Balanced activation in a simple embodied neural simulation

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Acknowledgements and Supplementary Materials

The code/implementation (in Unity) and compiled versions of the model are available to at https://github.com/c3nl-neuraldynamics/Avatar. Special thanks go to Eva Papaeliopoulos for getting us started in making computer games.
Abstract

In recent years, there have been many computational models exploring how spontaneous neural dynamics arise out of the brain’s structural connectome. Most of these models have focused on the resting state (with no external input) or investigate the effects of simple sensory inputs. However, neural systems exist to process incoming information from the environment and to guide motor output. Here, we explore a very simple neural network model (similar to that used previously to model dynamics at rest) and allow it to control a virtual agent in a very simple environment. This setup generates interesting brain-environment interactions that suggest the need for homeostatic mechanisms to maintain rich spontaneous dynamics. We investigate roles for both local homeostatic plasticity as well as macroscopic “task negative” activity (that compensates for “task positive”, sensory input) in regulating activity in response to changing environment. Our results suggest complementary functional roles for both balanced local homeostatic plasticity and balanced task-positive/task negative activity in maintaining simulated neural dynamics in the face of interactions with the environment. This work highlights the challenges that feedback between environment and brain presents to neural models as well as the different roles that balancing mechanisms may play; it also suggests a functional role for macroscopic task negative systems in the brain that may be of relevance to understanding large-scale brain systems such as the default mode network.
Introduction

At the macroscopic scale, whilst performing a task we observe both regional increases in neural activity (task positive regions, TP), as well as regional reductions in activity (task negative regions, TN) (Raichle et al, 2001; Shmuel et al, 2006). Unlike TP patterns of activity (e.g., activity in areas of occipital cortex evoked by a visual stimuli), there is surprisingly little theoretical consensus as to the functional role of TN. Often, TN have been viewed as either artifacts in functional imaging, ignored, or more recently, interpreted as relating to task-independent thought (Buckner et al, 2008).

One alternative approach to understanding TN patterns of activity, is by analogy with smaller-scale systems, such as the excitatory/inhibitory homeostatic interactions that regulate cortical dynamics by maintaining locally balanced activation across the brain. At the neuronal level, experimental and theoretical work suggests that the balance of local inhibition and excitation (E/I) has important computational properties (van Vreeswijk & Sompolinsky, 1996; Doiron et al. 2016), and can be maintained by homeostatic inhibitory plasticity (e.g., Vogels e al, 2011, Woodin et al, 2003; Hass et al, D'Amour et al.). At the macroscopic level, there is evidence from fMRI that there is some level of balance with networks of brain regions showing increased activity matched by other networks showing reduced activity, dependent on the specific task (e.g., Geranmayeh et al, 2014; Xu et al, 2015). Computational simulations also hint at a role for TN networks in supporting balanced dynamics (e.g., Deco et al, 2009). In our previous work, we have provided evidence that the role of task negative networks may act as a spatial or temporal counterbalance to task activation in other brain regions (Leech et al, 2014; Braga & Leech, 2015; Scott et al, 2015). This may constitute (at the macroscopic scale) an analogue of inhibitory computational mechanisms (Turkheimer et al, 2015) seen at smaller scales.

In the work presented here, we expand on these ideas about the regulatory role of homeostatic mechanisms, by combining a simplified local homeostatic mechanism with a macroscopic (task positive and negative networks) balancing mechanism, in a simple embodied system, which displays some interesting properties. We defined a simple model based on the Greenberg-
Hastings model (Greenberg & Hastings, 1978), incorporating information about human structural connectivity (Hagmann et al, 2008) that has been previously shown to approximate empirical functional connectivity patterns (Haimovici et al, 2013). In the model, a node was set to the ON state, with either a small random probability or if incoming activity was greater than a local threshold value (analogous to the amount of local inhibition).

In order to explore the interaction between brain and environment, we embodied the computational model in a simple environment. We began by defining an ‘agent’ that can move within a 2-dimensional plane, bounded by surrounding walls (see Figure 1). Within this framework, we defined a group of task-positive nodes (TP), which activated in response to simulated “sensory” input. Two pairs of bilateral nodes reacted to “visual” input from the environment to the simulated brain, and a pair reacted to “somatosensory” input: if input was detected these nodes were set to ON. A further pair of nodes simulated “motor” output from the simulated brain to the agent in the environment; their activity determined the movements of the agent.

