Supervised Learning with Complex Spikes and Spike-Timing-Dependent Plasticity

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Abstract
One distinctive feature of Purkinje cells is that they have two types of discharge: in addition to simple spikes they fire complex spikes in response to input from the climbing fibers. These complex spikes have an initial rapid burst of spikes and spikelets followed by a sustained depolarization; in some models of cerebellar function this climbing fiber input supervises learning in Purkinje cells. On the other hand, synaptic plasticity is often thought to rely on the timing of pre-synaptic and post-synaptic spikes. It is suggested here that the period of depolarization following a complex spike, combined with a simple spike-timing-dependent plasticity rule, gives a mechanism for the climbing fiber to supervise learning in the Purkinje cell. This proposal is illustrated using a simple simulation in which it is seen that the climbing fiber succeeds in supervising the learning.

Introduction
In addition to weak inputs from parallel fibers and local inhibitory cells, a Purkinje cell receives a strong input from a single climbing fibre [1,2]. The Purkinje cell has two distinct types of spike; simple spikes, in response to the aggregated signal from many weak inputs from the parallel fibers, and complex spikes, in response to an input from the climbing fiber. The simple spikes are similar to the spikes found throughout the nervous system whereas complex spikes have a distinctive structure with a leading spike followed by a series of partial spikes called spikelets and a sustained period of depolarization during which the cell is refractory [2–6].

It has been known for nearly two hundred years that the cerebellum is important for motor coordination [7,8] and, while there are non-motor symptoms [9,10], damage to the cerebellum is most closely associated with motor problems: intentional tremor, a want of harmony in movement, a loss of kinetic melody, a distinctive wide stance and an unsteady ataxic gait [11–14]. However, the precise role of the cerebellum in movement is still debated: it is proposed, for example, that the cerebellum is a computational engine for deciding precise levels of muscle activation [17–20], or an organ of prediction, predicting the sensory consequence of movement [21–24] or that its has a role in proprioception [25,26]. Of course, these putative functions may co-exist, or may be co-dependent, but there is no definitive description of what the function of the cerebellum is and there certainly no theory of cerebellar function which would predict its distinctive structure.

There are also many compelling ideas as to what the role of complex spikes may be. Some ideas relate to signalling: it has been proposed, for example, that complex spikes may provide a timing signal for motor control. [15,16]. Other ideas relate to learning: in the Marr-Albus-Ito model of the cerebellar cortex, climbing fiber spikes are a supervisory error signal [17–19,27]. Similarly, in models where the cerebellum is seen as predicting sensory outcomes, the climbing fiber input communicates significant deviations from the sensory prediction, a deviation which is incorporated into future predictions, if it is repeated with sufficient frequency. As with the overall function of the cerebellum, the complex spike is likely to subserve multiple functions. However, there is evidence that acting as learning signal is one these function: an increase in the frequency of complex spiking is associated with depression in the synapses connecting the parallel fibers to the Purkinje cells and a decrease is associated with potentiation of those synapses [28–30]. This paper proposes a mechanism for this plasticity. Long term plastic changes to these synapses are likely to be mediated by rather complex mechanisms. Here, however, a very simple mechanism is proposed, it is suggested that the refractory pause caused by a complex spike plays a crucial role in plasticity.

This proposal is made in the context of spike-timing-dependent plasticity (STDP). STDP describes models of long term plasticity in which the changes to synaptic strengths depend on millisecond-scale differences in spike times. In STDP models changes to synapse strengths depend on the timing difference between pre-synaptic and post-synaptic spikes. Frequently for excitatory synapses, this dependence is anti-symmetric, so, if a pre-synaptic spike precedes a post-synaptic one the synapse is potentiated, but if the order is reversed, the synapse is depressed. In other words, long term depression (LTD) and potentiation (LTP) depend on the timing of pre and post-synaptic spikes as well as the coincident activity of the pre and post-synaptic neurons.

