

# Neural Correlates of Anticipation in Cerebellum, Basal Ganglia, and Hippocampus

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**Abstract.** Animals anticipate the future in a variety of ways. For instance: (a) they make motor actions that are timed to a reference stimulus and motor actions that anticipate future movement dynamics; (b) they learn to make choices that will maximize reward they receive in the future; and (c) they form memories of behavioral episodes such that the animal's future actions can be predicted by current neural activity associated with those memories. Although these effects are clearly observable at the behavioral level, research into the mechanisms of such anticipatory learning are still largely in the early stages. This review, intended for those who have a computational background and are less familiar with neuroscience, addresses neural mechanisms found in the mammalian cerebellum, basal ganglia, and the hippocampus that give rise to such adaptive anticipatory behavior.

## 1 Introduction

Anticipatory behavior occurs when actions depend not only on past and present but also on predictions, expectations, or beliefs about the future [4]. While the volume in your hands is largely concerned with the computational or theoretical aspects of anticipatory behavior, the researchers who participate in the Anticipatory Behavior in Adaptive Learning Systems meeting are also interested in the processes that allow animals to generate such behavior. This paper will provide an overview of some types of neural activity in the mammalian brain that are highly correlated with particular forms of anticipatory behavior. Behaviorally correlated neural activity is generally interpreted as evidence that the brain area where the activity appears is involved in producing the behavior. Therefore, the study of activity correlated with anticipatory behavior can potentially reveal the neural mechanisms underlying behavioral production.

### 1.1 The Neural Correlates of Behavior

Behavioral neuroscience<sup>1</sup> is the study of how the mechanisms of the nervous system give rise to the behaviors observed at the level of the whole organism.

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<sup>1</sup> Some people prefer terms such as biological psychology, systems neuroscience, or cognitive neuroscience to describe roughly the same idea.

Questions of mechanism are always difficult to address, but the nature of the nervous system, with its nearly-innumerable interacting components (humans have on the order of  $10^{15}$  synapses) and its largely unknown molecular machinery, is particularly difficult. Creating a theory about the mechanism of a behavior, or the contribution a brain region makes to a behavior, is often only achieved by piecing together several indirect lines of evidence. The most common forms of evidence to look at are:

1. **Anatomy:** It is fairly clear where sensory and motor information arrives at or departs from the central nervous system. Neurons that are only a few synapses away from a sensory or a motor neuron are most likely to process that kind of information. Likewise, once a theory of function has been well-established for a particular brain region, it naturally may suggest functions of other areas that produce input for that region or receive output from it.
2. **Lesion studies:** Brain-damaged human patients or targeted lesions created in experimental animals can define which brain areas are at least necessary for a behavior. However, the problem arises that there are often multiple, parallel systems performing similar functions that can be difficult to disassociate from each other. In addition, knowing that a lesion to a particular region disrupts a behavior does not mean the region is responsible for the behavior. For example, the signals responsible for producing the behavior could merely transit through that region rather than originating or being processed there. Also the lack of disruption when a lesion occurs does not indicate the region is not involved in the behavior under some circumstances — parallel pathways could be compensating for the damaged region.
3. **Imaging studies:** Using magnetic resonance imaging, positron emission tomography, or similar techniques the neural activity of an entire region may be studied at once. This allows one to look for behaviors that correspond to activity in the region being studied; such correlated activity would at least suggest that the region is involved in that behavior. However, these methods suffer from the problem that it is not always clear what is going on at a mechanistic level. For instance measuring blood flow in an MRI only tells how metabolically active that brain region is, not whether it is affecting other regions and, if so, whether its effect is inhibitory or excitatory.
4. **Single-unit recording:** Electrodes are inserted into the brain and the action potentials fired by a single neuron are recorded, thus allowing one to study behavioral correlates of single neurons. However, the problem here is the inverse of the one above — it is difficult to record enough neurons simultaneously to understand much about how activity progresses through the nervous system.

