Discovering and sharing models with ModelDB

Using ModelDB and ModelView

Twitter: @modeldb

ModelDB Login Browse Analysis More Search

Amyloid beta (IA block) effects on a model CA1 pyramidal cell (Morse et al. 2010)

Overview | Files | ModelView | Citations

The model simulations provide evidence oblique dendrites in CA1 pyramidal neurons are susceptible to hyper-excitability by amyloid beta block of the transient K+ channel, IA. See paper for details.

Model Type: Neuron or other electrically excitable cell

Cell Type(s): Hippocampus CA1 pyramidal GLU cell

Currents: I Na,t; I L high threshold; I N; I T low threshold; I A; I K; I h; I K,Ca

Model Concept(s): Dendritic Action Potentials; Active Dendrites; Detailed Neuronal Models; Pathophysiology; Aging/Alzheimer`s

Simulation Environment: NEURON

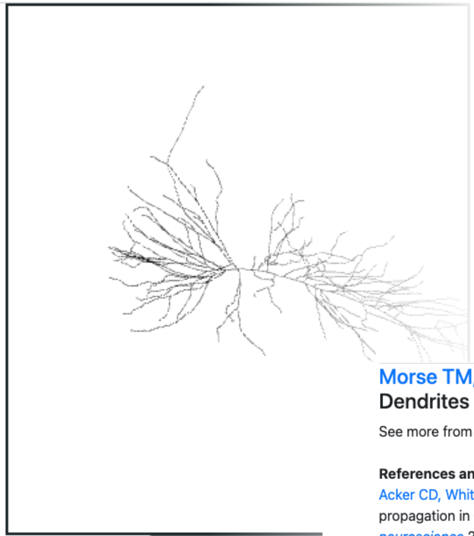
Implementer(s): Carnevale, Ted [Ted.Carnevale at Yale.edu]; Morse, Tom [Tom.Morse at Yale.edu]

References:

Morse TM, Carnevale NT, Mutalik PG, Migliore M, Shepherd GM. (2010). Abnormal Excitability of Oblique Dendrites Implicated in Early Alzheimer's: A Computational Study. *Frontiers in neural circuits* 4 [PubMed]

```
This is the readme for a model used in the paper

Morse TM, Carnevale NT, Mutalik PG, Migliore M, Shepherd GM (2010)
Abnormal excitability of oblique dendrites implicated in early
```



Show Diameter

Simulation Platform

View on GitHub

```
from neuron import h, gui, rxd

# initial values for concentrations, voltage; fixed value for temperature
h.nai0_na_ion = 4
h.nao0_na_ion = 151
h.v_init = -75
h.celsius = 30


class FleidervishNeuron:
    """Neuron of Fleidervish et al, 2010 without Na+ diffusion or accumulation"""
    def __init__(self, _id=None, x=0, y=0, z=0):
        '''Instantiate FleidervishNeuron.

        Parameters:
            x, y, z -- position offset
            _id -- cell id
        '''
        self._x, self._y, self._z = x, y, z
        self._id = _id
        self._setup_morphology()
        self._insert_mechanisms()
        self._discretize_model()
        self._set_mechanism_parameters()

    def __str__(self):
        if self._id is None:
            return 'FleidervishNeuron'
        return 'FleidervishNeuron[{}]'.format(self._id)

    def _setup_morphology(self):
        self._create_sections()
```

Morse TM, Carnevale NT, Mutalik PG, Migliore M, Shepherd GM. (2010). Abnormal Excitability of Oblique Dendrites Implicated in Early Alzheimer's: A Computational Study. *Frontiers in neural circuits* 4 [PubMed]

See more from authors: Morse TM · Carnevale NT · Mutalik PG · Migliore M · Shepherd GM

References and models cited by this paper

Acker CD, White JA. (2007). Roles of IA and morphology in action potential propagation in CA1 pyramidal cell dendrites. *Journal of computational neuroscience* 23 [PubMed]

- Roles of I(A) and morphology in AP prop. in CA1 pyramidal cell dendrites (Acker and White 2007) [Model]

Anderton BH et al. (1998). Dendritic changes in Alzheimer's disease and factors that may underlie these changes. *Progress in neurobiology* 55 [PubMed]

Andrásfalvy BK, Makara JK, Johnston D, Magee JC. (2008). Altered synaptic and non-synaptic properties of CA1 pyramidal neurons in Kv4.2 knockout mice. *The Journal of physiology* 586 [PubMed]

Canepari M, Djurisic M, Zecevic D. (2007). Dendritic signals from rat hippocampal CA1 pyramidal neurons during coincident pre- and post-synaptic activity: a combined voltage- and calcium-imaging study. *The Journal of physiology* 580 [PubMed]

Chen C. (2005). beta-Amyloid increases dendritic Ca2+ influx by inhibiting the A-type K+ current in hippocampal CA1 pyramidal neurons. *Biochemical and biophysical research communications* 338 [PubMed]

Colbert CM, Magee JC, Hoffman DA, Johnston D. (1997). Slow recovery from inactivation of Na+ channels underlies the activity-dependent

References and models that cite this paper

Culmone V, Migliore M. (2012). Progressive effect of beta amyloid peptides accumulation on CA1 pyramidal neurons: a model study suggesting possible treatments. *Frontiers in computational neuroscience* 6 [PubMed]

- CA1 pyramidal neurons: effects of Alzheimer (Culmone and Migliore 2012) [Model]

McDougal RA, Dalal I, Morse TM, Shepherd GM. (2019). Automated Metadata Suggestion During Repository Submission. *Neuroinformatics* 17 [PubMed]

- Automated metadata suggester (McDougal et al 2018) [Model]

McDougal RA, Morse TM, Hines ML, Shepherd GM. (2015). ModelView for ModelDB: Online Presentation of Model Structure. *Neuroinformatics* 13 [PubMed]

- ModelView: online structural analysis of computational models (McDougal et al. 2015) [Model]

Romani A et al. (2013). Computational modeling of the effects of amyloid-beta on release probability at hippocampal synapses. *Frontiers in computational neuroscience* 7 [PubMed]

- Amyloid-beta effects on release probability and integration at CA3-

modeldb.science

McDougal, R. A., Morse, T. M., Carnevale, T., Marenco, L., Wang, R., Migliore, M., ... & Hines, M. L. (2017). Twenty years of ModelDB and beyond: building essential modeling tools for the future of neuroscience. *Journal of computational neuroscience*, *42*(1), 1-10. doi:10.1007/s10827-016-0623-7

On reproducibility

"Non-reproducible single occurrences are of no significance to science."
- Karl Popper in *The logic of scientific discovery*. 1959.

What is needed for a model to be reproducible?

Model

- an approximation of the system of interest
  e.g. a model organism or a complete statement of the properties of the model in mathematical or computable form

