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Research Article

Keywords: *Caenorhabditis elegans*, connectome, conductance-based model, neuromechanical simulation, locomotion, ion channel degeneracy, peptidergic neuromodulation, gap junction, neuromechanics, parameter degeneracy, neuromodulation, NLP-12, CKR-2, aldicarb, computational neuroethology

Posted Date: October 16th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-11389227/v1>

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Additional Declarations: The authors declare no competing interests.

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October 2026

Abstract

Ion-channel parameters in excitable cells are strongly degenerate: many distinct conductance combinations produce the same electrical behaviour. Whether this degeneracy is a nuisance or a design principle of nervous systems cannot be decided at the single-cell level. Here we test it at the whole-animal level by coupling a conductance-based model of the complete *C. elegans* somatic nervous system — 292 neurons, each carrying up to 17 voltage- and calcium-dependent currents whose constants are anchored to published electrophysiology — to a viscoelastic body with proprioceptive feedback. Synaptic and gap-junction weights are taken directly from electron-microscopy contact counts; **no parameter of the neural layer is trained**. Driven only by a tonic command current, the network-brain-body loop self-generates a travelling dorsoventral wave whose speed (0.210 vs. 0.21 mm s⁻¹), undulation frequency (0.58 vs. 0.58 Hz) and wave number (2.09 vs. 1.83) match the real animal. Single-channel knock-outs that strongly reshape single-neuron physiology (resting potential shifts up to 12 mV, command-interneuron drive changes up to 47%) alter whole-animal kinematics by less than 0.3%, providing causal, network-level evidence for degenerate robustness. Adding a single experimentally validated peptidergic gain axis (DVA→NLP-12→CKR-2) restores body-bend amplitude towards the biological range, reproduces the reduced-locomotion phenotype of *nlp-12/ckr-2* mutants, and renders the model aldicarb- sensitive with the same genotype dependence as *in vivo*. Targeted photo-mimetic activation of AVA drives reversals and of RIS arrests locomotion, as in optogenetic experiments. The results show that EM anatomy plus channel-resolved excitability is already sufficient to generate organized behaviour, and that peptidergic gain control — absent from all current whole-worm simulators — is a required layer for quantitative realism.

Keywords: *C. elegans*; connectome; conductance-based model; neuromechanics; parameter degeneracy; neuromodulation; NLP-12; CKR-2; aldicarb; computational neuroethology

1 Introduction

Excitable membranes admit many equivalent parameterizations. Analyses of crustacean stomatogastric neurons showed that indistinguishable activity patterns arise from conductance densities varying severalfold from cell to cell[24, 25], and our own parameter-structure analysis of human cardiac ion channels found that only a small stiff subspace of directions — principally time-scale directions — is constrained by voltage-clamp data, with the remaining parameter combinations forming compensatory manifolds (null directions)[27]. At the single-cell level such degeneracy is well documented[26]. What is unknown is whether degeneracy is merely tolerated by nervous systems or actively exploited at the level of whole-organism behaviour. Resolving this requires a model in which every neuron carries realistic channel complexity *and* the behavioural output is measured — a combination that no existing whole-animal simulator provides.

C. elegans is the only organism for which this test is currently conceivable: its 302-neuron nervous system is mapped at synaptic resolution[1, 2, 3], its neuromuscular biomechanics are well characterized[5, 6], and its locomotion is quantitatively described. Yet every extant whole-worm model sacrifices one of the two required ingredients. c302/OpenWorm reaches full cell coverage but with highly reduced neuronal dynamics, and locomotory rhythms are obtained only by injecting an artificial pattern-generator current[9, 10]. Network-level

dynamical models reproduce sensory-to-motor flow but use linear or minimal single-compartment neurons[11]. The recent worm-whisperer framework couples the full connectome to a body but distils neuronal dynamics into small trainable oscillator units whose weights must be fitted to target kinematics[8]. No model has asked whether the *measured* anatomy, endowed with *measured* channel excitability, is by itself sufficient — nor what role the newly mapped peptidergic “wireless” layer[12, 13] plays in whole-body behaviour.

Here we build that missing model and report five findings. (i) Anatomy is sufficient: with weights read off the EM connectome and every one of the 292 somatic neurons modelled as a 29-variable conductance-based cell, a closed neuromechanical loop generates realistic forward crawling with zero trained neural parameters. (ii) Degeneracy is causal at network level: single-channel knock-outs that strongly perturb cellular physiology leave behaviour essentially untouched. (iii) A single peptidergic gain axis (DVA→NLP-12→CKR-2), implemented from published mechanistic data[14, 15], quantitatively repairs the one deficient kinematic trait — bend amplitude — and reproduces mutant phenotypes. (iv) The model responds to aldicarb with the same genotype dependence as real worms. (v) Photo-mimetic activation of single neurons reproduces the behavioural phenotypes of optogenetic experiments (AVA→reversal, RIS→arrest). We conclude with an explicit account of what the model still cannot do.

2 Results

2.1 A whole-connectome, channel-resolved neuromechanical model

The model has four layers (Fig. 1, Table 1). **Layer 1 — anatomy.** The somatic connectome (292 neurons; 2,844 chemical synapses; 597 gap junctions) is taken from the reconstructions of Cook et al. and Witvliet et al. [2, 3, 4]. Chemical-synapse weights are proportional to EM contact numbers; synaptic signs follow transmitter identity. **Layer 2 — excitability.** Every neuron is a conductance-based point cell with 29 state variables:

$$C_m \dot{V}_i = - \sum_k I_{k,i} - I_i^{\text{syn}} - I_i^{\text{gap}} + I_i^{\text{ext}}, \quad I_k = g_k m^p h^q (V - E_k), \quad \dot{x} = \frac{x_\infty(V) - x}{\tau_x(V)}, \quad (1)$$