This simple setup allows us to observe interesting interactions between the simulated neural network and the environment. Specifically, the following environment/agent closed-loop interaction occurs: (i) different parts of the environment evoke different amounts of visual and sensory stimulation, which subsequently (ii) alter regional/microscopic neural activity, (iii) leading to altered neural dynamics macroscopically, across the entire model; (iv) these altered dynamics in turn change the motor output from the model, v) changing the agent’s trajectory in the environment which in turn alters subsequent sensory input. This presents a challenge for network models, especially models focusing on spontaneous, rich dynamics, that often require careful parameterization to remain in a specific dynamic regime, and so typically are investigated in static situations (i.e., where the input to the model is stationary, such as Guassian noise). In such models, changes to the model input typically lead to destabilization of the dynamics (i.e., a shift to either random, saturated, or absent patterns of activity).

Balanced activity has been shown to facilitate a rich, spontaneous dynamical regime that is robust to different parameter values, (Deco et al, 2014; Hellyer
et al, 2016); this has not been explored in non-stationary scenarios, such as occur in this type of embodied models where brain-model interactions can occur. Therefore, we incorporated two mechanisms in the model to balance activity: first, a simplified version of the local (i.e., within-node) homeostatic plasticity rule presented in Vogels et al, 2011 and employed in a similar macroscopic neural model in Hellyer et al, 2016. This tuned the local threshold at each node, balancing against incoming excitatory activity from other nodes, and so driving time-averaged local activity to approximate a pre-specified, small target activity rate. In addition to the local mechanism, we also employed a non-local, macroscopic balancing mechanism, such that the activity of the task positive nodes (i.e., the six “sensory” nodes) was balanced over time by bilateral task negative nodes (TN). The choice of the TN nodes was loosely based on task-evoked relative deactivations from fMRI/PET, which we have previously suggested, may constitute a macroscopic balancing system (Leech et al, 2014).

Here, we explore the complementary roles that these two balancing systems may play in maintaining flexible dynamics in an embodied situation with sensory input from and motor output to the environment. In particular, we assess the agent's neural dynamics and trajectory through the environment, and demonstrate that these balancing mechanisms allow the agent to escape constrained environment-brain feedback loops, and more completely traverse the environment.
Figure 1: Illustration of the brain model (left) and two perspectives of the model-controlled agent (grey figure, with its colored trail over time) placed in a very simple environment (right). “Visual” or “somatosensory” sensory input to the agent, depends on proximity to the wall around the edge of the environment. The agent moves based activity in “motor” nodes of the model. The model (either compiled or as a unity project) can be downloaded from the GitHub repository: https://github.com/c3nl-neuraldynamics/Avatar).

Results

We start by considering simulated neural and movement dynamics, without the explicit task negative mechanism. Consistent with previous results (Hellyer et al, 2016), we observe that over time the homeostatic model adapts the threshold weights such that time-averaged excitatory activation approximates the pre-specified target activity, $\rho = 0.1$ (Figure 2A). Moreover, the model demonstrates high levels of persistent variability around this average activity value (Figure 2A and 2C), even though there is relatively little intrinsic noise in the system. The model also displays reliable but relatively weak positive correlations between nodes (Figure 2D), consistent with the presence of rich asynchronous dynamics. This is consistent with previous findings that a homeostatic mechanism (if $\rho$ is set appropriately) results in rich
simulated dynamics, displaying some hallmarks of self-organized criticality (Hellyer et al, 2016).

**Figure 2:** How the simulation with local plasticity changes over time (results are presented from averaged 1000 epochs of simulation). **A:** mean activation approaches the pre-specified target value $\rho=0.1$. **B:** this is accomplished by reductions and then gradual increases in local thresholds (similar to inhibitory plasticity reported in Hellyer et al, 2016). **C:** We observe an accompanying increased variability of activity (standard deviation of activity over time, averaged across nodes). Finally, **D:** we also observe an increase in connectivity across the network (measured as mean correlation between nodes). (Results are presented for a single 100,000 long training run, although qualitatively similar results were found for different initial conditions and random seeds).