A causal structure was implicit in the original formulation of Hebbian plasticity [31], a remarkable piece of prescience: in the

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Funding: The author is grateful to the James S. McDonnell Foundation for financial support through a Scholar Award in Human Cognition. The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

Competing Interests: The author has declared that no competing interests exist.

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nineties a series of papers pointed to experimental evidence for timing effects in plasticity [32–36] including a definitive demonstration of a spike-order dependent plastic changes in vitro in [33], the observation of asymmetric STDP in vivo in developing Xenopus in [37] and a clear graph of the time dependence of plastic changes in vitro in [38]. STDP has also been observed in vivo in mammal in, for example, [39–43] and, in fact, STDP has now been observed across animal models, brain regions and cell types. The asymmetric causal window is the most common STDP rule, but there is considerable variability, sometimes even within the same neuron, for a review see [45]; [44] gives an historical account.

Interest in putative functional roles for STDP preceded biological evidence for it [46] and a wide range of possible roles are reviewed in [47]. One particular strength of STDP rules is that they are capable of doing unsupervised learning of spike-spike correlations in the input [48–50]. Roughly speaking, if spikes from a group of pre-synaptic neurons tend to arrive at roughly the same time, they are more likely to cause a post-synaptic spike, resulting in potentiation.

It is proposed here that the learning role of complex spikes is to give a pause during which there is no post-synaptic spike. This will mean any pre-synaptic spikes will be in an anti-causal relationship with the initial spike of the complex spike. According to the standard STDP rule, this will cause long-term depression. In short, it is proposed that a standard STDP rule governs plasticity in synapses from the parallel fibres and local inhibitory neurons and that the combination of this rule and the refractory pose caused by the complex spike results in supervize learning. This is broadly in line with the models of cerebellar function in which climbing fiber complex spikes result in supervise learning. This is broadly in line with the models of cerebellar function in which climbing fiber complex spikes result in supervize learning. This is broadly in line with the models of cerebellar function in which climbing fiber complex spikes result in supervize learning.

...supervised learning in response to an error signal carried by the climbing fibre. In fact, the proposal described here can be thought of as the synapse strength which may be changed by synaptic plasticity.

To implement the STDP rule each synapse has two potentials [47]. One potential, \( x_i \), increases in response to pre-synaptic spikes and satisfies

\[
\tau_+ \frac{dx_i}{dt} = -x_i + \sum_j \delta(t-t_j^+)
\]

where the postsynaptic spike times are \( t_j^+ \). The other potential, \( y \), increases in response to post-synaptic spikes and satisfies

\[
\tau_- \frac{dy}{dt} = -y + \sum_j \delta(t-t_j^-)
\]

where the postsynaptic spike times are \( t_j^- \). The evolution of the synapse strength is now

\[
\frac{dw_i}{dt} = \eta_+ \, x_i(t) \sum_n \delta(t-t_n) - \eta_- \, y(t) \sum_j \delta(t-t_j)
\]

with hard lower and upper bounds on \( w_i \):

\[
0 \leq w_i \leq w_{\text{max}}
\]

where \( w_{\text{max}} = 0.015 \).

The choice of \( \tau_+ \) and \( \tau_- \) effects the temporal extent of the correlation that can be learned; here \( \tau_+ = \tau_- = 20 \) ms. The choice of \( w_{\text{max}} \) sets a synaptic scale; \( \eta_+ = 0.005w_{\text{max}} \) and \( \eta_- \) is set so that

\[
B = \frac{\eta_-}{\eta_+ + \tau_+} = 1.05
\]

which means \( \eta_- = 0.00525w_{\text{max}} \) and is larger than \( \eta_+ \) so if there is no relationship between pre- and post-synaptic spikes at a synapse its strength falls.

Thus, the STDP used here is additive with an exponentially decaying plasticity window and, in a biologically appropriate way, the individual spike-spike pairs are not tabulated, rather, a running...
account is maintained using potentials. The ability of this version of the STDP to learn structure in the input data is demonstrated in [50] by dividing the retinal neurons into two correlated groups.

