

Lecture 1

Introduction, Nernst, GHK and
Maintaining Gradients

Neuroengineering I: Neural Signals (BMES 477/710)

Instructor

Catherine von Reyn, PhD

crv33@drexel.edu

Office Hours

Friday 2:00 pm-3:00 pm

Location: Bossone 601 or via zoom (email)

TAs

Haley Croke

hrc43@drexel.edu

Kaitlin Raselsey

ker343@drexel.edu

Office Hours

Monday 9 am – 10 pm (Kaitlin)

Monday 1 pm – 2 pm (Haley)

Location: Zoom

Lecture

Tuesday and Thursday

5:00 – 6:20 pm

Labs and Activities

During the 2nd half of class, when indicated

Bring your laptop/tablet (MATLAB)

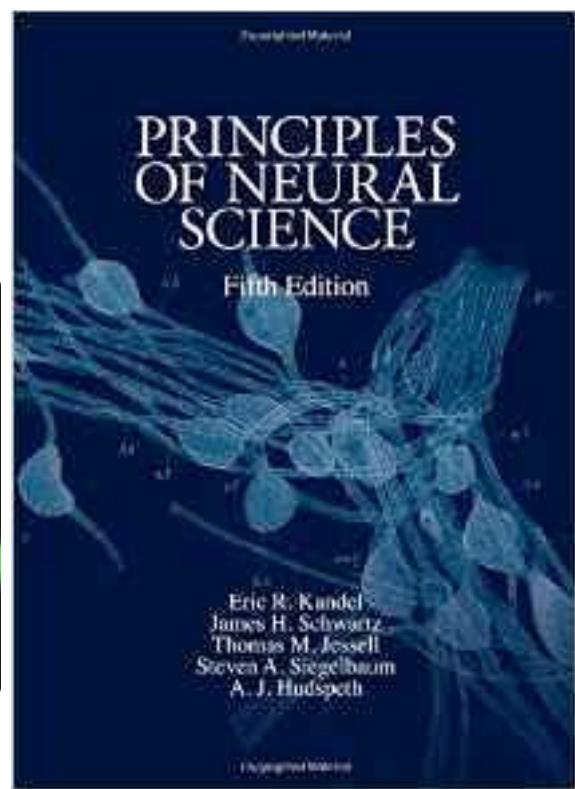
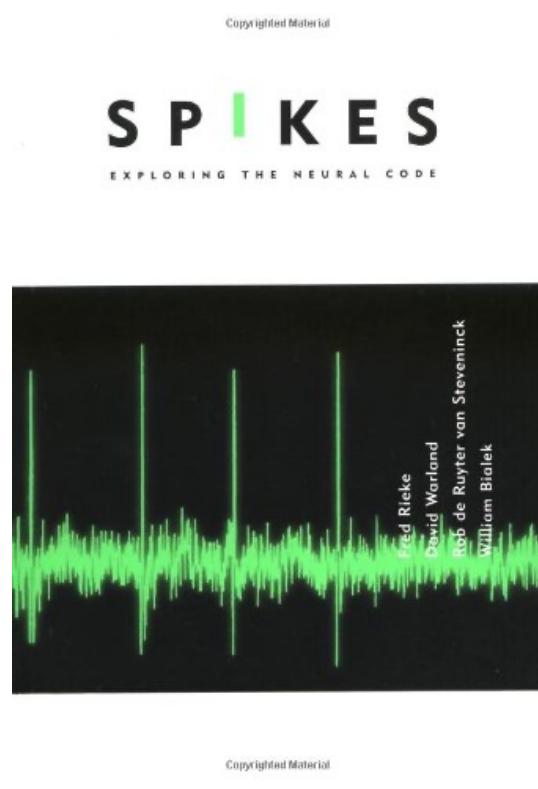
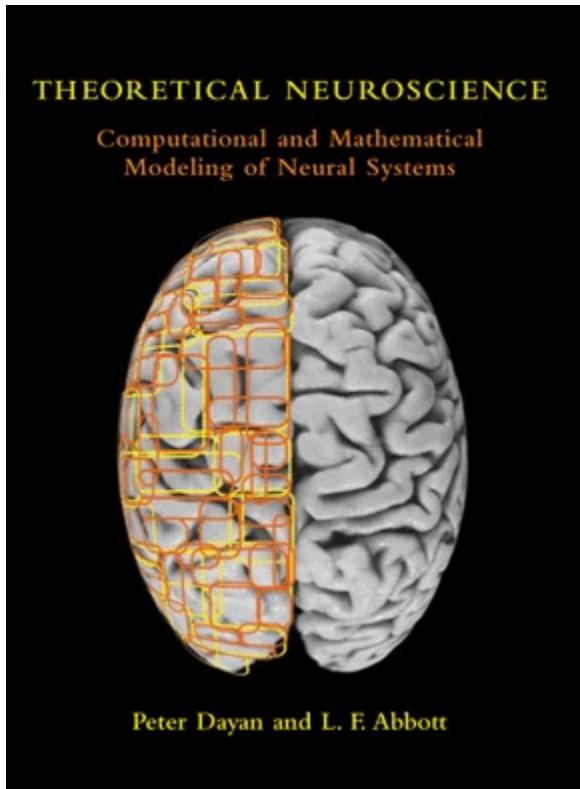
Required Text

Paul Miller

An Introductory Course in
**COMPUTATIONAL
NEUROSCIENCE**



Supplemental Texts



Objectives

1. To learn neural signaling techniques and the models used to describe them
2. Use programs to model generator potentials, equilibrium potentials, action potentials, chemical signaling and electrical signaling
3. Understand cable theory of passive dendritic activity
4. Derive the Hodgkin-Huxley description of action potential generation
5. Use compartmental models to simulate neural activity
6. Learn how networks of neurons organize
7. Apply computational algorithms to real neural data to model how neurons encode information

Grading and Requirements

Assignments (35%):

Lab Assignments: Students will be required to attend class time dedicated to lab and learn to apply and generate MATLAB functions for neural simulations and data analysis. Lab assignments include questions based on the theory and application of these functions. Assignments not finished during the lab period must be completed as homework. ***Lab assignments are given on Tuesday and are due on Monday at 11:59PM.***

Reading Assignments: Students will be required to write commentaries on journal articles. Commentaries must be 300 words or less and should highlight the key finding(s) of the article and address issues/limitations for interpreting data presented in the article. ***Reading assignments are posted on Monday and are due before class on Thursday.***

No late assignments will be accepted.

Quizzes (10%):

A short quiz will be given every Thursday morning at the beginning of class. Quizzes will cover topics from past lectures and reading assignments.

Midterm Exam (25%):

A midterm exam will be given during the ***second lecture of week 6***. The problems will include mathematical modeling and short answer questions.

Modeling Paper (30%): Students will be required to investigate a topic in neural engineering that applies neural models learned in this course. Students will select one paper with a modeling component that addresses the topic. Students will evaluate this model by running the code themselves. Students will then modify the code to ask their own specific question and report the results. They will then submit their working code and a paper (limited to 5 pages) that includes the following:

- **Intro (5%):**
 - the underlying biological questions asked by the authors and asked by the student
- **Methods (10%):**
 - the mathematical model being used to solve these questions
 - how the mathematical model has been derived or altered to represent the biological system (what did the authors alter and then what did the student additionally alter)
- **Results (10%):**
 - brief summary of the authors' results
 - the results generated by the student (need to include figures)
- **Discussion (5%):**
 - the student's evaluation of whether the model has been applied appropriately and the model limitations
 - the overall impact of the research paper as well as the overall impact of the student's modeling work

Topics and selected papers are due the day of the midterm and the final paper with working code is due TUESDAY on the week of exams.

