

# ModelDB

<http://modeldb.yale.edu>

ModelDB promotes discoverability and reproducibility of computational neuroscience research by serving as a platform for curated sharing and visualization of published models.

ModelDB

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

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Model Information

Accession: 87284

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

Reference:

1. 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 [PubMed]

Model Information (Click on a link to find other models with that property)

Model Type:	Neuron or other electrically excitable cell;
Brain Region(s)/Organism:	
Cell Type(s):	Hippocampus CA1 pyramidal cell;
Channel(s):	I Na,t; I L high threshold; I N; I T low threshold; I A; I K; I h;
Gap Junctions:	
Receptor(s):	
Gene(s):	
Transmitter(s):	

Simulation Platform 3D Print

Morse et al. 2010

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  
kap (kapprox.mod)  
kdr (kdrca1.mod)  
na3 (na3n.mod)

root: soma

X-Y X-Z Y-Z

Distance from root

0.313714

```
from neuron import h, rxd
import neuron.rxd.node as node
from matplotlib import pyplot
import time
```

```
h.load_file('stdrun.hoc')
```

```
soma = h.Section()
soma.L = 10
soma.diam = 10
soma.nseg = 11
dend = h.Section()
dend.connect(soma)
dend.L = 50
dend.diam = 2
dend.nseg = 51
```

```
def print_nodes():
    print ', '.join(str(v) for v in node._states)
```

```
print 'defining rxd'
region = rxd.Region(h.allsec(), nrn_region='i')
ca = rxd.Species(region, name='ca', d=1, charge=2, initial=
reaction = rxd.Rate(ca, -ca * (1 - ca) * (0.3 - ca))
```

```
print 'initializing'
h.finitialize()
```

```
print 'before:'
print_nodes()
print
```

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 [PubMed]

#### References and models cited by this paper

Acker CD, White JA (2007) Roles of I(A) and morphology in action potential propagation in CA1 pyramidal cell dendrites. *J Comput Neurosci* 23(2):201-16 [Journal] [PubMed]

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

Anderton BH, Callahan L, Coleman P, Davies P, Flood D, Jicha GA, Ohm T, Weaver C (1998) Dendritic changes in Alzheimer's disease and factors that may underlie these changes. *Prog Neurobiol* 55:595-609 [PubMed]

Andrasfalvy BK, Makara JK, Johnston D, Magee JC (2008) Altered synaptic and non-synaptic properties of CA1 pyramidal neurons in 6x4 Sliney mice. *J Neurosci* 28:1111-1121 [Journal] [PubMed]