We also observed the simulated trajectory: over time the model moves into a regime with generally higher levels of movement (i.e., left/right rotation and/or forward motion) (Figure 3A), although there is considerable variability (i.e., the mean level of movement and activity varies considerably over time). Several examples (each 1000 epochs long) are presented in (Figure 3B).
Figure 3: A Change in “motion” output (either nodes linked to “forward” output to the agent or nodes linked to “turn” over time calculated from the activity of the “motor” nodes). Note that this figure presents the average output generated by the model rather than the motion of the actual agent, which can be impeded by obstacles (i.e., walls) in the environment: i.e., the simulated brain could be sending a move forward command, but this cannot be achieved because of the wall. B Example trajectories of the agent over nine
randomly chosen time windows of 1000 epochs. Light colors are earlier in the time window, warm colors are later in the time window.

However, we observe that the dynamic regimes of the simulated neural activity and motion are not stationary, displaying instead periods of high and low dynamics. The model can be approximately characterized as alternating between periods of high and low activity, with correspondingly high and low amounts of movement: it never settles down into a stable regime, with a single distribution of movements/activity (see Figure 4 for an illustrative shorter 1000 epoch time period). This alternating pattern reflects the result of how the agent interacts with the environment. The level of sensory input: i) alters the level of simulated activity, which ii) alters the level of motor output, iii) that manifests in agent movement which in turn, iv) may alter subsequent sensory input.

**Figure 4:** An illustration of the brain/environment interaction. **A:** At short time periods, there are short, but sustained periods of elevated activity substantially above the target rate. **B:** The homeostatic rule is continuously adapting to the level of activity, tightly matched sustained changes in the local threshold. **C:** Model alternating between low and high average threshold
levels (or inhibition). **D:** The sustained changes are being driven by the sensory input the model receives, which closely match overall activity; **E:** This demonstrates how sensory input and activity are related to how close the agent is to a wall (where sensory input is highest, given triggering of “visual” and “somatosensory” sensors. **F:** Strong relationship between proximity to the wall and level of activity. **G/H** the threshold change (G) and the sensory input (H) (left axes) relate to average movement (right axis), over time.

A simple illustration of this feedback is presented in Figure 4 (and supplementary video); there are high levels of simulated visual and somatosensory input at the edges of the environment (that is touching and/or looking at the wall) and low or no sensory input in the center. Therefore, high sensory input triggers higher average activity which leads to elevated motor output, which on average moves the agent forward into the wall and in turn keeps the input activity high. This feedback cycle means the agent remains trying to run into the wall, trapped next to the wall and with sustained elevated activity patterns. In contrast, away from the wall, where there is little or no sensory feedback, the levels of activity remain low, and motor output and movement are either low or not present, meaning that the agent remains in a very low simulated neural activity and movement regime. The presence of homeostatic plasticity mechanism that tunes the threshold at each node to balance excitatory input from connected nodes ensures that the agent does not stay trapped in either state for long. As the threshold for activity (varying depending on the local homeostatic mechanism) at individual nodes increases (in the high activity state) or decreases (in the low activity state), the average activity level adapts to the target level. This results in the agent escaping the ‘trap’, with resulting activity levels closer to the target level $\rho$ and, consequently, more stable simulated neural and movement dynamics.

The model without local homeostasis is typically unable to cope with the sensory/motor feedback system. Local thresholds can be chosen to allow interesting dynamics (i.e., variable movements/neural activity); however, these must be chosen to either allow rich dynamics in the presence of sensory input (i.e., with higher local inhibition) or dynamics in the absence of sensory input (i.e., with lower local inhibition). Therefore, over time the agent
will tend to either: a) remain approximately stationary in a low-sensory area with local thresholds too great to allow much exploration (i.e., near stationary, see Figure 5C); or b) become trapped in a high-sensory area running into the wall (e.g., a corner) (see Figure 5A and 5B).

Figure 5: Example trajectories (from initialization) of the agent without local homeostatic plasticity, and threshold weights set uniformly at 5, 7 or 9 and run for 1000 epochs. With lower weights, the agent receives high levels of excitatory activity across the brain, and walks into the wall and is trapped. With higher weights, activity within the network is very low and driven mainly by random excitation rather than activity propagating through connections; as a result the agent moves very little over the course of the 1000 epochs. Cool colors are earlier in the time window, warm colors are later in the time window.