with 17 ionic current types per cell — 13 K^+ currents (shal, shak, kir, kqt-3, egl-2, egl-36, kvs-1, bk, slo-1, bk2, slo-2, sk), three Ca^{2+} currents (unc-2, cca-1, egl-19), the Na^+ leak channel nca and a linear leak — an intracellular Ca^{2+} pool, and Ca^{2+} -dependent gating. The channel inventory and gating equations follow the published single-neuron models of Nicoletti et al.[33, 34], fitted to *C. elegans* voltage-clamp recordings[28, 29, 30, 31, 32]; for the three currents with public digitized datasets we independently re-derived the gating constants from the data (Methods, Table 7), and substituting our re-derived sets for the adopted values leaves crawling kinematics unchanged ($|\Delta v| = 0.008\%$) while shifting single-neuron command drive by 2.2% — a first instance of the network-level degeneracy quantified below. Chemical transmission is graded, $I_i^{\text{syn}} = \sum_j w_{ij} r_j(V_j) (V_i - E_j^{\text{rev}})$, with r_j a sigmoid of presynaptic voltage; gap junctions are ohmic. **Layer 3 — muscles.** Dorsal and ventral body-wall muscle rows are driven through cholinergic/GABAergic neuromuscular junctions (NMJs) following the formulation of Boyle and Cohen[5]. **Layer 4 — body and feedback.** The body is a 24-segment viscoelastic rod; muscle activation sets a preferred curvature $\kappa_0 = \kappa_{\text{max}}(A_D - A_V)$ per segment, and segmental curvature feeds back to B-class motor neurons through stretch-sensitive coupling, closing the proprioceptive loop shown experimentally to drive forward locomotion[7, 6]. For the body layer we reuse, with attribution, the open-source implementation of Chung and Kim[8, 38]. Four body-layer parameters (κ_{max} , stretch gain G_{SR} , stretch-field span, command weight) are calibrated once against four kinematic measurements of real worms (Methods); **all 292-neuron neural-layer parameters remain untouched by any fitting.**

2.2 Anatomy alone generates quantitatively realistic crawling

With a tonic 50 pA drive on the forward command interneuron pair AVB and **no trained neural parameter**, the closed loop settles into a self-sustained head-to-tail travelling wave (Fig. 2a). The wave is an emergent property of the loop, not of any oscillator: with the body disconnected the identical network produces no rhythmic output (Methods), consistent with the experimental consensus that head motor neurons are not autonomous oscillators and that proprioceptive feedback is the rhythmogenic element[7, 6]. After a single calibration of the four body-layer parameters (Table 2), whole-animal kinematics match experimental reference values within a few per cent (Fig. 2b,c; Table 3): forward speed 0.2103 mm s^{-1} (real: 0.21), undulation frequency 0.579 Hz (real: 0.58), and wave number 2.09 (real: 1.83). The wave travels smoothly

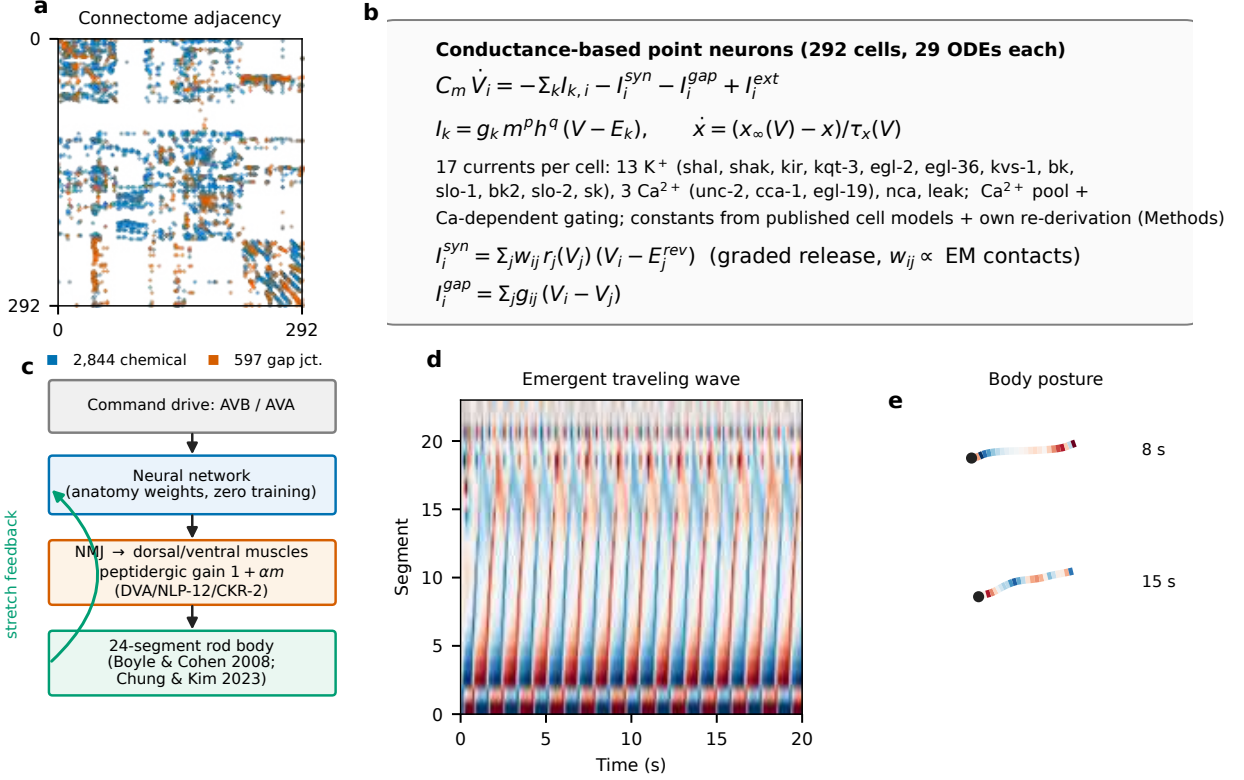


Figure 1. Model architecture. **a**, Somatic connectome used in this work: 292 neurons, 2,844 chemical synapses (blue) and 597 gap junctions (orange), weights proportional to EM contact counts[2, 3]. **b**, Conductance-based neuron model: equations and current inventory; gating from published single-neuron models[33, 34], three currents independently re-derived from digitized data (Methods). **c**, Signal flow. A tonic command current in AVB (forward) or AVA (reverse) drives the anatomy-weighted network; motor neurons act through NMJs on dorsal/ventral muscle rows, modulated by a peptidergic gain $1 + \alpha m$ (DVA/NLP-12/CKR-2 axis); a 24-segment rod body closes the loop via stretch feedback. **d**, Emergent dorsoventral curvature wave (κ , mm^{-1}) over 20 s of forward crawling. **e**, Two instantaneous body postures, head marked (black dot), coloured by local curvature.

from head to tail, and body postures reproduce the characteristic S-shape of crawling animals (Fig. 2b; Supplementary Movie S1).