COURSE SCHEDULE

WEEK	DATE		TOPIC	TEXT CHAPTERS	ASSIGNMENT
1	26-Sep	T	Intro, Nernst and Goldman; Lab	1.1-1.3; 2.1	Lab 1 Due 10/02
	28-Sep	Th	Equivalent Circuits	2.2	Quiz 1
2	3-Oct	T	Cable Theory	Remote	Lab 2 Due 10/09
	5-Oct	Th	Lab	Optional	Quiz 2 Online
3	10-Oct	T	Action Potentials, Channels and Currents		
	12-Oct	Th	Integrate and Fire Model; Lab	2.3-2.7	Quiz 3
4	17-Oct	T	Hodgkin and Huxley Model	1.4.1; 4.1-4.6	Lab 3 Due 10/23
	19-Oct	Th	Compartment Based Models; Lab	4.7-4.9	Quiz 4
5	24-Oct	T	Synapses	5.1-5.3	
	26-Oct	Th	Synaptic Plasticity	8.1-8.3	Commentary 1 Due 10/30
6	31-Oct	T	Networks; Review for Midterm	5.5-5.8	
	2-Nov	Th	MIDTERM		Topic and Modeling Paper Due
7	7-Nov	T	Recording Methods: Electrodes; PSTH	3.1	Lab 4 Due 11/13
	9-Nov	Th	The Somatosensory System		Quiz 7
8	14-Nov	T	Spike Train Statistics; Lab	3.1-3.7	Lab 5 Due 11/20
	16-Nov	Th	Auto and Cross Correlations		Quiz 8
	21-Nov	T	No Class		
	23-Nov	Th	No Class		
9	28-Nov	T	Information Theory; Lab	1.4.3	
	30-Nov	Th	Population Codes		Quiz 9
10	5-Dec	T	Place Cells and Decoding		Commentary 2 Due 12/06
	7-Dec	Th	Recording Methods: Imaging		

Final Modeling Paper Due Exam Week

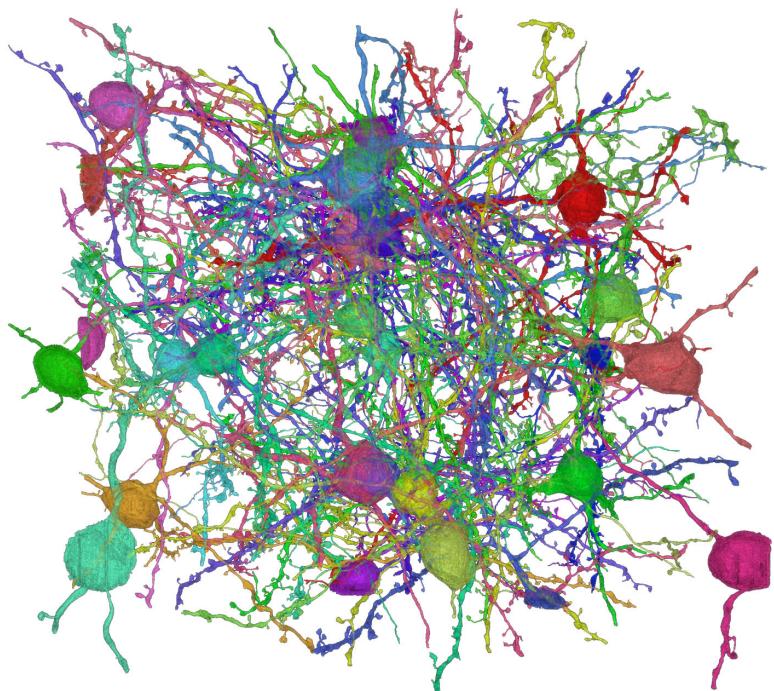
Why do we care about neural signals as engineers?





<https://www.youtube.com/watch?v=wZZ4Vf3HinA>





“Our goal is to leverage Google expertise and resources to advance understanding of the structure and function of the brain.”

“Human-Computer Interaction and Visualization... Google’s HCI researchers invent, design, build and trial large-scale interactive systems in the real world. We declare success only when we positively impact our users and user communities, often through new and improved Google products”

<https://research.google/>

4

Broad-impact, long-term human health and technology goals can be attained

Broad-impact, long-term human health and technology
goals can be attained

.....by monitoring, modeling, and manipulating neural
signals.

J Neurophysiol 118: 1292–1309, 2017.
First published May 31, 2017; doi:10.1152/jn.00149.2017.

REVIEW | *Biology of Neuroengineering Interfaces*

Neurophysiology and neural engineering: a review

Arthur Prochazka

Department of Physiology, University of Alberta, Edmonton, Alberta, Canada

Submitted 1 March 2017; accepted in final form 30 May 2017

Monitor

- Electrophysiology
 - Intracellular, extracellular
 - Single and multi-electrode probes
- Calcium and voltage indicators
- Functional magnetic resonance imaging (fMRI)
- Functional near infrared spectroscopy (fNIRS)

Model

- Synapse
- Neuron
- Circuit
- Large networks

Manipulate

- Optogenetics
- Transcranial magnetic stimulation (TMS)
- Deep brain stimulation
- Brain-machine interfaces

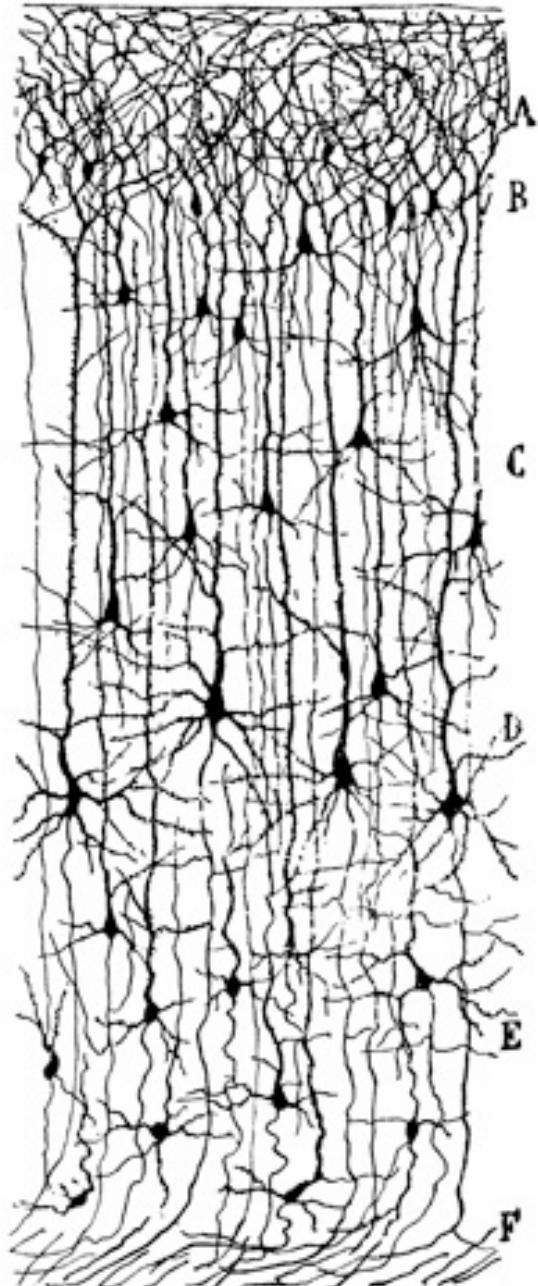
A very brief introduction to neuroanatomy



Santiago
Ramón y
Cajal

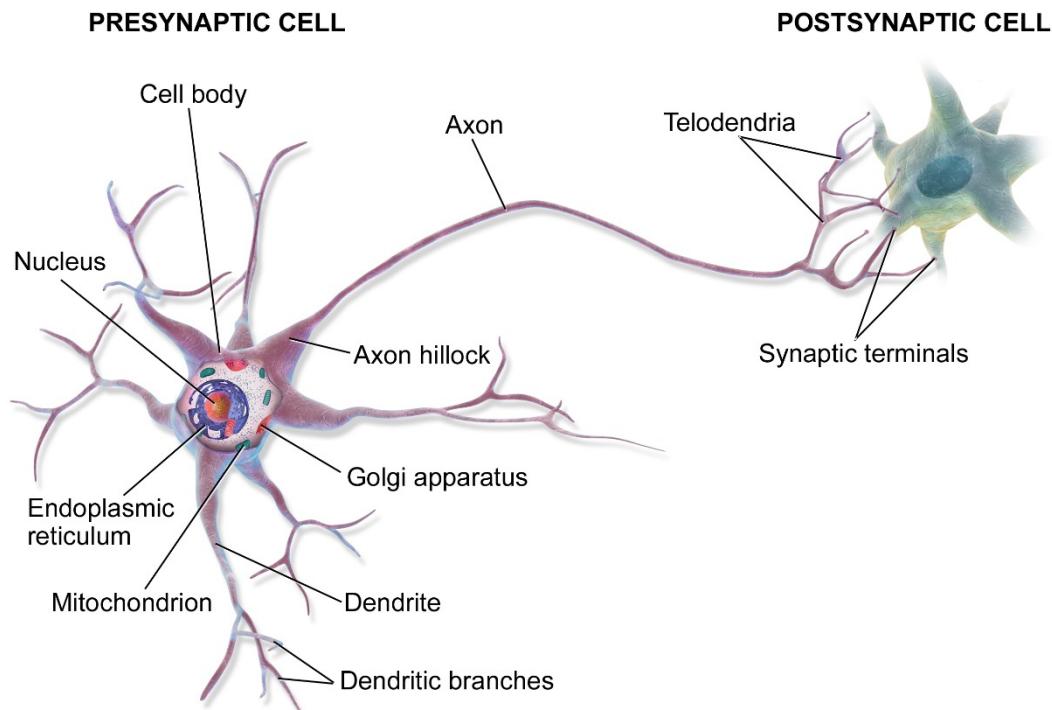
Image from **Neurocomic** by
Matteo Farinella

