THE KOHA CODE
A BIOLOGICAL THEORY OF MEMORY

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ABSTRACT

This work introduces the Koha model, a new theory that aims to explain two unresolved phenomena within biological neural networks: How information is processed and stored within neural circuits, and how neurons learn to become pattern detectors. In the Koha model, the dendritic spines of a neuron serve as computational units that scan for precise spike patterns in their synaptic inputs. The model proposes the existence of a temporal code within each dendritic spine, which is used for the dampening or amplification of signals, depending on the temporal information of incoming spike trains. Compelling evidence is provided and a concrete process is described for how signal filtration occurs within spine necks. A competitive learning algorithm is then proposed that describes how neurons use their internal temporal codes to become pattern detectors.

Keywords Memory · Dendritic Spines · neural code · competitive learning · associative memory

1 Introduction

1.1 Form follows function

The ability to form and store memories, recall past events, make inferences and deductions, are all examples of "emergent algorithms" that arise from neural circuits. Emergence occurs when a system exhibits characteristics that its constituents do not possess but emerge because of rules, or interactions between its parts. Parts of an emergent system often adhere to very basic principles. When those parts interact with one another, complexity can arise. In the case of the brain, neural circuits serve as the physical substrate for emergent algorithms, which arise as a result of neuronal interactions. Any modification to a neural circuit or structure may thus affect an existing algorithm or, in certain instances, result in the creation of a new algorithm. If a useful algorithm forms as a result of a mutation in one of its underlying neural circuits, the organism with the altered algorithm will have a competitive advantage over other organisms. Over time, evolutionary forces will shape the underlying circuits in a way that makes the emergent algorithm more efficient. This means that by examining the optimized "hardware" of organisms, it might be possible to deduce some of the existing emergent algorithms in nature. If a certain pattern repeats across many species, we can be very certain that the repeating pattern plays a significant role. By looking at the optimised morphology, structures, and processes of various neurons, we can start to guess their purpose in the context of the mind. After all, in biology, form follows function.

1.2 Patterns in the substrate

One of the most notable excitatory neurons is the pyramidal neuron. It is the most common type of excitatory neuron in the mammalian cortical structures, as well as one among the largest neurons in the brain. Each mammal, as well as birds, fish, and reptiles, has pyramidal neurons. Apart from its wide occurrence in nature, the pyramidal neuron exhibits also many noteworthy physical features, particularly when contrasted to inhibitory interneurons. The following is a list of key observations that are relevant for the Koha model presented in section 2.
• Studies in the layer 2/3 of the rat neocortex have shown that pyramidal neurons posses about 10 times more local connections with fast-spiking inhibitory neurons, than with other pyramidal neurons, with most of the connections being reciprocal [1].

• Pyramidal neurons have two kinds of dendrites. Basal dendrites, which come out of the soma and branch extensively. And a single, long, dendrite that emerges from its soma, also known as the apical dendrite. It can extend for several hundred microns before branching into many smaller dendrites (see figure [1]) also known as the apical tuft. Basal dendrites tend to receive their input from nearby neurons, whereas the apical dendrite receives its inputs from more distant regions [2]. While pyramidal neurons have an apical dendrite, inhibitory interneurons do not.

![Diagram of pyramidal neurons](image1)

Figure 1: The variable structure of pyramidal neurons across different parts of the brain. Even though the pyramidal neuron differs slightly across layers, it is characterized by its stereotypical morphology - the presence of an apical dendrite and the presence of basal dendrites. Adapted from Spruston, 2008 [3].

• The apical dendrite and basal dendrites of pyramidal neurons are completely covered with tiny protrusions structures known as dendritic spines (see figure [2]). It is believed that dendritic spines serve as memory storage components for neurons [4]. In contrast to excitatory neurons, most interneurons do not posses, or posses very few dendritic spines, i.e. most interneurons are essentially spineless [4].

![Diagram of dendritic spines](image2)

Figure 2: Two dendrites covered with dendritic spines. Adapted from Spruston, 2008 [3].