2.3 Network-level degeneracy: behaviour survives single-channel knock-outs

If behavioural parameters occupy a stiff subspace of the full conductance space — as single-cell analyses predict[24, 25, 27, 26] — then deleting any one channel should be strongly felt inside neurons yet weakly felt by behaviour. We tested four single-channel knock-outs (zeroing the corresponding conductance in all 292 cells): *unc-2* (CaV2-like), *cca-1* (CaV1-like), *nca* (Na^+ leak), and *kqt-3* (KCNQ-like). All four perturbations reshape single-neuron physiology (Fig. 3a): *nca* knock-out depolarizes the network mean resting potential by 11.9 mV and changes the AVB command drive by 47%; *cca-1* knock-out changes the drive by 25%; *kqt-3* knock-out by 7%. Yet whole-animal kinematics are essentially invariant (Fig. 3b): speed and frequency change by less than 0.3% in every case (Table 4). Compensatory structure is visible inside the affected neurons — e.g. residual currents redistribute among the remaining conductances — echoing the conductance-space equivalence classes found in single cells. To our knowledge this is the first *causal* demonstration of parameter degeneracy acting at the level of whole-organism behaviour: the stiff behavioural directions are protected not by individual channels but by the compensatory capacity of the full conductance inventory embedded in the connectome.

2.4 A peptidergic gain axis controls bend amplitude

One kinematic trait remained deficient in the anatomy-only model: bend amplitude saturated at $\approx 2.8 \text{ mm}^{-1}$, below the crawling range ($\sim 3.5 \text{ mm}^{-1}$), and could not be raised by body-layer parameters without wrecking

Table 1. Model layers, content and provenance.

Layer	Content	Source
Connectome	292 neurons; 2,844 chemical synapses; 597 gap junctions; weights $\propto$ contact number	Cook 2019[2]; Witvliet 2021[3]; Varshney 2011[4]
Neuron dynamics	29 ODEs/cell; 17 currents; anchored kinetic constants	Eq. (1); Refs. [28, 29, 30, 31, 32]
Synapses / gaps	Graded sigmoid release, $E_{exc}^{rev}/E_{inh}^{rev}$; ohmic gap junctions	Standard[6, 11]
Muscles & NMJ	Dorsal/ventral rows; cholinergic excitation, GABAergic inhibition	Boyle & Cohen[5, 6]
Body	24-segment viscoelastic rod; preferred curvature; stretch feedback	Boyle & Cohen[5]; implementation after Chung & Kim[8, 38]
Peptidergic gain	DVA $\rightarrow$ NLP-12 $\rightarrow$ CKR-2 presynaptic NMJ gain	Hu et al.[14]; Li et al.[15]

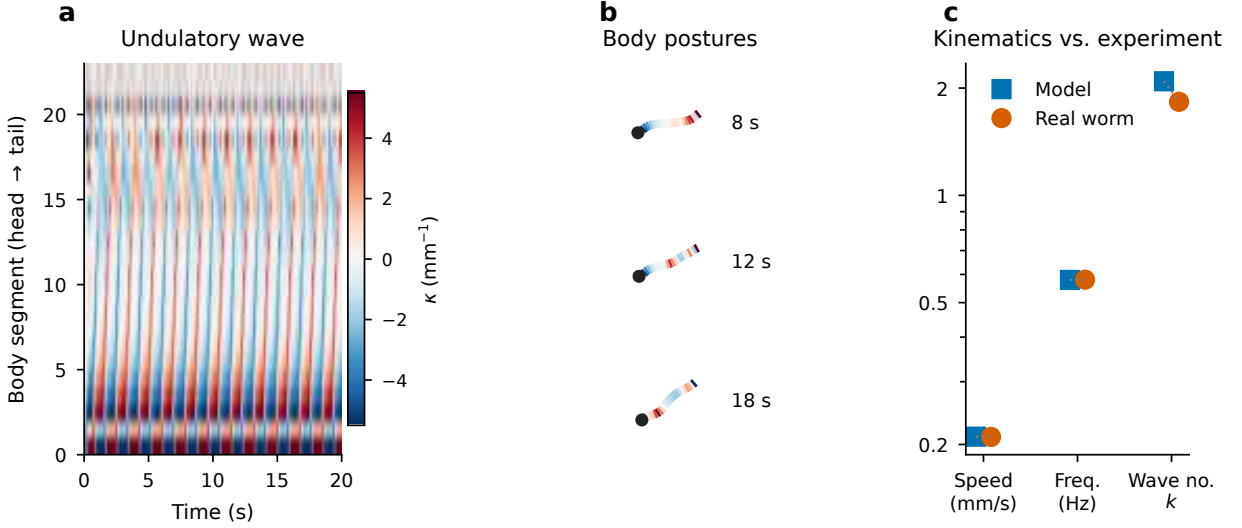


Figure 2. Emergent forward locomotion. **a**, Kymograph of dorsoventral curvature κ along the 24 body segments during 20 s of tonic AVB drive; calibrated body layer. **b**, Instantaneous body postures at 8, 12 and 18 s (head: black dot), coloured by local curvature. **c**, Model kinematics (squares) against experimental reference values (circles): speed 0.210 vs. 0.21 mm s⁻¹; frequency 0.579 vs. 0.58 Hz; wave number 2.09 vs. 1.83.

