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A false regime and its correction: the cortical microcircuit and its metabolism

A cortical column considered validated worked only in the first second. The defect was in the single neuron, and connecting the network to energy revealed a second limitation.

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Validated, open limits

2 test benches · 19 failed network repairs · confirmation on unseen seeds

5/5	19	5/6	0/5
column criteria at steady state after the fix, on seeds 6–11 too	attempts to repair the network, all failed: the defect was in the neuron	criteria of the metabolism coupled to the network	depolarisation block attempted and not adopted

SUMMARY

The spiking cortical column with active dendrites was considered validated with 5 criteria out of 5. Coupling it to metabolism revealed that the result had been measured in the **first second**, before the network reached its regime: at steady state the column slid into a hyperactive state that sustained itself. Nineteen repair attempts at the network level failed. A chain of hypotheses written in advance located the defect in the **single neuron**: the calcium/NMDA dendritic plateau had no termination mechanism and, once switched on, stayed on for ever. A potassium current activated by the plateau corrects it; with a declared scan of three parameters the column again passes 5 criteria out of 5 at steady state, also on seeds never used in the choice. The metabolism coupled to the corrected column passes 5 criteria out of 6 and closes the energy–network loop; two extensions (depolarisation block and ischaemia timing) failed and were not adopted.

1 Why it matters

Phase 2 of the organism, with populations of spiking neurons, rests on this column. Test F5 of the theory, which asks whether the organism's defence preserves inhibition when energy fails, requires a network coupled to metabolism. A non-stationary network would have produced results that depend only on when one looks.

2 The model

The column uses Izhikevich neurons with conductance-based synapses, integrated every 0.5 ms: excitatory pyramidal cells with 4 active dendritic branches (AMPA, NMDA and calcium plateau) and two classes of inhibitory interneurons, PV and SST. The assemblies have reinforced connectivity within them, as in the model of Litwin-Kumar and Doiron (2012). The key criterion, C4, requires a brief stimulus to switch on **localised** persistent activity in the stimulated assembly.

3 How we found the defect

step	result
coupling to metabolism	interneuron rate 40.5 Hz, above the limit: the column is not stationary
same criteria at steady state (3 s of transient discarded)	**3 out of 5**: localised persistent activity in 0 assemblies out of 6; the "validated" status is withdrawn
12 network variants (input, connections, NMDA, clustering, PV)	none restores C4
assembly-specific inhibition (Rost and colleagues, 2018), 7 variants	saturation or the same active state; not adopted
plateau hypothesis (three hypotheses written in advance)	two confirmed, one refuted: cutting every input makes firing **rise**
isolated neurons	30 ms of stimulus lock the plateau on for ever: the soma fires at about 556 Hz without any input
diagnosis	in the real neuron the plateau lasts tens or hundreds of ms and ends through potassium conductances (Schiller and colleagues, 2000; Major and colleagues, 2008)
potassium fix, unchanged configuration	185 ms plateau, no locking; the network becomes silent or bistable: 3 out of 5
declared scan of 20 variants on seeds 0–2	external input, clustering and NMDA weight chosen
run 2	**5 out of 5** at steady state