• There are various stages of dendritic spines, the exact borders of which are a bit blurry. Traditionally spines are classified into thin, stubby, and mushroom spines [5] (see figure [3]). Thin spines have a small head and a long neck that connects to the parental dendrite. Stubby spines have no neck at all and are more expressed in infants [6]. Whereas mushroom spines have a wide neck with a large head and are more expressed in adult brains [6, 7]. Because of the properties each spine category has, it is believed that thin spines may play a role in "learning", whereas mushroom spines in long-term memory [8]. Thin spines can dynamically appear and disappear throughout life, while most mushroom spines remain stable for a lifetime [8, 9]. The stability of the mushroom
spine and its high expression in adult brains, make it an ideal candidate as a long term "memory unit".

Figure 3: The three traditional types of dendritic spines.

- We now know that dendritic spines receive most of the excitatory inputs in neurons, and that practically every dendritic spine has an excitatory synapse on its head [10][11][12]. In other words, excitatory axons prefer for some reason to terminate particularly on spines, they almost never connect directly to dendritic shafts (the body of the dendrite) [12]. We can see the opposite behavior in inhibitory axons. Inhibitory axons almost always connect directly to dendritic shafts, rather than on spines [13]. The consistency of this pattern suggests a functional value.

- Imaging experiments have demonstrated that dendritic spines behave indeed like electrical and biochemical compartments [14][15][16][17][18][19]. The narrow neck of the spine can create an isolated compartment in which the biochemical signals of the spine head do not spread along the parent dendrite. Changes in the length and narrowness of the spine neck, has a direct effect on the compartmentalization of the spine [19].

- Experiments also show that the most significant filtering of local potentials in a neuron does not occur along the dendrite, but instead at the spine neck [20]. The spine neck is somehow able to modulate the amplitude of incoming signals. Further studies have also shown that spines, additionally to filtering out local potentials, are also able to amplify them [21].

- Evidence is now overwhelming that dendritic spines can change their physical structure in seconds, following an appropriate stimulus [22][23][24][25][26]. Spine necks have an actin cytoskeleton (see figure 4) that can dynamically change shape depending on its interactions with several actin regulators. This change in geometry has a direct affect on local voltage amplification and biochemical compartmentalization [27]. The strength of incoming signals can therefore be dampened, or amplified, depending on the length and width that the spine neck takes [28].

Figure 4: A simplified model of the organization of actin in dendritic spines. Reprinted from [29].

- Some dendritic spines also posses an enigmatic organelle known as the spine apparatus, a specialized formation of stacked endoplasmic reticulum that occupies a large portion of the spine neck. Not all spines have a spine apparatus, but almost all mushroom spines have one [30]. We now know that the role of the spine apparatus is closely linked with local calcium trafficking [31][32][33]. Due to the large surface area of the organelle, it is believed that the spine apparatus may act as a calcium buffer [31]. Studies now show that the spine apparatus is also able to quickly release $Ca^{2+}$ [34][35]. The spine apparatus is thus acting as an intracellular calcium store [31], serving as both calcium source and calcium sink, depending on internal feedback.

- The spine apparatus also appears to be closely associated with synaptopodin [36], which is an Actin-associated Protein. In an experiment, synaptopodin-deficient mice were associated with a loss of the spine apparatus and showed a reduction in hippocampal long-term potentiation, suggesting that the spine apparatus may play an important role in synaptic plasticity [37]. Synaptopodin’s preferential location in spines, and its close association with the spine apparatus and the actin cytoskeleton, suggests that synaptopodin is somehow involved in the rapid plasticity of the spine’s actin cytoskeleton. In fact, one study suggested that synaptopodin...
may act as an actin-bundling molecule in the spine neck, and that it may influence calcium release from the spine apparatus [38].

• An even more enigmatic organelle is the cisternal organelle, found at the axon initial segment of pyramidal neurons. The cisternal organelle is also a formation of stacked endoplasmic reticulum and shares many structural similarities with the spine apparatus. Very little is known about this organelle. There is now evidence that it may serve as an intracellular calcium store [39]. The cysternal organelle, just as the spine apparatus, also appears to be closely associated with synaptopodin. Experiments showed that synaptopodin-deficient mice were also associated with a loss of the cisternal organelle at the axon initial segment [40].

