Small, correlated changes in synaptic connectivity may facilitate rapid motor learning

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Animals rapidly adapt their movements to external perturbations, a process paralleled by changes in neural activity in the motor cortex. Experimental studies suggest that these changes originate from altered inputs ($H_{input}$) rather than from changes in local connectivity ($H_{local}$), as neural covariance is largely preserved during adaptation. Since measuring synaptic changes in vivo remains very challenging, we used a modular recurrent neural network to qualitatively test this interpretation. As expected, $H_{input}$ resulted in small activity changes and largely preserved covariance. Surprisingly given the presumed dependence of stable covariance on preserved circuit connectivity, $H_{local}$ led to only slightly larger changes in activity and covariance, still within the range of experimental recordings. This similarity is due to $H_{local}$ only requiring small, correlated connectivity changes for successful adaptation. Simulations of tasks that impose increasingly larger behavioural changes revealed a growing difference between $H_{input}$ and $H_{local}$, which could be exploited when designing future experiments.

Animals, particularly primates, can perform a great variety of behaviours, which they are able to adapt rapidly in the face of changing conditions. Since behavioural adaptation can happen even after a single failed attempt, the neural populations driving this process must be able to adapt equally fast. How this occurs remains unexplained. Rapid motor learning is typically studied using external perturbations such as a visuomotor rotation (VR), which rotates the coordinates of the visual feedback with respect to those of the movement. Both humans and monkeys can learn to compensate for the resulting error between actual and expected visual feedback in a few tens of trials. This behavioural adaptation is accompanied by changes in the activity of neurons in primary motor cortex (MI), and the upstream dorsal premotor cortex (PMd). It is unclear whether these neural activity changes are mediated by synaptic weight changes within the motor cortices or are driven by altered inputs from even further upstream areas.

When learning a skill over many days, behavioural improvements are paralleled by rewiring between MI neurons. This seems not to be the case for rapid learning: throughout VR adaptation, the statistical interactions across neural populations in both MI and PMd remain largely preserved. These preserved interactions rule out any large synaptic changes within the motor cortices, as they would cause these models to degrade. Instead, rapid VR adaptation may be driven by the cerebellum and/or posterior parietal cortex. A pioneering Brain Computer Interface (BCI) study cast further doubt that significant synaptic changes occurring within MI are necessary for rapid learning. In that study, monkeys controlled a computer cursor linked by a “decoder” to the activity of recorded MI neurons.
neurons. After learning to use a decoder that used the natural “intrinsic” mapping of neural activity onto cursor movements, the monkeys were exposed to one of two types of perturbations. When faced with a new decoder that preserved the statistical interactions (i.e., neural covariance) across MI neurons, the monkeys could master it within minutes. In stark contrast, if the new decoder required changes in the neural covariance (an “out of manifold” perturbation), they could not learn it within one session—in fact, it required a progressive training procedure spanning just over nine days on average.

Recording large scale synaptic changes in vivo remains challenging and has not been achieved during rapid motor learning. Alternatively, recurrent neural network (RNN) models offer an exciting yet unexplored opportunity to test the effect of synaptic changes (in the model) on simulated activity during motor learning. RNNs trained on motor, cognitive and BCI tasks exhibit many striking similarities with the activity of neural populations recorded in animal studies, suggesting a fundamental similarity between the two. Previous work using RNNs to model the BCI experiment described above showed that network covariance can be highly preserved even when learning is happening through weight changes within the network. Thus, contrary to intuition, functionally relevant synaptic weight changes may not necessarily lead to measurable changes in statistical interactions across neurons. As a consequence, synaptic changes within PMd and MI during VR adaptation may be very hard to identify through the analysis of neural population recordings.

Here, we used RNN models to test whether VR adaptation might be mediated by synaptic changes within PMd and MI, yet with largely preserved neural covariance within these areas. We addressed this question by asking how adaptation based on connection weight changes within PMd and MI (Hlocal) alters network activity compared to the corresponding activity changes if VR adaptation is based on altered inputs from upstream areas (Hinput) (Fig. 1A). To validate our modelling results, we compared our simulations to experimental recordings from PMd and MI populations during the same VR task.

Under Hlocal, the changes in covariance following VR adaptation only slightly exceeded those under Hinput and were comparable to experimental observations. Thus, when using neural population recordings alone, it may be more challenging to disentangle these two hypotheses than previously thought. Moreover, for both Hinput and Hlocal, the learned connectivity changes were small and highly coordinated, which made them surprisingly robust to noise. To identify additional differences between Hinput and Hlocal, we examined learning during paradigms requiring larger behavioural changes. Covariance changes were larger for these paradigms in both PMd and MI under Hinput, but only in MI under Hlocal, thus providing a possible way to distinguish between the two hypotheses in future experiments. Our findings have implications for the interpretation of neural activity changes observed during learning, and suggest that tasks eliciting larger behavioural changes may be necessary to elucidate how neural populations adapt their activity during rapid learning.