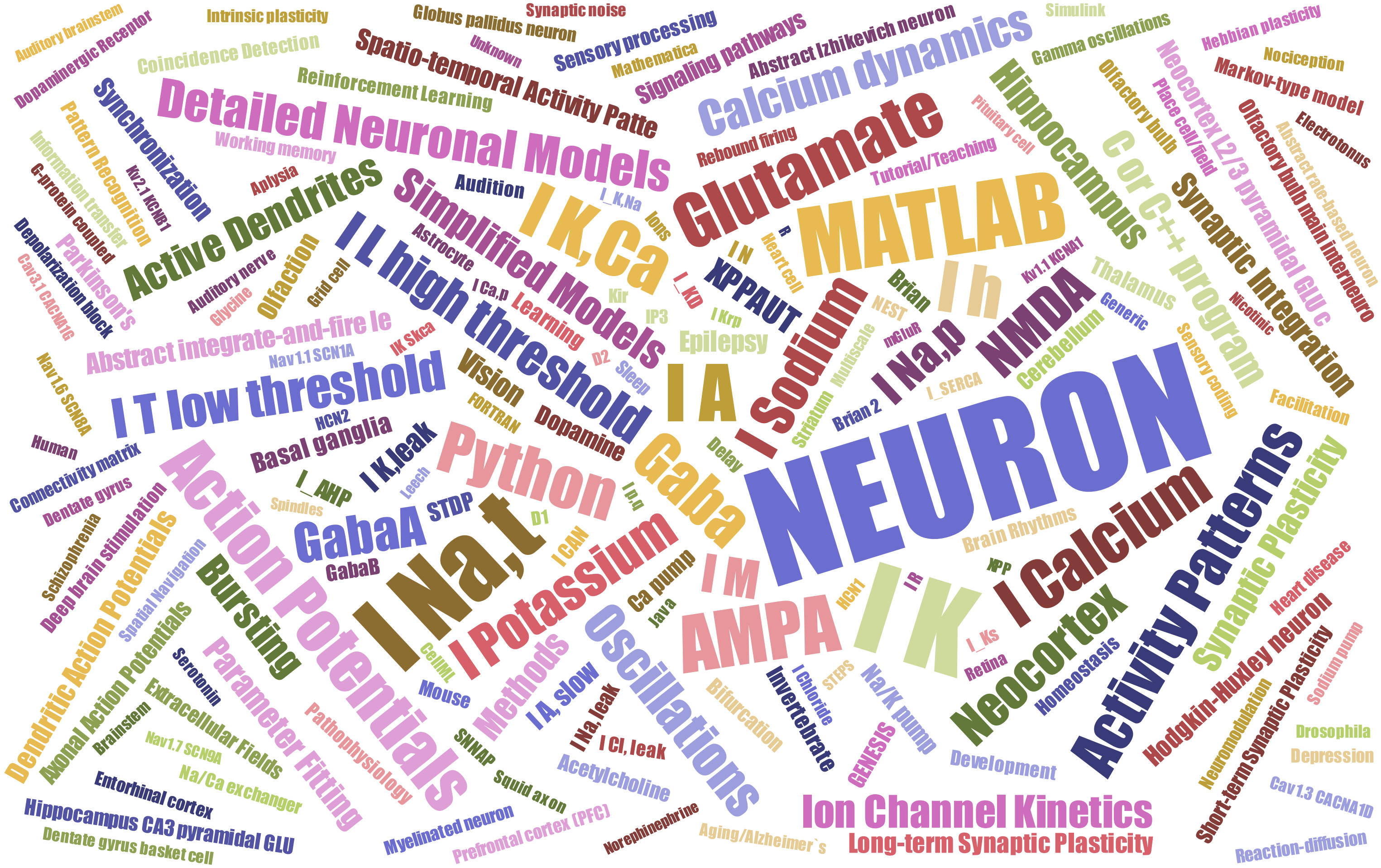
Experimental protocol

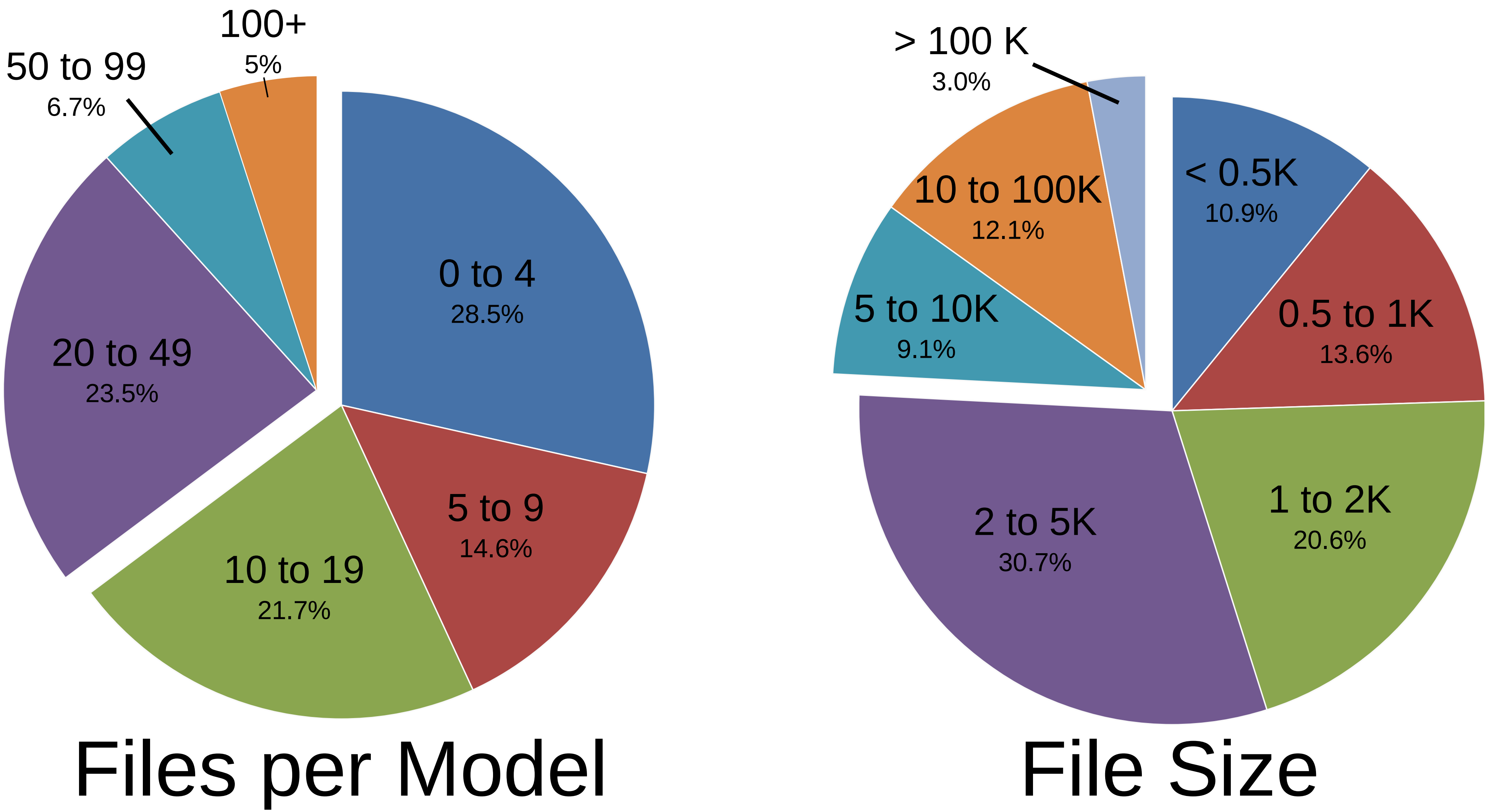
- what was done with the model to produce the data

Science builds upon previous work; in order to do that, the previous work needs to be reproducible.

What is in ModelDB?

- Models for:
  - 181 cell types
  - 21+ species
  - 63 ion channels, pumps, etc.
  - 214 topics (Alzheimer's, STDP, etc.)
  - 25+ mammalian brain regions
- 1808 published models from 110 simulators/programming languages
- 815 NEURON models.
- 546 network models.





Distributions from ModelDB, Fall 2013. A model was counted as having 0 files if it was not hosted on ModelDB.

Only reuse what you understand

The easiest way to replicate someone else's results – a first step toward building on them – is to get their model code from a repository such as ModelDB.

But beware:

- They may be solving a different problem than you (with respect to species, temperature, age, etc).
- Their code may have bugs.

To reduce the risk of problems:

- Read the associated paper.
- Compare the model and results to other similar models.
- Examine the model with ModelView and/or psection.
- Test ion channels individually.
- Collaborate with an experimentalist.

Amyloid beta (IA block) effects on a model CA1 pyramidal cell (Morse et al. 2010)

Overview | Files | ModelView | Citations

CA1_abeta / cal2.mod

```
TITLE l-calcium channel
: l-type calcium channel


UNITS {
        (mA) = (milliamp)
        (mV) = (millivolt)

        FARADAY = 96520 (coul)
        R = 8.3134 (joule/degC)
        KTOMV = .0853 (mV/degC)
}

PARAMETER {
        v (mV)
        celsius         (degC)
        gcalbar=.003 (mho/cm2)
        ki=.001 (mM)
        cai = 50.e-6 (mM)
        cao = 2 (mM)
        q10 = 5
        mmin=0.2
        tfa = 1
        a0m =0.1
        zetam = 2
        vhalfm = 4
        gmm=0.1
        ggk
}

NEURON {
        SUFFIX cal
```

This mechanism is potentially temperature dependent.

Reused in 14 other models:

- CA3 pyramidal neuron: firing properties (Hemond et al. 2008)
  - ca3b/cal2.mod
- CA3 pyramidal neuron (Safiulina et al. 2010)
  - develop/cal2.mod
- A model of unitary responses from A/C and PP synapses in CA3 pyramidal cells (Baker et al. 2010)
  - ca3-synresp/cal2.mod
- CA1 pyramidal neuron: effects of R213Q and R312W Kv7.2 mutations (Miceli et al. 2013)
  - kv72-R213QW-mutations/cal2.mod
- Distinct current modules shape cellular dynamics in model neurons (Alturki et al 2016)
  - AlturkiEtAl2016/1_Hemond/Original/cal2.mod
- Firing patterns of CA3 hippocampal neurons (Soldado-Magraner et al. 2019)

Amyloid beta (IA block) effects on a model CA1 pyramidal cell (Morse et al. 2010)

Overview | Files | ModelView | Citations

CA1_abeta / kaprox.mod

```
TITLE K-A channel from Klee Ficker and Heinemann
: modified to account for Dax A Current --- M.Migliore Jun 1997
: modified to be used with cvode  M.Migliore 2001

UNITS {
        (mA) = (milliamp)
        (mV) = (millivolt)
}

PARAMETER {
        v (mV)
        celsius         (degC)
        gkabar=.008 (mho/cm2)
        vhalfn=11   (mV)
        vhalfl=-56   (mV)
        a0l=0.05      (/ms)
        a0n=0.05    (/ms)
        zetan=-1.5    (1)
        zetal=3    (1)
        gmn=0.55   (1)
        gml=1   (1)
        lmin=2  (mS)
        nmin=0.1  (mS)
        pw=-1    (1)
        tq=-40
        qq=5
        q10=5
        qtl=1
        ek
}

NEURON {
```

This mechanism is potentially temperature dependent.