1.3 Receptive fields and invariances

The portion of sensory space that can cause a neuron to fire when stimulated is known as the neuron’s receptive field. There are visual receptive fields, auditory receptive fields, olfactory receptive fields, and somatosensory receptive fields. In the 1950s and 1960s, Hubel and Wiesel demonstrated that different visual stimuli have different effects on the firing pattern of a neuron [41, 42]. Hubel and Wiesel categorized the visual neurons in their experiments into two groups: simple cells and complex cells.

Simple cells are neurons that fire whenever stimulated with a specific input pattern. In the case of visual neurons, simple cells specialize to activate to specifically oriented edges, or bars. These neurons are basic pattern detectors that activate only to one specific input (see figure 5). In other words, if the neuron “sees” its pattern, it will fire rapidly. If however the pattern does not fully align with its receptive field, the neuron fires less frequently, or not at all. The same principle of basic pattern detection functions for other types of receptive fields as well. Simple cells with auditory receptive fields for example respond selectively to sound frequencies.

Figure 5: The response of a simple cell with a visual receptive field, when stimulated with four different patterns. The first example shows a series of action potentials, also known as spike train, when stimulated with an input pattern with the optimum size, position, and orientation. The second example shows a response to an input pattern that has an optimal orientation and size, but a slightly unaligned position with the receptive field. The neuron emits fewer action potentials. In the third example, the orientation of the input pattern does not align at all with the receptive field of the neuron, causing the neuron not to respond. In the last example, the input pattern represents a large fully illuminated region, which does not align with the neuron’s receptive field, and results in no firing.

Complex cells on the other hand, as the name suggests, have more complex receptive fields. In the case of visual receptive fields, some complex cells fire whenever stimulated with a specific input pattern, regardless of its position in the receptive field (see figure 6A). In other words, similarly to artificial convolutional neural networks (CNNs) [43, 44], the receptive field of these cells is said to have transition invariance. Other complex cells might have a receptive field with a directional preference (see figure 6C). These cells fire whenever stimulated with an input pattern that moves at a specific direction regardless of time, but do not fire if the same input pattern moves across the receptive field in the opposite direction. Similarly to artificial recurrent neural networks (RNNs) [45, 46], the receptive field of these cells are said to have temporal invariance. Some complex cells fire when stimulated with a specific pattern, regardless of the pattern’s size inside the receptive field (see figure 6D). These complex cells have a receptive field that is scale invariant.

With every successive processing stage, the receptive field of a neuron grows in size and complexity. In the early processing stages, the receptive field of neurons is specialized towards very specific input patterns, as seen in simple cells. The receptive field of neurons in "deeper" processing stages start to form various invariant abilities, such as transition invariance, scale invariance, rotational invariance, etc., as seen in complex cells with visual receptive fields. At a given stage, the receptive field of complex cells can reach even more advanced abilities, such as recognizing objects regardless of viewing angle (see figure 6B). The receptive field of these complex cells are said to have viewpoint invariance.
invariance. How neurons form their given receptive field and the various kinds of invariances remains unclear. It seems as if the architecture of biological neural networks already contains the necessary inductive biases in its design, to enable the learning of generalized invariances.

1.4 Lateral inhibition

Besides excitatory pyramidal neurons, the brain also contains a wide spectrum of specialized inhibitory interneurons. While pyramidal neurons are quite large, have an apical dendrite, and can form connections with distant regions of the brain, interneurons are smaller in size, do not have an apical dendrite, and usually form many local connections.

Neural circuits containing both pyramidal neurons and interneurons can organize in several ways, giving rise to networks with complex properties (see figure[7]). In this work, we are interested in the organization that enables lateral inhibition. Lateral inhibition occurs when a pyramidal neuron activates an inhibitory interneuron, which in turn suppresses the activity of surrounding pyramidal neurons. Networks with lateral inhibitory configurations (from now on simply referred to as "competitive circuits") form a "Winner take all" competition between the pyramidal neurons within an area. The neuron that fires before the other neurons "wins" the competition, by inhibiting the rest.