The retinal neurons are divided into two equal sized groups with the firing rates for each neuron chosen randomly, but in such a way as to give correlated firing inside each group. Specifically, for each group all the neurons are given a firing rate $r_i \sim 10(1 + \zeta f z_i j_i) \left( 10 \right)$ where both $j_i$ and $f$ are chosen from normal distributions with mean zero and variance one. While the same $f$ is used for all neurons in the group, each $j_i$ is chosen separately. These firing rates are held fixed for a time interval chosen randomly using an exponential distribution with rate 50 Hz, this corresponds to an average interval length of 20 ms, with the intervals determined separately for each group. At the start of each new interval a new $f$ and new $j_i$ are chosen, giving the rates, $r_i$, for the neurons in the group.

One of the two groups of neurons wins out over the other group. Small initial variations give one group a slightly greater tendency to control the post-synaptic neuron. The synapses in this group get stronger, reducing the proportion of post-synaptic spikes that are in a causal relationship with the pre-synaptic spikes from the other group. This means that the strength of these synapses will diminish. This process is slow, but with time, one group has synapses with strengths close to zero, the other, strengths close to $w_{\text{max}}$.

This Song-Abbott model exhibits unsupervised learning; the STDP rule allows the simple network to learn a correlation pattern in the input, but it does not determine which pattern it learns. Small initial differences decide which of the two groups wins out. The idea here is to show how a neuron modelled on the climbing fiber in the cerebellar cortex can act as a supervisor and determine which group ends up with stronger synapses.

The goal here is to illustrate the proposed learning mechanism using the network from [50] described above. To illustrate the proposal a single extra neuron has been added to the existing simulation. To make it easy to compare to the original model, nothing else has been changed, however, the pre-synaptic layer neurons are now interpreted as corresponding to parallel fibers and the post-synaptic neuron is interpreted as being a Purkinje cell. This is shown in Fig. 2B: the input later neurons are now considered to model granule cells rather than retinal cells, the V1 neuron has been replaced by a Purkinje cell and there is now a climbing fiber whose firing rate puts it in one of the two groups of granule cells.

Thus there is now an extra input, the climbing fiber is considered to belong to one of the two groups of neurons in the pre-synaptic layer; its rate is set at $10(1 + \zeta + \xi)$ where $\xi$ is the same value chosen for the other neurons in its group and $\zeta$ is chosen specifically for this neuron from the usual normal distribution. In this way, this neuron resembles the other pre-synaptic neurons in its own spiking, however, its effect on the post-synaptic neuron is
very different: if the climbing fiber neuron fires so does the post-
synaptic neuron and, after it fires, the conductance in the post-
synaptic neuron is fixed at zero for the refractory period of
20 ms. This mimics a complex spike. Figure 3 shows an
example of spiking activity in the model.

Results

The climbing fiber succeeds in supervising the learning, in every
one of a 100 trials the group with the climbing fiber is the one that
ends up with very low synapse strengths. This shows that the
supervision completely lifts the ambiguity in which group ends up
with low synaptic strengths and which ends up with high.

Supervised learning is also much faster. In Fig. 4 the average
value of \( d_g/w_{\text{max}} \) for the two groups is plotted against time. The

Figure 2. A summary of the model. A shows the network used in [50]: 1000 retinal layer neurons feed forward to a V1 neuron. The V1 neuron is
modelled as an integrator and fire neuron without refractory period. The retinal layer neurons are modelled as Poisson processes; they are divided into
two equal groups and the firing rates in each group are correlated with each other, but not with the firing rates for neurons in the other group. B shows
the same model adapted to the cerebellum, what was the retinal layer is now the granule layer so their feed forward projections are now
parallel fibers (PFs), the V1 neuron is now a Purkinje cell (PC). There is an extra neuron whose projection models the climbing fiber (CF); its firing rate
is correlated to the firing rates in one of the two groups of granule cells but the effect of spikes in the climbing fiber are different, it causes the
Purkinje cell to spike and then undergo a refractory period.