- The basic cellular unit of the brain: the neuron
- The human brain is a ridiculously complex network (10^{11} neurons, 10^{14} connections)



Sparse labeling of cortical neurons by Ramon y Cajal

Anatomy

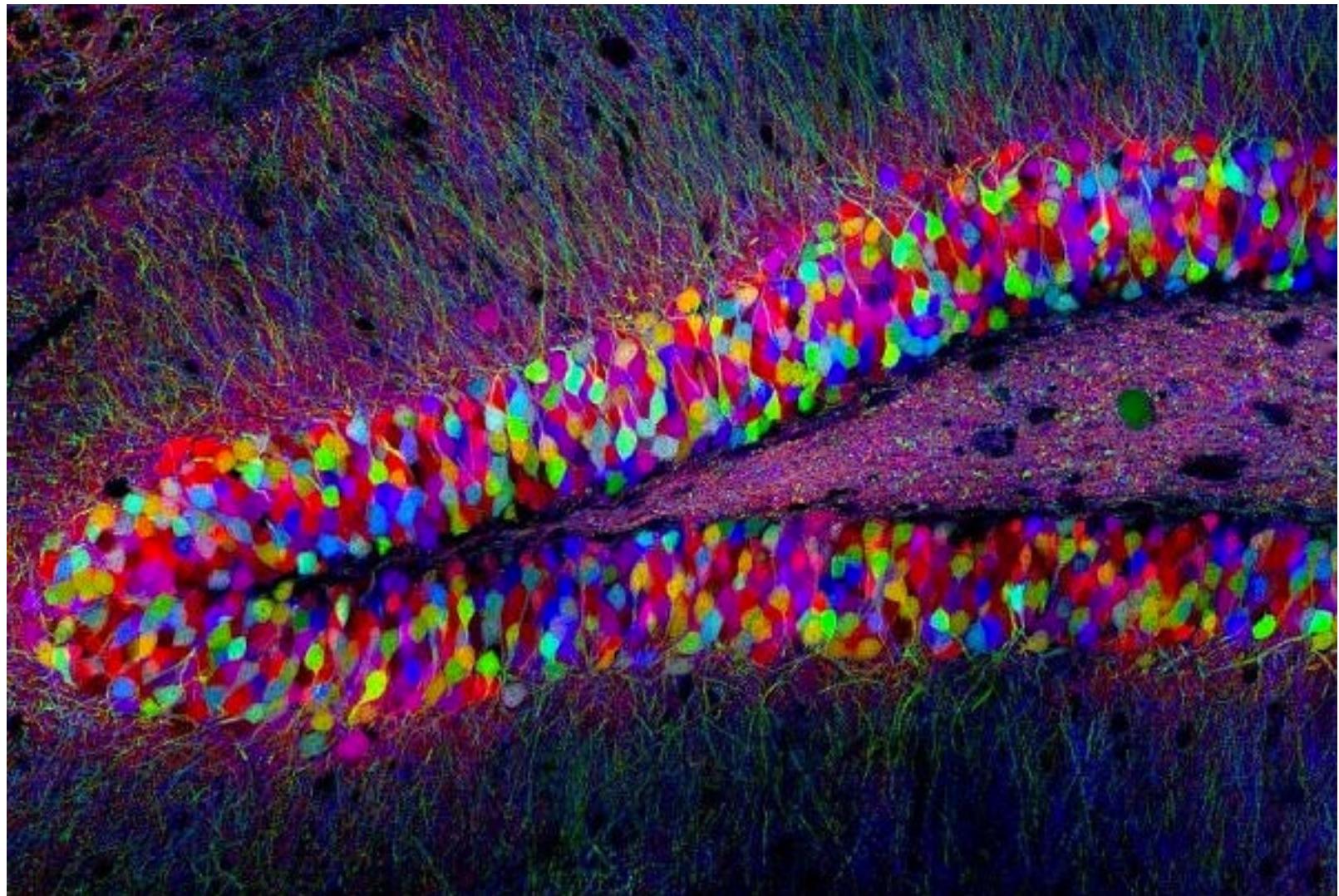


<http://en.wikipedia.org/wiki/Neuron>

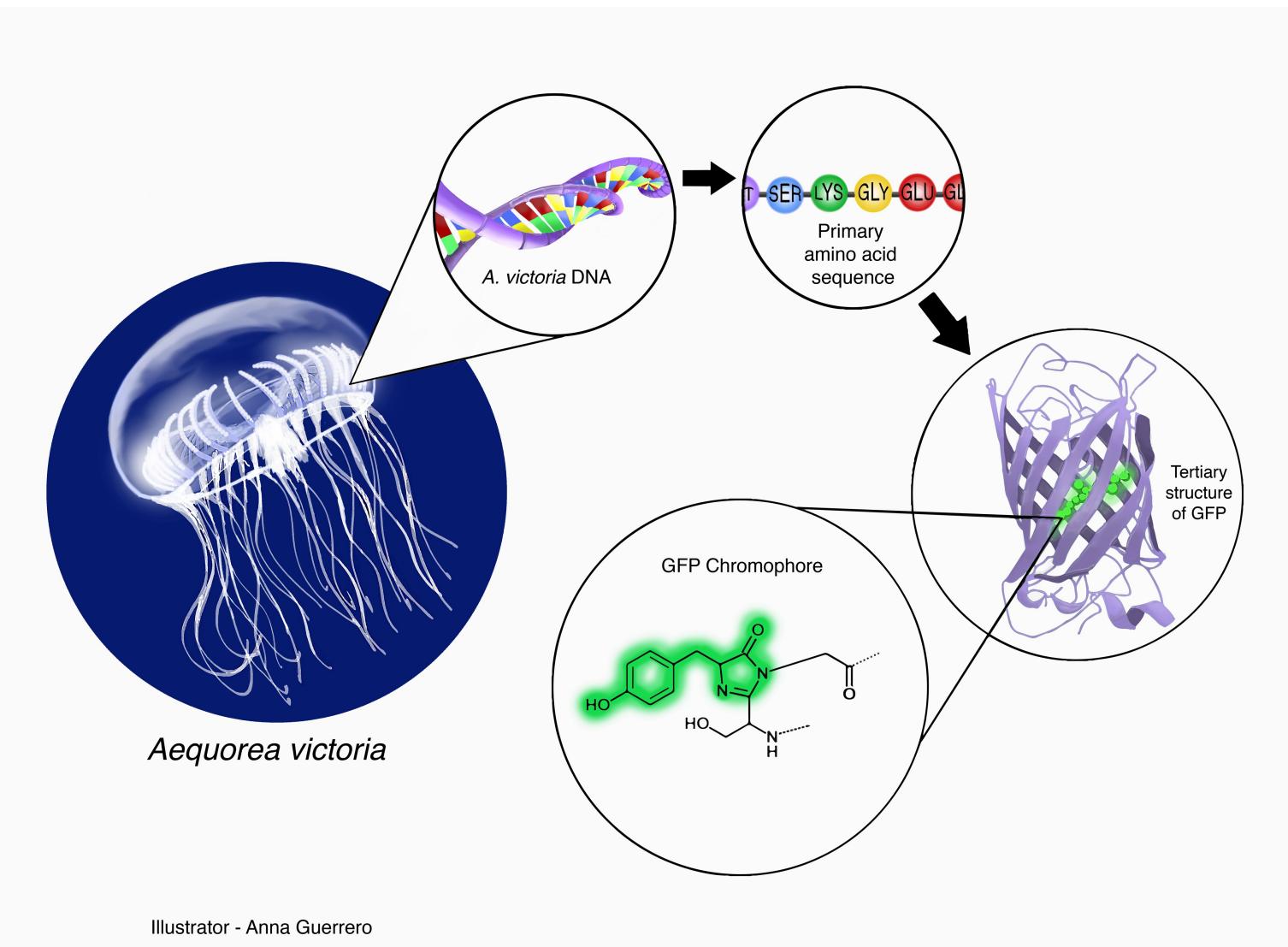
Probing neuroanatomy

- We have made substantial advancements in technologies to probe neuroanatomy from the days of Ramon y Cajal

