particular, that it arises from processes that determine how sensory (and other) data are interpreted by the brain. We start by outlining and comparing these two frameworks.

Attention as a regulator of sensory representations

Attention is most often described as a causal agent that exerts its effects on the sensory side of the complex cascade of sensory–motor processes in the brain (Figure 1A). This perspective was first described explicitly in the filter model of Broadbent (1958), which posited that only a limited subset of sensory signals reached later stages of processing. The original model placed the filter directly after the extraction of basic stimulus features, prompting a vigorous debate about the location of the filter [1]. There is now a general consensus that the filter-like property of attention limits but does not fully exclude basic features from further elaboration and that the curating of sensory data may occur either early or late in sensory processing [5,6].

The idea that sensory data are actively filtered has been strikingly corroborated by results from neurophysiology experiments. It is well documented that neurons in sensory areas of the cerebral cortex modulate their firing depending on how attention is allocated and that this effect occurs both early and late in processing. For example, in the visual system, modulation with attention is known to occur both at relatively early stages of visual processing, such as among edge-detecting neurons in the primary visual cortex, and also at later stages where more complex features are represented [7,8].

These physiology experiments have also identified a central principle for achieving the filtering of sensory data – competition for representation within the neocortex (Figure 1A). As demonstrated in several influential models [7,9–13], computations occurring in neocortical circuits can implement competition between sensory inputs that results in the enhanced representation of some signals at the expense of others, consistent with the filter-like properties of attention.

Moreover, this competition is believed to be regulated by feedback signals from later stages of processing – in particular, the frontal and parietal cortex [13–15], and also the superior colliculus (SC) in the midbrain [16]. These brain regions provide 'priority' signals that bias competition for representation in sensory cortex, establishing routes for both top-down and bottom-up control of attention. By actively filtering the representation of sensory signals, these cortical attention mechanisms control which data is then available to drive perception, action, and memory.

Attention as an effect of interpreting sensory (and other) data

Our alternative framework views attention as an effect rather than a causal agent. The central premise of this framework is that attention arises as a functional consequence of circuits centered on the basal ganglia involved in value-based motor and non-motor decision making (Figure 1B). Here we introduce the key features of this framework; in the next section, we present some lines of evidence in its favor.

Good decision making depends crucially on properly identifying the current state of the animal and its environment. If the state cannot be identified, the subject is left confused and indecisive. Defining the 'state' is complex, and involves interpreting many diverse sources of information – not only the sensed features of the external world, but also the internal status of the subject, their prior knowledge, and their ongoing needs. At each moment,

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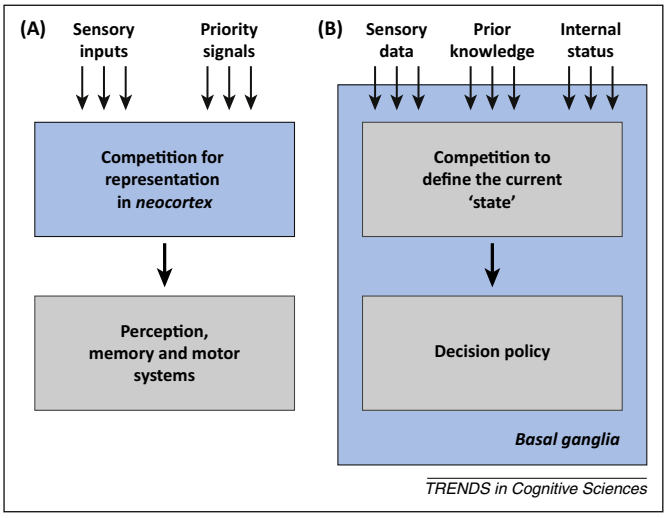

Figure 1. Two frameworks for thinking about attention. **(A)** Attention as a regulator of sensory representations. In this commonly accepted framework, attention acts by regulating how sensory inputs are represented in sensory areas of the neocortex. Sensory inputs compete with one another for representation and this competition is biased by priority signals from other cortical areas. Perception, memory, and motor systems are then driven by the resulting filtered sensory signals. **(B)** Attention as an effect of interpreting sensory and other data. In this alternative framework, attention is a byproduct of circuits centered on the basal ganglia involved in value-based decision making. Here, competition does not affect how sensory inputs are represented but instead determines which estimate of the 'state' provides the best match to the current sensory data, prior knowledge, and internal status of the subject. The dominant estimate of the state then determines which decision policy is followed.

the subject must consider several possible estimates of the state; these different estimates could be generated by differentially weighting the possible inputs using something akin to Bayesian inference [17–19].

There are good reasons to presume that this process of state estimation involves the input nuclei of the basal ganglia, especially the striatum [19,20]. The striatum receives converging inputs from many sources, prominently from the cerebral cortex, but also from the thalamus, the amygdala, and elsewhere [21–24]. The striatum also receives dopamine signals that guide reinforcement learning; the learned associations between a particular state and the set of actions with the highest expected values establish a decision policy that guides the subject's behavior [25–29].

Thinking about context-dependent decision making in this way leads us to a very different model. Specifically, this alternative framework centers on competition between possible interpretations of the current state by the basal ganglia (Figure 1B) rather than competition to determine how sensory data is represented in the neocortex (Figure 1A). Because each possible state differs in the weights it assigns to the various inputs, each state can be viewed as a candidate template [30], with the best-matching template dominating the competition. As circumstances change, a candidate state that provides a poor match in one round of competition could emerge as predominant just moments later. This results in a linked chain of states, where the transition from one to the next is precipitated by some event, or change in an internal variable, that gives the new state more support than the preceding one and carries along with it a new decision policy (also see Box 1).