Finally, it is important to note that while behavioral neuroscience is striving to understand the functioning of the nervous system in producing behavior, this does not imply that the goal is to assign one function to each and every neuron or brain region. Clear behavioral correlates are usually obtained only once the experiment has been highly simplified and the subject over-trained.

Neural activity is much messier when performing natural tasks, because most parts of the brain are likely to be involved in many behavioral functions. Yet clear progress can be, and has been made in understanding several aspects of behavioral production in the mammalian nervous system. It just requires the steady accumulation of multiple lines of evidence.

## 1.2 Scope

There is strong evidence that several brain regions are important in anticipating the future over different time-scales. This review focuses only on anticipation over time-scales of seconds or less. Long time-scale (minutes and longer) cognitive planning and circadian timing of behaviors is beyond the scope of this review, although the former are typically seen as involving frontal cortical areas of the brain [39,17], and the latter are believed to be primarily related to genetic transcription/translation auto-regulatory loops and involve a brain area known as the suprachiasmatic nuclei [17].

The brain areas and behavioral functions that will be reviewed in this paper are listed below:

**Cerebellum:** involved in many aspects of motor learning. This review looks at its involvement in both timing motor movements in relationship to stimuli, and in computing forward kinematic models that predict the results of motor commands.

**Basal ganglia:** involved in learning, timing, and sequential action selection. Most strikingly, parts of the basal ganglia contain neurons whose activity correlates with predictions of the reward structure of the environment.

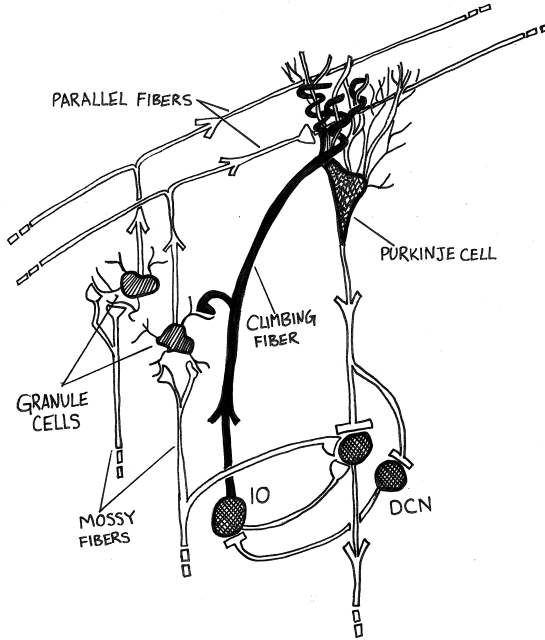
**Hippocampus:** (and surrounding areas) involved in learning and memory. Particularly, the hippocampus is necessary for creating memories that are sequential in nature, such as memories of route navigation. There are strong activity correlates of these kinds of memories in the hippocampus, and these correlates encode information about future events in the behavioral sequence.

## 2 Cerebellum - Motor Actions and Timing

The cerebellum is located to the rear of the brain underneath the cerebral cortex. Although it contains only about 10% of the brain's volume it has roughly half of all neurons. It has a regular, layered organization that basically consists of repetitions of the same circuit. Yet it contains several distinct regions, each receiving input from different parts of the brain, thus suggesting that the cerebellum is performing the same kind of calculations, but on a variety of data.

A cerebellar circuit (see figure 1) receives its input through two pathways: climbing fibers that excite single Purkinje cells directly, and mossy fibers that excite the parallel fibers joining cerebellar circuits together. The Purkinje cells excite deep cerebellar nuclei which in turn produce the cerebellum's output. The parallel fibers also stimulate inhibitory cells (not shown in the figure for clarity) that prevent Purkinje cells from firing; this produces a center excitation

and surrounding inhibition effect. Only Purkinje cells that receive high levels of input from their climbing fibers as well as the parallel fibers will fire. Nearby circuits will be prevented from firing due to the inhibitory cells that take their input from the parallel fibers.