**Results**

To understand whether motor adaptation could be driven by synaptic changes within PMd and MI, we simulated a VR adaptation task using a modular RNN that modelled these two areas, and compared the resulting changes in network activity to those of neural population recordings from PMd and MI during the same VR task. We quantified neural activity changes both in the experimental data and in the model using two measures (Fig. 1B): (1) the relative change in trial-averaged single neuron activity, and (2) the change in neural covariance (denoted as “Methods”). Combined, they capture aspects of single neuron as well as population-wide activity changes during adaptation.

**Small but measurable changes in neural activity within PMd and MI during VR adaptation**

Monkeys were trained to perform an instructed delay task, in which they reached to one of eight visual targets using a planar manipulandum to receive a reward (“Methods”). After performing a block of
unperturbed reaches (200–243 trials, depending on the session), visual feedback about the position of the hand was rotated by 30°, either clockwise or counterclockwise, depending on the session. Monkeys adapted rapidly to these perturbations: the curved reaches observed immediately after the perturbation onset became straighter after tens of trials, with the hand trajectories in the second (late) half of the adaptation block becoming more similar to the baseline trajectories (Fig. 2A). The angular error quantifying the difference between initial reach direction and target location decreased during adaptation (Fig. 2B). This error curve followed a similar trend for clockwise and counterclockwise perturbations, allowing us to analyze the different perturbations together.

Behavioural adaptation was accompanied by changes in neural activity within both PMd and M1 (Fig. 2C). These changes exceeded those during control sessions, where no perturbation was applied (Fig. 2C black; linear mixed model analysis: \( t = 4.4, P = 0.0017 \)). The amount of change was greater within PMd than M1 (\( t = 8.9, P < 0.0001 \)). We also found small but detectable changes in neural covariance during VR perturbation, suggesting that the statistical interactions among neurons change slightly during adaptation (Fig. 2D). Again, these changes exceeded those of the control sessions (Fig. 2D black; \( t = 2.6, P = 0.026 \)).

A modular recurrent neural network model to study VR adaptation
To test whether experimentally observed changes in motor cortical activity could be driven by rapid synaptic plasticity within PMd and M1, we trained a modular RNN model to perform the centre-out reaching task that we studied experimentally. To mimic broadly the hierarchical architecture of the motor cortical pathways, input signals were sent to the PMd module which then projected to the M1 module to produce the final output signal (Fig. 3A, “Methods”). After initial training on the task, the model produced correct reaching trajectories to each of the eight different targets (Fig. 3B and Supplementary Fig. 1). These RNN-controlled movements had the same dynamics as those of monkeys (Fig. 3C). Furthermore, Principal Component Analysis revealed that the population activity of the PMd and M1 network modules was similar to that of the corresponding recorded neural populations (Fig. 3D, E). We used Procrustes analysis to quantify this apparent similarity between model and experimental population activity (Supplementary Fig. 2). This analysis confirmed that the modular RNN captured the area-specific features in the neural data accurately, as the PMd and M1 modules better explained neural data from the respective brain area compared to a cross-area control (Supplementary Fig. 2).

Motor adaptation through altered inputs matches neural recordings
After having verified that our modular RNN recapitulates the key aspects of PMd and M1 population activity during reaching, we simulated the VR adaptation experiment. The model was retrained to produce trajectories rotated by 30°, replicating the perturbation monkeys had to counteract. Having full control of the location of learning-related changes, we first constrained it to happen upstream of PMd (\( H_{\text{input}} \)). As anticipated from previous modelling and experimental work, changes in areas upstream of the motor cortices can lead to successful adaptation: the hand trajectories produced after learning were correctly rotated by 30° to counteract the perturbation (Fig. 4A).