View Ion Channel Genealogy entry

Not reused in any other models.

The following explanation has been generated automatically by AI and may contain errors.

Biological Basis of the Computational Model

The code provided is a computational model of the K-A (A-type potassium) channel, which is a specific type of potassium ion channel known for its role in regulating neuronal excitability. The model is designed for integration into neural simulations, such as those used in the NEURON simulation environment, to study the dynamics of ionic currents in neurons.

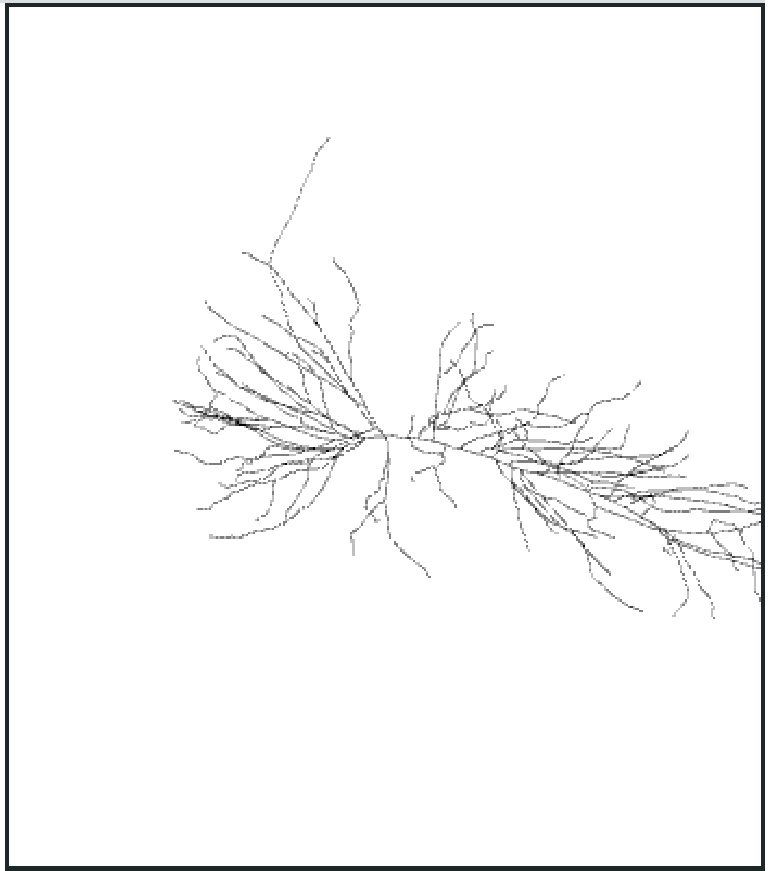
Key Biological Concepts

Ion Channels and Ionic Currents

Amyloid beta (IA block) effects on a model CA1 pyramidal cell (Morse et al. 2010)

Overview Files ModelView Citations

The model simulations provide evidence oblique dendrites in CA1 pyramidal neurons are susceptible to hyper-excitability by amyloid beta block of the transient K+ channel, IA. See paper for details.



Show Diameter

Model Type: Neuron or other electrically excitable cell

Cell Type(s): Hippocampus CA1 pyramidal GLU cell

Currents: I Na,t; I L high threshold; I N; I T low threshold; I A; I K; I h; I K,Ca

Model Concept(s): Dendritic Action Potentials; Active Dendrites; Detailed Neuronal Models; Pathophysiology; Aging/Alzheimer`s

Simulation Environment: NEURON

Implementer(s): Carnevale, Ted [Ted.Carnevale at Yale.edu]; Morse, Tom [Tom.Morse at Yale.edu]

References:

Morse TM, Carnevale NT, Mutalik PG, Migliore M, Shepherd GM. (2010). Abnormal Excitability of Oblique Dendrites Implicated in Early Alzheimer's: A Computational Study. *Frontiers in neural circuits* 4 [PubMed]


```
This is the readme for a model used in the paper

Morse TM, Carnevale NT, Mutalik PG, Migliore M, Shepherd GM (2010)
Abnormal excitability of oblique dendrites implicated in early
```

Simulation Platform

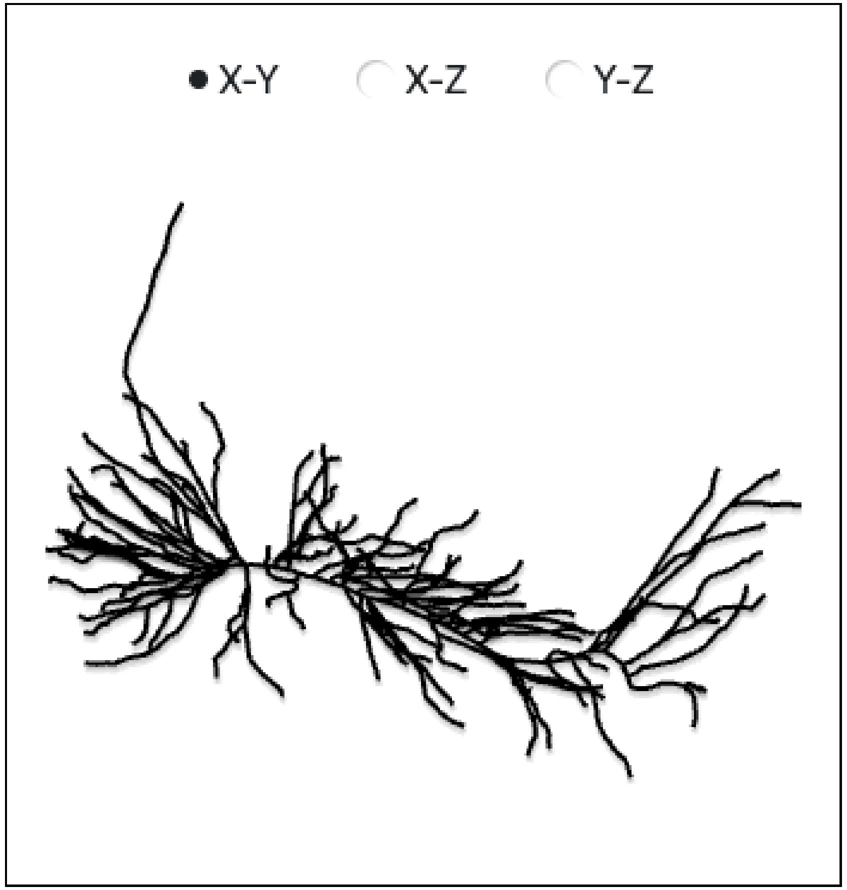
View on GitHub

Amyloid beta (IA block) effects on a model CA1 pyramidal cell (Morse et al. 2010)