1.5 Temporal codes

"Individual nerve cells were formerly thought to be unreliable, idiosyncratic, and incapable of performing complex tasks without acting in concert and thus overcoming their individual errors. This was quite wrong, and we now realise their apparently erratic behaviour was caused by our ignorance, not the neuron’s incompetence."[48]

Neurons transmit and receive information via sequences of action potentials, also known as spike trains. We now know that the number of action potentials within spike trains is not the only variable used to encode information; the timing
Figure 7: The various network configurations of inhibition. Circles represent interneurons, whereas triangles represent pyramidal neurons. Upward arrows indicate the activation of a neuron, whereas downward arrows indicate the inhibition of a neuron. A sequence of vertical lines represents a spike train. In a feedback network, the activation of a pyramidal neuron \( A \), results in the activation of an interneuron, which results in the inhibition of \( A \). This network organization might serve as a regulatory mechanism for a neuron’s firing pattern. In a feed-forward network, the activation of an interneuron results in the inhibition of another pyramidal neuron. A lateral inhibitory configuration occurs when several pyramidal neurons share one or more common interneurons. In this network configuration, pyramidal neurons compete with one another by trying to fire before the other pyramidal neurons, thereby activating their common interneurons and inhibiting the rest. Illustration reprinted from [47].

of the emitted spikes can also be part of the code [49, 50, 51, 52, 53]. Recent evidence supports the existence of precise spike patterns with millisecond-level precision (see figure 8) [54, 55, 56, 57, 58]. These patterns have been found in the olfactory system [59, 50], auditory system [60, 50], visual system [61, 52], somatosensory system [62, 63], and within other sensory systems [64].

Figure 8: The detection of spatiotemporal spike patterns. The Y-axis of the plot represents 100 neurons, 50 of which take part in a repeating precise spike pattern. The X-axis represents time. The dots within the plot are spikes. The darker dots represent spikes that are part of a precise spike pattern. The bottom panel plots the overall spike counts over 10 ms time intervals. The right panel plots the spike counts of each neuron over the whole period. Reprinted from [65]. Modified from [66].

The role and mechanism behind the precise spike patterns remains however enigmatic. Figure 8 perfectly demonstrates the elusiveness of the problem: While looking at each neuron’s spike train, the spikes within the spike trains seem completely random. At first glance, it looks as if there’s no temporal information encoded within the spike trains.
Only after performing an exhaustive search, do the repeating millisecond-level precise spike patterns become apparent (represented as black dots).

2 The Koha Model

The previous section addressed some of the biological observations required for the Koha Model. This section on the other hand is speculative in nature. In this section, I present a model which aims to explain two phenomena:

1. How information is processed and stored within neural circuits.
2. How neurons learn to become pattern detectors in an unsupervised way.

2.1 The Koha code

We have seen in section 1.2 that dendritic spines serve as electrical and biochemical compartments, able to amplify, as well as dampen incoming signals ([21, 22]). In fact we now know that most of the filtering of local potentials in a neuron happens within the spine neck and not along the dendrite ([20]). The spine is somehow able to know when to let information pass through its neck and when not. The amplification, or dampening of incoming signals inside dendritic spines directly depends on the length and narrowness of the spine neck. Changes in the length and narrowness of the spine neck therefore, have a direct effect on the compartmentalization of the spine ([19]). We also know that due to the spine neck’s highly dynamic actin cytoskeleton, dendritic spines can change their physical structure in seconds, following an appropriate stimulus ([22, 23, 24, 25, 26]). This change in shape is governed by actin-associated proteins such as drebrin ([67], α-actin ([68]), gelsolin ([69]), fodrin ([70]), etc. The properties of these proteins in turn depend on the internal calcium levels within a spine ([38]). Increases in Intracellular calcium levels might therefore change the properties of microfilament-associated proteins, which then interact with the actin cytoskeleton of the spine, which results in the reshaping of the spine neck, which in turn influences the compartmentalization of the spine.