**Fig. 1.** A schematic depiction of a single cerebellar circuit. Input goes through the inferior olive (IO) via the climbing fibers to the Purkinje cells, which excite the deep cerebellar nuclei (DCN) that produce the cerebellum's output. Another input pathway goes via the mossy fibers to the granule cells and their parallel fibers that lead to other cerebellar circuits. The parallel fibers also excite cells (not shown for clarity) which inhibit Purkinje cells.

The cerebellum has long been implicated in the feedback control of motor activity [22], but it may also be involved in anticipatory forms of motor control. This section contains a review of the cerebellum's possible role in timing motor actions and in predicting the kinematic results of those actions. These are problems of anticipation in the millisecond to second range, resulting from the need of the animal to produce smooth, controlled behavior while interacting with the physical world.

The cerebellum is involved in the timing of motor actions that are repetitive, such as rhythmic tapping. An ability to accurately reproduce a rhythm is a basic form of motor timing which can be used for producing anticipatory motor behavior. One way to measure this ability is to start a test subject tapping in time

to a metronome and ask them to continue the beat after the metronome stops. Patients with cerebellar damage are impaired at this task [14,15]. Patients with damage to medial parts of the cerebellum have deficits that could be described as execution (motor) error, and patients with damage to lateral parts of the cerebellum had deficits in timing their tapping but not in executing the motor action. This demonstrates that the cerebellum is involved not only in producing the motor performance, as would be predicted by the standard theory of cerebellar function, but is also involved in timing the rhythm.

The cerebellum is also involved in learning the timing of motor actions that are not repetitive, but must be executed in precise temporal relation to an external stimulus. The classic example of this is eyeblink conditioning [24,48], where a tone cue (conditioned stimulus, CS) begins at a fixed interval (100 ms - 3 s) before a puff of air (noxious unconditioned stimulus, US) is delivered to the eye. Rabbits learn that the CS predicts the appearance of the US, and shut their eyelids in response to the CS after 100-200 trials. After conditioning they shut their eyes with a delay such that the response peaks on average at the time the US is presented. Cerebellar lesions abolish the proper timing of a conditioned eyeblink response.

Another form of prediction that seems to be performed in the cerebellum is the computation of where a limb will be in the near future — this is known as a forward model. Optimal motor control theory requires the presence of a forward computation of what the effect of a motor command will be given the current state and motor commands [51]. A forward model provides the nervous system with a prediction of what the body state will be like in the near future. This prediction can be used to more accurately estimate actual body position.

Alternatively, a forward model can allow the production of movements that are faster than using only feedback control [17]. Animals have muscles arranged in antagonistic pairs so producing a desired motion without overshoot requires applying an early breaking force. While this calculation can in theory be performed using only feedback control, the long delays in the nervous system and the dynamic properties of muscles and proprioception favor the use of forward motion models for choosing when to apply the breaking force.

The cerebellum may coordinate eye and hand movements through the use of forward models [27]. Maximum eye-hand coordination is achieved by having the eyes slightly lead the hand, and the motor system is therefore hypothesized to use forward-model information from the ocular motor system to increase accuracy in producing hand movements. Imaging studies have demonstrated cerebellar involvement in these types of tasks [28]. Several studies reviewed in [2] demonstrate that patients with cerebellar damage have difficulty adapting to predictive (forward) motor control problems, but are equivalent to controls when adapting to reactive (feedback) motor control problems.

Mauk and Buonomano [23] hypothesize that both interval timing and feed-forward control deficits in cerebellar patients may be due to the same mechanism. They argue that tasks that involve stopping and starting, such as tapping and blinking, would have particularly noticeable effects if the feed-forward

models controlling antagonist muscle timing were disturbed. Thus they offer us the controversial possibility that both types of deficits result from a disturbance of the forward model.

It should be noted that the cerebellum is not the only brain region thought to be involved in creating forward motion models in the brain. The parietal cortex and various motor regions have neural activity that appears to encode limb positions in a variety of coordinate systems. They also have neural activity that encodes limb kinematics and dynamics in the near future; i.e., forward models. A good review of this material can be found in [45]. This does not, however, conflict with the view that the cerebellum is involved with computing forward models. It appears that the cerebellum is particularly involved with adapting these models during behavior — all of the citations offered above regarding cerebellar forward models have that flavor to their results.