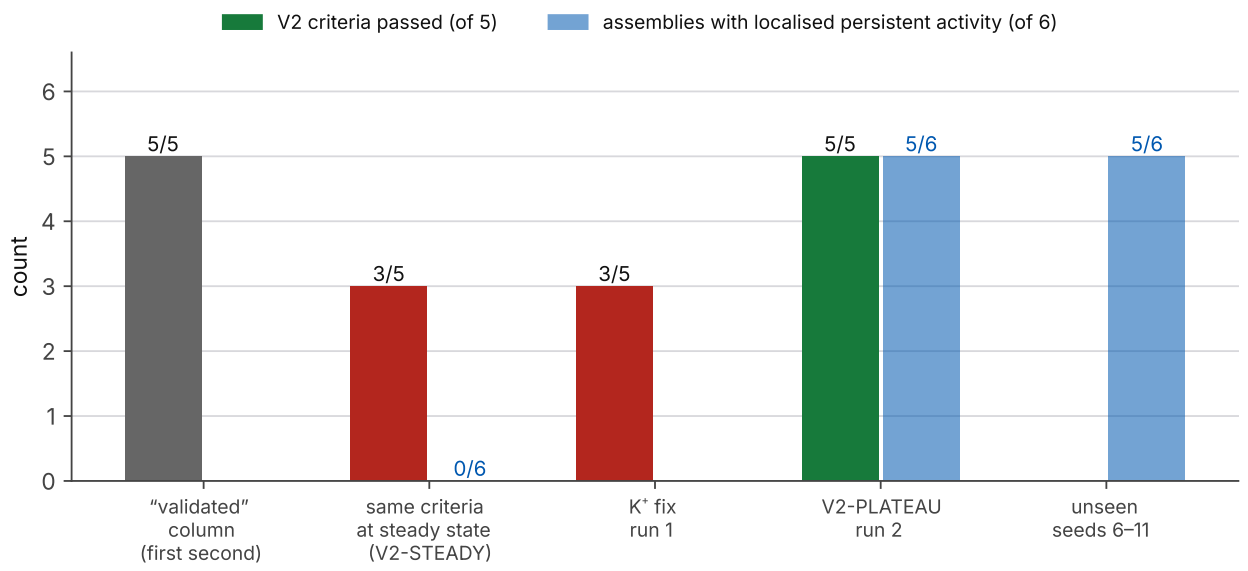


Figure 1. Criteria passed and assemblies with localised persistent activity along the diagnostic chain. The initial "5 out of 5" was measured in the first second; at steady state the column drops to 3 out of 5 and no assembly keeps localised activity. After the plateau fix and the parameter scan the column is back to 5 criteria out of 5, with localised activity in 5 assemblies out of 6, also on seeds 6–11 never used to choose the parameters.

The fix adds to the dendritic branch a current g·k·(E_K − v), with a variable k that follows the plateau with a time constant of 150 ms, conductance 2.0 and potassium reversal potential at −90 mV. The historical benches remain fixed to the previous model so that they can be reproduced.

4 The metabolism coupled to the network

The metabolic substrate (ATP, ADP, cerebral flow, astrocyte–neuron shuttle) existed but was not connected to any network. We coupled it to the corrected column with three compartments, pyramidal, PV and SST, and different energy costs per population taken from the literature: 21% of the signalling budget to action potentials and 50% to postsynaptic receptors (Howarth, Gleeson and Attwell, 2012); a cost per spike for PV cells 1.6 times that of pyramidal cells (Carter and Bean, 2009). When ATP falls, vitality drops, the sodium–potassium pump fails and synaptic release is reduced.

criterion	outcome
Q0 isolation	passed: with the feedback off, 6238 spikes identical bit for bit
Q1 stable rest	passed: ATP 2.82–2.94 mM, drift at most 0.46%
Q2 balance	passed: maximum error 5.0×10^{-16}
Q3 demand → supply	**failed**: total demand rises by 5–7% against the 10% required
Q4 supply → network	passed: during hypoxia 105.7 Hz with the feedback against 8.5 without
Q5 audit	passed

Criterion Q3 fails because of how it was written, not because of the model: baseline consumption is about two thirds of the total, so +6% of the total corresponds to about +20% of signalling. The criterion was not rewritten.