In short, if the dendritic spine could control its internal calcium levels in response to incoming signals, then it could also dampen, or amplify those signals by changing its shape. This is the main idea of the Koha model. Almost all mushroom spines possess an enigmatic organelle known as a spine apparatus ([30]). Spine appartus are now known to be intracellular calcium stores that are able to quickly absorb, as well as release $\text{Ca}^{2+}$, depending on internal feedback ([31, 34, 35]). A fascinating study showed that synaptic stimulation can cause the spine apparatus to release its internal calcium stores ([71]). According to the proposed hypothesis, I argue that there is a molecular unit within the dendritic spine that encodes a temporal pattern in the form of a sequence of “on” states and “off” states. Whenever a dendritic spine receives a sequence of inputs, it will compare the spike train’s temporal pattern, with its internal temporal code. If the received spike train’s temporal pattern is similar to the spine’s internal code, information flows from the spine to its dendrite freely. If on the other hand the spike train’s temporal pattern is significantly dissimilar from the spine’s internal code, the spine apparatus gets notified to increase intracellular calcium levels, which changes the shape of the spine and dampens the incoming signals. Signal processing on a local level is therefore controlled mechanically at the spine neck, the behavior of which depends on the temporal pattern of the incoming spike train.

2.2 Specialization through competition

To summarize, every dendritic spine on the apical dendrite of a pyramidal neuron computes a distance measure between the temporal pattern of the input signal and that of its internal code. When a neuron receives inputs at its dendritic spines, it is essentially in a competition with all other neurons that receive the same inputs. Neurons with internal codes that are substantially different from the input signals attenuate the signals (by altering the spine necks of their spines) and delay the propagation of the signals from the apical tuft to the soma of the neuron (see figure 10). The neuron with the internal codes most similar to the input signals, will allow the signals to move freely from the apical dendrite to the soma. The “best matching neuron”, that is, the neuron whose signals past through the apical dendrite to the soma the fastest, will fire before the other pyramidal neurons. If the neurons are organized as a competitive circuit (see section ??), the best matching neuron will activate neighboring inhibitory interneurons, which then inhibit the other pyramidal neurons in that participated in the competition.

We now know that whenever a neuron fires an action potential, another impulse from the soma is also generated that propagates backwards through the apical dendrite of the neuron. This process is also known as Neural backpropagation. When a backpropagating action potential contacts previously activated dendritic spines, a superlinear rise in internal calcium levels occurs inside the dendritic spines ([23, 22]). Neighboring spines that were not activated prior to the backpropagating signal would be unaffected. I argue that the backpropagating action potential can be viewed as a mechanism to inform dendritic spines that their action resulted in a best matching neuron. That is, it informs the
Figure 9: A - The response of a dendritic spine when stimulated with a spike train whose temporal code is similar to that of the spine’s internal code. The spine neck remains unchanged and the signal can pass freely. B - The response of a dendritic spine when stimulated with a spike train whose temporal code is not similar to that of the spine’s internal code. The spine neck becomes longer and narrower, dampening the strength of the incoming signal in the process.

dendritic spines down the apical dendrite that their neuron won the competition (see figure 11). Every dendritic spine will then modify their internal code to become slightly more similar to the temporal code that they received during the competition. In a way the backpropagating signal can be seen as a message to all the dendritic spines - “We won! Update your codes slightly so that we can win the next time even faster!” This process can be viewed as a form of competitive learning [72], which is a variant of Hebbian learning [73]. The internal codes of each neurons' dendritic spines change over time, becoming in the process more specialized towards a specific input. This in turn makes it more likely for a neuron to win the next competition again, if the same given input is provided.

It is worth noting that the model so far explains several organizational and morphological observations: We know that dendritic spines receive most of the excitatory inputs in neurons, and that practically every dendritic spine has an excitatory synapse on its head [10, 11, 12]. Excitatory axons prefer to terminate on spines and almost never connect directly to dendritic shafts (the body of the dendrite) [12]. This consistent observation makes sense, if we assume that dendritic spines serve as computational units that try to find specific temporal patterns within excitatory inputs. Inhibitory axons on the other hand almost always connect directly to dendritic shafts, rather than on spines [13]. This also makes sense, if we assume that one of the roles of inhibitory neurons is to signal the end of a neural competitions. Connecting directly onto dendritic shafts allows for faster propagation, than propagating an inhibitory signal through the spine neck. It also explains why most observed interneurons are essentially spineless [14]. In the Koha model, most inhibitory neurons serve as signal relayers, not as information processing units. The competitive circuit also explains why pyramidal neurons posses about 10 times more local connections with fast-spiking inhibitory neurons, than with other pyramidal neurons, with most of the connections being reciprocal (in the layer 2/3 of the rat neocortex at least) [1].