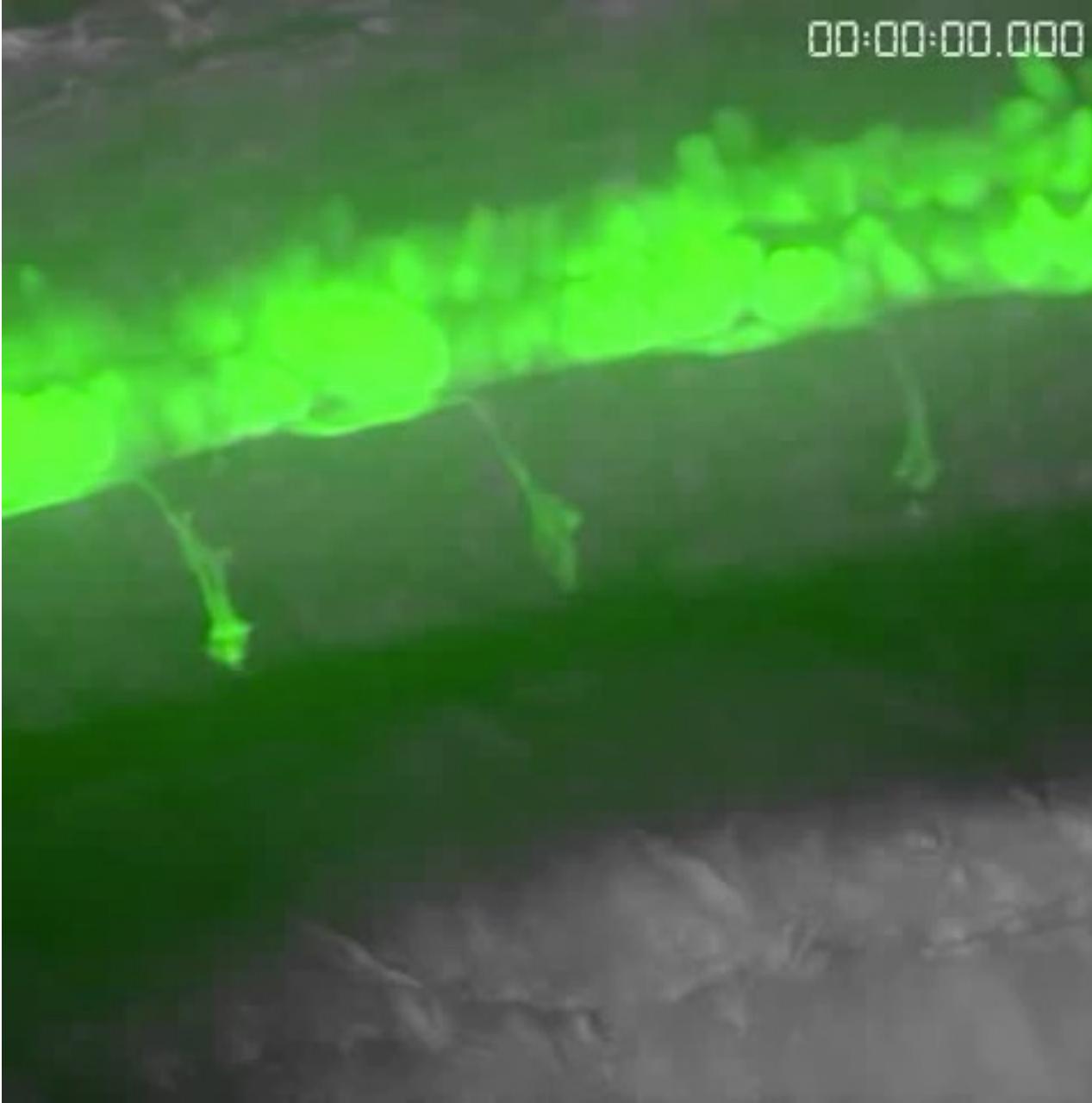
Current tools to visualize individual neurons: genetically encoded, fluorescent proteins