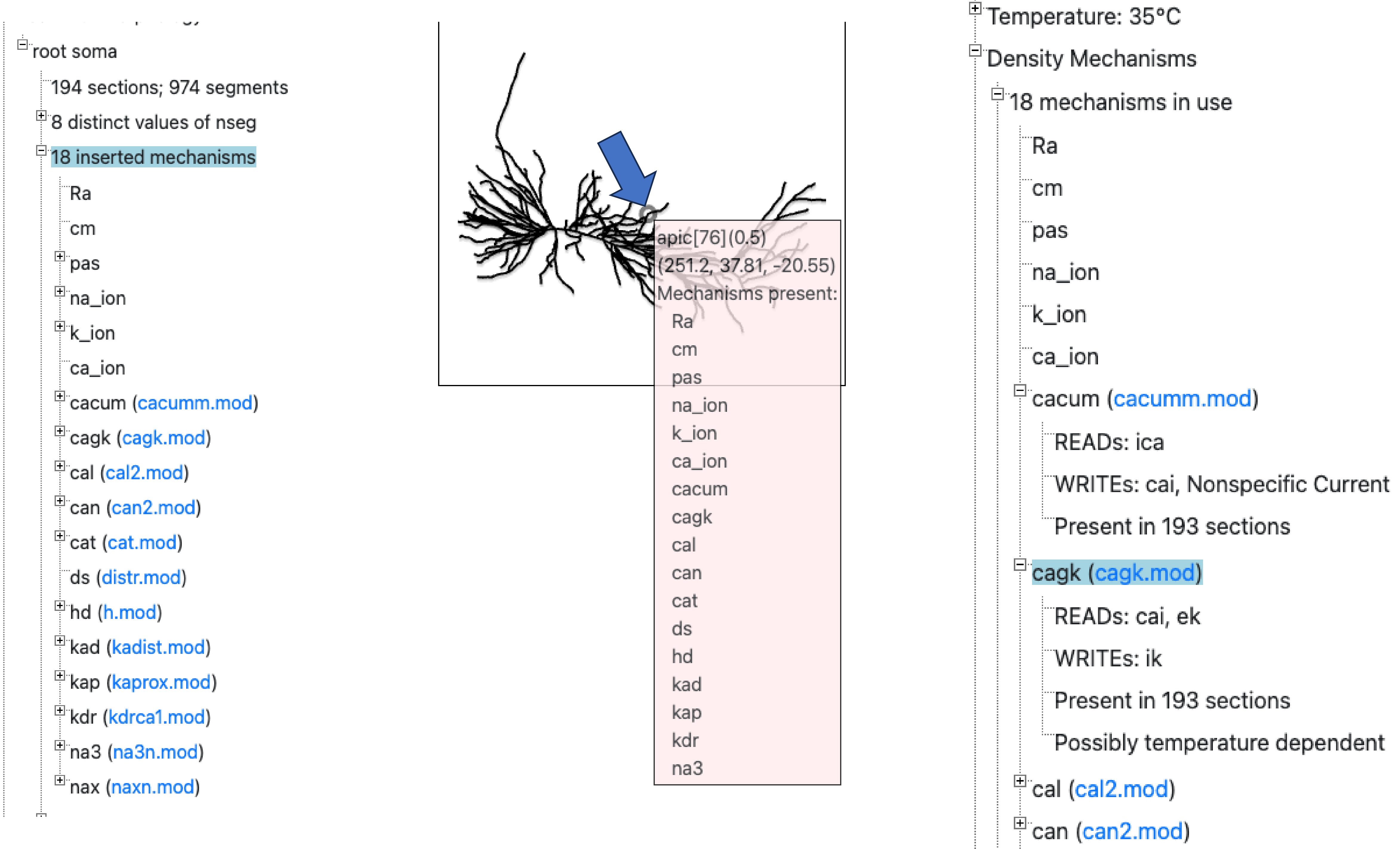
Overview | Files | ModelView | Citations

Figure 1, 2

- 194 sections; 974 segments
- 1 cell with morphology
  - root soma
- 0 artificial cells
- 0 NetCon objects
- 0 LinearMechanism objects
- Temperature: 35°C
- Density Mechanisms
- 1 point processes (0 can receive events) of 1 base classes
- 7 files shared with other ModelDB models
- References



McDougal et al, *Neuroinformatics* 2015

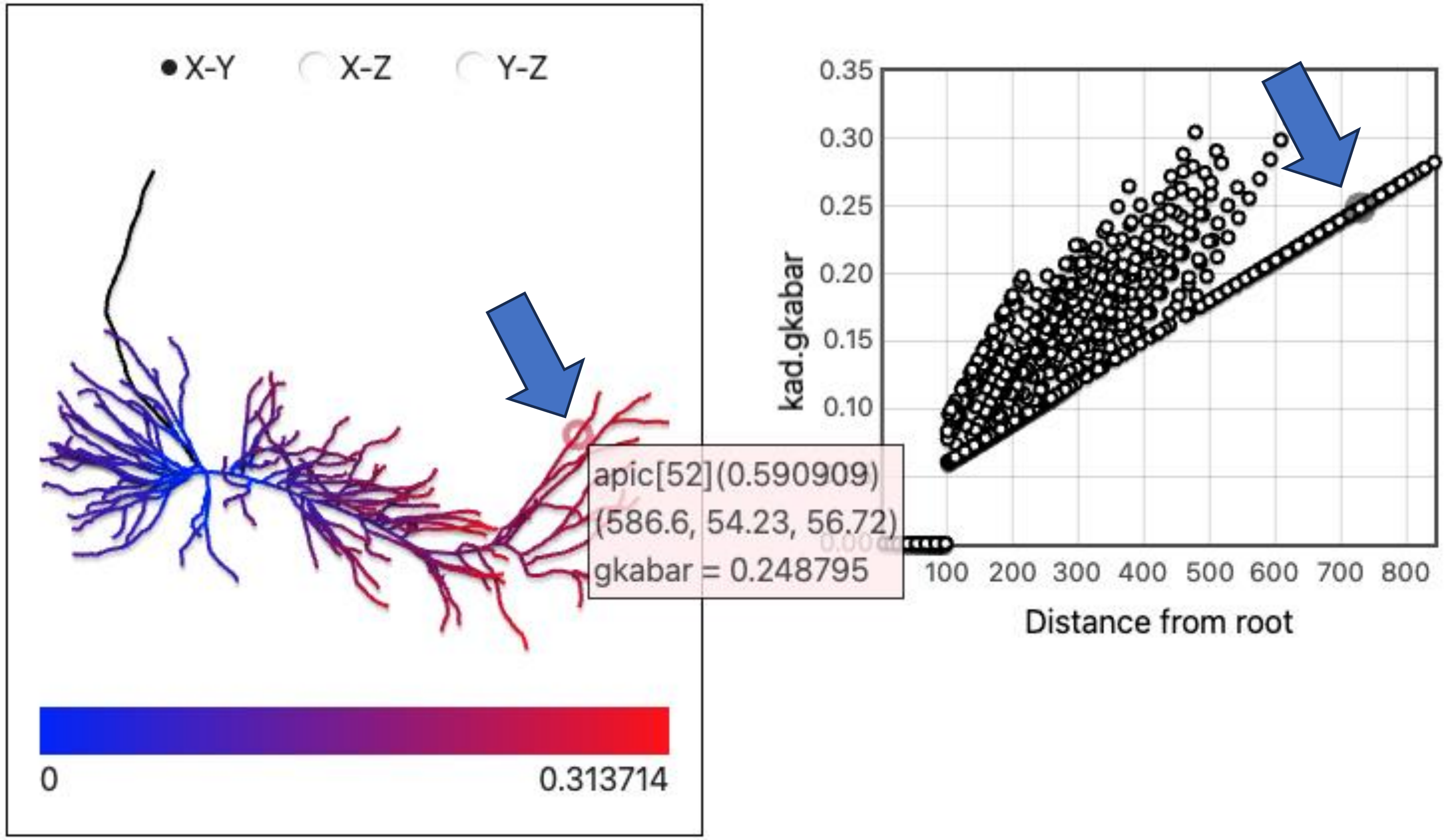


root soma
194 sections; 974 segments
8 distinct values of nseg
18 inserted mechanisms
Ra
cm
pas
na_ion
k_ion
ca_ion
cacum (cacumm.mod)
cagk (cagk.mod)
cal (cal2.mod)
can (can2.mod)
cat (cat.mod)
ds (distr.mod)
hd (h.mod)
kad (kadist.mod)
kap (kaprox.mod)
kdr (kdrca1.mod)
na3 (na3n.mod)
nax (naxn.mod)
apic[76](0.5)
(251.2, 37.81, -20.55)
Mechanisms present:
Ra
cm
pas
na_ion
k_ion
ca_ion
cacum
cagk
cal
can
cat
ds
hd
kad
kap
kdr
na3
Temperature: 35°C
Density Mechanisms
18 mechanisms in use
Ra
cm
pas
na_ion
k_ion
ca_ion
cacum (cacumm.mod)
READs: ica
WRITEs: cai, Nonspecific Current
Present in 193 sections
cagk (cagk.mod)
READs: cai, ek
WRITEs: ik
Present in 193 sections
Possibly temperature dependent
cal (cal2.mod)
can (can2.mod)

Overview Files ModelView Citations

Figure 1, 2

- 194 sections; 974 segments
- 1 cell with morphology
  - root soma
    - 194 sections; 974 segments
    - 8 distinct values of nseg
    - 18 inserted mechanisms
      - Ra
      - cm
      - pas
      - na_ion
      - k_ion
      - ca_ion
      - cacum (cacumm.mod)
      - cagk (cagk.mod)
      - cal (cal2.mod)
      - can (can2.mod)
      - cat (cat.mod)
      - ds (distr.mod)
      - hd (h.mod)
      - kad (kadist.mod)
        - gkabar



- References
  - Paper in Front. Neural Circuits
  - ModelDB Entry
  - Run Protocol
    - Compiling
      - cd CA1_abeta
      - nrnivmodl
    - Launching NEURON
      - nrngui -python
    - Running
      - from neuron import h, gui
      - h.load_file("mosinit.hoc")
      - h.fig1and2()

Amyloid beta (IA block) effects on a model CA1 pyramidal cell (Morse et al. 2010)

Overview | Files | ModelView | Citations

CA1_abeta / kadist.mod

```
TITLE K-A channel from Klee Ficker and Heinemann
: modified to account for Dax A Current ----------
: M.Migliore Jun 1997

UNITS {
        (mA) = (milliamp)
        (mV) = (millivolt)
}

PARAMETER {
        celsius
        v (mV)
        gkabar=.008 (mho/cm2)
        vhalfn=-1   (mV)
        vhalfl=-56   (mV)
        a0l=0.05      (/ms)
        a0n=.1    (/ms)
        zetan=-1.8    (1)
        zetal=3    (1)
        gmn=0.39   (1)
        gml=1   (1)
        lmin=2  (mS)
        nmin=0.2  (mS)
        pw=-1    (1)
        tq=-40
        qq=5
        q10=5
        qtl=1
        ek
}


NEURON {
        SUFFIX kad
        USEION k READ ek WRITE ik
```

This mechanism is potentially temperature dependent.