### 3 Basal Ganglia — Dopamine and Reward

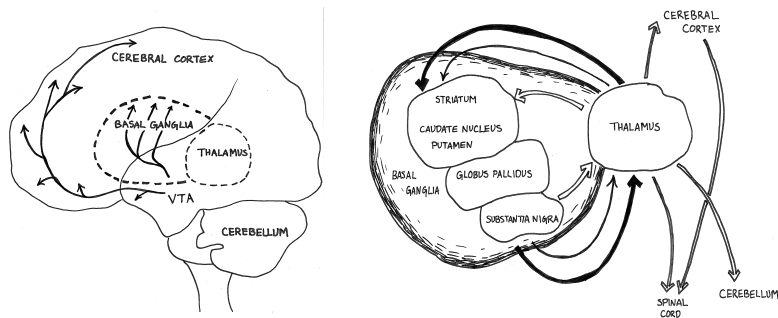
The basal ganglia are a collection of midbrain structures, including the regions that are the primary topics of this section: the striatum, the substantia nigra, and the ventral tegmental area. The basal ganglia is involved in learning and action selection, and disorders of this region lead to devastating diseases such as Parkinson's, Huntington's, and Attention Deficit Hyperactivity Disorder.

The basal ganglia are also interesting because of their unique anatomy and physiology, which is illustrated in figure 2. Input converges on the striatum from many different cortical locations. The striatum in turn projects to several other structures inside the basal ganglia. Information flows in several anatomically segregated, parallel loops through the basal ganglia before projecting back out to the cortex by way of the thalamus [26].

This region is the primary source of the neurotransmitter dopamine in the brain. The dopaminergic neurons, named after the neurotransmitter they release when firing, are located in the substantia nigra pars compacta of the basal ganglia and in the nearby ventral tegmental area (VTA). From these small structures the dopaminergic neurons project their axons widely throughout the brain, but primarily to striatum, where most of their input originates, and frontal cortical regions. The disorders mentioned above in relation to the basal ganglia are all disorders of dopamine function.

The basal ganglia's involvement in learning may come from neurons in this region whose activity seems related to the computational theory of reinforcement learning. Reinforcement learning is in turn based on observations of animal behavior at the cognitive level, thus completing a nice circle from observation of behavior, through computational theory, to observations of neural mechanisms that seem to be responsible for the behavior.

Reinforcement learning is of interest from the anticipatory learning viewpoint because the animal is learning to predict rewards that it will receive in the future in order to make decisions about actions in the present to obtain the best possible reward. Thorndike [49] originally formulated the principle of reward learning in



**Fig. 2.** *Left:* Dopaminergic neurons located in the substantia nigra of the basal ganglia and the adjacent ventral tegmental area project widely (arrows) across the brain. Dopamine release is associated with rewarding or pleasurable stimuli. *Right:* Information flows through the basal ganglia in segregated loops that pass through the thalamus and onward through sensory and motor cortical regions and also directly to motor neurons in the spinal cord. Connections within basal ganglia (not shown) maintain the segregation of the loops.

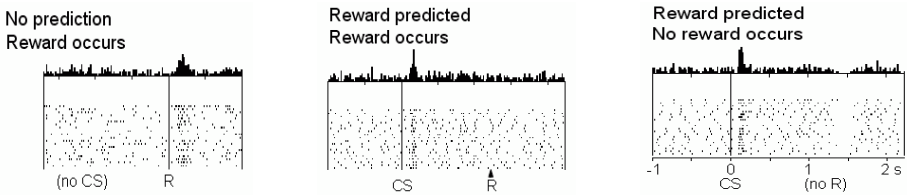
the following way, “Any act which in a given situation produces satisfaction becomes associated with that situation so that when the situation recurs the act is more likely than before to recur also.” This principle was further elaborated on in the Rescorla-Wagner model of Pavlovian conditioning [38]. This is an error-correction model where the associative strength between conditioned stimuli and the unconditioned stimulus is increased only to the extent to which the US is unpredicted by the CS. Learning slows progressively as the US becomes more predicted by the appearance of the CS. The temporal difference (TD) learning rule [47] is an extension of this principle to the operant conditioning domain<sup>2</sup>. TD learning is also an error-correction model based on the animal’s experiences. The TD rule is particularly powerful because learning can still take place even when the reward does not arrive every time the correction action is taken, and even when the reward arrives at an arbitrary delay after the correct action.