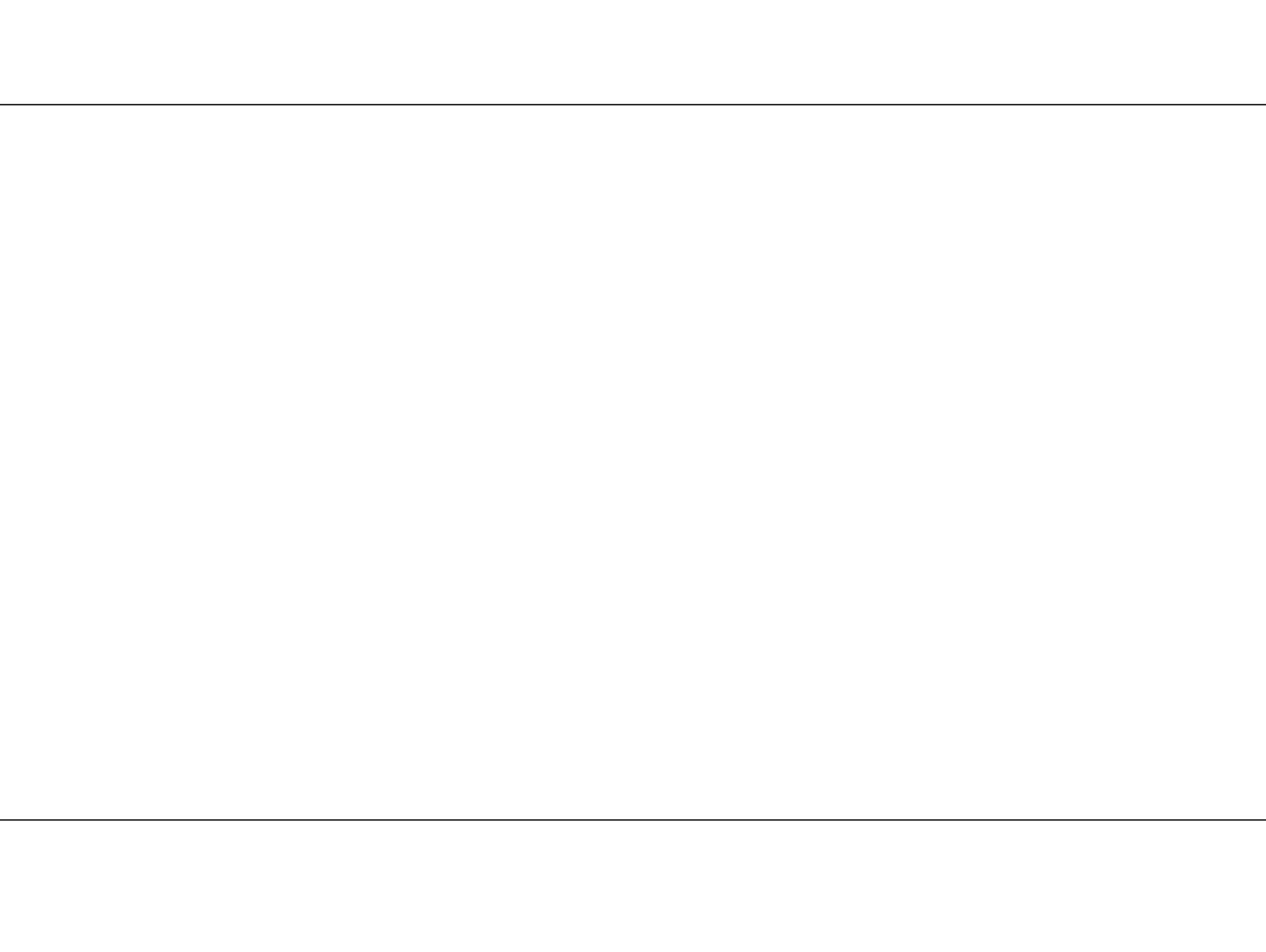


Green Fluorescent Protein

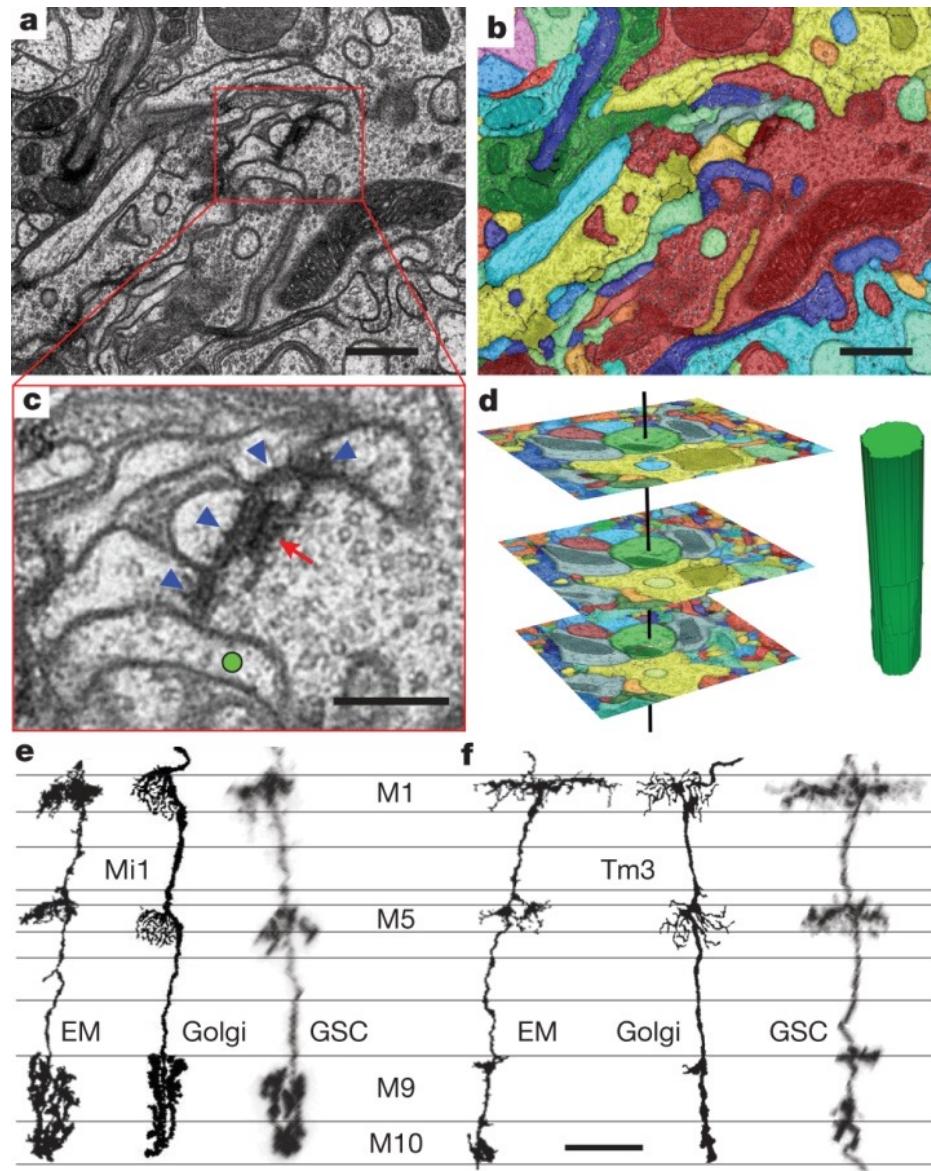


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Current tools to visualize neurons and circuits: electron microscopy (EM)