doi:10.1371/journal.pone.0099635.g002

Figure 3. An example of spiking activity in the model. This
shows the spiking activity in the Purkinje cell for one simulation trial;
the activity between 10 s and 10.5 s is given, in other words, it shows
the activity before plasticity has had any substantial effect on the
behavior of the cell. The simple spikes are marked with short black
vertical dashes, the complex spikes by tall green vertical dashes; the
horizontal dashes mark the refractory periods which follow each
complex spike. During these period 481 spikes arrive from the input
neurons correlated with the climbing fiber activity and 354 spikes from
the other group.
doi:10.1371/journal.pone.0099635.g003

Figure 4. The progress of supervised learning in the supervised
and unsupervised cases. The value of \( w_i/w_{\text{max}} \) is averaged across
each group during the simulation; the simulation is repeated for 100
trials and averaged. The curves for the supervised learning are marked
\( a_+ \) with \( a_- \) indicating the group correlated to the climbing fiber. In the
case of the unsupervised learning the group that wins out varies from
trial to trial, but the average is taken across matching groups. \( b_+ \) mark
the unsupervised learning groups, with \( b_- \) the average for the group
that ends up with the larger average value. The black lines trace the
average, the grey lines show the size of trial-to-trial variation, marking
\( \pm \sigma \).
doi:10.1371/journal.pone.0099635.g004
Supervised learning reaches an equilibrium after 2500 seconds but the unsupervised learning only reaches it after 7500 seconds.

Averaged over 100 trials the group correlated with the climbing fibre has \( \langle w_i/w_i^{\text{max}} \rangle = 0.063 \pm 0.006 \) after 7500 s; the other group has final value \( \langle w_i/w_i^{\text{max}} \rangle = 0.918 \pm 0.005 \) where the plus-or-minus gives the standard deviation. The same values for unsupervised learning are \( \langle w_i/w_i^{\text{max}} \rangle = 0.097 \pm 0.055 \); averaged over the group that ends up lower and \( \langle w_i/w_i^{\text{max}} \rangle = 0.932 \pm 0.043 \) for the other group. Thus, the averages are similar but the variation greater; however, the most significant difference is that supervision increases the speed of learning and determines which group is which.

The success of supervision is robust, it does not rely on a precise choice of climbing fibre parameters. The rate of learning does become slower if the refractory component of the complex spike is reduced, for example. Fig. 5 shows the effect of reducing the refractory period to 5 ms, the learning remains reliable but is slower to reach equilibrium. Increasing the refractory period to 40 ms has no discernible effect on learning.

To investigate robustness of the supervised learning another simulation was performed with very different parameter values, values chosen to mimic the behavior of cells in the cerebellum which are based on the description given in [51–53]. In particular, in this simulation the network topology is unchanged, but the firing behavior of the input layer was chosen to make it bursty, in imitation of the real behavior of granule cells.

The firing rates of the input neurons are determined by a more complicated Cox process with two mean firing rates, a low rate \( \lambda_0 = 1 \) Hz and a high rate \( \lambda_1 = 75 \) Hz. As before, the rate for each of two groups of neurons is piece-wise constant. At the start of each interval it is designated a high or low rate interval with equal probability. The actual rate at each synapse is then set as

\[
p = \lambda_0 + \sigma_v \varepsilon
\]

where \( \varepsilon \) is drawn separately from a normal distribution for each input neuron in the group. If the group is beginning a high rate interval the high rate values \( \lambda_1 \) and \( \sigma_1 \) are used with probability 0.1, otherwise the low rate values \( \sigma_0 \) and \( \sigma_0 \) are used; for a low rate interval the low rate values are always used. The value of \( \sigma_0 \) is different for low and high rate intervals chosen with \( \sigma_0 = 0.25 \) Hz and \( \sigma_1 = 20 \) Hz. Finally, low and high rate intervals have different average length, with the low rate intervals having an average length of 75 ms and the high rate intervals an average length of 25 ms. The climbing fibre is given a firing rate using the same formula as the corresponding group but it has a higher chance of being set to the high rate; \( p = 0.25 \). The dynamics of the synapses is the same as before. Background noise is also included as a Poisson process with rate 750 Hz, synapse strength \( g = 0.096 \) and time constant \( t_v = 5 \) ms. An example of spiking activity in this model is given in Fig. 6.