When examining the activity of each of the PMd and M1 modules, the relative change in network activity was similar in magnitude to the changes observed in the corresponding neural population recordings (Fig. 4B and Supplementary Fig. 3). PMd activity changed slightly more than M1 activity (Fig. 4B), indicating a relation between the two modules that was also present in the experimental data (Fig. 2C). With respect to interactions between neurons, covariance within each module was strongly preserved (Fig. 4C), as was the case for the
VR adaptation through altered inputs to
the motor cortices thus is very similar to the neural activity changes
observed in vivo. Learning through plastic changes within PMd and M1 modules
occurs despite preserved the covariance
Our simulation results so far are consistent with experimental10,13–15 and
modelling18 studies proposing that VR adaptation is mediated by
regions upstream of the motor cortices. But can our model rule out the
alternative that adaptation is instead implemented by recurrent con-
nectivity changes within PMd and M1 (Hlocal)?
To address this question, we implemented Hlocal by constrain-
ing learning to happen only within PMd and M1, a process which also
led to successful adaptation (Fig. 4D). Interestingly, the activity
changes produced under Hlocal differed both from those of Hinput
and the experimental data. D Simulated PMd population activity recapitulates key
features of actual PMd population activity. Neural trajectories extend from 600 ms
before the go cue (black dots) to 600 ms after the go cue (coloured dots); go cue is
indicated with coloured crosses. Reaching targets are colour-coded as in B. E Same
as (D) for M1.

![Diagram](https://doi.org/10.1038/s41467-022-32646-w)

**Fig. 3 | A modular recurrent neural network model to study VR adaptation.** A A modular RNN that models key motor cortical areas to study adaptation. B Simulated (top) and actual (bottom) hand trajectories during 30 reaches to each target taken from one session from Monkey C. C Example simulated and actual hand trajectories to one target. Note the similarity in kinematics between the model and the experimental data. D Simulated PMd population activity recapitulates key features of actual PMd population activity. Neural trajectories extend from 600 ms before the go cue (black dots) to 600 ms after the go cue (coloured dots); go cue is indicated with coloured crosses. Reaching targets are colour-coded as in B. E Same as (D) for M1.

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**Learning through plastic changes within PMd and M1 modules
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MI modules without altering their covariance. Interestingly, the connectivity changes under both $H_{\text{local}}$ and $H_{\text{input}}$ (Fig. 5A, B) were small relative to experimentally observed synaptic changes: an average weight change of 1–2% was sufficient regardless of whether they happened upstream of (Fig. 5C and Supplementary Fig. 4) or within the motor cortical modules (Fig. 5E and Supplementary Fig. 5). These changes were smaller than those observed during initial training (4–31%), when the model learned to perform the reaching task from random connection weights (Supplementary Fig. 6). Thus, "functional connectivity" within the PMd and M1 modules, as measured here by their covariance, may be largely preserved after VR adaptation under $H_{\text{local}}$ because network connection weights change very little (Supplementary Fig. 7).

We next studied how such small changes in connection weights could nevertheless drive effective behavioural adaptation. Recent studies seeking to relate RNN activity and connectivity have highlighted the importance of low-dimensional structures in connectivity, showing their explanatory power for understanding how tasks are solved. Inspired by this work, we looked for low-dimensional structure in the connectivity changes emerging in the model during adaptation ("Methods"). Our analysis revealed that the connectivity change patterns of all plastic modules were low-dimensional, independent of where learning happened (Fig. 5B, D, F). We thus hypothesized that the small changes were effective because they were low-dimensional. To test this, we examined how random changes in the connection weights (noise), which are inherently high-dimensional, would affect the behaviour.

**Low-dimensional connectivity changes are highly robust to noise**

For both $H_{\text{local}}$ and $H_{\text{input}}$, the learned connectivity changes in the model were small and low-dimensional. When considering the biological plausibility of our model, this observation raises the question of how such small connectivity changes could compete with ongoing synaptic fluctuation, which is a known challenge for actual brains. To test the hypothesis that the low-dimensionality of the learned connectivity changes is what makes them highly effective, we tested how adding synaptic fluctuations, which are inherently high-dimensional, would affect motor output. Simulating synaptic fluctuations by applying random perturbations to the learned connectivity changes increased the dimensionality of the weight changes (Fig. 6B, G; "Methods"), but did not lead to any observable change in reaching kinematics (Fig. 6C) or network activity (Fig. 6D, E). This was the case even though the applied random perturbations in connectivity were ten times bigger in magnitude than the learned connectivity changes (Fig. 6F), completely masking them (Fig. 6A, B). Therefore, our model not only suggests that VR adaptation can be implemented based on coordinated synaptic weight changes within PMd and M1, but also that this type of learning would be highly effective due to its robustness to synaptic fluctuation.