Takemura et al., Nature 2013

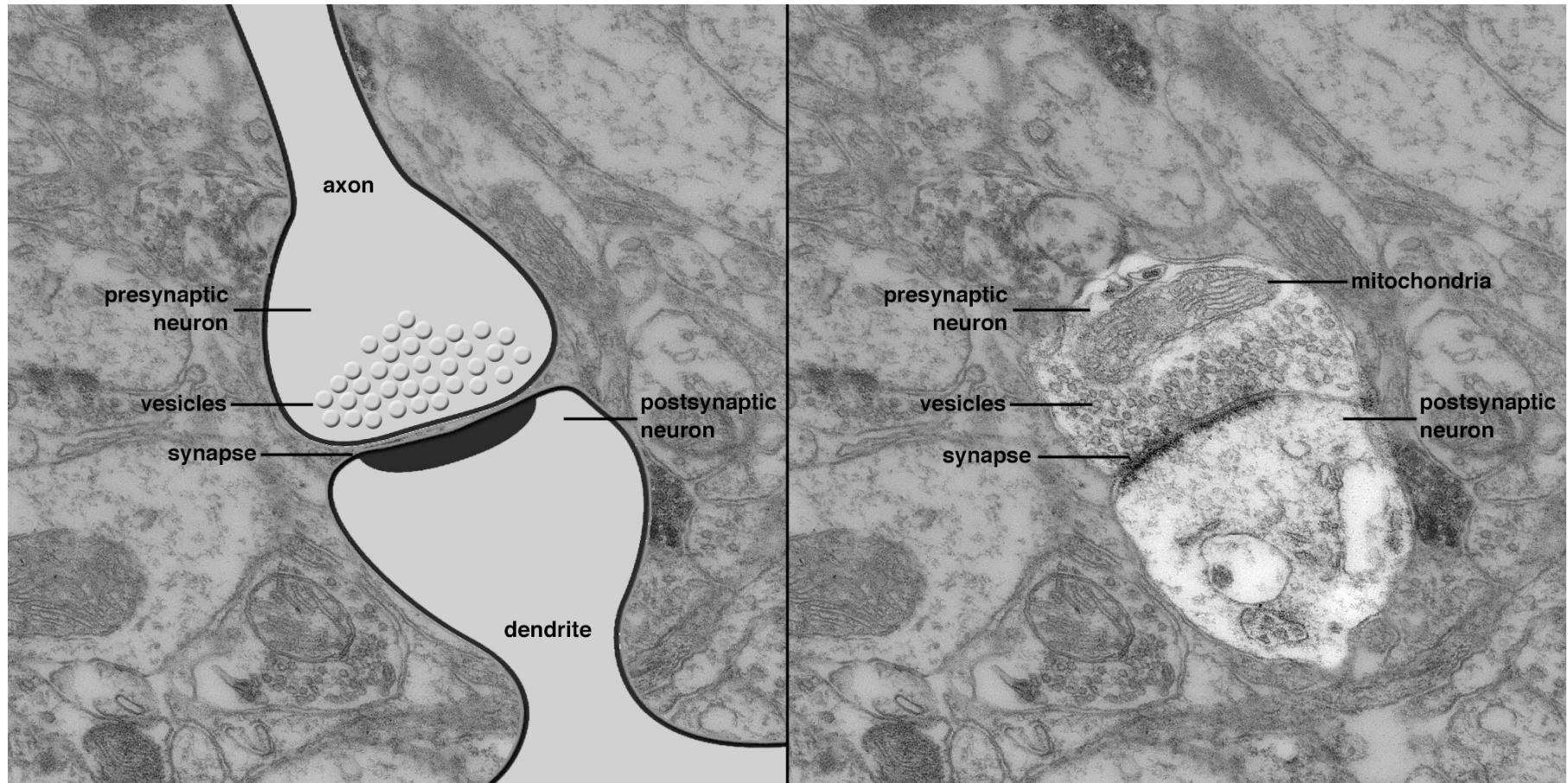
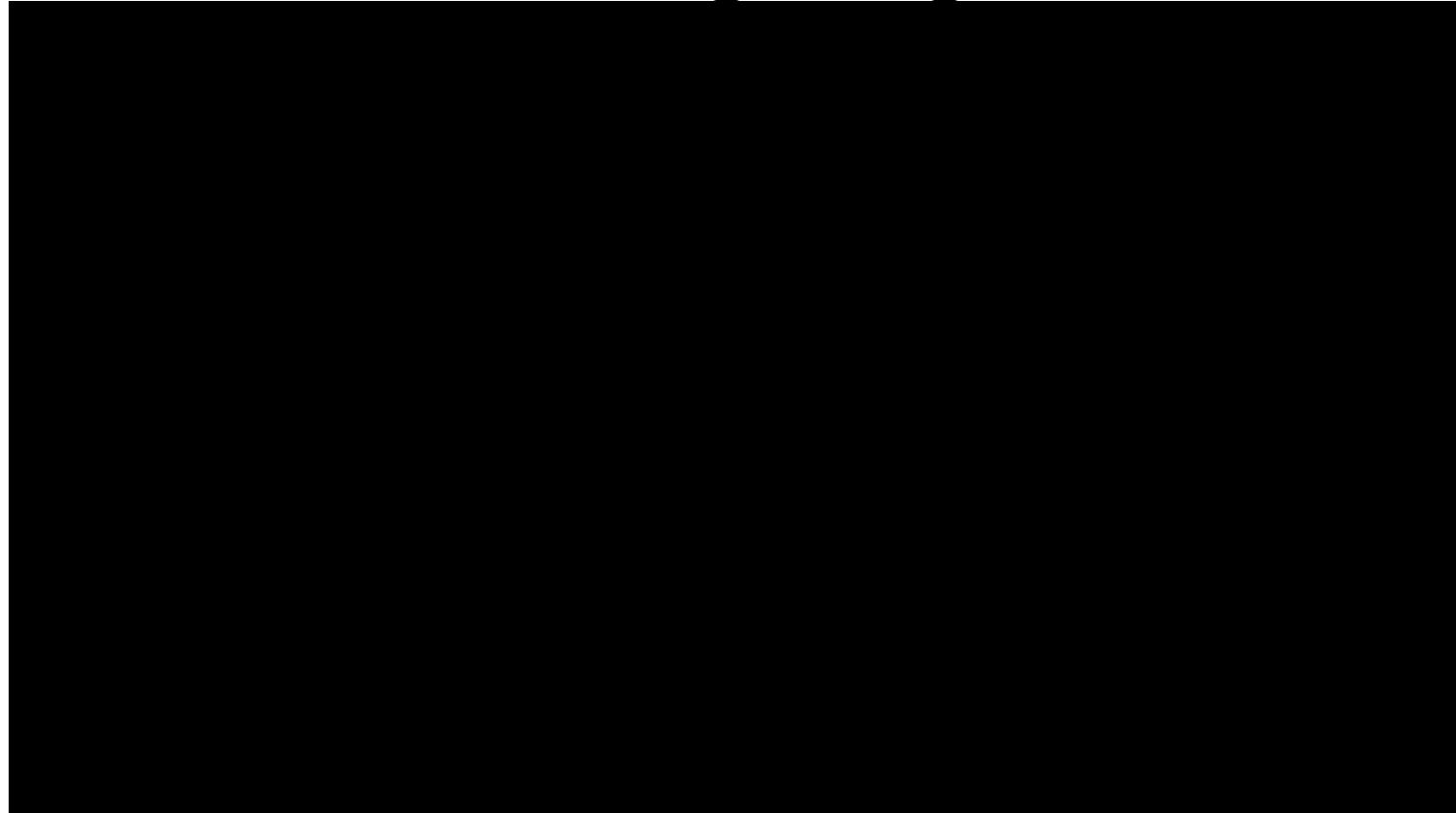
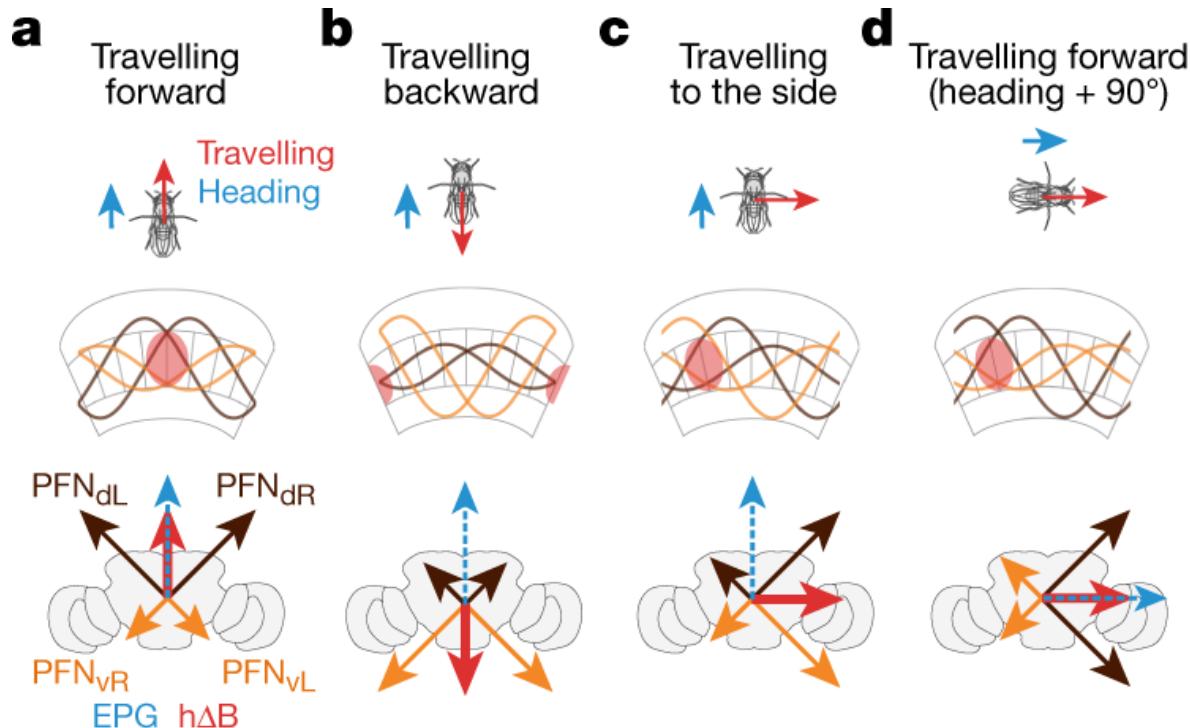


Image from <http://blog.nervousencounter.com> by Melanie King

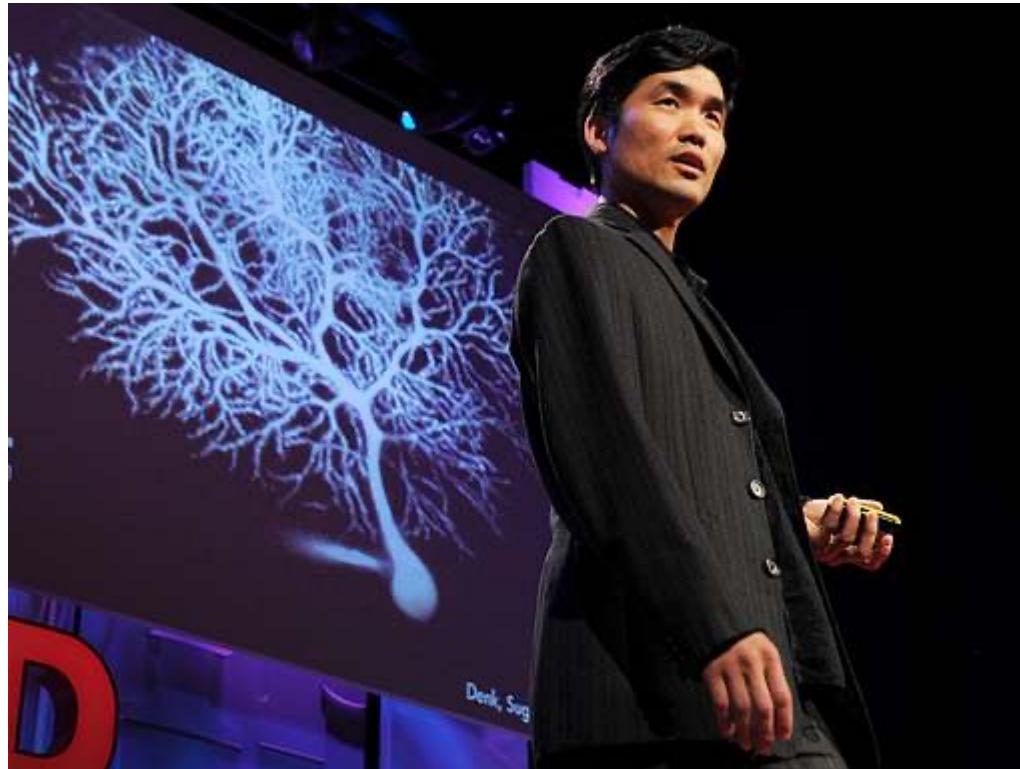
A circuit's wiring diagram..



..is the basis for its function.



“I am my connectome”



https://www.ted.com/speakers/sebastian_seung

Neurodevelopmental disorders – a wiring problem?