3 The Koha model as an associative memory network

The Koha model can be viewed as an associative memory model. The idea of an associative memory network is to map an input to the most similar internal pattern. Machine learning models such as the Hopfield Network [45], and the self organizing map [72] are classical associative memory models. Recently self-attention-based architectures, such as Transformers [75] have gained wide popularity in the machine learning space. The newer Modern Hopfield Network [76], an artificial network that uses the Transformer’s attention mechanism as an update rule, is of particular interest.
1. The dendritic spines of the apical tuft receive excitatory signals from a previous layer. Each spine computes the similarity between the temporal pattern of the input signal and that of its internal code (see section 2.1). 2. The processed signals move from the apical tuft, through the apical dendrite to the soma. The signal of each neuron will have to cross this distance. 3. The neuron whose propagating signals reach the soma first, will fire a series of action potentials, which activate surrounding inhibitory interneurons (shown as circles). The activated interneurons then inhibit all the other neurons within the competition.

Figure 11: The process of backward propagation within the Koha model. 1 - The winning neuron fires a series of action potentials to the next layer, as well as to its neighboring interneurons (shown as circles), which inhibit the surrounding neurons. In parallel to that, the winning neuron fires a series of backpropagating action potentials in the direction of its apical dendrite. 2 - The propagating signals move from the winning neuron’s soma, through its apical dendrite, all the way to the individual dendritic spines at the apical tuft. 3 - Each dendritic spine that participated in the competition updates its internal code, so as to become slightly more similar to the temporal pattern of the spike train that each dendritic spine received (see section 2.1).

The Modern Hopfield Network shares many parallels with the Koha hypothesis of biological memory and could be considered as a continuous implementation of the biological hypothesis. The "HopfieldLayer" implementation of the Modern Hopfield Network can be defined as:

$$Z = \text{softmax} \left( \beta RW^T_K \right) W_V$$

The network stores learnable patterns in a weight matrix $W_K$, also referred to as the "key weight matrix". The matrix $R$ is the input set, where every row represents a data point. $\beta$ is a parameter for controlling the "temperature" of the model, whereas $W_V$ is the "value weight matrix".
From a biological perspective, we can imagine every column of $\mathbf{W}_K$ to be a neuron with its temporal codes representing the values of the column. When computing the dot product of a normalized input $\mathbf{R}_i$ with the key weight matrix $\mathbf{W}_K$, we essentially compute the cosine similarity between the input and every pattern of the weight matrix. The resulting vector of cosine similarities represents a score that tells us which neuron is more similar to the given input. Passing the computed scores to the softmax function, creates a probability distribution in which patterns more similar to the input are closer to 1, and dissimilar patterns closer to 0. The described process can be viewed as a neural competition. $\mathbf{W}_V$ represents the output spike train of the neurons, which in themselves would contain precise spike patterns. If dendritic spines truly possess the ability to scan for precise spike patterns, than it is natural to assume that every neuron has a specific temporal "ID", that is, a spike pattern that it transmits. If the activity of presynaptic neuron is too much out of sync with its original "temporal ID", the dendritic spines of postsynaptic neurons will dampen the signal, and therefore weaken the presynaptic neuron’s vote in the dendritic computation of the postsynaptic neuron’s temporal pattern. In fact, an ingenious experiment by [77] showed that the temporal order of a spike train emitted by a neuron, does not get computed in the soma, or even at the axonal initial segment, but rather in the dendritic tree of the neuron.