Attention can be explained by this competition between possible states. Because different sensory inputs and types of knowledge contribute unequally to different states, their influence on perception and action will be limited by the strength of their contribution to the state that currently dominates the competition. From this viewpoint, it is not necessary to change how sensory signals are represented to generate the phenomenology of attention. The filter-like properties associated with attention result from the particular weights applied to the sensory and non-sensory inputs that define the current state. The pattern of weighted inputs gives the appearance of limited sensory resources, because increasing the weight of one input necessarily involves reducing the proportional weight of all of the other inputs.

This framework also provides a definition for shifts of attention – they correspond to transitions from one dominant state to the next. If the transition is triggered by unexpected sensory data, this might be considered a stimulus-driven or bottom-up shift of attention; if it is prompted by a change based on internal state or knowledge, this might be an endogenous or top-down shift of attention. However, these categories are somewhat arbitrary [31]. In principle, shifts of attention could come in many different flavors, because transitions between states could be prompted by changes along any of the dimensions represented by the diverse inputs to the striatum.

Some arguments in favor of this new framework

We start with results from recent physiology studies of the midbrain that are difficult to explain with the conventional view of attention and then consider several other types of observations that implicate a centralized decision mechanism in the basal ganglia.

The SC regulates attention but not through the visual cortex

The SC is a highly conserved midbrain structure that contains a retinotopically organized map of the visual world. The primate SC is best known for its role in controlling orienting movements of the eyes and head [32], but recent experiments have confirmed that the SC is also necessary for the control of spatial attention [33]. For example, when activity in the SC is locally and reversibly suppressed (Figure 2A,B), animals have difficulty performing attention tasks for stimuli placed in the affected part of the visual field [34]. The deficit resembles clinical cases of extinction [35]: animals mostly ignore cued stimuli in the inactivated region when they compete with irrelevant stimuli placed elsewhere, but discrimination ability is largely intact when stimuli appear alone, even in the affected region.

Taken by themselves, these findings seem consistent with the established idea that attention works by controlling how sensory data are represented in the brain: the SC could be the source of 'priority' signals that bias competition for representation in sensory cortex. However, when directly tested, this interpretation was found to be incorrect. The test involved recording from neurons in cortical areas necessary for processing the sensory signals used in the task, at the same time that activity in the SC was suppressed (Figure 2C,D). The unexpected finding was that the enhanced responses of sensory neurons to attended stimuli (Figure 2E,F) were preserved during

Box 1. An explanation for attention task performance

During a typical attention task involving the detection of a visual probe and a button-press response, the subject would transition between several different states (Figure I). At first **(A)**, the subject might have had a neutral state that weighted inputs approximately equally and placed slightly higher value on not pressing the button to avoid restarting the trial **(B)**. After the spatial cue **(C)**, the subject would shift to a state (e.g., 'cued motion on the left') that more heavily weighted inputs about motion and remembered cues on the left and that placed high value on not pressing the button **(D)** to avoid committing a false alarm. Finally, the occurrence of the motion change **(E)** would trigger a transition to a belief state (e.g., 'the relevant change just happened') that heavily weighted inputs about motion changes on the left side and that placed very high value on pressing the button **(F)** to collect the reward at the end of the trial.

This scheme can explain the deficits in attention caused by SC inactivation. Focal inactivation temporarily suppresses spatially specific signals that provide crucial support for states involving those parts of the visual field **(G)**. Without those inputs from the SC, it becomes much more difficult for those states to win against competing states, even if all of the other inputs, including those from the visual cortex, remain intact. As a result, the subject ends in an erroneous state that leads to incorrect decisions **(H)**. This would be consistent with the idea that the SC is part of a spatial indexing system that identifies which signals elsewhere in the brain should be used to guide actions and determine the content of perception [33].