Temporal difference learning is algorithmically simple:  $\Delta V(t) \propto V(t - 1) - R(t)$ , where  $t$  is time,  $V$  is the predicted reward, and  $R$  is the actual reward received. The  $\Delta V$  term is the reward prediction error, and is used in a reinforcement learning algorithm to drive the system to change its action selection mechanism in a way that allows the learner to obtain more reward over time.

Dopaminergic neurons have activity which can be related to the reward prediction error of TD learning [41,50]. Dopamine is released phasically with rewarding stimuli; that is, midbrain neurons release dopamine with short latency after the rewarding stimulus, and this increase in dopamine release persists for only a

<sup>2</sup> In Pavlovian conditioning reward always occurs after the CS is presented, whereas in operant conditioning the animal must make a correct action or it will receive no reward.

short time. The release of dopamine does not appear, however, to be just related to reward or pleasure [29]. The phasic dopamine signal has several properties that make it a candidate for carrying a TD-style reward prediction error, as is shown in figure 3. Dopamine is released when the animal receives an unexpected reward, which would correspond to a positive reward prediction error. Dopamine is no longer released for a rewarding stimulus once the animal has learned to expect reward in a particular situation — the reward would be fully predicted in this case and the prediction error should be zero. Finally, if the animal does not receive the reward when it expects to receive it, then there is a depression of dopamine release, which is consistent with the negative prediction error that would occur in that situation.



**Fig. 3.** Rastergrams showing how the activity of dopaminergic neurons in VTA is similar to the reward prediction error of TD learning. VTA neurons respond strongly if the animal receives an unpredicted reward (*left*). When the animal has learned that CS predicts reward then VTA neurons respond strongly at the CS and not at the US (*middle*). When the animal does not receive the reward as expected, the firing rates of VTA neurons depresses at the time of the expected reward (*right*). Each dot is an action potential fired by the neuron being recorded, and each row represents one task trial. Time is on the x-axis. The top of each graph is a histogram showing the number of action potentials that occurred over all trials at each time point. This figure is modified from [http://scholarpedia.org/article/Reward\\_Signals](http://scholarpedia.org/article/Reward_Signals)

The dopaminergic system is also an interesting candidate for carrying a reward prediction error because it projects widely across many brain areas in a uniform fashion. Dopamine producing neurons fire synchronously and do not have any systematic differences in the locations to which they project. Therefore the dopamine signal reaches many brain regions with no systematic difference in the signal from brain region to brain region. This is consistent with usefulness of a reward prediction error in any situation in which the animal is trying to maximize the amount of reward it receives.

However, not everyone believes that the short-latency dopamine signal is a reward prediction error. An alternative hypothesis is that the short-latency dopamine response is involved in novelty-detection and attentional mechanisms. It has been suggested that the initial burst of dopaminergic firing could represent an essential component in the process of switching attentional and behavioral



selections to unexpected, behaviorally important stimuli [36]. This fits in with the role of dopamine dysfunction in Attention Deficit Hyperactivity Disorder. Note that this theory of function is also of interest from an anticipatory learning standpoint; one possible mechanism that can be used in deciding which stimuli are novel and worth giving attentional resources is to pick ones which cannot be predicted [8,40,18].