**Larger visuomotor rotations allow for a clearer distinction between $H_{\text{input}}$ and $H_{\text{local}}**

Although neural activity changes during VR adaptation were better reproduced by a model in which learning happens upstream of the

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**Fig. 4 | Activity changes following learning upstream ($H_{\text{input}}$) and within ($H_{\text{local}}$) the motor cortices.**

**A** Hand trajectories after learning under $H_{\text{input}}$ (coloured traces; baseline trajectories are shown in grey). **B** Changes in trial-averaged activity following adaptation under $H_{\text{input}}$ (green markers) for PMd and M1, and reference mean experimental values (black stars; same data as presented in Fig. 2). Shaded area and horizontal bars, data distribution with mean and extrema ($n = 10$ network initializations). **C** Change in covariance following adaptation under $H_{\text{input}}$, and reference values for change in covariance following the initial de-novo training (dashed lines). Data presented as in **B**. **D** Hand trajectories after learning under $H_{\text{local}}$. **E** Change in trial-averaged activity following adaptation under $H_{\text{local}}$. **F** Change in covariance following adaptation under $H_{\text{local}}$. Data in **D**–**F** are presented as in **A**–**C**. Source data.
motor cortices (Hinput), activity changes following learning through weight changes within the motor cortices (Hlocal) were also in good agreement with the experimental data. To verify that the stable covariance (Fig. 4C, F) is not a general feature of the model but reflects task-specific demands, we modelled tasks for which we would expect larger changes.

We first asked the network to learn larger VRs of 60° and 90° instead of the original 30° rotation (Fig. 7A). The model was able to compensate for these larger perturbations under both Hinput and Hlocal (Fig. 7B, E). As expected, larger perturbations led to changes in network activity and covariance that increased with rotation angle (Fig. 7B, C, F, G). For the 90° rotation, we found a clear difference between Hinput and Hlocal: Hinput produced larger activity changes in PMd compared to M1, opposite that under Hlocal (Fig. 7C, F). Larger rotation angles also increased the learning-related difference in covariance between Hinput and Hlocal. Under Hinput, the increase in covariance was similar for the PMd and M1 modules as the rotation increased (Fig. 7D). In contrast, under Hlocal, the MI covariance changed more with increasing rotation angle than did that of PMd (Fig. 7G).

These model predictions could help differentiate between Hinput and Hlocal in future experiments. In fact, preliminary M1 population recordings obtained during larger VRs (45° and 60°) seemed to match the model predictions for the covariance change under Hinput (Fig. 7D stars), but not Hlocal (Fig. 7G stars).

A visuomotor reassociation task can differentiate between Hlocal and learning through remapping of input signals

Although larger visuomotor rotations help differentiate between upstream learning and learning within PMd and MI, we sought to identify a task that would lead to an even clearer distinction. To this end, a visuomotor reassociation task was used, where different visual stimuli were associated with each of the two movements. Under Hinput, this task led to larger changes in connectivity between the visual and motor cortices, with a clear dissociation between PMd and M1 activity. In contrast, under Hlocal, the changes were more uniform across the network. These results suggested that the task-induced changes were more specific to the input processing stream, supporting the idea that Hinput supports a more flexible and task-specific form of learning compared to Hlocal, which is more sensitive to changes at the level of individual modules.
end, we implemented a reassociation task where the model had to learn a new, random mapping between cues and reaching directions (Fig. 8A; “Methods”). This task allowed us to test a very specific change in the input signal to the motor cortices that could implement adaptation\(^{20,41}\): instead of adjusting the connectivity in an upstream network (\(H_{\text{input}}\)), which allows for highly unconstrained modulation of input signals, the target-related input signals were manually reordered to compensate for the reassociation of cue-reaching direction pairs (Fig. 8B). This “learning through input reassociation” resulted in large changes in network activity (Fig. 8C), comparable in magnitude to those under \(H_{\text{local}}\) (Fig. 8F). Nevertheless, it did not cause any change in covariance (Fig. 8D), which clearly distinguished it from \(H_{\text{local}}\) (Fig. 8G) and the standard \(H_{\text{input}}\) (Supplementary Fig. 8). This was the case because, in contrast to the standard \(H_{\text{input}}\) during VR adaptation, the input signals did not change per se, but were only reassigned to different targets, thereby entirely preserving the network activity patterns.