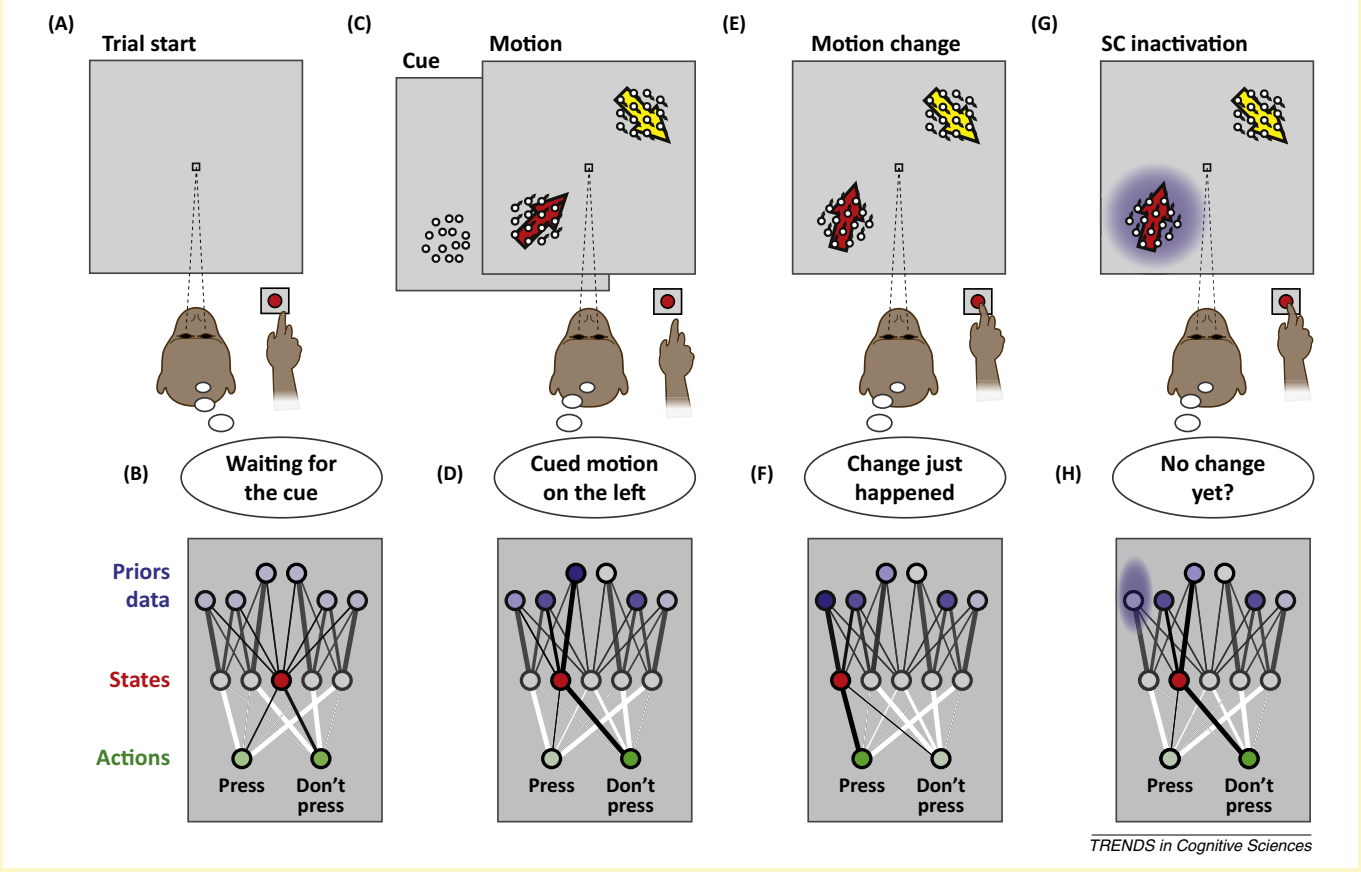

Figure I. Illustration of possible state changes during the performance of a typical attention task by an intact (A–F) and impaired (G–H) subject.

SC inactivation [36]. Indeed, all of the well-known signatures of attention in the cortex (i.e., changes in firing rate, Fano factor, interneuronal correlations) remained intact and unchanged, despite concomitant behavioral deficits in the covert attention task.

These findings directly contradict the conventional view of attention: if attention operates by regulating how sensory signals are represented in the neocortex, some aspect of cortical activity should have changed to cause the deficits in attention performance.

Subcortical routes lead to the basal ganglia

If the SC does not affect attention through the visual cortex, which routes does it use? Several prominent possibilities involve pathways through the thalamus that project to the striatum. One route out of the SC is to the medial dorsal nucleus of the thalamus, which provides corollary discharge signals about saccadic eye movements to the frontal eye fields in the prefrontal cortex [37]. The medial dorsal nucleus also conveys signals to the striatum, both directly [38] and indirectly through its widespread projections to the prefrontal cortex [39].

Another important route through the thalamus is from the intermediate layers of the SC to the parafascicular nucleus (Pf) [40], which in turn projects broadly throughout the caudate nucleus [41,42]; indeed, the Pf appears to be the predominant source of thalamic inputs to both the direct and indirect pathways in the striatum [38]. Activity in this circuit has been directly implicated in the performance of spatial attention tasks: neurons in the Pf are

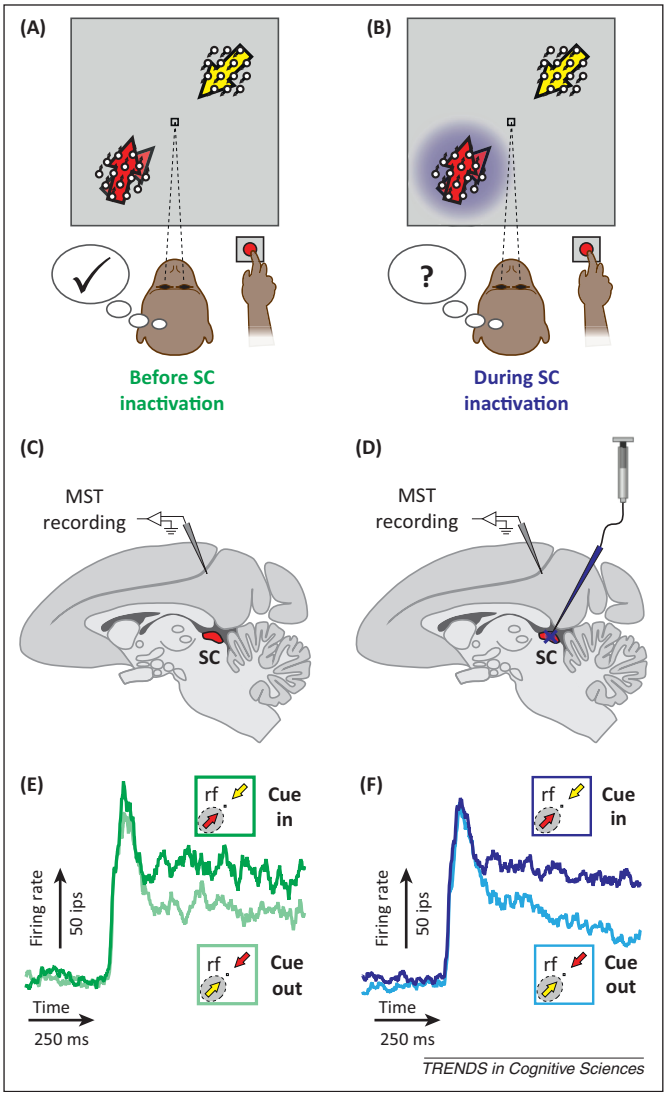

Figure 2. Attention-related modulation in the visual cortex remains intact during superior colliculus (SC) inactivation, despite SC inactivation causing large deficits in attention. **(A)** Before SC inactivation, the animal was able to correctly detect the change in motion direction, reported by pressing a button while maintaining fixation. The red arrow schematically indicates the cued motion patch that should be monitored to obtain a reward at the end of the trial. The yellow arrow indicates the irrelevant motion patch that should be ignored. These colored arrows were not present during the task. **(B)** During SC inactivation, the animal showed large and spatially specific deficits in their ability to detect changes in the cued stimulus. The blue shaded region indicates the part of the visual field affected by the SC inactivation. Neuronal activity was recorded in the medial superior temporal area (MST) before (**C**) and during **(D)** SC inactivation. Despite the large deficits in task performance, attention-related modulation of MST neurons by spatial cues was present both before **(E)** and during **(F)** SC inactivation. This modulation is indicated by the elevated activity when the cued stimulus was inside versus outside the MST neuron's receptive field (rf). Adapted from [36].