#### 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 *Front Comput Neurosci* 6:52 [Journal] [PubMed]

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

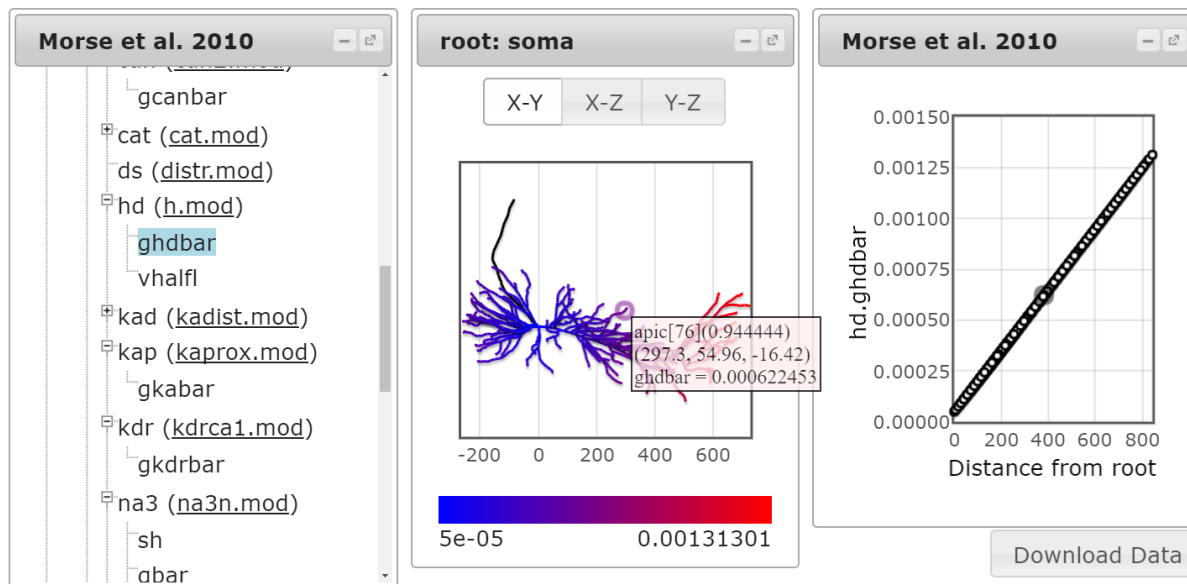
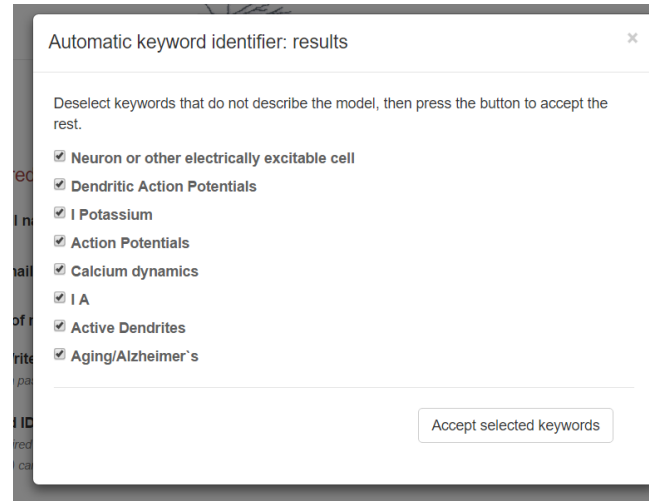
McDougal RA, Morse TM, Hines ML, Shepherd GM (2015) ModelView for ModelDB: online presentation of model structure *Neuroinformatics* 13(4):459-70 [Journal] [PubMed]

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

Over 1200 models · 76 simulation environments · 178 cell types · 145 topics (Alzheimer's, STDP, etc) · 16+ species · 54 ion channels, pumps, etc · 24+ mammalian brain regions

# Ongoing ModelDB projects

*Improving quantity and quality of model entries by actively identifying new modelling literature and providing NLP tools to assist entry of descriptive metadata.*



*Model visualization tools make models more accessible by allowing insight into the model structure without reading code.*

Model Information **Model File** Citations Model Views 3D Print

Download the displayed file **ICGenealogy**

/

- CA1\_abeta
  - translate
  - readme.html
  - cacumm.mod
  - cagk.mod \***
  - cal2.mod \*
  - can2.mod \*
  - cat.mod \*

TITLE CaGk  
: Calcium activated K channel.  
: Modified from Moczydlowski and Latorre (1983)

UNITS {  
    (molar) = (1/liter)  
}

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

#### General data

- **ICG id:** 2464
- **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:** Q4 (slow)
- **Subtype:** not specified

#### Metadata generic

- **Age:** 7-14 weeks old.
- **Authors:** M Migliore.
- **Comments:** Calcium activated k channel, modified from moczydlowski and latorre (1983). From hemond et al. (2008), model no. 101629, with no changes (identical mod file). Animal model taken from chen (2005) which is used to constrain model. Channel kinetics from previous study on hippocampal pyramidal neuron (hemond et al. 2008)

Amyloid beta (1A block) effects on a model CA1 pyramidal cell (Morse et al. 2010)

**Better model context**  
through partnerships with  
external neuroinformatics  
resources like the Ion  
Channel Genealogy (above)  
and through identifying  
repeated patterns within  
ModelDB itself (right).

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Model Information **Model File** Citations Model Views Simulation Platform 3D Print

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/

- CA1\_abeta
  - translate
  - readme.html**
  - cacumm.mod
  - cagk.mod \*
  - cal2.mod \*
  - can2.mod \*
  - cat.mod \*
  - distr.mod \*
  - h.mod
  - lpulse2.mod \*
  - kadist.mod
  - kaprox.mod
  - kdrca1.mod
  - na3n.mod
  - naxn.mod \***
  - zcaquant.mod
  - aBeta.hoc

This is the readme file for the model. It contains information about the model, its authors, and how to use it. 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 channel models) and modifications and interaction with other models to be installed.

To recreate figure 1, auto-launching the model is recommended.

Under unix systems, the command "python3 run.py" will open the simulation window.

Under Windows systems, the command "python3 run.py" will open the simulation window.