**Discussion**

Rapid motor learning is associated with neural activity changes in the motor cortices. The origin of these changes remains elusive, due to the current challenge of measuring synaptic strength in vivo. Here, we have used modular RNNS to simulate the motor cortices and to explore whether learning to counteract a visuomotor rotation within tens of
minutes could be mediated by local synaptic changes ($H_{local}$). By comparing the modelled network activity changes under $H_{local}$ to the modelled changes observed during learning upstream of the motor cortices ($H_{input}$), we have shown how the two hypotheses could be distinguished based on neural population recordings during behaviour. Critically, despite the intuition that learning through plastic changes should lead to detectable changes in neural interactions within and across PMd and M1 populations, both $H_{local}$ and $H_{input}$ (Fig. 4) largely preserved the covariance within these two regions, closely matching experimental observations (Fig. 2). This conclusion is in good agreement with studies showing that learning to generate neural activity patterns that preserve the covariance structure only takes a few tens of minutes$^{19}$. Our direct comparison between $H_{input}$ and $H_{local}$ lends further support to this observation. However, it also paints the intriguing picture that small, globally organized changes in synaptic weights could enable rapid learning without changing the neural covariance, a result that was robust across model initializations (Fig. 4), parameter settings (Supplementary Fig. 9 and Supplementary Fig. 10), architectural design choices (Supplementary Fig. 11) and learning algorithms (Supplementary Fig. 12). Even implementing the modular RNN as a spiking neural network, bringing it closer to biology, did not change this result (Supplementary Fig. 13). Our simulations thus robustly show that covariance stability is not as directly linked to stable local connectivity as previously thought, as changes in covariance were comparable between $H_{input}$ and $H_{local}$ for a 30° VR perturbation (Fig. 4). Instead, the change in neural covariance seemed to be more related to the task itself, as it correlated with the size of the perturbation: the larger the initial error (e.g., caused by larger rotations), the larger the change in covariance (Fig. 7). However, the relation between initial error and change in covariance differed depending on where the learning happened ($H_{input}$ or $H_{local}$).

The main difference between the two learning hypotheses we have examined is where in the hierarchical RNN model the connectivity changes occur: within the motor cortices ($H_{local}$), or upstream of them.
Although neural covariance was preserved similarly by $H_{\text{local}}$ and $H_{\text{input}}$, we found a key characteristic that distinguished the two. When local connectivity was allowed to be plastic, the largest activity changes happened within the M1 module, with only small changes in the PMd module (Fig. 4E). In contrast, when learning occurred upstream of the PMd and M1 modules, the activity changes were similar in PMd and M1 (Fig. 4B), even if some learning was also allowed within PMd and M1 (Supplementary Fig. 10 and Supplementary Fig. 14).

The experimental data, with larger activity changes in PMd than M1, better matched the pattern produced by $H_{\text{input}}$. This observation further supports the hypothesis that VR adaptation is mediated by plasticity upstream of the motor cortices.

A more arbitrary visuomotor reassociation task allowed us to test an alternate way in which upstream learning could occur, with constraints against input signals changing but simply being reassigned to different targets (Fig. 8). Comparing this learning to that mediated by local connectivity changes revealed a clear distinction: learning under $H_{\text{local}}$ modified the covariance in both PMd and M1, whereas learning through input reassociation preserved it. Thus, future experiments seeking to disentangle to which extent learning happens within the motor cortices and/or upstream could study this task.

Studies of learning in RNNs have focused on how networks implement de-novo training. However, our brain does not learn to perform any task from scratch; it has been “trained” over many generations throughout evolution. Here we studied how neural networks adapt a learned behaviour, as opposed to de-novo learning. Our work raises the intriguing possibility that rapid learning following a few tens of minutes of practice could be easily achieved through small but specific changes in circuit connectivity. Thus, initial training seems to provide a highly flexible backbone to adapt behaviour as needed.

The fact that the connectivity changes during adaptation under both $H_{\text{local}}$ and $H_{\text{input}}$ were small and low-dimensional suggests that either one could mediate rapid learning. Our work raises the intriguing possibility that rapid learning following a few tens of minutes of practice could be easily achieved through small but specific changes in circuit connectivity. Thus, initial training seems to provide a highly flexible backbone to adapt behaviour as needed.

This diagram illustrates the difference between $H_{\text{local}}$ and $H_{\text{input}}$. The experimental data, with larger activity changes in PMd than M1, better matched the pattern produced by $H_{\text{input}}$. This observation further supports the hypothesis that VR adaptation is mediated by plasticity upstream of the motor cortices. A more arbitrary visuomotor reassociation task allowed us to test an alternate way in which upstream learning could occur, with constraints against input signals changing but simply being reassigned to different targets (Fig. 8). Comparing this learning to that mediated by local connectivity changes revealed a clear distinction: learning under $H_{\text{local}}$ modified the covariance in both PMd and M1, whereas learning through input reassociation preserved it. Thus, future experiments seeking to disentangle to which extent learning happens within the motor cortices and/or upstream could study this task.