While the model with local homeostasis is able to deal with this sensory-motor interaction, the addition of an explicit task-negative system, alongside the homeostatic learning rule, facilitates simulated neural dynamics and trajectories through the environment. This occurs because the task negative system balances changes in external input to the model so that the number of activated units (sensory nodes or task negative nodes) remains constant irrespective of interactions with the environment.

To demonstrate the complementary role of the two homeostatic systems, we compared the model with just the local homeostatic mechanism with the model with the local homeostatic mechanism and the task-negative system on a range of measures assessing the model’s dynamics (simulated neural activity, simulated movement, and threshold changes). We see that for both
types of model, there are similar levels of mean simulated activity across the network (excluding sensory or task-negative nodes), approximately equal to the target rate (Figure 6A). However, we see that activity and threshold weight changes are less variable for the TN (Figure 6C). This is the case when considering both the standard deviation and the coefficient of variation (s.d./mean). Also, there is a strong negative relationship between distance from the wall and the amount of activity in the local homeostasis model (Spearman Rho=−0.45), whereas this relationship becomes much smaller in the task-negative model (Spearman Rho=−0.25), suggesting the feedback loop between brain/environment is less influential.

Figure 6: Comparing simulations with and without TN (blue is without, red is with TN). We see that while the level of activation is similar in the two types of model (A, mean activity for non-TN or task positive nodes), there are higher average thresholds (B, again for non-TN or TP nodes), possibly because there are never completely quiescent periods; importantly there is substantially reduced variability in activity over time for the TN model (C); and lower average correlations between nodes (D). All results presented here and
in the text comparing simulations with and without TN were highly statistically significant ($t > 3$), with data sampled from each of the 1000 epoch blocks. Also, (E) image entropy from plotted trajectories and (F) fractal dimension from plotted trajectories calculated over 1000 epoch blocks.

When considering motor output (i.e., “motor signals” sent to control movement of the agent, e.g., turn left, go forward), we observe that there is significantly higher entropy for the plotted trajectories of the TN model. Similarly, when observing the movement of the agent we see that there is more movement in general (Figure 7A,B), and that the path of the agent has a higher fractal dimension and higher entropy (taking the 2d entropy of the image of the path over 1000 epochs) for the TN model (and has visited significantly more of all possible locations, see Figure 7C), suggesting it enjoys a more complex pattern covering more of the environment and is less affected by the feedback system.

![Graphs and images](image.png)

**Figure 7:** A and B (top) present actual forward motion (i.e., average distance moved per epoch), and absolute average turning over time for both the TN (red) and non TN (blue) simulations. We see that there is generally more movement for the TN model. C, (bottom), location of the agent averaged across the last 50,000 epochs contrasting the TN model with the non-TN model (smoothed with a Gaussian kernel, sigma=0.5). We see that in general the TN model visits more of the environment than the non-TN (i.e., pixels with warm colors, indicate more frequent visit TN; cold colors are the reverse).

We initially chose the nodes to have fixed locations of sensory input systems and motor output systems, and the approximate location of core task-negative nodes. However, given the coarse resolution of the parcellation that it is not
based on functional data, the assigning of sensory or motor labels to nodes is inherently limited and very approximate, and we do not wish to draw conclusions about any brain regions or networks on the basis of any of the specific locations we used. However, it is interesting to look at the relative properties of task positive and task negative nodes, to make inferences about how these might relate to one another. Therefore, we repeated the simulations, this time randomly varying the location of the task negative nodes. We ran 65 simulations that were the identical to the simulations detailed previously except: 1) they were shorter (5000 epochs, for practical reasons); 2) the TN were randomly chosen from any of the nodes that had not been defined as “sensory” or “motor”. We observed that the stability of the model is dependent on the choice of the individual TN nodes. Specifically, we see that the solution is more stable when TP are linked by more walks to TN nodes (calculated using the Brain Connectivity Toolbox, Rubinov & Sporns, 2011). This was assessed by counting the number of walks (of <6 steps) between TP and TN nodes: there were three distance measures (assuming left and right symmetric): somatosensory TP-TN, and two visual TP-TN measures. These three measures were entered into general linear models to predict measures of neural dynamics; the standard deviation and coefficients of variation for threshold weights and activation as well as the correlation between distance to wall and activation were all significant at p<0.01 (F(3,63)>4.5).
Discussion

This model is unequivocally not intended to represent either a detailed model of real embodied cognition or actual sensorimotor systems; instead, in both regards, it is highly simplified. This simplification allows us to consider the interactions between macroscopic brain networks, neural dynamics and the environment. We acknowledge that there have been many arbitrary design choices, and do not intend this to be a definite presentation of how to model brain/environment/behavior.