The climbing fibre successfully supervises the learning, this can be seen in Fig. 7; the group that is correlated with the climbing fibre has an average synaptic strength \( \langle w_i/w_i^{\text{max}} \rangle = 0.066 \pm 0.009 \), the other group has \( \langle w_i/w_i^{\text{max}} \rangle = 0.956 \pm 0.005 \). Learning occurs quickly, the low group has equilibrated after 1250 s, the other group after 1000 s. Learning does not occur without supervision; if there is no climbing fibre input the synapses in both groups equilibrate at \( \langle w_i/w_i^{\text{max}} \rangle = 0.937 \pm 0.005 \) and if one group has a larger value of \( w_i/w_i^{\text{max}} \) at a given time, the probability it belongs to the same group 50 s later is 0.55. Fig. 8 shows how the distribution of synapse strengths differ in the supervised and unsupervised case.

The bursty input is very effective in driving the output; this is why there is no learning without the climbing fibre input: all the synapses approach the maximum value. In fact, the synapses that are suppressed by the climbing fibre input recover if that input is turned off. This is shown in Fig. 9 where the spike rate is also plotted, it increases when the climbing fibre input is switched off. This behavior is reminiscent of that observed in Purkinje cell when the inferior olive is lesioned [30,54–56].

In these simulations the climbing fibre has a firing rate of about 2.5 Hz; this is comparable with the 1.5 Hz firing rate observed experimentally in quiescent cat [57] or the observation of a 0.9 Hz background rate in anaesthetised rat, with a high rate in response to stimulation [60]. The spiking rate of the Purkinje cell is also realistic [28,58,59], after learning it has an average firing rate of 62 Hz, this is the average of more and less active periods driven by the bursty input in the parallel fibers, during the more active periods it fires at 127 Hz. Changing the climbing fibre firing rate and the refractory period, \( t_v \), that follows complex spikes does effect learning, Fig. 10A and Fig. 10B, but learning is reasonably robust. However, learning only occurs for a small window in \( \theta \), the ratio between depression and potentiation in the STDP rule.

**Figure 5. The effect of shortening the refractory period following a complex spike.** The solid black line graphs the synapse strengths for the two groups in a simulation where the refractory period following climbing fibre input is reduced to 5 ms. As before, the result has been averaged over 100 trials. Learning is still effective in the sense that the same group always wins; learning is, however, slower. For comparison, the same graph for the original refractory period of 20 ms has been include in dashed grey and the graph for unsupervised learning in solid grey.

doi:10.1371/journal.pone.0099635.g005

**Figure 6. Example spike behavior.** This shows the spiking activity in the Purkinje cell for one simulation trial with the parameters chosen to reflect the behavior of cells in the cerebellum. The activity between 10 s and 10.5 s is given, before plasticity has had any substantial effect. The simple spikes are marked with short black vertical dashes, the complex spikes by tall green vertical dashes; the horizontal dashes below the spikes mark the refractory periods which follow each complex spike. The horizontal lines above the spikes mark the high firing periods for the two groups, below the dotted line corresponds to the group correlated with the climbing fibre activity, above to the other group.

doi:10.1371/journal.pone.0099635.g006
This window is made broader if the climbing fiber firing rate is increased, accounting for the strong depolarization associated with the complex spike by using higher values of $A_z$ and $A_{\pm}$ for complex spiking has a similar effect. In short, the amount of learning depends on the detail of the simulation and any precise predictions would require a much more extensive simulation. However, the increased depression observed with the higher rate of climbing fibre input is broadly consistent with the experimental observations noted in [61].