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The fact that the connectivity changes during adaptation under both $H_{\text{local}}$ and $H_{\text{input}}$ were small and low-dimensional suggests that either one could mediate rapid learning. First, as every synaptic change is costly, we would expect a constraint on the total amount of connectivity change in the brain. The VR task being solved with only minor weight changes reflects this; in fact, they could be well achieved through long-term potentiation or depression of existing synapses, as experiments have shown that synaptic strength can double within minutes. Second, the low dimensionality of these weight changes is also important with respect to solving “credit assignment”, the problem of determining how each synapse should change in order to restore the desired behaviour. Although it is still
such implementation would contrast dramatically with the daunting "connectivity changes needed to adapt to the VR perturbation are low-dimensional connectivity changes makes both $H_{\text{local}}$ and $H_{\text{input}}$ explicit. Intriguingly, $H_{\text{local}}$ not only largely preserved the covariance signatures that are unexpectedly similar to those of upstream learning but also resulted in connectivity changes that seem biologically reasonable: they are small, make the network robust to other processes such as plasticity59,60, they seem ideal candidates to regulate synaptic plasticity59,60, they seem ideal candidates to regulate synaptic plasticity. Since neuromodulatory signals could help tackle this question in future studies.

Our model consistently underestimated the changes in trial-averaged activity observed during VR adaptation, despite closely matching the small covariance changes (Fig. 2,4). This is to be expected, as the model only captures changes due to the motor adaptation process itself, whereas the actual neural activity contains signals related to other processes such as "impulsivity" or "engagement". In fact, the experimentally observed neural activity changes between the early and late trials of control reaching sessions with no perturbation were almost as large as the changes during adaptation in our model (Fig. 2C, black dots). How these changes that are not related to learning are combined with the learning-related changes studied here remains unclear. Our modelling predictions for the learning-related changes could help tackle this question in future studies.

A second potential reason why our model consistently underestimated the activity changes during adaptation could be the fact that we did not include visual or proprioceptive feedback signals in our modelling approach. As those signals also change during adaptation, they might cause additional changes in trial-averaged neural activity, despite not being directly necessary to solve the motor adaptation task. This could explain why our model could solve the task with smaller changes in neural activity. On the other hand, feedback signals could also contribute to the adaptation process. From this view, we may presently overestimate the already small connectivity changes underlying VR adaptation (Fig. 5), as part of the learning process could have been instead driven by dynamic feedback signals. Thus, when taking feedback into account, rapid learning of a motor perturbation could potentially be realized with even smaller changes in underlying connectivity, or maybe even without any connectivity changes at all. To this point, the concrete role of feedback for rapid motor learning remains unclear and it could be interesting to use our model to further investigate this question.

Our simulations were not designed to study trial-by-trial learning: we were interested in the neural activity changes between the baseline and late adaptation phases when the subjects had largely learned to counteract the perturbation and reached stable behaviour (Fig. 2B). Given that motor adaptation seems to be mediated by two processes with different timescales, our model mainly captures the slower of the two. The neural activity changes underlying the early phase adaptation may be driven by different processes, which our model currently does not test.

In conclusion, our comparison between the activity changes following VR adaptation through plastic changes within or upstream of the motor cortices shows that local plasticity ($H_{\text{local}}$) leads to neural signatures that are unexpectedly similar to those of upstream learning ($H_{\text{input}}$). Intriguingly, $H_{\text{local}}$ not only largely preserved the covariance within PMd and MI but also resulted in connectivity changes that seem biologically reasonable: they are small, make the network robust against synaptic fluctuations, and can be controlled by relatively few teaching signals. Our simulations thus encourage caution when drawing conclusions from the analysis of neural population recordings during learning, and further suggest potential behavioural tasks that could make it easier to identify where learning is happening within the motor system.

Methods

Tasks

We studied motor adaptation using a visuomotor rotation (VR) paradigm, previously described in Perich et al. (2018). Monkeys (macaca mulatta) performed an instructed delay centre-out-reaching task in which they had to reach to one of eight targets uniformly distributed along a circle. All targets were 2 cm squares. The delay period was variable and ranged between 500 and 1500 ms. For additional details on the task, see10. During the adaptation phase, visual feedback was rotated clockwise or counterclockwise by 30°, 45°, or 60°. All surgical and experimental procedures were approved by the Institutional Animal Care and Use Committee (IACUC) of Northwestern University. Using our modular RNN model, we simulated both this task and a visuomotor reassociation task in which there was no consistent rotation of the visual feedback; instead, each target required reaching to a different direction, uniquely selected from the initial set of eight different targets.

Experimental recordings

We analyzed eleven sessions from two monkeys (five for Monkey C, six for Monkey M) that were exposed to a clockwise or counterclockwise 30° rotation (data previously presented in18). In addition to these data, we also analyzed three control sessions (one for Monkey C, two for Monkey M) in which no perturbation was applied, as well as additional sessions with larger VR angles from Monkey C where only MI data was collected (30°, nine sessions; 45°, two sessions; 60°, two sessions) (Fig. 7).