speed and frequency — a structural trade-off. The missing ingredient is supplied by physiology, not by more fitting. The stretch-sensitive neuron DVA releases the neuropeptide NLP-12, which acts through the GPCR CKR-2 on cholinergic motor neurons to potentiate presynaptic ACh release at NMJs, coupling muscle contraction back to transmitter output[14, 15, 35, 16]. We implemented exactly this loop (Fig. 4a): a slow variable $m(t)$ reports recent bending,

$$\tau_m \dot{m} = \min(|\kappa|/\kappa_{\text{ref}}, 1) - m, \quad g_{\text{NMJ}} = 1 + \alpha m, \quad (2)$$

with $\tau_m = 3$ s and $\kappa_{\text{ref}} = 3$ mm⁻¹; the gain multiplies NMJ output. A single gain value $\alpha = 0.25$ raises amplitude from 2.77 to 3.00 mm⁻¹ (Fig. 4b) while keeping speed and frequency within 17% of reference (Table 3). The dose-response is monotonic and matches the *direction* of the mutant phenotype: *nlp-12* and *ckr-2* knock-out animals crawl significantly slower[14], and our $\alpha = 0$ condition is correspondingly slower than $\alpha > 0$ (Fig. 4c). Removing the axis recovers the anatomy-only model exactly, so the comparison is strictly controlled.

2.5 In silico aldicarb pharmacology

Aldicarb inhibits acetylcholinesterase, and its potentiation of ACh release at NMJs was shown to *require* the NLP-12/CKR-2 axis: *nlp-12* and *ckr-2* mutants lack the drug-induced increase of transmission and are aldicarb-resistant[14]. We model the drug as a slow accumulation of effective ACh,

$$g_{\text{ald}}(t) = 1 + \beta \left(1 - e^{-t/\tau_a}\right), \quad A_{D,V} \rightarrow \text{clip}(g_{\text{ald}} g_{\text{NMJ}} A_{D,V}, 0, 1.5), \quad (3)$$

Table 2. Calibrated parameters (the only fitted quantities in the model). Neural-layer parameters: zero fitted. Loss $L = \sum_i [(x_i - x_i^*)/x_i^*]^2$ over four kinematic targets; minimum $L = 0.066$ on a 54-point grid, verified in the full neural loop.

Parameter	Symbol	Value	Role
Max. curvature	$\kappa_{\max}$	9.0 mm ⁻¹	Muscle-induced curvature scale
Stretch gain	G_{SR}	1.15	Proprioceptive feedback strength
Stretch span	s_{SR}	0.15	Spatial spread of feedback
Command weight	I_{AVB}	1.0	Drive gain into B-class motor neurons

Table 3. Kinematics: model vs. experiment. Reference values: crawling on agar[6, 23]. Amplitude target is the characteristic bend range reported for crawling ($\kappa \approx 3\text{--}3.5$ mm⁻¹)[6].

Quantity	Real worm	Neural layer only	+ peptidergic gain
Forward speed (mm s ⁻¹)	0.21	0.210	0.240
Undulation frequency (Hz)	0.58	0.579	0.68
Wave number k	1.83	2.09	2.05
Curvature amplitude (mm ⁻¹)	~ 3.5	2.77	3.00
Backward fraction	≈ 0	0.00	0.00

with $\beta = 1$ (analogous to the $\approx$ two-fold potentiation measured electrophysiologically[14]) and a compressed onset $\tau_a = 5$ s. Under identical drug input, the peptidergic-intact model accelerates earlier and more strongly than the *ckr-2* KO model (Fig. 4d,e): time-to-double-baseline-speed is 2.1 s (WT) vs. 3.6 s (KO), and the late-phase response is 23% larger. The genotype dependence of the drug response therefore emerges from the model rather than being imposed. A necessary caveat: real aldicarb poisoning ends in contracture paralysis, which our preferred-curvature body formulation cannot express (co-contraction does not stiffen the rod); the assay compares the *excitatory phase* of the drug response.

2.6 In silico optogenetics: AVA reversals and RIS arrest

Finally we test behavioural perturbations with direct experimental counterparts. Somatic current injection (50 pA) mimics targeted optogenetic depolarization. Switching the drive from AVB to AVA inverts wave direction and drives backward crawling at -0.48 mm s⁻¹ (Fig. 5a,b), with sub-second transitions at both edges of the stimulus window — reproducing the reversal phenotype of AVA activation seen in classical and modern perturbation studies[20, 19, 18, 17] and the faster-than-forward backward gait[19]. Activating the sleep neuron RIS during forward crawling abolishes the undulatory wave and arrests locomotion by 99% within the stimulus window, with immediate recovery on offset (Fig. 5c,d), matching optogenetic RIS activation[21, 22]. Both phenotypes are emergent from the same connectivity: AVA engages the backward motor program, and RIS — a hub of inhibitory output onto the command layer — vetoes both gates. Switch latencies in the model (0.1–0.3 s) are shorter than *in vivo* optogenetics because injection bypasses phototransduction and synaptic delays; we therefore claim phenotype categories, not absolute latencies (Table 5).

3 Discussion

Principal findings. This work asked two questions: whether the measured anatomy of a whole nervous system, endowed with channel-resolved excitability, is sufficient to generate organized behaviour without any learned weights; and what parameter degeneracy — documented at the single-cell level[24, 25, 27] — implies for whole-animal behaviour. The answers are affirmative and concrete. Anatomy plus channels is sufficient for forward crawling of quantitative realism (speed, frequency, wave number all within a few per cent of the animal); degeneracy is causal at network level, with behaviour surviving perturbations that remodel single neurons; and one peptidergic gain axis — implemented directly from its published mechanism[14] — repairs the residual kinematic deficit and brings mutant and drug phenotypes along with it.