Under MAC OS X: the command "python3 run.py" will open the simulation window.

Other models using cagk.mod:

- A model of unitary responses from A/C and PP synapses in CA3 pyramidal cells (Baker et al. 2010)
- CA1 pyramidal neuron: effects of R213Q and R312W Kv7.2 mutations (Miceli et al. 2013)
- CA3 pyramidal neuron (Safuлина et al. 2010)
- CA3 pyramidal neuron: firing properties (Hemond et al. 2008)
- Neuronal dendrite calcium wave model (Neymotin et al. 2015)

Other models using naxn.mod:

- CA1 pyramidal neuron: effects of R213Q and R312W Kv7.2 mutations (Miceli et al. 2013)
- CA1 pyramidal neuron: functional significance of axonal Kv7 channels (Shah et al. 2008)
- CA1 pyramidal neuron: rebound spiking (Ascoli et al. 2010)
- CA1 pyramidal neuron: schizophrenic behavior (Migliore et al. 2011)
- CA1 pyramidal neuron: signal propagation in oblique dendrites (Migliore et al. 2005)
- CA1 pyramidal neurons: binding properties and the magical number 7 (Migliore et al. 2008)
- CA1 pyramidal neurons: effect of external electric field from power lines (Cavarretta et al. 2014)
- CA1 pyramidal neurons: effects of Alzheimer (Culmone and Migliore 2012)
- CA1 pyramidal neurons: effects of Kv7 (M-) channels on synaptic integration (Shah et al. 2011)
- CA1 pyramidal neurons: effects of a Kv7.2 mutation (Miceli et al. 2009)
- CA1 pyramidal neuron: reduction model (Marasco et al. 2012)
- Effect of the initial synaptic state on the probability to induce LTP and LTD (Migliore et al. 2015)
- Effects of electric fields on cognitive functions (Migliore et al. 2016)
- Neuronal morphology goes digital ... (Parekh & Ascoli 2013)
- Spine head calcium in a CA1 pyramidal cell model (Graham et al. 2014)

# Open Source Brain

<http://www.opensourcebrain.org>

Open Source Brain is...

*One slide giving an overview of the resource, example datasets, etc.*

*One slide discussing current  
work/future plans*

*Optional 3<sup>rd</sup> slide...*

*General requests:*

- Please favour graphical content over text where possible*
- Please don't use animation (use multiple slides if required)*
- Try to keep to font Arial*
- Save slides as .pptx (Powerpoint 2007-2013); slides will be concatenated into a single PDF presentation*



# NeuroML

<http://www.neuroml.org>

NeuroML is a language for expressing models in computational neuroscience in a simulator independent, standardised format. It can express models from integrate and fire cells to complex networks of multicompartmental neurons.

Standardised XML language for  
computational neuroscience  
Version 1.x allowed specification of:

- Detailed neuronal morphologies
- Ion channels
- Synapses
- 3D network structure