TABLE 1 Summary of Genes Associated with NDDs That May Be Involved in Steps of Synapse Assembly

Step in synapse assembly	Gene	Chromosome	Protein function	Alleles	NDD diagnosed	Prevalence	Reference
Neuronal contact	NLGN3	Xq13.1	Synaptic CAM	Mutations	ASD	0.26% (1774) [2]	Jamain <i>et al</i> (2003); Glessner <i>et al</i> (2009)
	NLGN4X	Xp22	Synaptic CAM	Mutations	ASD, ID, TS	2.65% (498) [2]	Jamain <i>et al</i> (2003)
	NLGNI	3q26.31	Synaptic CAM	CNV	ASD	*5.01% (2195) [1]	Glessner <i>et al</i> (2009)
	NRXN1	2p16.3	Synaptic CAM	CNV, mutations, microdel.	ASD, SCZ	0.42% (2380) [3]	Awadalla <i>et al</i> (2010); Reichelt <i>et al</i> (2012)
	CADM1	11q23.2	Synaptic CAM	Mutations	ASD	1.00% (194) [1]	Zhiling <i>et al</i> (2008)
Transport	MAPT	17q21.31	Microtubule associated	CNV	ASD, SCZ, ID	0.14% (14270) [2]	Rovelet-Lecrux <i>et al</i> (2012)
	KIF17	1p36.12	Motor protein	Nonsense mutation	SCZ	0.29% (710) [2]	Awadalla <i>et al</i> (2010); Tarabeux <i>et al</i> (2010)
	GSK3B	3q13.33	Kinase	SSM	SCZ	NA (459) [1]	Blasi <i>et al</i> (2013)
Recruitment and maintenance	SHANK1	19q13.3	Scaffolding protein	Microdeletions	ASD	0.31% (1614) [2]	Sato <i>et al</i> (2012)
	SHANK2	11q13.2	Scaffolding protein	Deletions, SSM	ASD, ID	3.41% (851) [2]	Berkel <i>et al</i> , (2010); Leblond <i>et al</i> (2012)
	SHANK3	22q13.3	Scaffolding protein	Mutations	ASD, SCZ, mild ID	0.74% (1466) [2]	Awadalla <i>et al</i> , (2010)
	CDK5R1	17q11.2	Kinase regulator	Mutations	ID	1.00% (100) [1]	Venturin <i>et al</i> (2006)
	CASK	Xp11.4	Scaffolding protein	Mutations	XLMR, MIC-PCH	**4.43% (557) [3]	Hackett <i>et al</i> (2009); Najm <i>et al</i> (2008)

Abbreviations: ASD, autism spectrum disorder; CAM, cell adhesion molecule; CNV, copy number variation; ID, intellectual disability; MIC-PCH, microcephaly and disproportionate pontine cerebellar hypoplasia; NA, not available; SSM, splice site mutation; TS, Tourette's syndrome; XLMR, X-linked mental retardation.

Prevalence is quantified as the average percentage of the sample's affected population with the identified alleles. The total sample size and the number of studies are in round and square parentheses, respectively. *NLGNI CNVs were detected in control populations, resulting in an odds ratio of only 1.47; **CASK mutations were determined using a very specific disease population, XLMR and MIC-PCH, and thus does not represent the prevalence in the NDD population.

Wiring issues may also underlie neurodegenerative diseases (or other neurological disorders)

Pediatric Neurology 123 (2021) 67–76



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journal homepage: www.elsevier.com/locate/pnu



Topical Review

Neurodevelopmental Clues to Neurodegeneration

Nina F. Schor, MD, PhD ^{a,*}, Diana W. Bianchi, MD ^b



^a National Institute of Neurological Disorders and Stroke, National Institutes of Health, Bethesda, Maryland

^b Eunice Kennedy Shriver National Institute of Child Health and Human Development, National Institutes of Health, Bethesda, Maryland

“our hypothesis, first proposed in 2010, that proteins implicated in neurodegenerative disorders play important roles in brain development”

<https://www.sciencedirect.com/science/article/pii/S0887899421001533>

Future EM (bigger brains)



<https://alleninstitute.org/materials-library/>

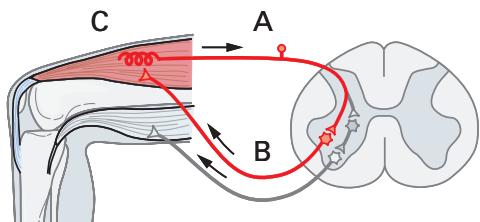
What can we learn from anatomy?

1. What hypotheses can we generate?
2. Where are we limited in our understanding?

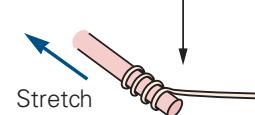
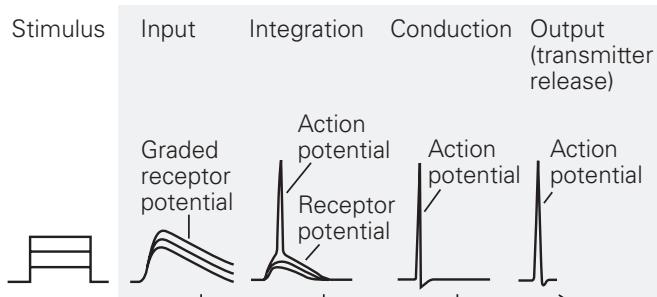
Our Focus

- What is the physical basis of neural signaling?
- How can we observe these processes?
- How can we model these processes?
- How can we then apply these understandings to engineer and interface with the nervous system?

The complexity of neural signals

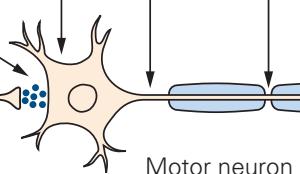
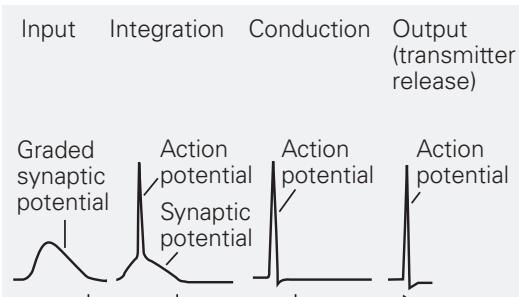


A Sensory signals



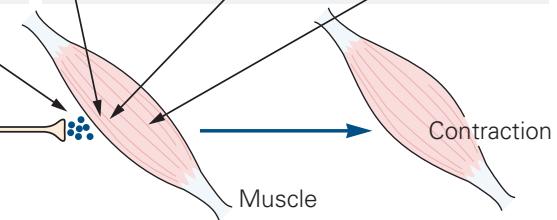
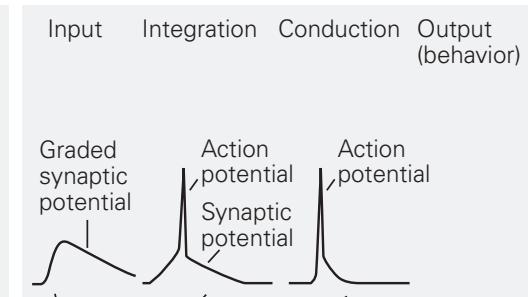
Muscle spindle