View Ion Channel Genealogy entry

Not reused in any other models.

General data

- **ICG id:** 2471
- **ModelDB id:** 87284
- **Reference:** *Morse TM, Carnevale NT, Mutalik PG, Migliore M, Shepherd GM (2010)*: Abnormal Excitability of Oblique Dendrites Implicated in Early Alzheimer's: A Computational Study.

Metadata classes

- **Animal Model:** rat
- **Brain Area:** hippocampus, CA1
- **Neuron Region:** unspecified
- **Neuron Type:** pyramidal cell
- **Runtime Q:** Q3
- **Subtype:** A

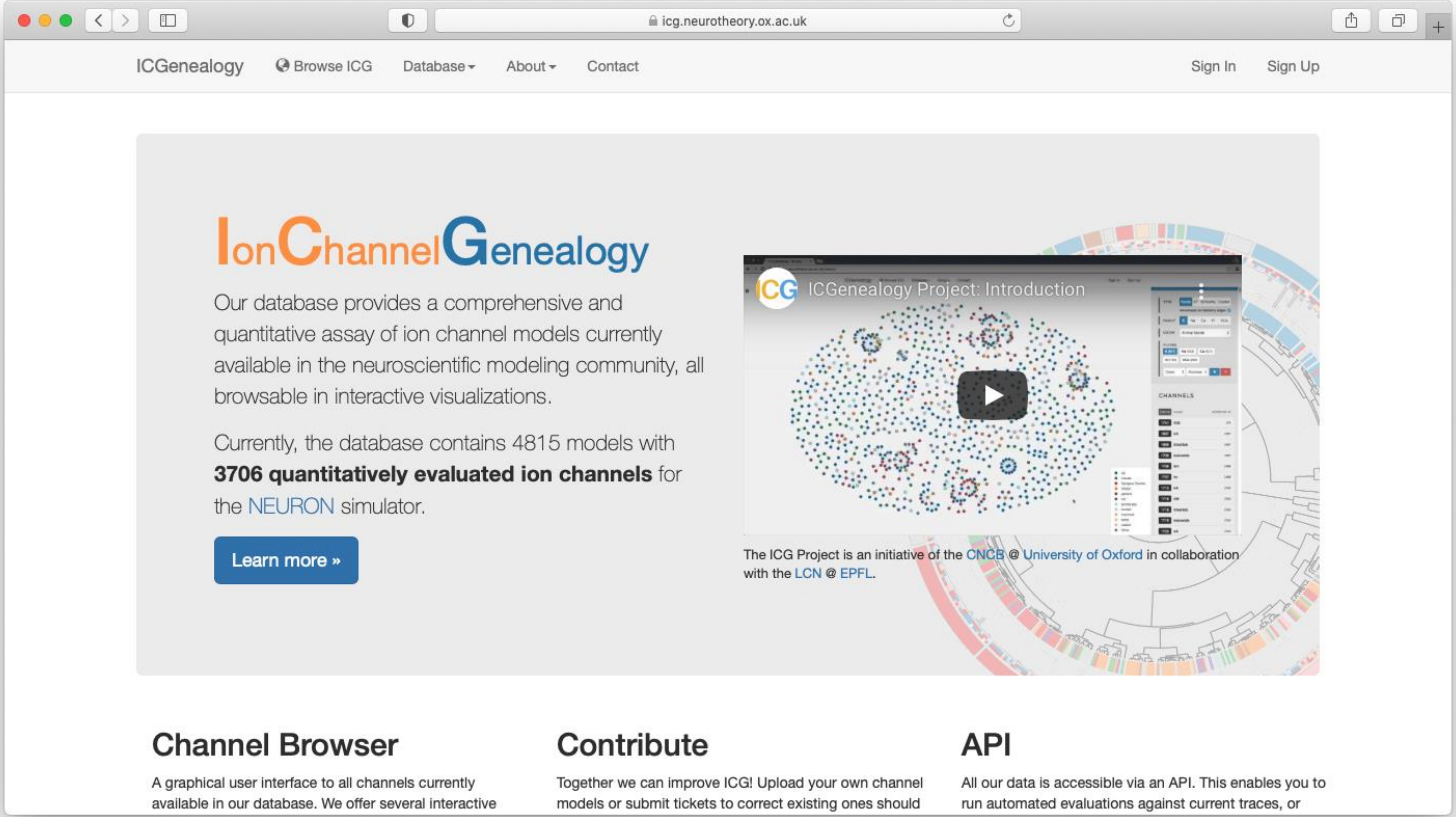
Metadata generic

- **Age:** 7-14 weeks old.
- **Authors:** Migliore M.
- **Comments:** K-a channel from migliore et al. (2005), model no. 55035, with no changes to kinetics. Animal model taken from chen (2005) which is used to constrain model. Channel kinetics from previous studies on hippocampal pyramidal neuron (migliore et al. 2005)
- **Runtime:** 7.979
- **Temperature:** Model has temperature dependence, q10=5.

Ion Channel Genealogy: ion channel metadata

When viewing many mod files describing an ion channel, a “View Ion Channel Genealogy”button appears. Clicking this button loads the corresponding page of the Ion Channel Genealogy which has derived information about the underlying data, etc) and response curves.

See Podlaski et al., 2017. doi:10.7554/eLife.22152.001


icg.neurotheory.ox.ac.uk
ICGenealogy
Browse ICG
Database
About
Contact
Sign In
Sign Up
IonChannelGenealogy
Our database provides a comprehensive and quantitative assay of ion channel models currently available in the neuroscientific modeling community, all browsable in interactive visualizations.
Currently, the database contains 4815 models with 3706 quantitatively evaluated ion channels for the NEURON simulator.
Learn more »
ICGenealogy Project: Introduction
The ICG Project is an initiative of the CNCB @ University of Oxford in collaboration with the LCN @ EPFL.
Channel Browser
A graphical user interface to all channels currently available in our database. We offer several interactive
Contribute
Together we can improve ICG! Upload your own channel models or submit tickets to correct existing ones should
API
All our data is accessible via an API. This enables you to run automated evaluations against current traces, or

ModelDB for literature review

- Every model can be considered a review of the literature.
- ModelDB reveals what has been modeled in each cell type.
- Comparing models shows what mechanisms are considered critical by the community.

Hippocampus CA1 Pyramidal Cells

IA

- 47 models: 2796, 7386, 9769, 19696, 20212, 32992, 44050, 55035, . . .

IK,Ca

- 11 models: 20212, 87284, 115356, 119266, 123927, 125152, . . .