modulated by spatial cues and inactivation of the Pf increases reaction times during attention tasks [43]. The Pf also appears in human functional MRI (fMRI) data as one of the most reliably activated sites in the thalamus during a covert attention task [44].

There is also a direct projection from the intermediate layers of the SC to the substantia nigra pars compacta (SNpc) [45–48], which in turn provides a dopaminergic projection to the striatum. Neurons in the SNpc are best known for their bursts of activity that provide reward prediction error signals that guide reinforcement learning and value-based action selection by the basal ganglia [25,26,49]. Among the several possible sources of inputs driving the phasic responses of dopaminergic SNpc neurons, activity from the SC could provide signals related to the detection of salient sensory events [50,51].

Thus, many of the anatomical routes out of the SC to other sensory–motor structures lead to the basal ganglia. Although these routes may seem exotic, they belong to a highly conserved circuit motif.

A conserved circuit motif for attention

Despite its prominence in current thinking about selective attention, the neocortex is not actually necessary. Many vertebrate species lacking a well-developed neocortex nonetheless demonstrate clear behavioral signs of attention, including pigeons [52], peacocks [53], owls, [54], frogs [55,56], salamanders [57], and zebrafish [58]. For example, zebrafish can perform the same type of complex multi-choice serial reaction-time task [59] that is commonly used to assess attention function in rodents [60].

Given this conservation in function, one might expect a corresponding conservation in brain organization, and a strong case can be made that this neural motif involves circuits through the basal ganglia [61,62]. Despite changes in brain size, the basal ganglia play a crucial role in value-based decision making in all vertebrates, even as the details of the circuits undergo dramatic revisions [61,63]. The changes in sensory inputs are especially relevant for thinking about attention. In amphibians, the basal ganglia receive inputs about salient sensory events directly from the dorsal thalamus and SC, whereas in reptiles and birds sensory signals undergo additional processing by being rerouted from the thalamus through the dorsal ventricular ridge, a distinctive laminar structure not present in other vertebrates. In mammals, the basal ganglia receive most of their inputs from the massively expanded neocortex, even while retaining inputs from the dorsal thalamus and the likely remains of the dorsal ventricular ridge, the claustroamygdalar complex. From this viewpoint, the neocortex is important for attention in mammals, not because it fundamentally changes the circuit, but because it is where the process of extracting salient features has been outsourced.

The first evolutionary branch with brains that support attention may have started even earlier – invertebrates (e.g., insects) also exhibit behavioral signs of attention. Honeybees can use probabilistic spatial cues to improve their discrimination between visual stimuli [64]. Similarly, *Drosophila* can use spatial cues to bias their optomotor flying response [65,66]. In *Drosophila*, the ability to interpret competing visual cues depends on the mushroom bodies, sensory–motor structures that receive dense dopaminergic inputs and play a central role in learned, context-dependent responses [67,68]. Thus, despite their evolutionary distance from primates, even invertebrate brains provide evidence of a conserved circuit motif for attention.

Artificial systems share a similar architecture

Work on artificial systems has generally converged on strategies similar to these biological solutions – a centralized process of state estimation linked to a set of decision rules. For example, cognitive architectures are designed to reproduce the intelligent, goal-directed behavior of

humans and typically include properties analogous to those outlined in Figure 1: the representation of knowledge and data, the use of knowledge based on rules, and mechanisms for acquiring new knowledge and rules [69].

In robotics, complex behaviors can be achieved using a distributed, layered architecture that builds up responses from many smaller components, even without a centralized controller [70]. However, if conflicts among the components are resolved through local mechanisms alone, the resulting behavior is fixed and inflexible, especially as the system becomes more complex. Introducing a centralized mechanism that includes state estimation is an efficient way to solve this problem [17,71].

Notably, these approaches do not involve explicitly adding attention; the filter-like properties of behavior are byproducts of the decision-making process.

Clinical evidence

Damage to the striatum (e.g., after stroke) causes deficits in attention. One common symptom is spatial neglect – the failure to explore or process objects adequately across space, especially in the presence of distracters. Spatial neglect is commonly associated with damage to the right parietal cortex, although subcortical lesions can also cause neglect, and the striatum in the right hemisphere, along with the thalamic pulvinar nucleus, is the primary subcortical structure associated with spatial neglect [72,73]. Damage to the striatum in the left hemisphere also causes deficits in attention tasks but not spatial neglect; these deficits tend to be problems with divided attention and working memory [74].

Parkinson's disease is primarily associated with problems in motor control, but patients also have cognitive impairments including deficits in attention and visual perception. The attention deficits are mostly observed during challenging tasks; for example, searching for an ambiguous visual target among distracters [75,76]. It is also not uncommon for patients to experience visual hallucinations and misperceptions [77,78] that are not just side effects of treatment [79]. Such hallucinations could be characterized as erroneous estimates of the current state.

Thus, disruptions of the basal ganglia cause problems with attention. Conversely, the most common disorders of attention also point to functions centered on the basal ganglia (Box 2).