We acknowledge that this is a simplified sensory/neural feedback system, and we make no claims that this particular example ever occurs in the real world as presented. We wish to emphasize that we have not embodied this simple neural model in order to model sensorimotor systems and as such this work should not be judged on this, and we acknowledge that there are many, far more sophisticated and better implemented simulated or real robotic setups. Instead, we are using simple embodiment to try to understand the functional roles of homeostatic systems in the brain. Equally, from a neural rather than computational point of view, there are many possible sensorimotor/neural setups we could have employed, with equal or greater merit. Nevertheless, we used the specific sensory-motor/neural system presented in the paper because: it is a) simple to present; and, b) simple to implement. In reality, such feedback systems are likely to be far more complex, and possibly only probabilistic or non-stationary. Moreover, they will also inevitably depend on the complexity both of the neural system, but also the complexity of the motor and sensory systems. Our intentions, considerations and choice of features outlined here simply render the work a useful toy example.

With all of the above as a strong caveat, our findings highlight the challenges that feedback between environment and brain presents to neural models. Further, our results suggest that modeling spontaneous dynamics at rest (e.g., Honey et al, 2009; Messe et al, 2014; Vasa et al, 2015) or with a simple task such as encoding a sensory stimulus (Hellyer et al, 2014; Ponce-Alvarez, 2015) is different to modeling sensori-motor interactions with an environment, and the existence of a closed-loop feedback made the roles of homeostatic mechanisms more important and obvious. In our case, we observed that
without the local homeostatic plasticity, the agent in the environment would become trapped in either a stationary state (with high thresholds, akin to local inhibition) or would be in a permanent state of motion (with too low local inhibition). Instead, we observe that plasticity is a constant feature of the system. Initially, there are large changes in local thresholds across time points, as the model approximately balances average incoming excitation at each node. As time progresses, however, the weight changes become smaller, but never drop to zero.

We also observed that local homeostatic plasticity could be complemented by adding a macroscopic, task-negative system to compensate for sensory-induced activity across the whole simulated brain. The simple system we implemented, modeled on patterns of task negative deactivation from the fMRI/PET literature (e.g., Leech et al., 2014) was designed so that there would be reduced destabilizing effects from the changing amount of sensory input to the model. Without the task negative system, the overall level of activity within the model is more dependent on the level of sensory input (i.e., “touching” or “seeing” the wall). This makes the task of the local homeostatic plasticity mechanism harder, since exogenous input to the system is varying considerably. Instead, the task negative system simply balances the level of exogenous activity to a constant amount, such that task negative input decreases as sensory input increases and visa versa. This means that the environment/brain feedback loop does not change the overall level of incoming activity to the model, therefore facilitating the local homeostatic plasticity to find a more stable solution, i.e., one that requires the smallest weight changes to approximate the target activation rate. Further, what we observe are different balancing systems operating at different spatial and temporal scales and with different specific mechanisms. This is consistent with the proposed account of normalization found in many neural systems (Carandini and Heeger, 2012), which provides a canonical computation across scales and specific mechanisms by improving neural coding efficiency and sensitivity.

From a traditional cognitive neuroscience perspective, this way of thinking about TN may sit somewhat uncomfortably. What we have been describing as
TN may provide a partial functional explanation for the default mode network. The default mode network is a well-characterized, frequently observed and relatively poorly understood macroscopic brain network located in areas of the brain not associated with sensorimotor activity; the default mode network has been observed across ontogeny (Doria et al, 2010), phylogeny (Mantini et al, 2011), and found across different cognitive and sensorimotor tasks (Gusnard & Raichle, 2001) and implicated through abnormal function in many disorders (Zhang & Raichle, 2010). According to our findings, the default mode network can be thought of as acting as a counterweight, or as an endogenous generator of neural activity that allows the neural system to remain relatively stable in an inherently unstable world. One analogy could be with the vascular system of warm-blooded animals, which attempts to maintain a constant body temperature, irrespective of the temperature outside, in order to maintain a stable environment for chemical reactions to take place, ultimately allowing more flexible behavior. (We note that the proposed balancing functional role for the TN does not preclude more traditional cognitive roles ascribed to them, such as internal mentation. We hypothesise that TN could have initially evolved to perform some basic neural function, such as balancing incoming sensory activity, and eventually been repurposed by exaptive evolutionary processes to perform more specific cognitive functions that occur when external input is not present).