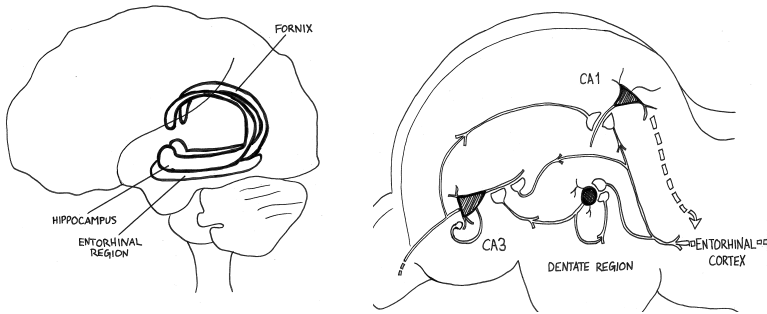
If we accept the hypothesis that the short-latency dopamine response is a reward prediction error, it still raises the question of how this signal is being used to produce behavior. One mechanism that is suggested by the analogy of dopamine and TD error is the use of an actor-critic architecture [47]. In this form of model the critic learns to predict the reward structure of the environment and produces a learning signal used by a separate actor to learn what actions will produce the most reward over time. The exact form and location of the actor and critic components has been the subject of several theories [13,16,35,31,12]. But most of these models point at the probable involvement of the striatum because it is both the largest input to and largest output from the dopaminergic neurons.

There are also neural activity correlates of reward terms in several areas outside the basal ganglia [42,43] including orbitofrontal cortex, prefrontal cortex, cingulate cortex, perirhinal cortex, parietal cortex, premotor cortex, frontal and supplementary eye fields, and the superior colliculus. This list includes regions involved in executive function, sensory processing, and even motor control. Many of these regions receive substantial dopaminergic projections. There is a broad range of reward-related activity in these regions, including stronger activity when rewarded, stronger activity when the reward is more certain, and a ramp-up of activity during the waiting period culminating in highest activity when rewarded. It is likely that all of these signals are useful in different ways in producing adaptive behavior that maximizes reward. It is also likely that there are multiple, parallel systems that use these signals just as there are multiple, parallel locations that carry this reward information.

The basal ganglia are also hypothesized to be involved in the problem of action sequencing. Neurons in basal ganglia show activity that encodes serial movement order, for instance when a rat is engaged in grooming actions that have a natural sequence [1]. Actor-critic models of the basal ganglia, such as those discussed above, possess the useful computational ability to learn action sequences that lead to reward at a time much later than the action. Interestingly, the interval timing task discussed in the cerebellum section can also be seen as an action sequencing problem. There is evidence that the basal ganglia are involved in interval timing performance as well [3]: patients with dopamine disorders have problems with interval timing tasks, and the basal ganglia are hypothesized to produce interval timing through coincidence detection of the relative phases of many oscillators located in different brain regions. This close relationship between the functions of basal ganglia and cerebellum in learning, timing, and motor control has been noted by others [6,15].

## 4 Hippocampus - Episodic Memory

Episodic memory can be defined as the integration of sensory events (“what”), over time (“when”), and space (“where”) [11]. In humans, episodic memory is autobiographical in nature, a memory of personal experience instead of just the dry knowledge of a fact. The medial temporal lobe, which includes the hippocampus and adjacent cortical structures (see figure 4), is necessary for the acquisition of episodic memories, as demonstrated by the deficits of patients that have damage to this brain area [44], and by imaging studies [46,30].

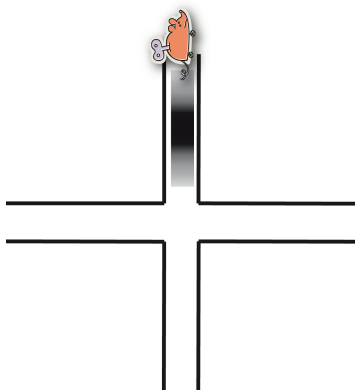


**Fig. 4.** *Left:* A cutaway of the medial temporal lobe including the hippocampus. Inputs from all over the brain converge onto the entorhinal region and from there enter the hippocampus. *Right:* A schematic of the hippocampal circuit. Input from the entorhinal cortex projects via the perforant path to dentate, CA3 and CA1 hippocampal subfields. Another pathway, the trisynaptic loop, projects from the entorhinal cortex to dentate to CA3 to CA1 and then back to the entorhinal cortex. Self-excitatory loops exist in both CA3 and dentate. The overall effect is that a great variety of signals pass through nested loops of various lengths — thus integrating different information streams over several time scales.