Box 2. Attention-deficit hyperactivity disorder

Attention-deficit hyperactivity disorder (ADHD) is one of the most prevalent disorders of attention, arising as a neurodevelopmental problem during childhood and often persisting into adulthood. ADHD has two main features – inattention and impulsivity [88]. Inattention refers to the tendency to be easily distracted, making it difficult to maintain the mental focus that is required to organize and complete tasks. Impulsivity refers to the tendency to be restless and impatient, leading to inappropriate bursts of activity; for example, failing to wait one's turn in conversation. Sensory processing *per se* appears to be mostly unaffected in ADHD.

Although the exact etiology of ADHD is uncertain, pharmacological treatments for ADHD likewise implicate circuits involved in regulating behavior rather than processing sensory inputs. The most commonly used drugs to treat ADHD are stimulants like methylphenidate, which increases the levels of dopamine in the basal ganglia by acting on the dopamine transporter and increases the levels of noradrenaline in the prefrontal cortex by blocking the noradrenaline transporter [89]. These changes in neurotransmitter action are hypothesized to improve the ability to detect important events and reinforce appropriate behavioral reactions [90]. Consistent with these drug effects, ADHD is associated with polymorphisms in many genes related to neurotransmitter action [91–93]. Several of these involve dopamine, such as the dopamine transporter and dopamine receptor genes, but non-dopamine genes have also been identified.

Animal models of ADHD also highlight the importance of dopamine and circuits related to organizing actions. The most commonly used animal model is the spontaneously hypertensive rat [94,95]. These animals exhibit the key features of ADHD – inattentiveness and impulsivity – without any obvious sensory impairment; they also possess unusual variations in the gene for the dopamine transporter. In mice, knocking out individual genes has identified possible relationships between particular gene products and aspects of behavior in ADHD. For example, mice lacking the gene for the dopamine transporter show deficits in learning and memory tasks as well as hyperactivity [96,97]. Similarly, knockout of the guanylyl cyclase receptor gene in midbrain dopamine neurons produce mice that exhibit ADHD-like behaviors [98].

Thus, much of what we know about the etiology of ADHD highlights the importance of circuits related to decision making and learning rather than sensory processing.

Possible implications

The primary result of this framework is that it does away with attention as a causal agent and instead explains attention as an effect of value-based decision making. This could be challenging. We are accustomed to using attention as an explanation and our descriptive metaphors encourage this way of thinking. However, viewing attention as an effect rather than a cause has the advantage of eliminating the need to find the homunculus that aims the spotlight of attention. Instead, we should be able to explain the functional circuits that give rise to the phenomenology of attention without using the word 'attention'.

This framework also emphasizes the relationship between attention and learning. We presume that the weights that define each candidate state – and that set the filter-like properties of attention – are not innate or fixed, but learned. Thus, the particular propensities of attention in an individual would develop over time through learning mechanisms that determine which states and their associated value functions are most likely to result in rewarding outcomes. This has implications for how we think about the performance of attention-demanding tasks, like reading, and could help refine the use of behavioral therapies in cases of attention disorders.

An important constraint in this framework is that, because each state is associated with a unique value function, adopting a different value function requires changing the state. This constraint implies that expanding one's behavioral repertoire requires adding new states – they are needed to provide novel points of association for the value functions. One function of the extensive inputs from the prefrontal cortex to the striatum in primates could be to provide additional non-sensory inputs to expand the number of states and associated value functions that can be acquired [80,81].

There are, of course, limits to the aspects of behavior that can be controlled through this mechanism. This is evident from the fact that decision policies bias behavior but do not dictate final outcomes. This presumably reflects the autonomy of downstream circuits to make decisions

based on information that needs to be handled quickly, is impractical to share, or is simply irrelevant for competition between states. Furthermore, not all instances of sensory–motor selection involve this type of flexible control; perhaps the most dramatic example is habit formation, which also depends on circuitry in the basal ganglia [82,83].

The contrast between goal-directed behavior and habits raises an important unresolved issue in behavioral neuroscience. We suspect that one of the more functionally significant aspects of the circuits we have outlined is that they provide a mechanism for switching how salient events are handled: instead of letting an event be swallowed by an existing state linked to a habitual response, circuits implicated in attention may open a window of opportunity for the event to define a novel state associated with new goal-directed outcomes [84–87].

Concluding remarks

We have presented a novel framework that does not treat attention as a cause but instead views it as an effect arising from value-based decision-making mechanisms centered on the basal ganglia. This viewpoint is supported by converging lines of evidence from physiology, neuroanatomy, modeling, and clinical findings. Although speculative, this framework provides new ways to think about the neuronal circuits, molecular mechanisms, and computational principles underlying this important mental function (Box 3).

Box 3. Outstanding questions

- What is the contribution of each pathway from the SC to the basal ganglia? Do they provide transient signals related to the detection of behaviorally relevant events, sustained signals related to memory and expectation, or both? Do the signals conveyed along these pathways arise from the same neurons in the SC or is there functional segregation?
- What is the functional connectome of the circuits within the striatum [99]? Which striate neurons are targeted by the inputs from the substantia nigra and parafascicular nucleus: medium spiny neurons, tonically active neurons, or both? How does local circuitry within the striatum, across matriosomes and striosomes, contribute to the establishment of a state?
- How does basal ganglia circuitry contribute to the properties of attention? What is the functional role of the direct and indirect pathways? How do inhibitory feedback circuits within the basal ganglia contribute to competition between states and the transition from one belief state to another? How do disruptions in dopamine and acetylcholine release alter the properties of attention?
- How does the topography of inputs to the striatum influence the number and type of states? Are certain states more likely simply because the anatomical arrangement of inputs makes them easier to support? Could different forms of attention be explained by the dominance of particular classes of inputs in determining the outcome from competition between states? For example, is spatial attention observed when retinotopic inputs dominate, whereas feature-based attention is observed when signals about particular features (e.g., color, orientation) are critical for resolving the competition?
- What is the function of cortical gain modulation? Does it help bias competition between states to achieve effects like those described in cortical models of attention [7,9]? Could it be due to feedback from the basal ganglia to the cortex, reinforcing sensory data that support a particular state and thereby also modifying the content of perception [100]?
- How are these brain mechanisms related to computational models of attention based on perceptual templates and signal detection theory [101,102]? Conversely, how might computational models be used to deduce the functional roles of cortical and subcortical circuits?
- Why are there priority maps in the cortex as well as the SC? Are cortical priority maps franchises of functions originally established in subcortical circuits? Do they address a functional limitation in the basal ganglia circuitry; for example, to help avoid flooding the competition with an excess of near-equally weighted states that might otherwise cause confusion and delay?