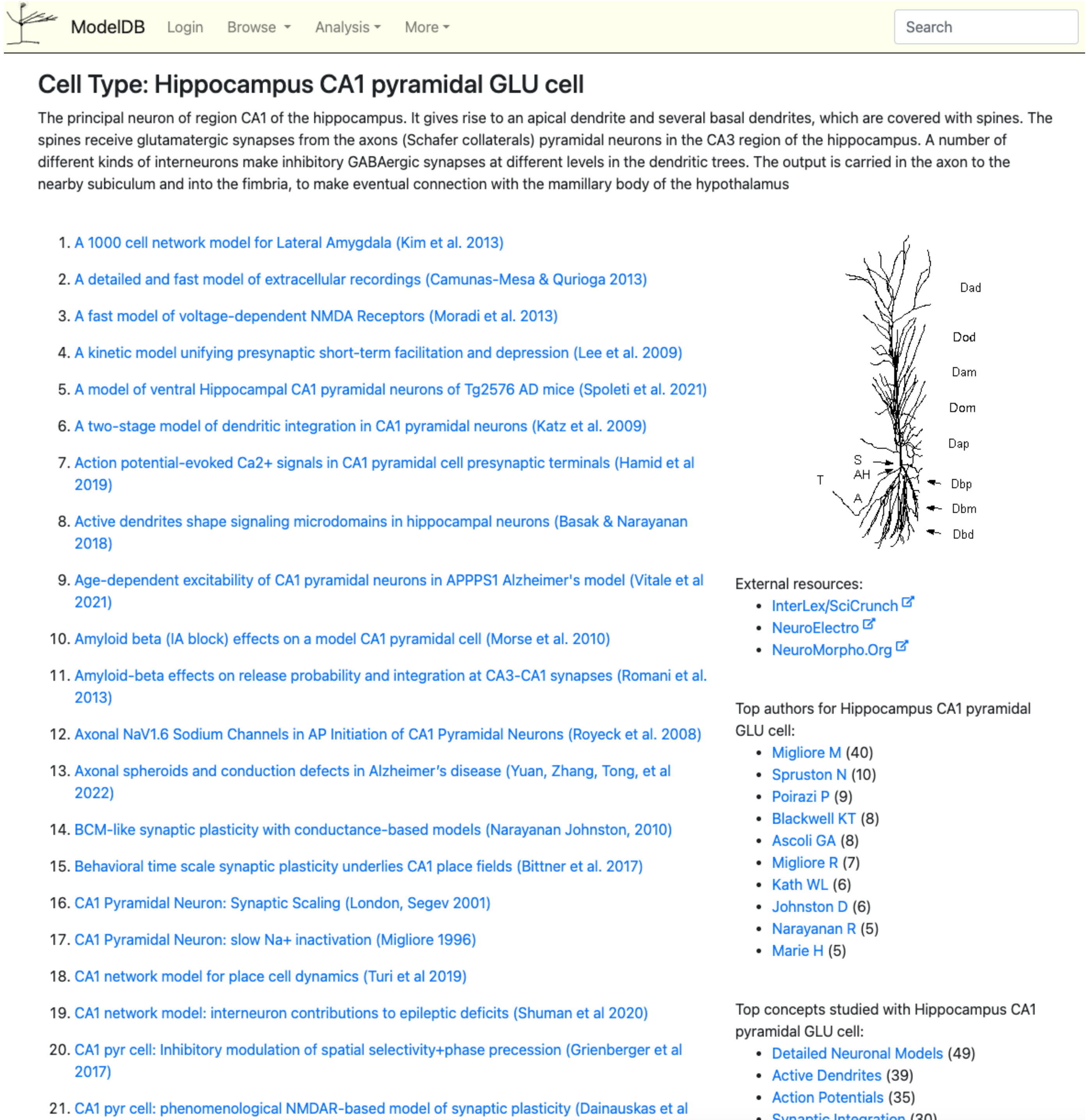
IM

- 16 models: 2937, 20212, 66268, 112546, 115356, 118986, 119266, . . .

26 currents, 6 transmitters, 10 receptors

ModelDB for literature review

Side panel includes:
- Links to resources
- Top authors
- Top concepts
- Top neurons
- Top currents
- Top references


ModelDB Login Browse Analysis More Search

Cell Type: Hippocampus CA1 pyramidal GLU cell

The principal neuron of region CA1 of the hippocampus. It gives rise to an apical dendrite and several basal dendrites, which are covered with spines. The spines receive glutamatergic synapses from the axons (Schafer collaterals) pyramidal neurons in the CA3 region of the hippocampus. A number of different kinds of interneurons make inhibitory GABAergic synapses at different levels in the dendritic trees. The output is carried in the axon to the nearby subiculum and into the fimbria, to make eventual connection with the mamillary body of the hypothalamus

1. A 1000 cell network model for Lateral Amygdala (Kim et al. 2013)
2. A detailed and fast model of extracellular recordings (Camunas-Mesa & Qurioga 2013)
3. A fast model of voltage-dependent NMDA Receptors (Moradi et al. 2013)
4. A kinetic model unifying presynaptic short-term facilitation and depression (Lee et al. 2009)
5. A model of ventral Hippocampal CA1 pyramidal neurons of Tg2576 AD mice (Spoleti et al. 2021)
6. A two-stage model of dendritic integration in CA1 pyramidal neurons (Katz et al. 2009)
7. Action potential-evoked Ca2+ signals in CA1 pyramidal cell presynaptic terminals (Hamid et al 2019)
8. Active dendrites shape signaling microdomains in hippocampal neurons (Basak & Narayanan 2018)
9. Age-dependent excitability of CA1 pyramidal neurons in APPPS1 Alzheimer's model (Vitale et al 2021)
10. Amyloid beta (IA block) effects on a model CA1 pyramidal cell (Morse et al. 2010)
11. Amyloid-beta effects on release probability and integration at CA3-CA1 synapses (Romani et al. 2013)
12. Axonal NaV1.6 Sodium Channels in AP Initiation of CA1 Pyramidal Neurons (Royeck et al. 2008)
13. Axonal spheroids and conduction defects in Alzheimer's disease (Yuan, Zhang, Tong, et al 2022)
14. BCM-like synaptic plasticity with conductance-based models (Narayanan Johnston, 2010)
15. Behavioral time scale synaptic plasticity underlies CA1 place fields (Bittner et al. 2017)
16. CA1 Pyramidal Neuron: Synaptic Scaling (London, Segev 2001)
17. CA1 Pyramidal Neuron: slow Na+ inactivation (Migliore 1996)
18. CA1 network model for place cell dynamics (Turi et al 2019)
19. CA1 network model: interneuron contributions to epileptic deficits (Shuman et al 2020)
20. CA1 pyr cell: Inhibitory modulation of spatial selectivity+phase precession (Grienberger et al 2017)
21. CA1 pyr cell: phenomenological NMDAR-based model of synaptic plasticity (Dainauskas et al

External resources:
- InterLex/SciCrunch
- NeuroElectro
- NeuroMorpho.Org

Top authors for Hippocampus CA1 pyramidal GLU cell:
- Migliore M (40)
- Spruston N (10)
- Poirazi P (9)
- Blackwell KT (8)
- Ascoli GA (8)
- Migliore R (7)
- Kath WL (6)
- Johnston D (6)
- Narayanan R (5)
- Marie H (5)

Top concepts studied with Hippocampus CA1 pyramidal GLU cell:
- Detailed Neuronal Models (49)
- Active Dendrites (39)
- Action Potentials (35)
- Synaptic Integration (30)

https://modeldb.science/ModelList?id=258

ModelDB Login Browse Analysis More Search

Top cited papers by ModelDB papers

filter

PaperID	Paper	Count
4461	Hines ML, Carnevale NT. (1997). The NEURON simulation environment. Neural computation 9	313
29126	HODGKIN AL, HUXLEY AF. (1952). A quantitative description of membrane current and its application to conduction and excitation in nerve. The Journal of physiology 117	275
80306	Hines ML, Carnevale NT. (2006). The NEURON Book	154
4252	Mainen ZF, Sejnowski TJ. (1996). Influence of dendritic structure on firing pattern in model neocortical neurons. Nature 382	100
38501	Hines ML, Morse T, Migliore M, Carnevale NT, Shepherd GM. (2004). ModelDB: A Database to Support Computational Neuroscience. Journal of computational neuroscience 17	88
24580	Hines ML, Carnevale NT. (2001). NEURON: a tool for neuroscientists. The Neuroscientist : a review journal bringing neurobiology, neurology and psychiatry 7	82
89145	Ermentrout GB. (2002). Simulating, Analyzing, and Animating Dynamical System: A Guide to XPPAUT for Researchers and Students Society for Industrial and Applied Mathematics (SIAM)	75
6271	Hoffman DA, Magee JC, Colbert CM, Johnston D. (1997). K+ channel regulation of signal propagation in dendrites of hippocampal pyramidal neurons. Nature 387	74
40104	Izhikevich EM. (2007). Dynamical Systems in Neuroscience: The Geometry of Excitability and Bursting	68
4640	Wang XJ, Buzsáki G. (1996). Gamma oscillation by synaptic inhibition in a hippocampal interneuronal network model. The Journal of neuroscience : the official journal of the Society for Neuroscience 16	60

Showing 1 to 10 of 100 rows 10 rows per page

‹ 1 2 3 4 5 ... 10 ›

Top references cited by Parkinson's models:

- Albin RL, Young AB, Penney JB. (1989). The functional anatomy of basal ganglia disorders. Trends in neurosciences 12 (20)
- Terman D, Rubin JE, Yew AC, Wilson CJ. (2002). Activity patterns in a model for the subthalamopallidal network of the basal ganglia. The Journal of neuroscience : the official journal of the Society for Neuroscience 22 (19)
- Bergman H, Wichmann T, Karmon B, DeLong MR. (1994). The primate subthalamic nucleus. II. Neuronal activity in the MPTP model of parkinsonism. Journal of neurophysiology 72 (16)
- Raz A, Vaadia E, Bergman H. (2000). Firing patterns and correlations of spontaneous discharge of pallidal neurons in the normal and the tremulous 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine vervet model of parkinsonism. The Journal of neuroscience : the official journal of the Society for Neuroscience 20 (15)
- Plenz D, Kital ST. (1999). A basal ganglia pacemaker formed by the subthalamic nucleus and external globus pallidus. Nature 400 (15)

How do people use ModelDB?