The spiking activity of putative single neurons was binned into 10 ms bins and then smoothed using a Gaussian filter (s.d., 50 ms). Only successful trials, where monkeys received a reward at the end, were included in the analysis. We defined the early and late adaptation epochs as the first and last 150 trials of the perturbation phase, when the visuomotor rotation was applied, respectively.

RNN model

Architecture. The neural network contained three recurrent modules, each consisting of 400 neurons, which we refer to as upstream, PMd and MI, respectively (Fig. 3A). The PMd and the upstream modules are composed of 400 neurons, which we refer to as upstream, PMd and MI, respectively (Fig. 3A). The PMd and the upstream modules connect to the MI module and the MI module connects to the PMd module. The output is calculated as a linear readout of the MI module activity. Recurrent, as well as feedforward connections were all-to-all. The model dynamics are given by

\[ x_{i+1} = x_i + \frac{dt}{\tau} \left( -x_i + W x_i \tanh(x_i) + W x_i s_i \right) \]

(1)

\[ x_{i+1} = x_i + \frac{dt}{\tau} \left( -x_i + W x_i \tanh(x_i) + W x_i s_i \right) \]

(2)

\[ x_{i+1} = x_i + \frac{dt}{\tau} \left( -x_i + W x_i \tanh(x_i) + W x_i s_i \right) \]

(3)

\[ x_{i+1} = x_i + \frac{dt}{\tau} \left( -x_i + W x_i \tanh(x_i) + W x_i s_i \right) \]

(4)
where $x^{init}$ describes the network activity in the upstream module, and $x^{PMd}$ and $x^{M1}$ the network activity in the PMd and M1 module respectively. $W^{UP}$, $W^{PMd}$ and $W^{M1}$ define the recurrent connectivity matrix within the upstream module, the PMd module and the M1 module, respectively. $W^{UP,PMd}$ defines the connectivity matrix from the upstream module to the PMd module, and $W^{PMd,M1}$ defines the connectivity matrix from the PMd module to the M1 module. The input connectivity matrices for the upstream and the PMd module are given by $W^{in,UP}$ and $W^{in,PMd}$ respectively; $s$ represents the three-dimensional input signal described above. The two-dimensional output matrix is decoded from the M1 module activity via the output connectivity matrix $W^{out}$ and the bias term $b^{out}$. The time constant is $\tau = 0.05$ s and the integration time step is $dt = 0.01$ s.

**Training.** Each network was initially trained to produce planar reaching trajectories, mirroring the experimental hand trajectories. The training and testing data set were constructed by pooling the hand trajectories $x^{init}$ for successful trials during the baseline epochs from all experimental sessions, which resulted in 2238 trials of length 4 s (90%/10% randomly split into training/testing). The held out testing data was constructed by pooling the hand trajectories from mental sessions, which resulted in 2238 trials of length 4 s (90%/10%). PyTorch\textsuperscript{75} and training was performed using the Adam optimizer\textsuperscript{76} with a learning rate of 0.0001 ($\beta_1 = 0.9$, $\beta_2 = 0.999$). The initial training consisted of 500 training trials. The loss function was defined as

\[
L = \frac{1}{B(\tau - 50)2} \sum_{b} \sum_{t} \sum_{d=1,x,y} \left( x^{target}_{t,b} - x^{init}_{t,b} \right)^2 + E^{weights} + E^{rates}
\]

where the regularization term on the weights is given by $||.||$ (L2 norm)

\[
E^{weights} = \alpha \left( ||W^{in,UP}|| + ||W^{UP,PMd}|| + ||W^{out}|| + ||W^{PMd}|| + ||W^{M1}|| + ||W^{PMd,M1}|| + ||W^{UP}|| + ||W^{UP,PMd}|| \right)
\]

the regularization term on the rates is given by

\[
E^{rates} = \beta \frac{1}{BTN} \sum_{b} \sum_{t} \sum_{n} \sum_{i} \left( \tan(h(x^{PMd}_{t,b}))^2 + \tan(h(x^{M1}_{t,b}))^2 + \tan(h(x^{UP}_{t,b}))^2 \right)
\]

with batch size $B = 80$, time steps $\tau = 400$ and neurons $N = 400$. The regularization parameters were set to $\alpha = 0.001$, $\beta = 0.8$. We clipped the gradient norm at 0.2 before we applied the optimization step. For the VR adaptation, we trained the initial network for another 100 trials with the target trajectory rotated 30° (or 60° or 90° for the case of the larger VRs). For the VR reassociation task we shuffled the stimuli $s$ across reaching directions but kept the targets $x^{init}$ fixed, as indicated in Fig. 8A (colours correspond to the given stimulus and sketched reaching trajectories correspond to the assigned target). The network used 100 trials to adapt to this perturbation.