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References

1 Treisman, A.M. (1969) Strategies and models of selective attention. *Psychol. Rev.* 76, 282–299
2 Eriksen, C.W. and St James, J.D. (1986) Visual attention within and around the field of focal attention: a zoom lens model. *Percept. Psychophys.* 40, 225–240
3 Posner, M.I. *et al.* (1980) Attention and the detection of signals. *J. Exp. Psychol.* 109, 160–174
4 Broadbent, D.E. (1958) *Perception and Communication*, Pergamon Press
5 Driver, J. (2001) A selective review of selective attention research from the past century. *Br. J. Psychol.* 92, 53–78
6 Carrasco, M. (2011) Visual attention: the past 25 years. *Vision Res.* 51, 1484–1525
7 Desimone, R. and Duncan, J. (1995) Neural mechanisms of selective visual attention. *Annu. Rev. Neurosci.* 18, 193–222
8 Reynolds, J.H. and Chelazzi, L. (2004) Attentional modulation of visual processing. *Annu. Rev. Neurosci.* 27, 611–647
9 Reynolds, J.H. and Heeger, D.J. (2009) The normalization model of attention. *Neuron* 61, 168–185
10 Deco, G. and Rolls, E.T. (2005) Neurodynamics of biased competition and cooperation for attention: a model with spiking neurons. *J. Neurophysiol.* 94, 295–313
11 Treue, S. and Martínez Trujillo, J.C. (1999) Feature-based attention influences motion processing gain in macaque visual cortex. *Nature* 399, 575–579
12 Bundesen, C. (1990) A theory of visual attention. *Psychol. Rev.* 97, 523–547
13 Kastner, S. and Ungerleider, L.G. (2000) Mechanisms of visual attention in the human cortex. *Annu. Rev. Neurosci.* 23, 315–341
14 Squire, R.F. *et al.* (2013) Prefrontal contributions to visual selective attention. *Annu. Rev. Neurosci.* 36, 451–466
15 Bisley, J.W. and Goldberg, M.E. (2010) Attention, intention, and priority in the parietal lobe. *Annu. Rev. Neurosci.* 33, 1–21
16 Fecteau, J.H. and Munoz, D.P. (2006) Salience, relevance, and firing: a priority map for target selection. *Trends Cogn. Sci.* 10, 382–390
17 Kaelbling, L.P. *et al.* (1998) Planning and acting in partially observable stochastic domains. *Artif. Intell.* 101, 99–134
18 Doya, K. (2007) Reinforcement learning: computational theory and biological mechanisms. *HFSP J.* 1, 30–40
19 Rao, R.P.N. (2010) Decision making under uncertainty: a neural model based on partially observable Markov decision processes. *Front. Comput. Neurosci.* 4, 146
20 Samejima, K. and Doya, K. (2007) Multiple representations of belief states and action values in corticobasal ganglia loops. *Ann. N. Y. Acad. Sci.* 1104, 213–228
21 Zorrilla, E.P. and Koob, G.F. (2013) Amygdalostriatal projections in the neurocircuitry for motivation: a neuroanatomical thread through the career of Ann Kelley. *Neurosci. Biobehav. Rev.* 37, 1932–1945
22 Selemon, L.D. and Goldman-Rakic, P.S. (1985) Longitudinal topography and interdigitation of corticostriatal projections in the rhesus monkey. *J. Neurosci.* 5, 776–794
23 Nakano, K. *et al.* (1990) Topographical projections from the thalamus, subthalamic nucleus and pedunculopontine tegmental nucleus to the striatum in the Japanese monkey, *Macaca fuscata*. *Brain Res.* 537, 54–68