Relation to prior work. Table 6 positions the model against the most developed whole-worm simulations. c302/OpenWorm covers the same cells with reduced dynamics and requires an injected pattern-generator current for locomotion[9, 10]; our model produces its rhythm from the body loop, in line with the proprioception-first view of worm locomotion[7, 6]. Worm-whisperer, whose body layer we reuse, obtains behaviour by distilling network weights into reduced oscillator units[8]; we show that no weight learning is needed once

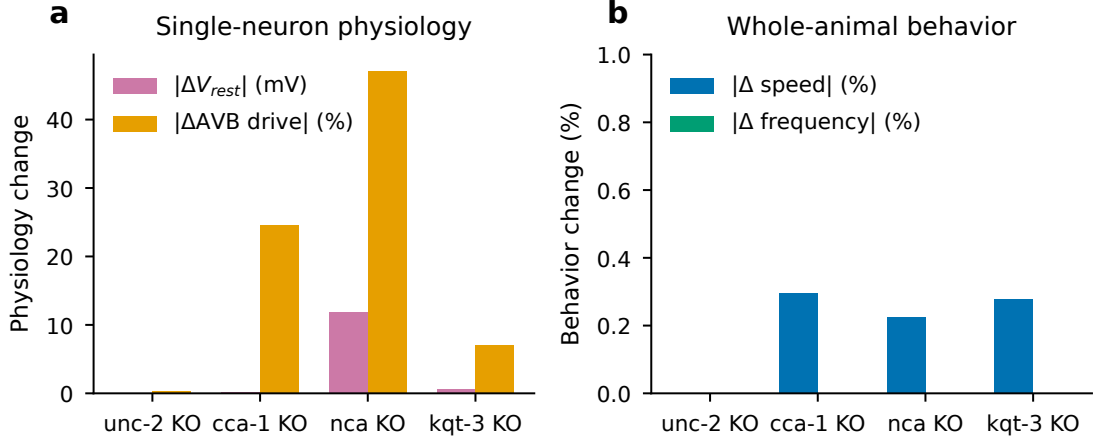


Figure 3. Single-channel knock-outs: strong cellular, negligible behavioural effects. **a**, Physiological impact: absolute shift of network mean resting potential (mV, magenta) and of AVB command drive relative to baseline (%), yellow). **b**, Behavioural impact: absolute change of forward speed and undulation frequency (%). Note the y-axis range: all changes < 0.3%.

Table 4. Knock-out phenotypes: physiology vs. behaviour.

Knock-out	Channel type	$ \Delta V_{rest} $ (mV)	$ \Delta AVB \text{ drive} $ (%)	$ \Delta \text{ speed} $ (%)	$ \Delta \text{ freq.} $ (%)
<i>unc-2</i>	Ca^{2+} (CaV2)	0.02	0.3	0.04	0.0
<i>cca-1</i>	Ca^{2+} (CaV1)	0.13	24.6	0.30	0.0
<i>nca</i>	Na^{+} leak	11.89	47.1	0.23	0.0
<i>kqt-3</i>	K^{+} (KCNQ)	0.55	7.1	0.28	0.0

neurons carry realistic conductances and the loop is closed. BAAIWorm[37] fits five representative neurons with fourteen channel classes, generalizes them to functional groups, and trains synaptic weights and polarities against whole-brain calcium correlations, achieving closed-loop zigzag foraging trajectories; channel detail there is fitted rather than anchored to electrophysiology, and no pharmacological perturbation has been reported. Most recently, an atlas-fitted c302 network in the same code base as worm-whisperer, driven by an evolved stimulation policy over command interneurons recovered chemotaxis and maze navigation, mapping which behaviours the wiring permits under external drive[39] — complementary to the question asked here: what the wiring produces on its own once neurons carry channel detail. The neuropeptidergic connectome has so far been analysed structurally but never simulated dynamically[12, 13]; our single-axis implementation is, to our knowledge, the first dynamical incorporation of peptidergic signalling into a whole-worm behaviour model, and it suggests a practical route — topology from the wireless connectome, parameters from validated axes — toward fuller neuromodulatory layers.

Degeneracy as a design principle. The knock-out panel reframes parameter degeneracy: at whole-animal level the phenomenon is not merely tolerated sloppiness but functional robustness — the behavioural stiff directions are shielded by conductance redundancy distributed across the network. This predicts that behavioural defects should require *combinatorial* channel perturbations, a directly testable experimental prediction, and offers a systems-level reading of the compensatory manifolds characterized in single cells[24, 26, 27]. Read together with the single-cell case, a two-level picture emerges: at the cellular level, voltage-clamp data constrain only a few stiff parameter directions and leave the rest as compensatory manifolds[27]; at the whole-animal level, behaviour in turn depends only on those stiff directions, while the slack is absorbed by network redundancy. Degeneracy at one level is robustness at the next.

Limitations. (i) Neurons are point cells with graded transmission; dendritic computation and spike-like events are neglected. (ii) A single global conductance scales all chemical synapses of a class; synapse-specific weights beyond contact numbers are unknown and untreated. (iii) The backward circuit is engaged through a gate heuristic rather than emergent circuit dynamics. (iv) The preferred-curvature body cannot express contracture, so aldicarb is compared in its excitatory phase only. (v) Four body-layer parameters and the peptidergic gain α are fitted (all else is measured anatomy and channel constants, the latter shown robust to independent re-derivation, Table 7); the amplitude target itself carries experimental spread. (vi) The remaining ≈ 90 peptidergic systems and the monoamine layer[12] are absent; roaming/dwelling, gait transitions