4 Discussion, Criticism, & Future Work

The current model proposes a mechanism for dendritic spines to scan incoming signals for temporal patterns. But the exact molecular mechanism remains unexplained. Synaptopodin’s preferential location at the spine neck, and its close association with the spine apparatus and the actin cytoskeleton, suggests that synaptopodin is somehow involved in the rapid plasticity of the spine’s actin cytoskeleton. One study suggests that synaptopodin could provide a link between the actin cytoskeleton of the spine neck, and the membrane of the spine apparatus [38]. Considering that some membrane-bound receptors have mechanosensitive properties [78], a mechanical interaction between synaptopodin and the receptor molecules of the spine apparatus might be possible. If correct, calcium release of the spine apparatus might be determined by the spine apparatus’ interactions with synaptopodin.

Another detail not explain in depth, is the role of thin spines. Thin spines in contrast to most mushroom spines do not have a spine apparatus. Because of this fact, one might argue that the spine apparatus cannot be the only mechanism for how dendritic spines filter information. Which is correct, calcium gates within the spine membrane play certainly an important role. It is however important to note that thin spines have significantly longer necks than mushroom spines, and are as a result biochemically more isolated. Thin spines might therefore not play as much of a role in dendritic computations as mushroom spines do. I argue that in the "neural democracy" of the dendritic tree, the "vote" of mushroom spines has more value than that of a thin spine. Considering that mushroom spines can remain stable for a lifetime, while thin spines can appear and disappear throughout life [8, 9], it would make sense to weigh the contribution of mushroom spines more.

The Koha model also argues for the existence of a code within dendritic spines, but it does not describe the physical structure of the hypothetical code. There are however good candidates. The talin protein, a highly conserved [79] mechanosensitive cytoskeletal protein in vertebrates, could be encoding the temporal information that the Koha model suggests exists. In an excellent paper [80], Goult proposes the MeshCODE hypothesis, in which he argues that talin has the right properties to serve as a memory molecule. The talin protein consist of 13 "switches" which can reside in two thermodynamically stable states. The MeshCODE hypothesis refers to these switch states as "on" an "off" states and proposes that information can be encoded on the molecule by either folding, or unfolding the talin switches (see figure 13). Considering that both talin and synaptopodin are tightly connected to the actin of the cytoskeleton, a switch controlled mechanical interaction between talin, actin, and synaptopodin might exist, and could trigger the release of the spine apparatus’ calcium store down the line. Note however that this is a hypothesis build on top of another hypothesis and that the correct answer might be something entirely else.

Another missing component of the Koha model, is the formation of complex cells. The described competitive learning process can give rise to simple cells with simple receptive fields, and even to cells with directional preferences (by adding
skip-connections), but it does not describe, or propose a biologically plausible way for the formation of "generalized invariances". That is, it does not describe how neurons become complex cells with invariant abilities, such as viewpoint invariance. There are several existing hypotheses for how complex cells form [81], but none seem compelling enough on a biological level, that is, none show a concrete process for how invariances are learned naturally. One component that has been purposefully put on the side in this work is the role of basal dendrites. The whole design of the proposed competitive learning process depends on the apical dendrite and its dendritic spines, but ignored the role of basal dendrites, which are in fact just as important. I argue that by incorporating a few more elements to the Koha model (such as basal dendrites and the nonlinearities introduced in the dendritic processing of apical dendrites), one can explain and model the formation of generalized invariances as well. This is also why the optimization criteria in the Modern Hopfield Network example was not covered in detail. The topic of generalized invariances deserves and requires a paper in itself. How biological neural networks learn invariances, and how generalized invariances can be used to design better performing artificial neural networks will be the focus of my subsequent paper.

5 Conclusion

This work introduces two novel ideas with widespread implications for the field of neuroscience:

On a micro level, it argues for the existence of a temporal code within every dendritic spine. Just as genes are the molecular unit of heredity, the Koha code is argued to be the unit of memory. It shows how dendritic spines use these temporal codes to scan for precise spike patterns in their synaptic inputs. It describes a biologically plausible process for how signal filtration occurs within spine necks, as well as provides compelling evidence for the existence of such a mechanism within dendritic spines.

On a macro level, it explains how neurons within competitive circuits can learn to become pattern detectors, through competitive learning. In this model, the chance of a neuron to become the "winning neuron" within a competitive circuit, directly depends on the temporal codes within a neuron's dendritic spines.

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