24 Peck, C.J. *et al.* (2013) The primate amygdala combines information about space and value. *Nat. Neurosci.* 16, 340–348
25 Schultz, W. (2010) Dopamine signals for reward value and risk: basic and recent data. *Behav. Brain Funct.* 6, 24
26 Lee, D. *et al.* (2012) Neural basis of reinforcement learning and decision making. *Annu. Rev. Neurosci.* 35, 287–308
27 Ito, M. and Doya, K. (2011) Multiple representations and algorithms for reinforcement learning in the cortico-basal ganglia circuit. *Curr. Opin. Neurobiol.* 21, 368–373
28 Graybiel, A.M. *et al.* (1994) The basal ganglia and adaptive motor control. *Science* 265, 1826–1831
29 Samejima, K. *et al.* (2005) Representation of action-specific reward values in the striatum. *Science* 310, 1337–1340
30 Brunelli, R. and Poggio, T. (1993) Face recognition: features versus templates. *IEEE Trans. Pattern Anal. Mach. Intell.* 15, 1042–1052
31 Awh, E. *et al.* (2012) Top-down versus bottom-up attentional control: a failed theoretical dichotomy. *Trends Cogn. Sci.* 16, 437–443
32 Gandhi, N.J. and Katnani, H.A. (2011) Motor functions of the superior colliculus. *Annu. Rev. Neurosci.* 34, 205–231
33 Krauzlis, R.J. *et al.* (2013) Superior colliculus and visual spatial attention. *Annu. Rev. Neurosci.* 36, 165–182
34 Lovejoy, L.P. and Krauzlis, R.J. (2010) Inactivation of primate superior colliculus impairs covert selection of signals for perceptual judgments. *Nat. Neurosci.* 13, 261–266
35 Vuilleumier, P.O. and Rafal, R.D. (2000) A systematic study of visual extinction Between- and within-field deficits of attention in hemispatial neglect. *Brain* 123, 1263–1279
36 Zenon, A. and Krauzlis, R.J. (2012) Attention deficits without cortical neuronal deficits. *Nature* 489, 434–437
37 Sommer, M.A. and Wurtz, R.H. (2008) Brain circuits for the internal monitoring of movements. *Annu. Rev. Neurosci.* 31, 317–338
38 Wall, N.R. *et al.* (2013) Differential innervation of direct- and indirect-pathway striatal projection neurons. *Neuron* 79, 347–360
39 Parthasarathy, H.B. *et al.* (1992) Distributed but convergent ordering of corticostriatal projections: analysis of the frontal eye field and the supplementary eye field in the macaque monkey. *J. Neurosci.* 12, 4468–4488
40 Harting, J.K. *et al.* (1980) Ascending pathways from the monkey superior colliculus: an autoradiographic analysis. *J. Comp. Neurol.* 192, 853–882
41 Sadikot, A.F. *et al.* (1992) Efferent connections of the centromedian and parafascicular thalamic nuclei in the squirrel monkey: a light and electron microscopic study of the thalamostriatal projection in relation to striatal heterogeneity. *J. Comp. Neurol.* 320, 228–242
42 Sadikot, A.F. and Rymar, V.V. (2009) The primate centromedian–parafascicular complex: anatomical organization with a note on neuromodulation. *Brain Res. Bull.* 78, 122–130
43 Minamimoto, T. and Kimura, M. (2002) Participation of the thalamic CM–Pf complex in attentional orienting. *J. Neurophysiol.* 87, 3090–3101
44 Hulme, O.J. *et al.* (2010) Spatially distributed encoding of covert attentional shifts in human thalamus. *J. Neurophysiol.* 104, 3644–3656
45 Dommett, E. *et al.* (2005) How visual stimuli activate dopaminergic neurons at short latency. *Science* 307, 1476–1479
46 Comoli, E. *et al.* (2003) A direct projection from superior colliculus to substantia nigra for detecting salient visual events. *Nat. Neurosci.* 6, 974–980
47 May, P.J. *et al.* (2009) Tectonigral projections in the primate: a pathway for pre-attentive sensory input to midbrain dopaminergic neurons. *Eur. J. Neurosci.* 29, 575–587
48 McHaffie, J.G. *et al.* (2006) A direct projection from superior colliculus to substantia nigra pars compacta in the cat. *Neuroscience* 138, 221–234
49 Doll, B.B. *et al.* (2012) The ubiquity of model-based reinforcement learning. *Curr. Opin. Neurobiol.* 22, 1075–1081
50 Redgrave, P. *et al.* (2008) What is reinforced by phasic dopamine signals? *Brain Res. Rev.* 58, 322–339
51 Schultz, W. and Romo, R. (1990) Dopamine neurons of the monkey midbrain: contingencies of responses to stimuli eliciting immediate behavioral reactions. *J. Neurophysiol.* 63, 607–624
52 Shimp, C.P. and Friedrich, F.J. (1993) Behavioral and computational models of spatial attention. *J. Exp. Psychol. Anim. Behav. Process.* 19, 26–37
53 Yorzinski, J.L. and Platt, M.L. (2014) Selective attention in peacocks during predator detection. *Anim. Cogn.* 17, 767–777
54 Johnen, A. *et al.* (2001) Spatial attention modulates sound localization in barn owls. *J. Neurophysiol.* 85, 1009–1012
55 Ingle, D. (1975) Focal attention in the frog: behavioral and physiological correlates. *Science* 188, 1033–1035
56 Traub, B. and Elepfandt, A. (1990) Sensory neglect in a frog: evidence for early evolution of attentional processes in vertebrates. *Brain Res.* 530, 105–107
57 Ruhl, T. and Dicke, U. (2012) The role of the dorsal thalamus in visual processing and object selection: a case of an attentional system in amphibians. *Eur. J. Neurosci.* 36, 3459–3470
58 Echevarria, D.J. *et al.* (2011) Assessing attention in the zebrafish. *Prog. Neuropsychopharmacol. Biol. Psychiatry* 35, 1416–1420
59 Parker, M.O. *et al.* (2012) Development and implementation of a three-choice serial reaction time task for zebrafish (*Danio rerio*). *Behav. Brain Res.* 227, 73–80
60 Higgins, G.A. and Breysse, N. (2001) *Rodent Model of Attention: The 5-Choice Serial Reaction Time Task*, John Wiley & Sons
61 Reiner, A. *et al.* (1998) Structural and functional evolution of the basal ganglia in vertebrates. *Brain Res. Brain Res. Rev.* 28, 235–285
62 Redgrave, P. *et al.* (1999) The basal ganglia: a vertebrate solution to the selection problem? *Neuroscience* 89, 1009–1023
63 Striedter, G.F. (2005) *Principles Of Brain Evolution*, Sinauer Associates
64 Eckstein, M.P. *et al.* (2013) Rethinking human visual attention: spatial cueing effects and optimality of decisions by honeybees, monkeys and humans. *Vision Res.* 85, 5–19
65 Sareen, P. *et al.* (2011) Attracting the attention of a fly. *Proc. Natl. Acad. Sci. U.S.A.* 108, 7230–7235
66 van Swinderen, B. (2012) Competing visual flicker reveals attention-like rivalry in the fly brain. *Front. Integr. Neurosci.* 6, 96
67 Zhang, K. *et al.* (2007) Dopamine–mushroom body circuit regulates saliency-based decision-making in *Drosophila*. *Science* 316, 1901–1904
68 van Swinderen, B. (2011) Attention in *Drosophila*. *Int. Rev. Neurobiol.* 99, 51–85
69 Langley, P. *et al.* (2009) Cognitive architectures: research issues and challenges. *Cogn. Syst. Res.* 10, 141–160
70 Brooks, R.A. (1991) New approaches to robotics. *Science* 253, 1227–1232
71 Prescott, T.J. *et al.* (1999) Layered control architectures in robots and vertebrates. *Adapt. Behav.* 7, 99–127
72 Karnath, H-O. *et al.* (2002) The subcortical anatomy of human spatial neglect: putamen, caudate nucleus and pulvinar. *Brain* 125, 350–360
73 Karnath, H-O. *et al.* (2004) The anatomy of spatial neglect based on voxelwise statistical analysis: a study of 140 patients. *Cereb. Cortex* 14, 1164–1172
74 Murakami, T. *et al.* (2014) Neuroanatomic pathway associated with attentional deficits after stroke. *Brain Res.* 1544, 25–32
75 Maddox, W.T. *et al.* (1996) Visual selective attention deficits in patients with Parkinson's disease: a quantitative model-based approach. *Neuropsychology* 10, 197
76 Horowitz, T.S. *et al.* (2006) Visual search deficits in Parkinson's disease are attenuated by bottom-up target salience and top-down information. *Neuropsychologia* 44, 1962–1977
77 Hepp, D.H. *et al.* (2013) Cognitive correlates of visual hallucinations in non-demented Parkinson's disease patients. *Parkinsonism Relat. Disord.* 19, 795–799
78 Botha, H. and Carr, J. (2012) Attention and visual dysfunction in Parkinson's disease. *Parkinsonism Relat. Disord.* 18, 742–747
79 Fénelon, G. (2008) Psychosis in Parkinson's disease: phenomenology, frequency, risk factors, and current understanding of pathophysiologic mechanisms. *CNS Spectr.* 13, 18–25
80 Seo, M. *et al.* (2012) Action selection and action value in frontal–striatal circuits. *Neuron* 74, 947–960
81 Miller, E.K. and Cohen, J.D. (2001) An integrative theory of prefrontal cortex function. *Annu. Rev. Neurosci.* 24, 167–202
82 Hikosaka, O. *et al.* (2013) Why skill matters. *Trends Cogn. Sci.* 17, 434–441
83 Graybiel, A.M. (2008) Habits, rituals, and the evaluative brain. *Annu. Rev. Neurosci.* 31, 359–387