30+ simulators/applications/  
databases/libraries support  
NeuroML

### **Simulators**

NEURON  
GENESIS  
MOOSE  
Brian

### **Interoperability**

PyNN  
neuroConstruct

### **Initiatives**

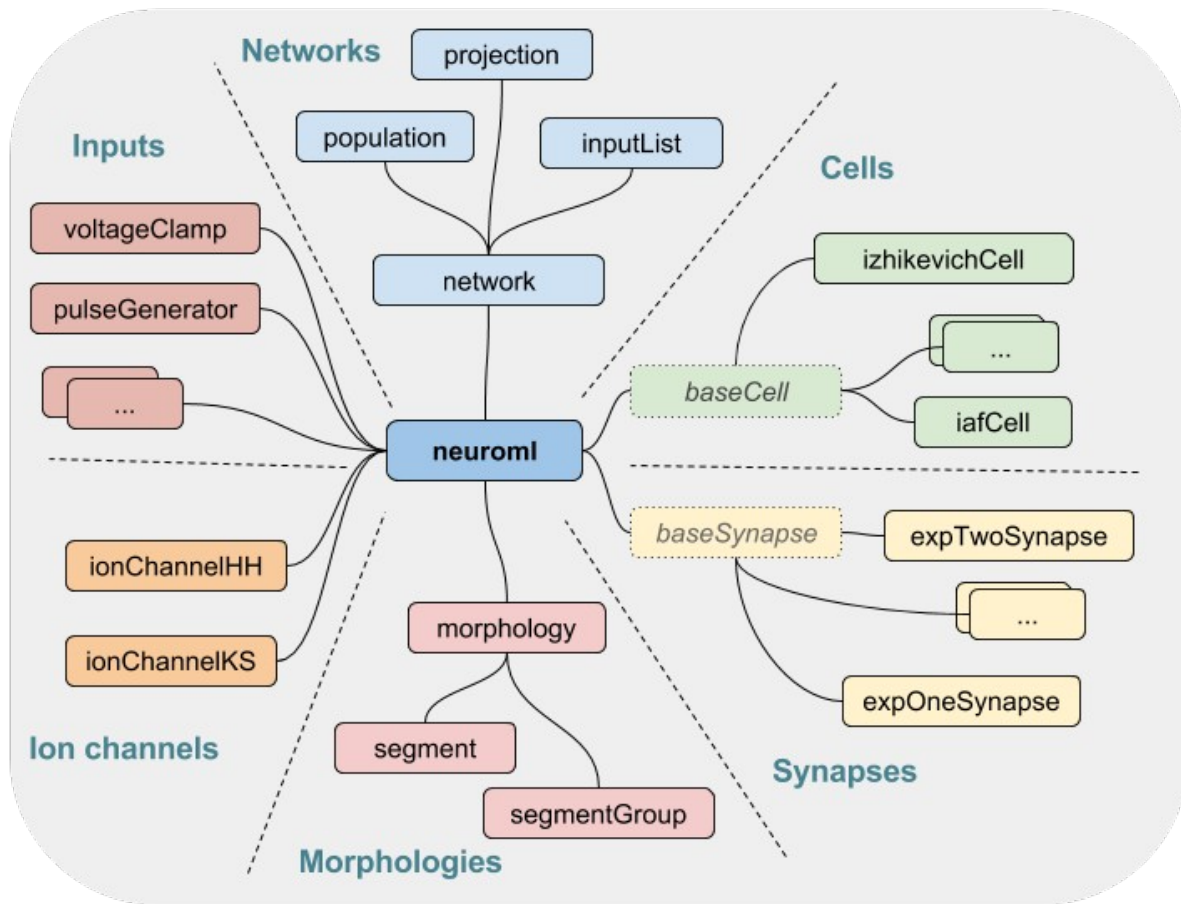
OpenWorm  
Open Source  
Brain

### **Databases**

Channelpedia  
BBP NMC  
NeuroMorpho  
Allen Institute  
Cell Types DB

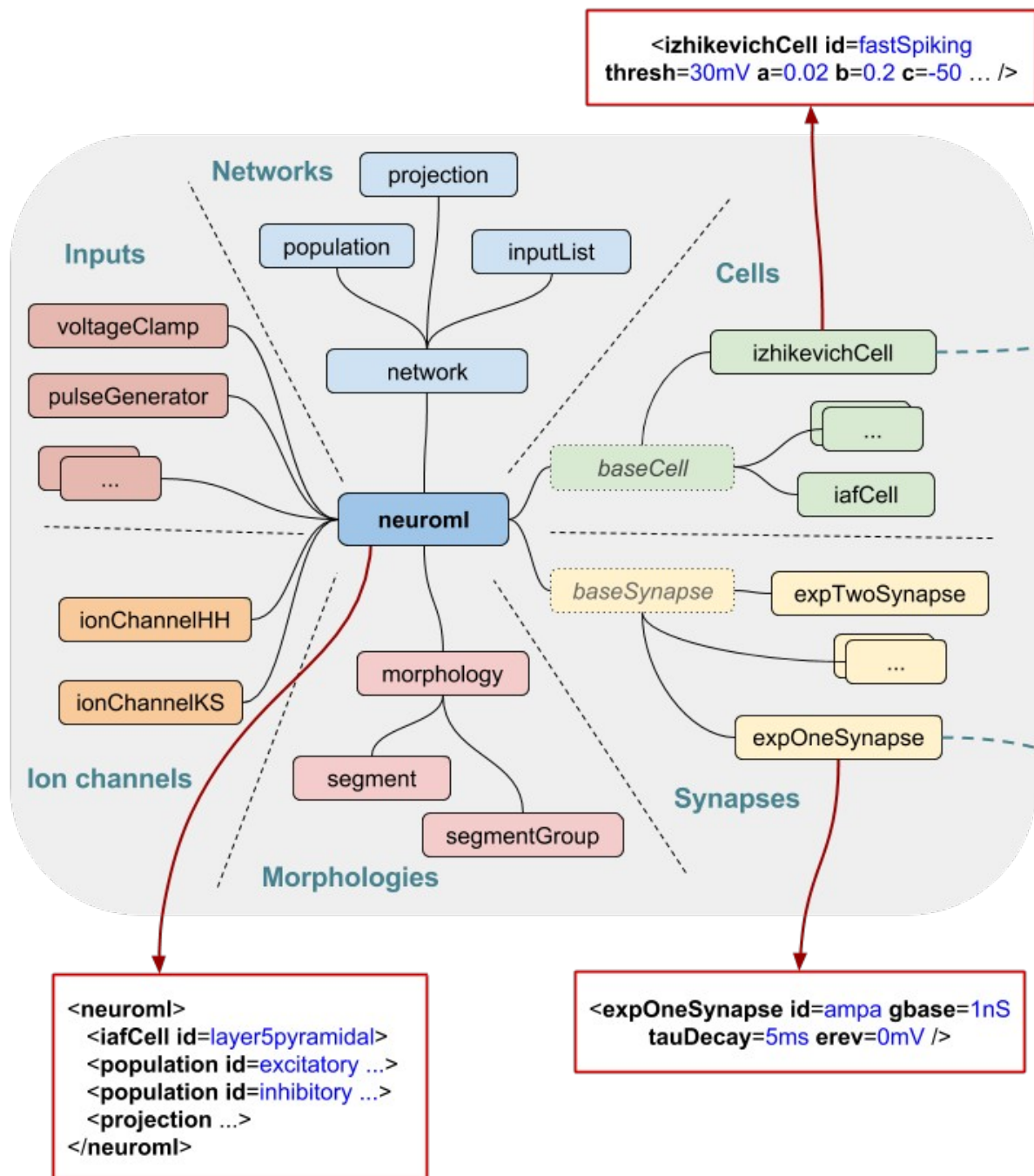
### **Morphological analysis/ generation**

Cx3D  
TREES Toolbox  
NeuGen



## NeuroML 2

## LEMS



### Standard NeuroML 2 ComponentType definitions

#### Cells.xml

ComponentType: **izhikevichCell**

Parameters: **thresh**, **a**, **b**, **c**, **d**, ...

Dynamics

StateVariables: **v**, **U**

TimeDerivatives:

$$dv/dt = 0.04 \cdot v^2 + 5 \cdot v + 140.0 - U$$

$$dU/dt = a \cdot (b \cdot v - U)$$

OnConditions:

$$v > \text{thresh} \Rightarrow$$

$$v = c$$

$$U = U + d$$

#### Synapses.xml

ComponentType: **expOneSynapse**

Parameters: **gbase**, **tauDecay**, **erev**

Dynamics

StateVariables: **g**

TimeDerivatives:

$$dg/dt = -g / \text{tauDecay}$$

DerivedVariables:

$$i = g \cdot (\text{erev} - v)$$

OnEvents:

$$g = g + \text{gbase}$$

Networks.xml

Inputs.xml

...

...

# ReScience

<http://rescience.github.io>

**Reproducible Science is good, Replicated Science is Better.**

ReScience is a scientific journal dedicated to the publication of replication in computational sciences.

## Journal Philosophy

ReScience is a peer-reviewed journal that targets computational research and encourages the explicit [replication](#) of already published research, promoting new and open-source implementations in order to ensure that the original research is [reproducible](#).

To achieve this goal, the whole publishing chain is radically different from other traditional scientific journals. ReScience lives on [GitHub](#) where each new implementation of a computational study is made available together with comments, explanations and tests. Each submission takes the form of a pull request that is publicly reviewed and tested in order to guarantee that any researcher can re-use it.

If you ever replicated computational results from the literature in your research, ReScience is the perfect place to publish your new implementation.

## Publishing fees

None. Zero. Nada. 0\$. 0€.

## **Criteria for Publication**

To be considered for publication in ReScience, any given submission must satisfy the following criteria:

- Replicability
- Rigorous methodology
- Original source code
- Substantial evidence for replication of the original results

Furthermore, you cannot submit the replication of your own research, nor the research of your close collaborators. We believe such restrictions will favor the cross-fertilization of research and the spread of knowledge.

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Reproducible science is good. Replicated science is better.