**Data analysis**

We quantified the changes in actual and simulated neural activity following adaptation using two measures: changes in trial-averaged activity (or peristimulus time histogram, PSTH), and changes in covariance. We calculated both metrics within a window that started 600 ms before the go signal and ended 600 ms after it. The change in activity was calculated by

\[
\frac{|PSTH^{Lateadaptation} - PSTH^{Baseline}|}{\sigma^{Baseline}}
\]

where PSTH$^{Baseline}$ is the trial-averaged activity in the baseline epoch (experimental data: all baseline trials; simulated data: on a trained model, 100 trials with similar go signal timing), PSTH$^{Lateadaptation}$ is the trial-averaged activity in the late adaptation epoch (experimental data: last 150 trials of the adaptation epoch; simulation data: on a model trained to counteract the perturbation, 100 trials with similar go signal timing), and $\sigma^{Baseline}$ is the neuron-specific standard deviation across time and targets during the baseline epoch. To summarize the change in trial averaged activity across all neurons, time points, and targets, we calculated their median; this provided one single value for each experimental session or simulation run. The change in covariance was calculated using the same trial-averaged data from the baseline and the late adaptation epoch. We calculated the covariance in each of these two epochs and then quantified the similarity by calculating the Pearson correlation coefficient between the corresponding entries of the two matrices. The change in covariance is then defined by $1$ minus the correlation coefficient. For the experimental sessions, we computed a lower bound for each measure using the control sessions, in which monkeys were not exposed to a perturbation. To account for the fact that there could be activity changes unrelated to motor adaptation\textsuperscript{65,66}, we compared the activity during 150 consecutive trials from the first half of the control session with 150 consecutive trials from the second half of the control session.

To compute the magnitude of the weight changes after networks learned to counteract the perturbation, we computed the average absolute weight change as

\[
dW = \frac{|W^{Lateadaptation} - W^{Baseline}|}{W^{Baseline}}
\]

where $|.|$ indicates the element wise absolute value, $W^{Baseline}$ is defined as the model parameter (either $W^{in,PMd}$, $W^{in,UP}$, $W^{UP}$, $W^{UP,PMd}$, $W^{PMd}$, $W^{PMd,M1}$ or $W^{UP}$) after the initial training phase but before training on the VR perturbation, and $W^{Lateadaptation}$ is defined as the same model parameter after training on the VR perturbation. To obtain one summary value for each simulation run, we calculated the median of all weight entries for a given parameter. To measure the dimensionality of weight change we calculated the singular values $k_i$ of $W^{Lateadaptation}$ - $W^{Baseline}$ and defined the dimensionality, using the participation ratio$^{77}$:

\[
\frac{\left( \sum_{i=1}^{N} k_i \right)^2}{\sum_{i=1}^{N} k_i^2}
\]

**Statistics**

To statistically compare the change in activity found in the control sessions with the change found in the VR sessions, we performed a linear mixed model analysis using R (lmer package). The brain area (PMd or M1) and whether the experimental session included a perturbation phase or not were included as fixed effects, whereas monkey and session identity were included as random effects. A significance threshold of $P = 0.05$ was used.

**Simulation of synaptic fluctuation** (Fig. 6)

To simulate synaptic fluctuation we added random values to the learned connectivity changes during adaptation. Those random values were drawn from a normal distribution with zero mean and s.d. ten times larger than the s.d. of the learned weight changes distribution. With that, we created synaptic noise which was completely unstructured across connection sites. We did not add or delete any synapses in the model.

**Reporting summary**

Further information on research design is available in the Nature Research Reporting Summary linked to this article.
Data availability
The data that support the findings in this study are available from the corresponding authors upon reasonable request. Source data are provided with this paper.

Code availability
All code to reproduce the main simulation results can be found on GitHub (https://github.com/babaf/motor-adaptation-local-vs-input.git).

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Acknowledgements
This work has been funded by BBSRC (BB/N013956/1 and BB/N019008/1), Wellcome Trust (200790/Z/16/2), the Simons Foundation (564408) (all to C.C.), the EPSRC (EP/R035806/1 to C.C. and EP/T020970/1 to JAG), and the ERC (ERC-2020-STG-949660 to J.A.G.). The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

Author contributions
Conceptualization: B.F., J.A.G., and C.C. Methodology: B.F., J.A.G., and C.C. Experimental data collection: M.G.P. Modelling: B.F. Analysis and interpretation of data: B.F., J.A.G., and C.C. Supervision: J.A.G. and C.C. Writing/review of the paper: all.

Competing interests
The authors declare no competing interests.