Discussion

The example model provided here is intended to illustrate the possibility that STDP and complex spiking can result in supervised learning in a natural way. A complete account of learning in Purkinje cells will be complicated. The model presented here ignores most of the complexity of the cerebellar cortex, there are no inhibitory input cells for example. Furthermore, it ignores the complexity of the complex spike itself. The complex spike, in vivo, is generated by a broad dendritic depolarization mediated by the climbing fiber input. It is associated with calcium spikes in the dendrites which are activated by the climbing fiber input [3,64] and these may be responsible for the sustained depolarization that causes the pause in spiking after the initial spike of a complex spike [63]. There is a very low density of sodium gates in the dendrites and they are not thought to support active back-propagation of axonal spikes, however the spikes do spread passively in the dendritic tree [5,62,65,66]. The spikelets which follow the initial spike of a complex spike are, in contrast, axonally generated and, like the simple spikes, are initiated in the axonal hillock [63].

These details have been ignored here but would need to be incorporated in further development of the model. For example, the passive back-propagation of axonal spiking is likely to filter the post-synaptic spike timing. It is also likely that calcium dynamics plays a role in modulating plasticity. Finally, the treatment here ignores all but the first spike in the complex spike; it is easy to check that naively including the spikelets as supplementary post-synaptic spikes increases the strength of the supervisory effect since it increases the number of post-synaptic spikes in an anti-causal relationship with any pre-synaptic spikes which arrive during the subsequent refractory period.

The model uses a particularly simple description of STDP, ignoring the considerable progress that has been made in exploring different STDP rules. For example [67] presents a STDP rule which relates the plastic changes not to the post-synaptic spiking behavior but to the local post-synaptic membrane potential. This rule accounts for frequency-dependent [33,68,69] and dendritic-location dependent [70–73] effects which are observed experimentally but which are not accounted for by the STDP rule used here, despite the likely importance of dendritic-location dependent effects in the case of Purkinje cells.

It might be expected that the strong depolarization of the Purkinje cell after a complex spike would increase the size of the effect seen here. However, although STDP has been observed in Purkinje-like cells [34], little is known about what form STDP in Purkinje cells might take. Certainly, most STDP rules share an association between causal spikes and potentiation and acausal spikes and depression; the advantage of the simple rule used here is that it abstracts this common feature. What the model described here does illustrate is a potential mechanism for supervised learning with complex spikes and STDP; it remains to be seen whether this mechanism is part of learning in Purkinje cells. In fact, STDP has been previously proposed for learning in Purkinje cells: in [80] the synaptic weight distributions associated with a typical, unsupervised, STDP rule is compared with the synapse strengths in Purkinje cells and found to be in good agreement; this is certainly a criterion that could be usefully applied to more
realistic STDP modelled developed from the supervised learning mechanism proposed here.

The cerebellum appears to have a role in classical conditioning [74]. This appears to support a motor learning view of cerebellar function [75] and the issue of timing, the lengthy delay between the conditioned and unconditioned stimuli, is certainly a challenge to other views of cerebellar function and for the sort of time windows normally associated with STDP. The Golgi cells are believed to act to create delays in cerebellar dynamics [76] but there is experimental evidence for a long plasticity windows directly at the parallel fiber synapses, with LTD occurring when parallel fiber input proceeds climbing fiber input by 100 ms or more [77]. The temporal ordering involved in this is the opposite to the one required for the supervised learning described here. In fact, the synaptic dynamics described in [77] are very complicated, the LTD that occurs when parallel fiber input proceeds climbing fiber input is associated with sparse parallel fiber activity; other experiments find LTD when climbing fiber activity narrowly proceeds parallel fiber activity [61,78].