Following this explanation of the task negative systems in general and the default mode network more specifically, may not strictly be “necessary” for accomplishing any task. Lesioning TN regions is unlikely to disturb any associated function entirely, and as such TN systems may appear to be epiphenomenal. However, just as a sailing boat does not require a keel to move (the keel counterbalances the forces on the sail, facilitating stability and allowing a wider range of movement and greater speed), the brain may have a greater range of neural state and potentially be more controllable, when it is properly counterbalanced. It might only be over longer time periods when initially adapting to a novel environment or across development that damage to TN systems becomes particularly disabling, failing to facilitate other adaptive systems as efficiently.
We find that the model finds a more stable solution if TN nodes are strongly linked to task positive nodes. This is consistent with the presence of multiple TN systems in the brain (Leech et al, 2012; Xu 2015) rather than a single TN. This would be consistent with the brain being configured to involve active counterbalancing systems, such that in the optimal case each task positive configuration would have a matched task negative one, to balance it. However, we acknowledge that there are likely to be trade-offs between having a perfectly balanced system and having a functional one. In our model, the nodes do not carry out any actual computations and are assigned unitary roles (in terms of sensorimotor function), which is unlikely to be true; both of these, and other (e.g., onto- or phylogenetic) considerations could affect the type of TN that evolution has arrived at.

Finally, in order to achieve a relatively stable solution with rich spontaneous dynamics and interactions with the environment, the system may have to encode (in the local thresholds) information about the world, and the agent’s movement in it. Given the simplicity of the environment in the current simulation, the presence of local thresholds is adequate to facilitate a relatively stable solution. However, as the environment (and sensory input systems) becomes more complex, it will be necessary to use more sophisticated models with more flexibility. If the repertoire of brain states is to be more fully explored in the face of this increasing complexity, then it will be necessary to capture more information about the environment/sensory systems. This leaves open questions about the roles of other types of learning (e.g., longer-distance excitatory and reinforcement learning) and their roles in supporting the system staying in a rich dynamical regime in a complex environment with complex sensorimotor systems and with more cognitive control mechanism.
Methods

Empirical Structural Connectivity

Simulated activity patterns were generated from a computational model constrained by empirical measures of white-matter structural connectivity between 66 cortical regions of the human brain, defined by diffusion tensor imaging (DTI) (Hagmann et al, 2008). This structural network has been used in a range of previous computational models to demonstrate emergent properties of resting state functional connectivity (Hellyer et al, 2016; Cabral et al., 2011; Hellyer et al., 2014; Messe et al., 2014). A full methodology, describing the generation of this matrix $\langle C \rangle$ is available in (Hagmann et al., 2008). In brief: measures of length and strength of stream-line based connectivity were estimated using Deterministic tractography of DSI datasets (TR=4.2s, TE=89s, 129 gradient directions max b-value 9000s/mm$^2$) of the brain in 5 healthy control subjects. A high-dimensional ROI based connectivity approach was projected though the 66 regions of the Desikan-Killianey atlas (FreeSurfer http://surfer.nmr.mgh.harvard.edu/), such that $C_{i,j}$ is the number of streamlines connecting nodes $i$ and $j$.