84 Bradfield, L.A. *et al.* (2013) The thalamostriatal pathway and cholinergic control of goal-directed action: interlacing new with existing learning in the striatum. *Neuron* 79, 153–166
85 Ding, J.B. *et al.* (2010) Thalamic gating of corticostriatal signaling by cholinergic interneurons. *Neuron* 67, 294–307
86 Pan, W-X. *et al.* (2013) Neural signals of extinction in the inhibitory microcircuit of the ventral midbrain. *Nat. Neurosci.* 16, 71–78
87 Minamimoto, T. *et al.* (2009) Roles of the thalamic CM–Pf complex–basal ganglia circuit in externally driven rebias of action. *Brain Res. Bull.* 78, 75–79
88 Dalsgaard, S. (2013) Attention-deficit/hyperactivity disorder (ADHD). *Eur. Child Adolesc. Psychiatry* 22 (1 Suppl.), 43–48
89 Morris, S.M. *et al.* (2012) The genetic toxicity of methylphenidate: a review of the current literature. *J. Appl. Toxicol.* 32, 756–764
90 Tripp, G. and Wickens, J.R. (2009) Neurobiology of ADHD. *Neuropharmacology* 57, 579–589
91 Thapar, A. *et al.* (2013) Practitioner review: what have we learnt about the causes of ADHD? *J. Child Psychol. Psychiatry* 54, 3–16
92 Dichter, G.S. *et al.* (2012) Reward circuitry dysfunction in psychiatric and neurodevelopmental disorders and genetic syndromes: animal models and clinical findings. *J. Neurodev. Disord.* 4, 19
93 Gizer, I.R. *et al.* (2009) Candidate gene studies of ADHD: a meta-analytic review. *Hum. Genet.* 126, 51–90
94 Russell, V.A. *et al.* (2005) Animal models of attention-deficit hyperactivity disorder. *Behav. Brain Funct.* 1, 9
95 Sagvolden, T. *et al.* (2005) Rodent models of attention-deficit/hyperactivity disorder. *Biol. Psychiatry* 57, 1239–1247
96 Gainetdinov, R.R. and Caron, M.G. (2000) An animal model of attention deficit hyperactivity disorder. *Mol. Med. Today* 6, 43–44
97 Trinh, J.V. *et al.* (2003) Differential psychostimulant-induced activation of neural circuits in dopamine transporter knockout and wild type mice. *Neuroscience* 118, 297–310
98 Gong, R. *et al.* (2011) Role for the membrane receptor guanylyl cyclase-C in attention deficiency and hyperactive behavior. *Science* 333, 1642–1646
99 Chuhma, N. *et al.* (2011) Functional connectome of the striatal medium spiny neuron. *J. Neurosci.* 31, 1183–1192
100 Carrasco, M. *et al.* (2004) Attention alters appearance. *Nat. Neurosci.* 7, 308–313
101 Lu, Z.L. and Dosher, B.A. (1998) External noise distinguishes attention mechanisms. *Vision Res.* 38, 1183–1198
102 Eckstein, M.P. *et al.* (2000) A signal detection model predicts the effects of set size on visual search accuracy for feature, conjunction, triple conjunction, and disjunction displays. *Percept. Psychophys.* 62, 425–451