Navigation can be seen, in this sense, as a special case of a more general episodic memory process, where sequences (when) of egocentric perception (what) are converted into an allocentric map (where) of the environment [37]. The hippocampus is known to be important for navigation ability in both humans [21] and rodents [33], which suggests both that navigation and episodic memory rely on the same processes, and that animals may constitute a valid model for episodic memory research (on this topic, see also [5]).

One of the most interesting and well-studied neural correlates of episodic memory is the place cell. In the rodent hippocampus there are excitatory pyramidal neurons whose firing is highly correlated with the animal being at a particular location in the environment [32]. Such cells fire action potentials at rates up to 40Hz when near the location they respond to, tapering off their firing rates gradually while moving away from that location, often in a Gaussian-like manner.

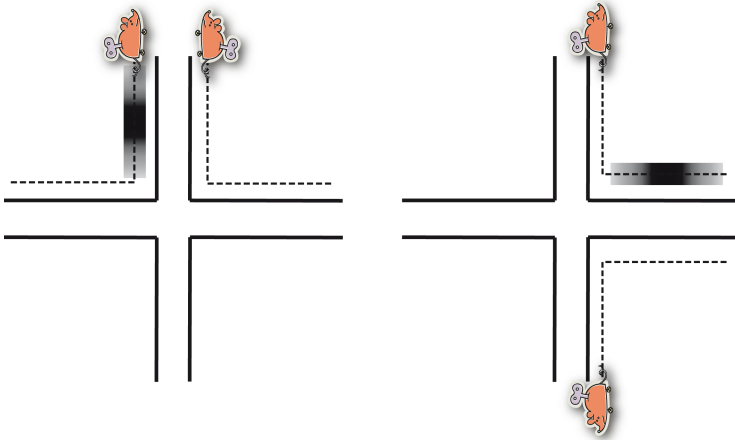
This Gaussian-like relationship between place and firing rate of a cell is known as a place field (see figure 5). When the animal is far from the center of the place field the cell may be completely silent or at most fire a few handfuls of action potentials over very long periods of time.



**Fig. 5.** A schematic example place field of a hippocampal neuron. When the animal is in a particular location in the maze the cell has high firing rate (dark). This firing rate drops away in a gradual fashion as the animal is further from the center of the place field (lighter color in the field map). Elsewhere the cell is totally silent.

Place cells also have several interesting characteristics from the standpoint of anticipatory learning. Such cells have a phase relationship with the theta rhythm that contains information about whether the animal is just approaching, at the center of, or departing from the place field [34]. The theta rhythm is an oscillation of 7-12 Hz observed in electro-encephalograms of the hippocampus. When a place cell in the hippocampus begins to fire action potentials as the rat enters the place field, it begins consistently at a particular phase relationship to the theta rhythm. Then as the rat traverses from the leading to the trailing edge of the place field, the phase shifts progressively forward on each theta cycle. It does so in a way that suggests it is actually location and not time that determines the phase relationship. Thus place cell firing contains not just information about how close the animal is to the center of a particular place cell, but also information about whether the animal is approaching the center of the field (a predictive encoding) or has already crossed the center and is departing the place field. There is evidence that phase relationships are indeed a fairly general mechanism used by several brain regions to encode the serial order of relationships [20]. Serial order encoding of stimuli is a necessary pre-condition before it is possible to produce behavior that anticipates a sequence.

Another interesting neural correlate of episodic memory that has a strong anticipatory flavor are hippocampal cells that have place fields only if the animal will take a particular path in the future, but have no firing field if the animal



**Fig. 6.** A schematic example of a retrospective (left) and prospective (right) hippocampal place field. When the animal is in a particular location in the maze the cell has a place field only if the animal has taken (retrospective, left) or will take (prospective, right) a particular path. Otherwise the cell is silent.

will take a different path [7,52,10]. A place field whose activity is dependent on the future path in this fashion is said to have prospective coding. There are also cells whose place fields exist only if the animal has arrived at the field location via a particular path, but not when another path is used [7,10]. A place field whose activity is dependent on the past in this fashion is said to have retrospective coding. A schematic example of such a firing field can be seen in figure 6. Somewhere between half [7] and two-thirds [52] of hippocampal place cells have prospective or retrospective coding.