Computational Model

Neural Dynamics

To simulate brain activity, we defined a simple model based on the Greenberg-Hastings model, which has been shown in previous work to approximate patterns of empirical functional connectivity (Haimovici et al, 2013). At each time point, $t$, each node, $i$, in the model can be in one of three states, $S_{i,t}$: excitatory (E), quiescent (Q), or refractory (R). Nodes changed state according the following simple probabilities: $p_i(E \rightarrow R) = 1$; $p_i(R \rightarrow Q) = 1$; $p_i(Q \rightarrow E) = 10^{-1}$. Importantly, nodes would also change from Q->E if the summed input from $n$ connected nodes, $j$, was greater than a local threshold value: $\sum_{j=1}^{n} C_{i,j} S_{j,t-1} > T_i$. The strength of the activation threshold, $T_i$, could be tuned to separately at each node (see below). $S_{i,t}$ was binarized so that E was coded as 1, R or Q as 0.
**Homeostatic plasticity**

For most of the simulations, we used a local homeostatic plasticity mechanism as follows: we allowed the activation threshold to vary by a small amount based on the activity in each node at the previous time-step, according to the following rule similar (but simplified) to that introduced in (Vogels et al., 2011) and used in (Hellyer et al, 2016) and with a similar (but simpler) effect of balancing incoming excitation from connected nodes:

$$\delta t_i = \alpha (S_{i,t} - \rho)$$

where $\rho$ is a target activation and $\alpha$ is a learning rate. Thus in the case that the activity of $i$ is 1, and $\rho < 1$ there is an increase in the threshold whereas, otherwise the threshold decreases. Therefore, the time-averaged activity of $S_i$ will approximate $\rho$.

**Environmental Embedding**

The motor activity (movement) of the agent was defined by two commands; Turn ($h$) in radians per update step and Move ($v$) which moved the agent forward $v$ world units. The activity within these two parameters at each time-step was determined by the simulated neural activity at four nodes (two rotate and two advance nodes) of the computational model. We chose these nodes to be bilaterally symmetrical such that they approximately correspond to motor related regions in the brain (n.b., we make no claims that this anatomical correspondence is correct or that the results are dependent on this). If a rotate node was active, the agent would attempt to turn $\approx 30^\circ$ in that direction. If both nodes were active, then the effect would cancel out this out. If a single forward node was active, the agent would move forward 1/10 of a unit the arbitrary world space, if both forward nodes were active, the unit would move forward 1 unit of world space. In addition, we added some temporal smoothing across time for activity within the move such that the move command described was 7/8 of the activity of the relevant assigned node, and 1/8 of the activity of the previous time step. (The amount of this smoothing and the values of how nodes translated into movement were chosen semi-
arbitrarily, to produce agent motion that appeared plausible, i.e., neither very fast or slow).

Sensory information ('visual' perception) from the environment was integrated into the computational model through the use of two horizontal ray-traces emanating from each “eye” of the agent and offset by ±10° from the vertical. A distance threshold was defined, such that if an object (i.e., the wall, in this simple environment) was less <2 units of world space then a specific node (“near visual”) of the model was set to the E state, if an object was detected between 2 and 10 world units away then the “far visual” node was set to excitatory. In addition, to “visual” input, we also defined a rudimentary “somatosensory” input, whereby if model had collided with any other object in the environment then specific “somatosensory” nodes for collisions on either of the Left or Right side of the agent within the computational model were set to the E state.

**Task-Negative nodes**

In order to explore the effect of balance between task positive (TP) and task negative (TN) networks, we defined for some simulations, a collection of TN nodes that were anti-correlated with the TP nodes described above. These TN nodes were defined as two (bilateral) pairs of task negative nodes approximately corresponding to regions that consistently show relative deactivation across many empirical fMRI tasks were chosen (although, this was still a relatively arbitrary decision and we do not wish to make any claims based on anatomical precision). These nodes were set to the E state if TN nodes (i.e., the “visual”, or “somatosensory” nodes were in the Q or R states, and Q, when the TN nodes were activated, such that TP and TN nodes were anti-correlated.

Further, given that TN activity is task specific (e.g., Shmuel et al, 2006; Leech et al, 2014; Seghlier et al, 2013), we defined two TN nodes (one on the left and its homologous region on the right) that were set to be excitatory when “visual” activity was not excitatory; and, a separate pair of nodes (again bilateral, homologous) were set to be activated when “somatosensory” activity was silent. For most simulations, the location of the task negative nodes were
kept constant. However, in an additional set of simulations, the task negative nodes were randomly re-positioned by picking random bilateral homologous pairs of nodes from the network. Unless stated otherwise, the results presented are from a single model run for 100,000 epochs. However, we repeated the model two further times with different random seeds (so different patterns of excitatory noise, resulting simulated dynamics and movements), replicating the results presented below.
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