The mechanism also raises a question about the dynamics of other neurons where inhibitory input or complicated spiking dynamics which cause variable periods of quiescence: is it possible that the complex spike with its extended pause in spiking may be a particularly direct example of a more widespread phenomenon where learning is supervised through the modulation of pauses in spiking? In other words, is it possible that the simple STDP rule is a plasticity Swiss army knife, producing Hebbian and anti-Hebbian learning in different neuronal systems through the modulations of pauses? The answer seems to be no. There are brain areas that resemble the cerebellum, such as the dorsal cochlear nucleus in mammals and the electro-sensory lateral line lobe of mormyrid electric fish: they have afferent structure that is similar to the parallel fibers and they are believed to calculate a ‘negative images’, a predicted sensory background which is removed from sensory input as part of processing [79]. However, [34] indicates that the electro-sensory lobe does not exploit the mechanism described here and, in fact, has a reversed STDP rule with causal spike pairs undergoing LTD. Thus, STDP appears to be variable and complicated mechanism and it remains to be seen whether the simple observation described here is useful in analysing its operation.

![Figure 9. Switching off the climbing fiber.](image)

Figure 9. Switching off the climbing fiber. In this simulation the climbing fiber input is switched off after 1000 s, the time marked with a vertical line. A plots the synapse strength, the group that had been correlated with the climbing fiber input increases steadily towards the other group. The spike rate also increases, this is shown in B. The results are averaged over 25 trials.

doi:10.1371/journal.pone.0099635.g009

A: synapse strength

B: spike rate

![Figure 10. Varying the simulation parameters.](image)

Figure 10. Varying the simulation parameters. In each of these graphs the difference in \( \langle w_i/w_{\text{max}} \rangle \) between the two groups after 1500 s is plotted. Thus, if \( \delta \langle w_i/w_{\text{max}} \rangle \) is near one the gap between the two groups is nearly as large as it could be, if it is near zero very little separates the two groups. In A the refractory period following a complex spike is varied between \( t_r = 10 \) ms and \( t_r = 80 \) ms. For \( t_r = 10 \) ms learning is reduced to \( \delta \langle w_i/w_{\text{max}} \rangle = 0.78 \); in fact the group correlated with the complex spike has \( \langle w_i/w_{\text{max}} \rangle = 0.18 \) and the other group has \( \langle w_i/w_{\text{max}} \rangle = 0.96 \). For all larger values of \( t_r \) the gap \( \delta \langle w_i/w_{\text{max}} \rangle \) is larger than 0.87. In B, \( p_c \), the probability that the climbing fiber is set to a high firing rate during the high firing period is varied; this does have a considerable effect on learning, at 0.05 \( \delta \langle w_i/w_{\text{max}} \rangle \) is 0.18 with \( \langle w_i/w_{\text{max}} \rangle \) = 0.18 values of 0.74 and 0.92. The overall firing rate of the climbing fiber changes with \( p_c \), for \( p_c = 0.05 \) is it 1.7 Hz, this rises to 3.3 Hz for \( p_c = 0.35 \). Finally, in C the value of \( b \), the ratio between the LTD and LTP in the STDP rule is varied. The black line plots \( \delta \langle w_i/w_{\text{max}} \rangle \) with \( p_c = 0.25 \), the value used elsewhere, for the grey line \( p_c = 0.5 \). If \( b \) is low \( \langle w_i/w_{\text{max}} \rangle \) is near one for both groups, if \( b \) is high, it is near zero for both; learning occurs in a window between the two. In all three plots there are ten trials for each value. Supervision was successful for all but one trial for \( b = 0.85 \) and \( p_c = 0.25 \): that is, in all but one trial the group correlated with the climbing fiber had a lower value of \( \langle w_i/w_{\text{max}} \rangle \) at the end of the supervision.

doi:10.1371/journal.pone.0099635.g010
Author Contributions
Conceived and designed the experiments: CH. Performed the experiments: CH. Analyzed the data: CH. Contributed reagents/materials/analysis tools: CH. Wrote the paper: CH.

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