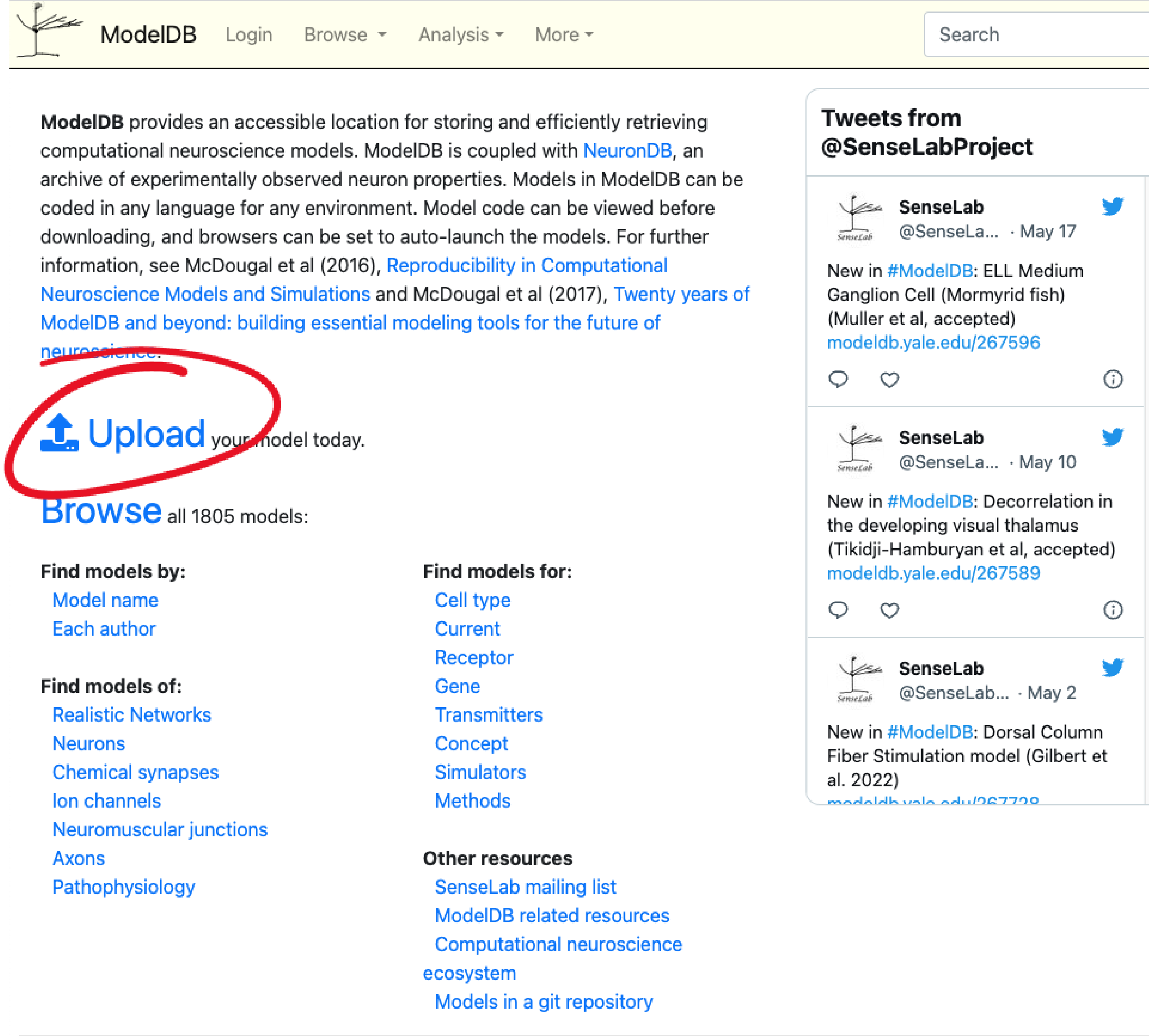
Find a model described in a paper, download it, and experiment to understand the model's predictions.

Find a model described in a paper. Use ModelView to understand the model's structure.

Locate models and modeling papers on a given topic.
Locate model components (e.g. L-type calcium channel) for potential reuse.

Search for simulator keywords (e.g. FInitializeHandler) to find examples of how to use them.

Sharing your models



Submit New Model

Only the information in this first section is required, however the more information you provide, the easier it will be for others to discover your model.

Your full name: Contributor name

Your email: Contributor email

Paper citation: Enter citation or PMID in any format
Required for public models.

Read/write access code: Access code
Allows viewing and editing your model before it is made public.

Zip file with model code: Choose File No file chosen

Model license:
A license tells people what they can and cannot do with your model.

Model overview and sharing

Model name: Model name, typically: model topic (author et al, year)
What is investigated and paper. Examples in model names list.

Model notes: Paper abstract or a more concise summary of the model, question, and results.
A concise summary of the model, question, and results. Some people provide the paper abstract, but a model-focused explanation is better. You might want to answer: what makes your model different from models of the same cell,

Model notes:

A concise summary of the model, question, and results. Some people provide the paper abstract, but a model-focused explanation is better. You might want to answer: what makes your model different from models of the same cell, disease, etc? What were the main predictions/explanations from your model?

Paper abstract or a more concise summary of the model, question, and results.

Experimental motivation:

Brief summary of key experimental findings driving modeling choices.

If applicable, e.g. why the channel distributions, connectivity rules, etc

Read only access code:

Allows others (e.g. reviewers) to read but not edit an unpublished model.

Read-only access code

Model properties

Properties autocomplete as you type or you can use the down arrow to see the full list.

Choose the properties of the model by keeping this guiding question in mind: "Do I want my model to appear when someone searches for all models with that(those) keyword(s)?" If one or more properties you would like to choose for your model does not already exist in the list(s) use the "Other tags" text box at the end to enter your suggestion. Most of the properties allow multiple selections.

Let us find ModelDB keywords for you!

Click the button to automatically find, approve, and populate model entry keywords based on your paper abstract.

Model type: Select model type

Neurons: Select neurons

Currents: Select currents

Model notes:

A concise summary of the model, question, and results. Some people provide the paper abstract, but a model-focused explanation is better. You might want to answer: what makes your model different from models of the same cell, disease, etc? What were the main predictions/explanations from your model?

Pa results.

Experimental motivation:

Brief summary of key experimental findings driving modeling choices.

If a

Read only access code:

Allows others (e.g. reviewers) to read but not edit an unpublished model.

Re

Model properties

Properties autocomplete as you type or you can us

Choose the properties of the model by keeping thi s for all models with that(those) keyword(s)?" If one or more properties you would like to choose fo e end to enter your suggestion. Most of the properties allow multiple selections.

Automatic keyword identifier

×

Please paste your paper abstract here.

The integrative properties of cortical pyramidal dendrites are essential to the neural basis of cognitive function, but the impact of amyloid beta protein (abeta) on these properties in early Alzheimer's is poorly understood. In animal models, electrophysiological studies of proximal dendrites have shown that abeta induces hyperexcitability by blocking A-type K+ currents (I(A)), disrupting signal integration. The present study uses a computational approach to analyze the hyperexcitability induced in distal dendrites beyond the experimental recording sites. The results show that back-propagating action potentials in the dendrites induce hyperexcitability and excessive calcium concentrations not only in the main apical trunk of pyramidal cell dendrites, but also in their oblique dendrites. Evidence is provided that these thin branches are particularly sensitive to local reductions in I(A). The results suggest the hypothesis that the oblique branches may be most vulnerable to disruptions of I(A) by early exposure to abeta, and point the way to further experimental analysis of these actions as factors in the neural basis of the early decline of cognitive function in Alzheimer's.