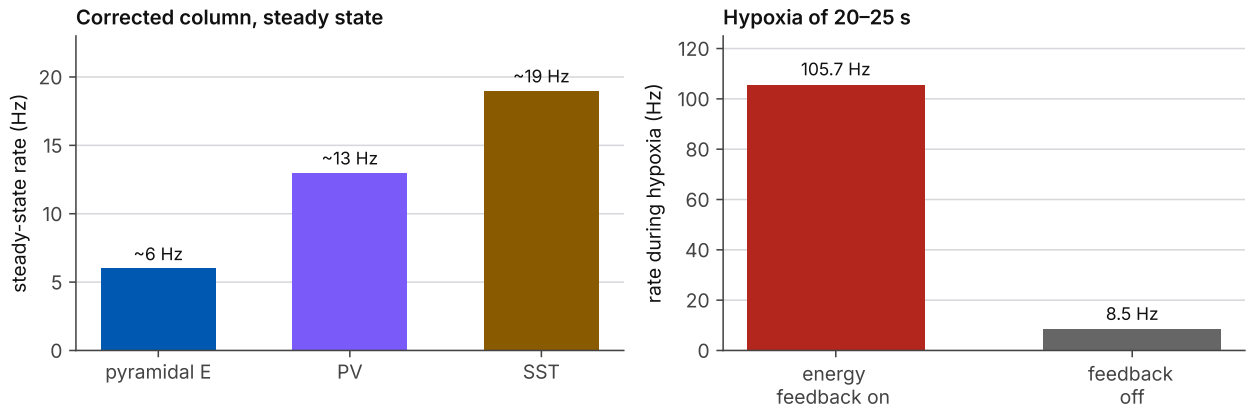


Figure 2. Left, the steady-state rates of the corrected column by population. Right, the network rate during 20–25 s of hypoxia with the energy feedback on and off: when ATP falls the network really changes, but in the wrong direction (see the critical limitation). Values from the logs of the V2-PLATEAU and V2-ENERGY-P2 benches.

5 The critical limitation: neurons that speed up instead of stopping

With ATP exhausted the model neurons fire at hundreds of hertz, whereas real neurons go into **depolarisation block** and stop firing. We tried to fix this twice:

- **Depolarisation block (0 out of 5).** A phenomenological gate on sodium availability was triggered even at rest, let the membrane rise to +30 mV irreversibly and did not stop the neurons with active dendrites. The code is off by default.
- **Ischaemia timing (1 out of 7).** With phosphocreatine and anaerobic glycolysis, and physiological reserves, ATP cannot last the roughly 2 minutes observed if consumption stays constant: the data imply that consumption falls during ischaemia. Two defects of the model also emerged. Original values restored.

Test F5 remains blocked until we have a neuron with a biophysical anoxic block, for example with sodium inactivation based on conductances or ion concentrations (Kager and colleagues, 2000).

6 What it shows and what it does not

It shows that the column now has a physiological stationary regime, that the defect was cellular and not in the network, and that the energy–network loop is exact and isolated, with per-population costs grounded in the literature.

It teaches a rule of method that we have applied to every module since: **every criterion is measured at steady state**, with the transient declared and discarded. It is a lesson the project had to relearn later, for example with the spiking workspace (NR-05).

It does not show quantitative fidelity to cortical data: the margins on persistent activity and on the gamma band are thin. It says nothing about the order in which populations fail during hypoxia, which depends on the artefact of firing at zero ATP.

> **Limitations.** Izhikevich neurons, not full conductance-based models; the oxidative capacity of PV cells (1.5 times that of pyramidal cells) is an assumption based on qualitative data; the parameter scan is declared but remains a choice; F5 is blocked.

References

- Litwin-Kumar, A. and Doiron, B. (2012). Slow dynamics and high variability in balanced cortical networks with clustered connections. *Nat. Neurosci.* 15, 1498.
- Rost, T., Deger, M. and Nawrot, M. P. (2018). Winnerless competition in clustered balanced networks. *Biol. Cybern.* 112, 81.
- Schiller, J. and colleagues (2000). NMDA spikes in basal dendrites of cortical pyramidal neurons. *Nature* 404, 285.
- Major, G. and colleagues (2008). Spatiotemporally graded NMDA spike/plateau potentials in basal dendrites. *J. Neurosci.* 28, 7625.
- Howarth, C., Gleeson, P. and Attwell, D. (2012). Updated energy budgets for neural computation. *J. Cereb. Blood Flow Metab.* 32, 1222.
- Carter, B. C. and Bean, B. P. (2009). Sodium entry during action potentials of mammalian neurons. *Neuron* 64, 898.
- Kann, O., Papageorgiou, I. E. and Draguhn, A. (2014). Highly energized inhibitory interneurons are a central element for information processing in cortical networks. *J. Cereb. Blood Flow Metab.* 34, 1270.
- Kager, H., Wadman, W. J. and Somjen, G. G. (2000). Simulated seizures and spreading depression in a neuron model incorporating interstitial space and ion concentrations. *J. Neurophysiol.* 84, 495.
- Internal documents: whitepapers WP-02 and WP-03; records `microcircuit_cortical_column`, `metabolic_microcircuit`.

How to cite

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Nadirion technical report, not peer-reviewed. The criteria of every test were written before running it; negative results are reported like positive ones. The tests measure functional roles.