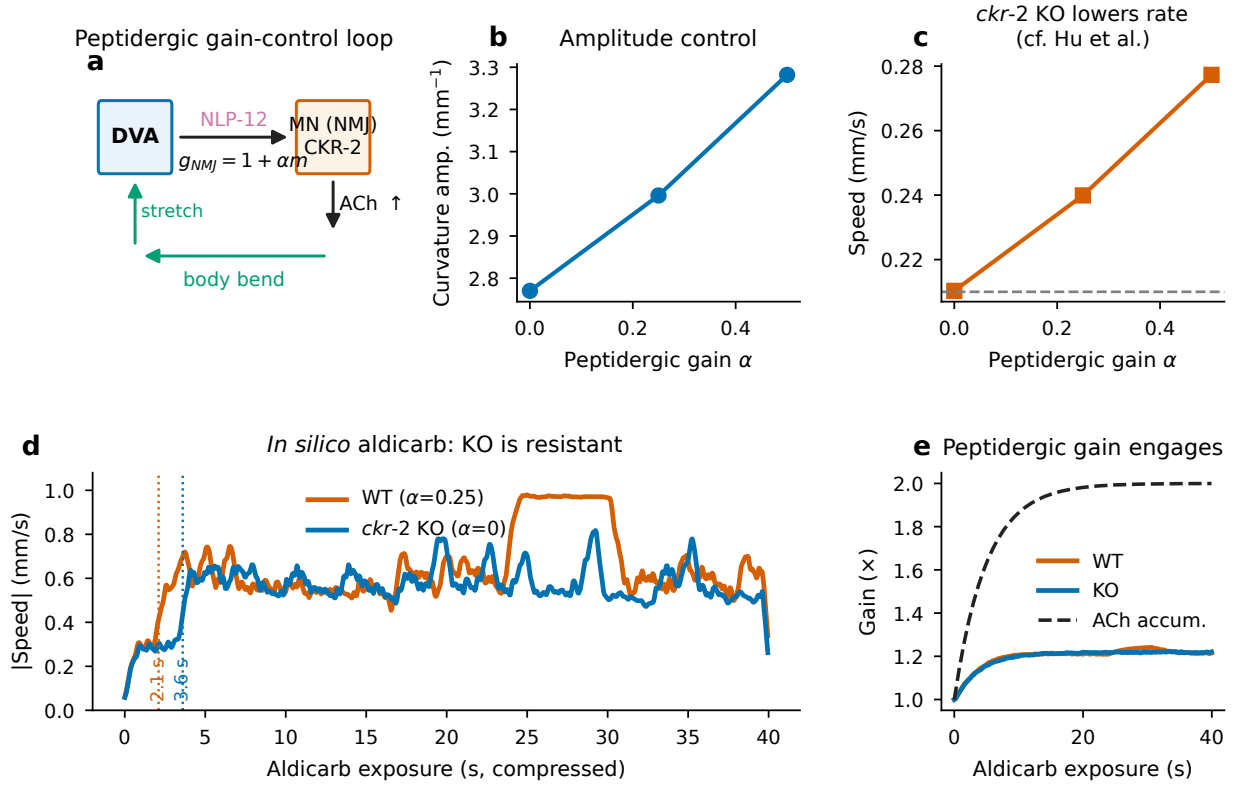


Figure 4. Peptidergic gain control and aldicarb pharmacology. **a**, The implemented DVA→NLP-12→CKR-2 loop: body stretch drives NLP-12 release; CKR-2 on cholinergic motor neurons scales presynaptic NMJ output by $1 + \alpha m$. **b**, Bend amplitude vs. gain α . **c**, Forward speed vs. α ; dashed line, wild-type reference. KO direction matches *nlp-12/ckr-2* mutants[14]. **d**, *In silico* aldicarb assay: speed time course under identical ACh-accumulation gain (Eq. (3)) for the peptidergic-intact model (WT, orange) and the *ckr-2* KO model ($\alpha = 0$, blue). WT responds earlier (2.1 vs. 3.6 s to double baseline) and stronger, reproducing the aldicarb resistance of *ckr-2/nlp-12* mutants[14]. **e**, Gain decomposition: ACh accumulation (dashed, both genotypes) and peptidergic gain $1 + \alpha m$ (solid).

and feeding-state modulation are out of scope. (vii) Whole-brain calcium imaging[18, 17] is not yet used as a constraint — comparing simulated and measured network activity patterns is the sharpest next test of the model.

What this enables. Because every one of the 292 neurons carries its full channel set, the model doubles as a measurement instrument and a screening device. First, it is an *in vivo* pharmacology predictor: channel mutations or blockers can be ranked *in silico* for their behavioural phenotype before any animal is touched — the knock-out panel (Fig. 3) and the aldicarb and *ckr-2* manipulations (Fig. 4) are proofs of principle, and the logic is the one validated in cardiac safety pharmacology[27], now ported to a nervous system. Second, it is a fully observable experimental platform: the state of every channel of every neuron at every millisecond is accessible, so compensation maps — which channels increase their activity when a homologous channel is removed — are directly computable, a measurement no current experimental technique can perform. Third, it supplies priors for optogenetics: the AVA and RIS stimulations of Fig. 5 were simulated before comparison with the experimental literature, and the same protocol can pre-screen candidate targets and illumination patterns.

Outlook. Three extensions follow naturally: combinatorial knock-out screens to map the boundary of the degenerate region; community-parameterized wireless layers built on the neuropeptidergic connectome[12]; and direct fitting of network dynamics to whole-brain imaging. That command-level stimulation suffices for chemotaxis in a reduced model[39] marks navigation as the natural next behaviour for the channel-detailed network. The model and all analysis code are openly available (Code availability), and the framework is designed so that future experimental constraints enter as data, not as re-engineering.