Cancel Submit

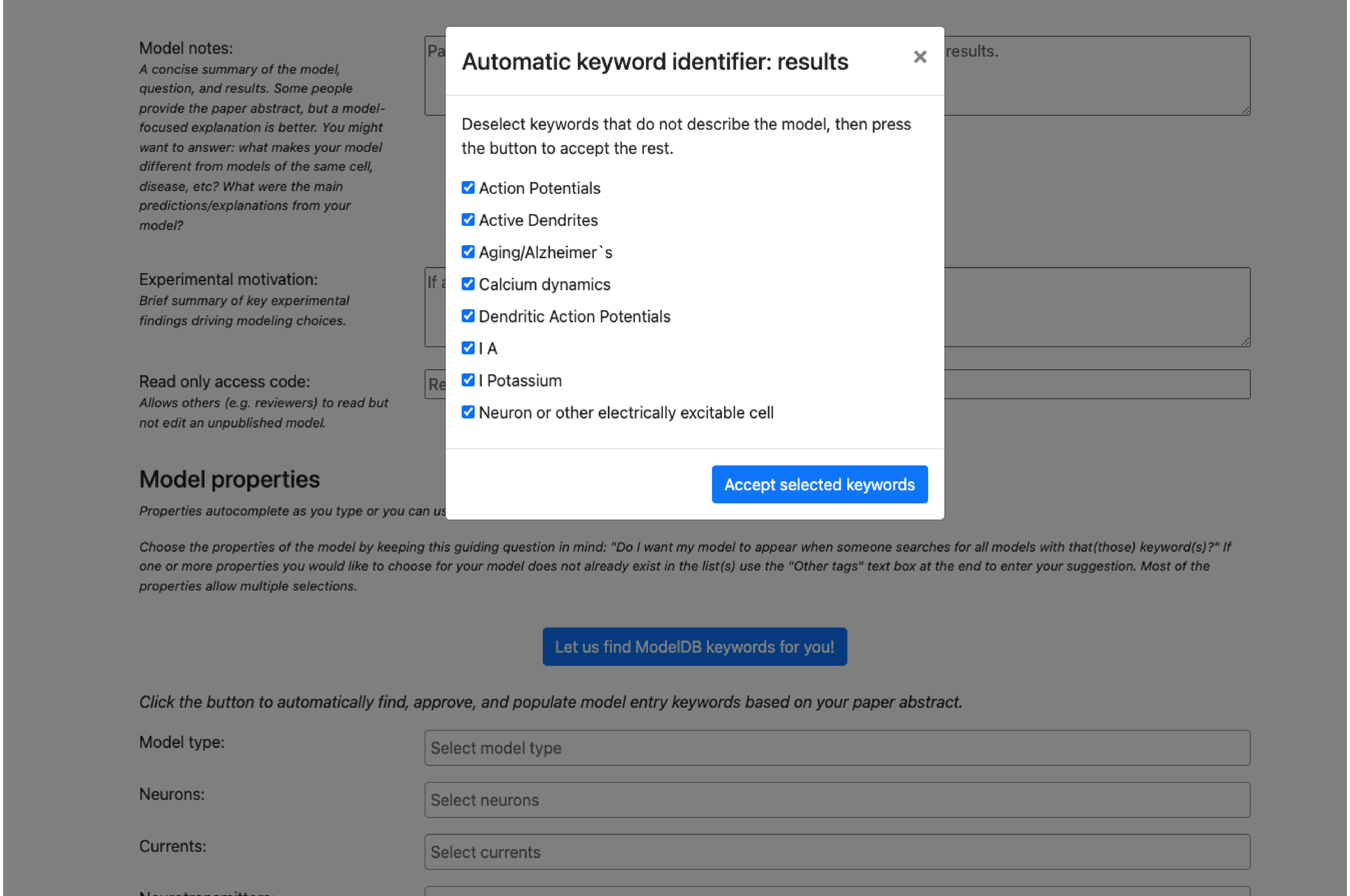
Click the button to automatically find, appr ct.

Model type: Se

Neurons: Select neurons

Currents: Select currents

Neurotransmitters:


Model notes:
A concise summary of the model, question, and results. Some people provide the paper abstract, but a model-focused explanation is better. You might want to answer: what makes your model different from models of the same cell, disease, etc? What were the main predictions/explanations from your model?
Experimental motivation:
Brief summary of key experimental findings driving modeling choices.
Read only access code:
Allows others (e.g. reviewers) to read but not edit an unpublished model.
Model properties
Properties autocomplete as you type or you can us
Choose the properties of the model by keeping this guiding question in mind: "Do I want my model to appear when someone searches for all models with that(those) keyword(s)?" If one or more properties you would like to choose for your model does not already exist in the list(s) use the "Other tags" text box at the end to enter your suggestion. Most of the properties allow multiple selections.
Let us find ModelDB keywords for you!
Click the button to automatically find, approve, and populate model entry keywords based on your paper abstract.
Model type:
Select model type
Neurons:
Select neurons
Currents:
Select currents
Automatic keyword identifier: results
×
Deselect keywords that do not describe the model, then press the button to accept the rest.
Action Potentials
Active Dendrites
Aging/Alzheimer`s
Calcium dynamics
Dendritic Action Potentials
I A
I Potassium
Neuron or other electrically excitable cell
Accept selected keywords

Let us find ModelDB keywords for you!

Click the button to automatically find, approve, and populate model entry keywords based on your paper abstract.

Model type: × Neuron or other electrically excitable cell

Neurons: Select neurons

Currents: × I A × I Potassium

Neurotransmitters: Select neurotransmitters

Receptors: Select receptors

Genes: Select genes

Concepts: × Action Potentials × Active Dendrites × Aging/Alzheimer`s × Calcium dynamics × Dendritic Action Potentials

Region or organism: Select region or organism

Simulation environment: Select simulation environment

Implementer(s): Implementers

Are there any other tags we should use to annotate your model?

Suggest other tags

Submit

Metadata queryable via API

ModelDB Login Browse Analysis More Search

ModelDB - API Overview

The prefix for accessing all API calls is `/api/v1`.

To see what types of data are available simply call /api/v1. This returns a JSON-encoded list that includes e.g. celltypes, models, ...

To find out which data objects are available for a given type, query that type. e.g. The list of all model object ids is /api/v1/models.

To get a specific instance, include the object id. e.g. The data for model 87284 is at /api/v1/models/87284.

You can also get a list of property values in the same order as the object ids by querying on the name of the property. For example, to get the "name" property for all the celltypes objects, query /api/v1/celltypes/name.

By default, the results are returned without pretty-printing, however you may get a pretty-printed version instead by specifying a number for the GET argument `indent`; e.g. /api/v1/models/87284?indent=4.

For the special cases of `/api/v1/models` and queries of specific properties, the set of models/property values returned may be filtered by other properties of the model using either the object_id or the object_name, e.g. /api/v1/models?model_concept=112047 shows the set of models with model concept 112047 (Alzheimer's), /api/v1/models/created?model_concept=112047 shows the creation date of these models, and /api/v1/models?modeling_application=NEURON shows the set of models using NEURON as a modeling application. Multiple fields may be filtered at the same time, but currently only one filter value for a given field is allowed, and only potentially multi-valued fields are supported.

All results are returned with mime type `application/json`.

modeldb.science/api

Example

- modeldb.science/87284

ModelDB Login Browse Analysis More Search

Amyloid beta (IA block) effects on a model CA1 pyramidal cell (Morse et al. 2010)

Overview Files ModelView Citations

The model simulations provide evidence oblique dendrites in CA1 pyramidal neurons are susceptible to hyper-excitability by amyloid beta block of the transient K+ channel, IA. See paper for details.

Model Type: Neuron or other electrically excitable cell

Cell Type(s): Hippocampus CA1 pyramidal GLU cell

Currents: I Na,t; I L high threshold; I N; I T low threshold; I A; I K; I h; I K,Ca

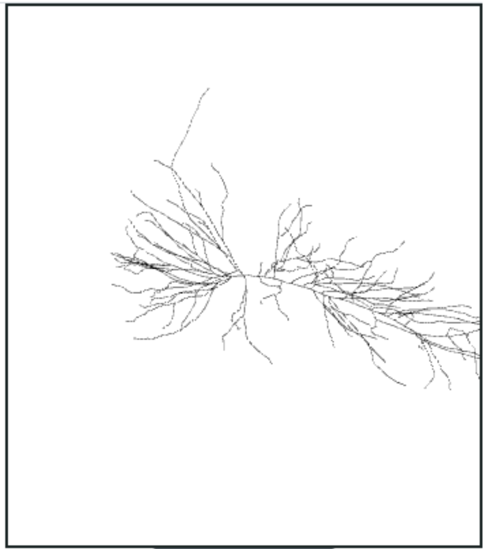
Model Concept(s): Dendritic Action Potentials; Active Dendrites; Detailed Neuronal Models; Pathophysiology; Aging/Alzheimer`s

Simulation Environment: NEURON

Implementer(s): Carnevale, Ted [Ted.Carnevale at Yale.edu]; Morse, Tom [Tom.Morse at Yale.edu]

References:

Morse TM, Carnevale NT, Mutalik PG, Migliore M, Shepherd GM. (2010). Abnormal Excitability of Oblique Dendrites Implicated in Early Alzheimer's: A Computational Study. *Frontiers in neural circuits* 4 [PubMed]



Show Diameter

Simulation Platform
View on GitHub

```
This is the readme for a model used in the paper

Morse TM, Carnevale NT, Mutalik PG, Migliore M, Shepherd GM (2010)
Abnormal excitability of oblique dendrites implicated in early
Alzheimer's: a computational study Front. Neural Circuits 4:16


The model code was contributed by Tom Morse.  It was created (see
paper for details) from earlier models (especially Migliore et
al. 2005 and calcium channels from Hemond et al. 2008) with
modifications and additions by Tom Morse and Ted Carnevale with
interaction with the other authors. It requires the NEURON simulator
to be installed (available at http://www.neuron.yale.edu).

To recreate figures from the paper, start the simulator by
auto-launching from ModelDB *OR*

Under unix systems:
-------------------
In the expanded archive's folder compile the mod files using the
command "nrnivmodl"
```