The existence of prospective and retrospective place fields implies that the hippocampus contains information not just about what, where and when, but also information that associates different wheres and whens together, which is an important ability for an anticipatory learning system. One of the most convincing elements establishing prospective activity as an observable correlate of an entire behavioral episode, is the discovery [7] of cells that fire in a prospective coding for one future path even if the animal makes a momentary error and begins traveling down the incorrect (non-coded) pathway before correcting itself and taking the correct path. This demonstrates that the prospective firing field is related to the whole path through the maze (the intended behavioral episode) and not the immediate decision that is about to be made at the next junction.

Why is it the hippocampus, and not some other brain region, that seems to be responsible for forming episodic memory? The unique anatomy and connectivity of the hippocampus is the likely mechanism by which episodic memories are formed [19,9]. The region receives many different inputs from both unimodal and polymodal sensory areas as well as higher association areas of the cortex. All this information is merged and flows through several nested loops of different

time-scales in the hippocampus. The hippocampus then projects broadly back out to many regions of the cortex. This convergence, looping, and divergence of information, seen in figure 4, is a mechanism that can form associations across space and time.

Perhaps because of its central role in memory, the hippocampus is also involved in a variant of the eyeblink conditioning task known as trace conditioning [48]. Trace eyeblink conditioning is different from regular eyeblink conditioning in that there is a delay period between CS and US, such that the tone is off (between 500ms and tens of seconds) before the air puff is delivered. The lack of stimuli predicting the noxious US in the delay period makes this a more memory-intense version of eyeblink conditioning. Rabbits with hippocampal lesions are unable to acquire trace conditioning and rabbits that have been trained and then lesioned lose the conditioning. Additionally, there are strong neural correlates of trace conditioning in the hippocampus; around one quarter of hippocampal excitatory neurons fire maximally when the US should appear even if it is omitted [25].

## 5 Conclusion

This review has presented results demonstrating the involvement of three brain regions with anticipatory behavior. There are, however, other regions of the brain that are beyond the scope of this review that are probably involved with these same anticipatory behaviors. In addition, the regions covered in this review have neural correlates of other anticipatory behaviors that were not discussed here. The references discussed in the previous sections can provide the reader with an entry point into this literature.

Although each brain region was presented at the beginning of the review as being associated with a particular form of anticipatory behavior, there were in fact considerable overlaps that were pointed out at various points in the text. The hippocampus is involved in some forms of eyeblink conditioning, as well as being required for episodic memory. Basal ganglia are involved in timing, as well as producing a primary learning signal. The cerebellum is involved in learning, as well as being important for timing of motor actions. This should give the reader a feeling for behavioral neuroscience research and an understanding of why a point was made earlier about the need to accumulate evidence from multiple sources to formulate theories of function.

Although it is still not possible to fully understand the mechanisms that produce even some fairly simple behaviors in mammals, this review did illuminate a principle that could be of use in designing anticipatory learning systems. All three of these brain regions have unique anatomy that is very likely the mechanism by which they compute the functions proposed in this paper.

- The cerebellar circuit has a two-input, center excitation, surround inhibition, multi-layer structure that undoubtedly plays a large role in its ability to process information about millisecond level timings.

- The basal ganglia contains multiple segregated loops that may allow it to use the reward predictions it generates to select an appropriate action from many possibilities.
- The hippocampal circuit contains multiple nested loops of varying time-scales that help to integrate information over time to produce memories.

Hopefully computational theorists in the anticipatory learning community will be able to draw on these principles to create useful adaptive systems, and will in doing so develop an abiding interest in producing biologically-grounded models of how these brain structures produce adaptive behavior.

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