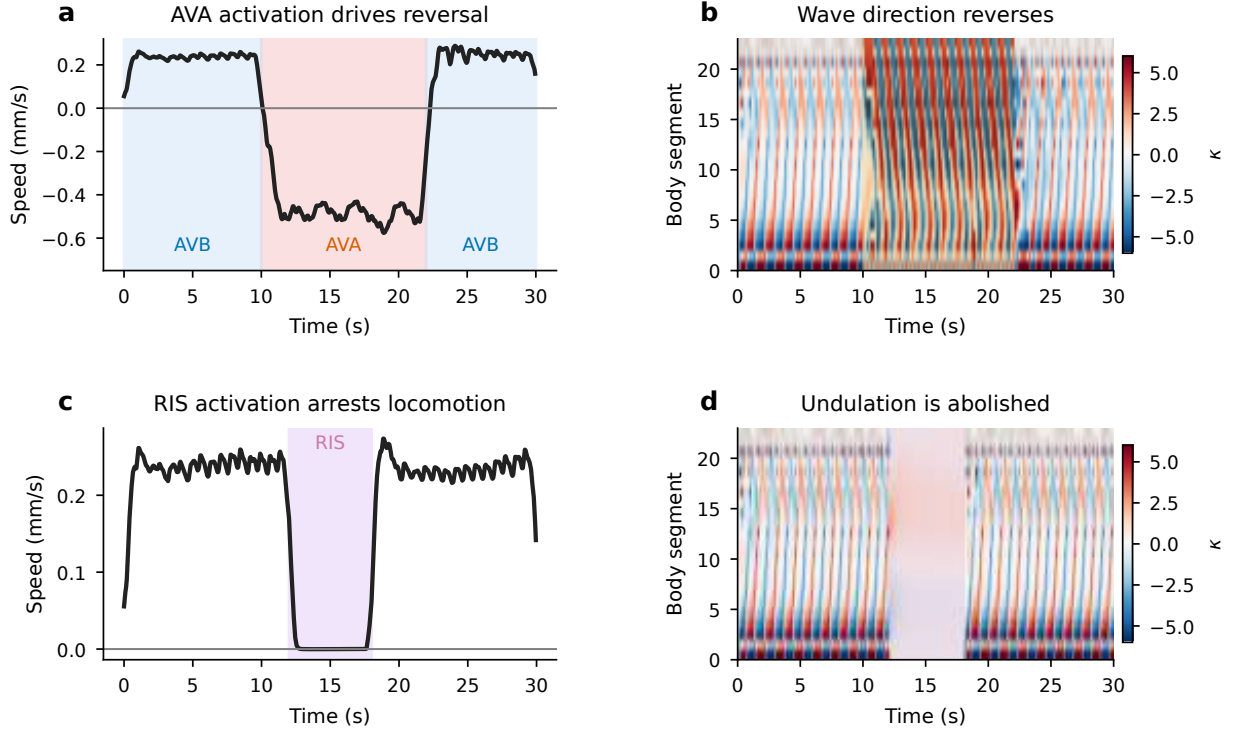


Figure 5. In silico optogenetic perturbations. **a**, Speed time course of the switch protocol: AVB drive (blue shading), then AVA activation 10–22 s (red), then AVB again. Reversal is immediate and sustained; backward speed exceeds forward speed, as in the animal. **b**, Curvature kymograph of the same trial: the wave reverses direction within the AVA window. **c**, RIS activation 12–18 s (magenta) during forward crawling arrests locomotion; recovery on offset. **d**, The undulatory wave is abolished during RIS activation.

4 Methods

4.1 Connectome and weights

The somatic nervous system (292 neurons; pharyngeal network excluded) was assembled from the adjacency data of Cook et al.[2] as updated by Witvliet et al.[3], using the public releases [4]. Chemical synapse $j \rightarrow i$ receives weight $w_{ij} = g_{\text{syn}} n_{ij}$ with n_{ij} the EM contact number; sign from transmitter identity (ACh, Glu excitatory, $E^{\text{rev}} = 0$ mV; GABA inhibitory, $E^{\text{rev}} = -90$ mV). Gap junctions are symmetric ohmic couplings with conductance proportional to contact number.

4.2 Neuron model

Each neuron integrates Eq. (1) with 17 currents and 29 state variables (voltage, 27 gating variables, intracellular Ca^{2+}). Activation/inactivation steady states are sigmoids of voltage; time constants are bell-shaped functions. The channel inventory and gating equations follow the published single-neuron models[33, 34]; provenance, an independent re-derivation of three currents from digitized voltage-clamp data, a substitution test and cell-state anchoring are detailed in §4.3. Resting potentials settle near -67 mV network-wide without any per-cell tuning. Integration uses a fixed-step explicit scheme at $\Delta t = 0.025$ ms, compiled with numba; a 20 s behavioural trial simulates in ≈ 3 min on one CPU core.

4.3 Channel constants: provenance and independent re-derivation

The 17-current inventory and the gating equations (sigmoid steady states, bell-shaped time constants, Ca^{2+} -nanodomain coupling of BK currents) follow the open-source single-neuron models of Nicoletti et al. (ModelDB accession 267187 and its successors)[33, 34], fitted in that work to published *C. elegans* voltage-clamp recordings[28, 29, 30, 31, 32]. On top of this adopted base we ran an independent re-derivation pipeline on the public digitized voltage-clamp datasets of the corresponding channels (Channelpedia CAEEL collection[36]):

Table 5. Perturbation phenotypes: model vs. experiment.

Perturbation	Model phenotype	Experimental counterpart
$\alpha = 0$ (<i>ckr-2</i> KO)	Speed -12 to -24%	<i>nlp-12/ckr-2</i> mutants crawl slower and bend shallower[14, 16]
Aldicarb, WT vs. <i>ckr-2</i> KO	WT faster (2.1 vs. 3.6 s) and stronger (+23%) response	Mutants aldicarb-resistant, potentiation abolished[14]
AVA activation	Reversal, -0.48 mm s^{-1} , wave inverts	Reversal on AVA stimulation[20, 19, 18, 17]
RIS activation	99% arrest, wave abolished	Arrest/quiescence on RIS activation[21, 22]
Single-channel KOs ($\times 4$)	Behaviour change $< 0.3\%$	(Prediction; untested at whole-animal level)

Table 6. Whole-worm models at a glance. Neuron-level dynamics, channel content, weight provenance, rhythm origin, behavioural validation and pharmacological capability of the most developed *C. elegans* simulations, and of the present model.

Model	Neuron dynamics	Channel detail	Weights	Wave origin	Behaviour-level validation	Drug screening
c302 / Open-Worm [9, 10]	Reduced single-compartment	Partial (K^+ , Ca^{2+})	Fitted	Injected pace-maker current required	Qualitative locomotion	None
Kunert et al. 2014 [11]	Linear / minimal point neurons	None	Analytic + optimized	Bifurcation to limit cycle	Sensory-to-motor flow, modes	None
worm-whisperer [8]	Distilled oscillator units	None	Trained to kinematics	Learned	Forward/reverse kinematics	None
BAAIWorm [37]	Multi-compartment (5 fitted, group-generalized)	14 fitted classes	Weights + polarities trained on calcium correlations	Trained network drives body	Zigzag foraging, structural lesions	None
Lee 2026 [39]	c302 reduced, atlas-fitted	None	Atlas-fitted; evolved stimulation policy	Learned command drive	Chemotaxis (91%), maze navigation	None
This work	Conductance-based point neurons, 29 ODEs/cell	17 currents per cell; anchored + re-derived constants	None trained (4 body + 1 gain)	Emergent: anatomy + body loop	Kinematics, channel knock-outs, aldicarb, optogenetics	Yes (Figs. 3, 4)

steady-state activation and inactivation were refit by least-squares Boltzmann functions with R^2 quality gates and bootstrap confidence intervals; activation curves were obtained from chord conductance rather than tail currents where the latter carry capacitive artefacts; reversal potentials were taken from the measured I - V zero-crossing with outlier exclusion; and total-conductance fits were converted to gate level for the exact gating powers of the model. Table 7 reports the re-derived constants against the adopted ones. Midpoint discrepancies of 10–17 mV appear, in both directions — consistent with the differing data sources and fitting conventions — so the frozen model retains the published single-cell-fit values and our re-derived sets serve as independent parameter bounds. Their behavioural (ir)relevance was then tested directly: substituting all three re-derived sets into the identical calibrated model (same four body-layer parameters, no re-fitting) changes forward speed by +0.008%, undulation frequency by $< 0.01\%$ and curvature amplitude by $< 0.01\%$ (maximal trajectory divergence $5.6 \mu\text{m}$ over 20 s), while the AVB command drive shifts by -2.2% . Single-channel constant uncertainty of this magnitude therefore does not propagate to behaviour, in line with the network-level degeneracy of §2.3. Cell-class state anchors were re-set to direct measurements rather than inherited from the source fits: leak reversal per class was solved (bisection) so that the isolated resting potential matches published recordings[34, 30]; the graded-release midpoint sits at the isolated equilibrium of each class plus a fixed offset (-10 mV); reversal potentials follow ionic conditions; and the CaV2 (*unc-2*) conductance in VA/DA motor neurons — an underdetermined direction of the source voltage-clamp fit — was restored and scaled by a single global factor (0.4 nS).

Table 7. Independently re-derived channel constants vs. adopted values. Re-derived from digitized public voltage-clamp data (Channelpedia CAEEL collection[36]) by least-squares Boltzmann fitting (R^2 in parentheses; 95% bootstrap CI of midpoints within ± 2 mV). Adopted values are the published single-cell-fit constants [33, 34] used in the frozen model († converted to gate level, m^3). Substituting the three re-derived sets into the calibrated model leaves behaviour unchanged (§4.3).

Current	Constant	Re-derived (this work)	Adopted[33, 34]
SHK-1 (shak)	act. $V_{1/2}$, k (mV)	+3.8, 7.18 (0.998)	+20.4, 7.7
	inact. $V_{1/2}$, k (mV)	-18.9, 4.29 (1.00)	-7.0, 5.8
SHL-1 (shal) [†]	act. $V_{1/2}$, k (mV)	-4.0, 24.6 (0.993)	-6.8, 14.1
	inact. $V_{1/2}$, k (mV)	-35.2, 6.94 (0.999)	-51.1, 8.3
EGL-19	act. $V_{1/2}$, k (mV)	+8.3, 8.34 (1.00)	-4.4, 7.5
	E_{rev} (mV)	+55.1	+60

4.4 Muscles, body and feedback

Muscle rows, preferred-curvature actuation, the 24-segment rod and stretch-receptor feedback follow the neuromechanical formulation of Boyle and Cohen[5, 6], reusing the open-source Python implementation of Chung and Kim (worm-whisperer)[8, 38]. The forward/backward motor programs are gated by depolarization of AVB/AVA command neurons relative to rest (threshold 3 mV), with RIS veto and SMD/RIV head-steering biases; body drag $C_n = 40$; motor time step 5 ms. Preferred curvature $\kappa_0 = \kappa_{\text{max}}(A_D - A_V)$ per segment.

4.5 Calibration

Only the four body-layer parameters of Table 2 were fitted, by minimizing $L = \sum_i [(x_i - x_i^*)/x_i^*]^2$ over targets $\{v = 0.21 \text{ mm/s}, f = 0.58 \text{ Hz}, \kappa_{\text{amp}} \approx 3.5 \text{ mm}^{-1}, k = 1.83\}$ on a 54-point grid ($\kappa_{\text{max}} \times G_{\text{SR}} \times s_{\text{SR}} \times I_{\text{AVB}}$) evaluated in the body-layer-only loop; the winner ($L = 0.066$) was re-verified in the full neural loop (values in Table 3). No neural-layer quantity was fitted at any stage.

4.6 Peptidergic layer and perturbations

The DVA→NLP-12→CKR-2 axis follows Eq. (2) with $\tau_m = 3 \text{ s}$, $\kappa_{\text{ref}} = 3 \text{ mm}^{-1}$, $\alpha = 0.25$ unless stated; results were unchanged within $\pm 3\%$ for $\tau_m \in [0.5, 10] \text{ s}$. Channel knock-outs set the corresponding conductance to zero in all cells before the 2 s baseline equilibration. Aldicarb follows Eq. (3) with $\beta = 1$, $\tau_a = 5 \text{ s}$. Photomimetic activation injects 50 pA into the named neuron class during the stated windows.

4.7 Metrics

Speed: axial centre-of-mass velocity along the instantaneous head-tail axis. Frequency: dominant FFT component of mid-body curvature. Wave number: fitted spatial phase gradient of the curvature wave. Amplitude: mean absolute curvature peak. All metrics are computed after a 1 s settling window.

4.8 Code and data availability

Simulation code, the connectome tables, calibrated parameters and figure scripts are available at <https://github.com/lihuanlz/worm-fullchannel> (release v1.0); the commands documented there regenerate every trajectory underlying the figures. Body-layer components are derivatives of worm-whisperer (<https://github.com/kairess/worm-whisperer>)[8]; the remainder is original code. Supplementary Movie S1 shows the calibrated forward-crawling trial.

Competing interests

The author declares no competing interests.

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