B Motor signals



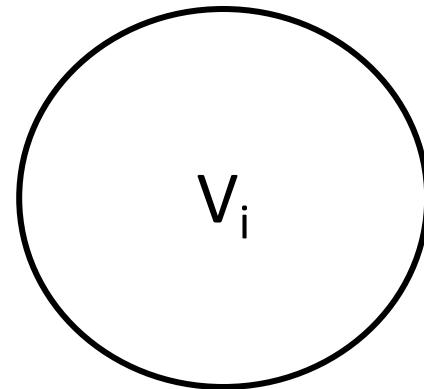
Sensory neuron

C Muscle signals



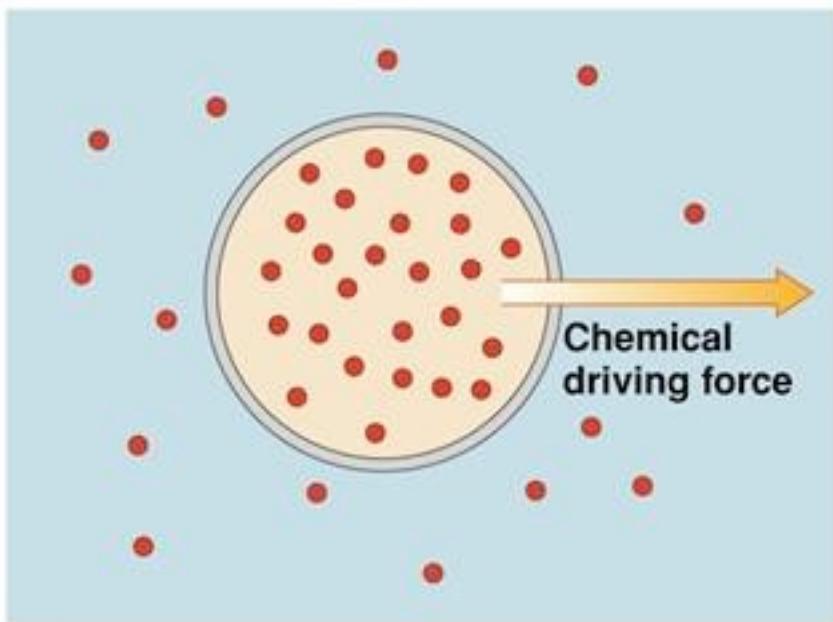
Muscle

A very basic, first model

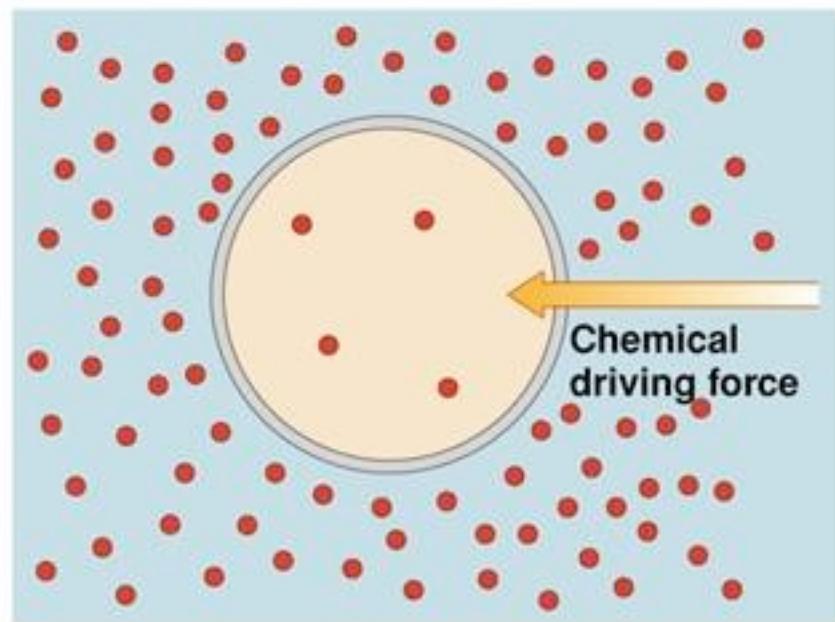


- Isopotential cell
 - Selectively permeable membrane
 - Single ion with different intracellular and extracellular concentrations

Chemical driving force



(a)

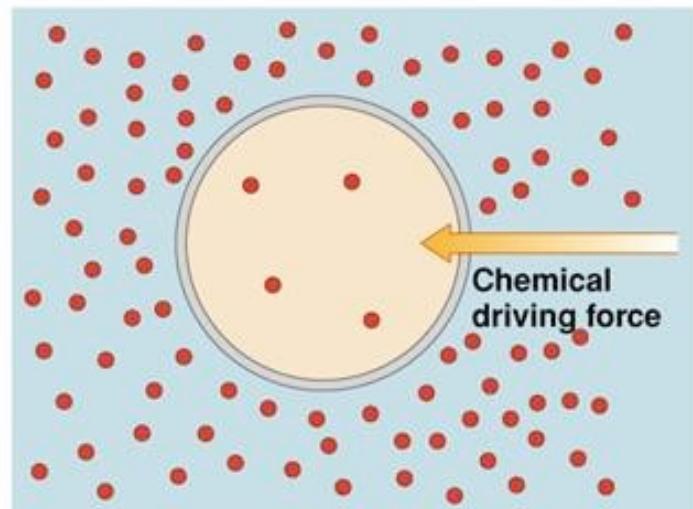


(b)

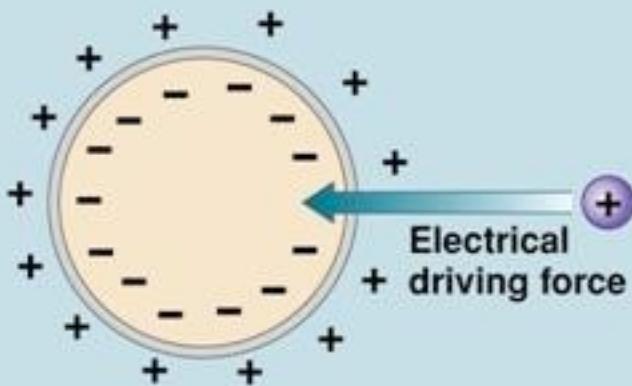
Fick's Law of Diffusion

$$\text{Ion Flux} = J_{\text{diffusion}} = - D \frac{\partial [C]}{\partial x}$$

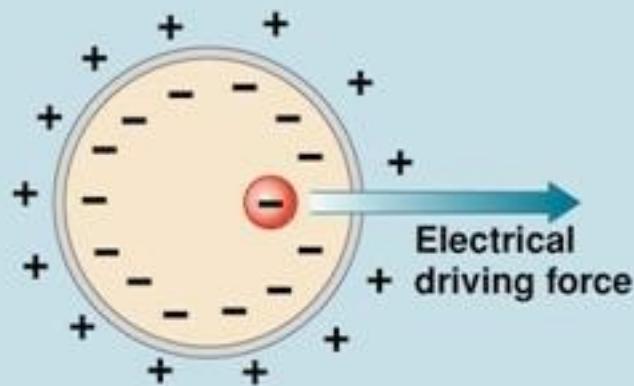
- $J_{\text{diffusion}}$ is the diffusion flux, amount flowing across a unit area per unit time (molecules/cm²s)
- D is the diffusion coefficient (cm²/s) that factors in temperature, the mass of the molecule, and the medium the molecule is diffusing in.
- C is the concentration of the ions (molecules/cm³)



Electrical driving force



(a)



(b)

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Ohm's Law of Drift

$$\text{Ion Drift} = J_{drift} = -z[C]\mu \frac{\partial V}{\partial x}$$

- J_{drift} is the drift flux, amount flowing across a unit area per unit time (molecules/cm²s)
- z is the ion valence
- C is the concentration of the ions (molecules/cm³)
- μ is the electric mobility of a charged particle (cm²/sV)
- V is the potential

Nernst-Plank Equation

The movement of ions across a semipermeable membrane can be exactly described by the linear combination of ion diffusion and drift flux:

$$J = J_{diffusion} + J_{drift}$$

$$J = -D \frac{\partial [C]}{\partial x} - z[C]\mu \frac{\partial V}{\partial x}$$

Einstein's relationship between drift and diffusion

Drift and diffusion are additive processes that can be described by the physical properties of a system:

$$D = \mu \frac{kT}{q} = \mu \frac{RT}{F}$$

- D is the diffusion coefficient (cm^2/s)
- μ is the electric mobility of a charged particle (cm^2/sV)
- q is the elementary charge ($1.6 \times 10^{-19} \text{ C}$)
- k is the Boltzmann constant ($1.3807 \times 10^{-23} \text{ J/K}$)
- T is the absolute temperature ($273.15 + T^{\text{Celsius}} \text{ K}$)
- R is the gas constant, kN_A , ($8.3145 \text{ J mol}^{-1} \text{ K}^{-1}$)
- F is the Faraday Constant, qN_A , ($9.6485 \times 10^4 \text{ C/mol}$)
- Remember, 1 mole contains Avogadro's number of particles ($N_A = 6.02 \times 10^{23}$)

Nernst-Plank Equation

$$J = J_{diffusion} + J_{drift}$$

$$J = -\mu \frac{RT}{F} \frac{\partial [C]}{\partial x} - z[C]\mu \frac{\partial V}{\partial x}$$

$$I = zFJ$$

$$I = -zF\mu \left(\frac{RT}{F} \frac{\partial [C]}{\partial x} + z[C] \frac{\partial V}{\partial x} \right)$$

- I is the current (Amps)

What happens to this equation when the flux is zero (equilibrium, no *net* current)?

$$0 = J_{diffusion} + J_{drift}$$

$$0 = -zF\mu \left(\frac{RT}{F} \frac{\partial [C]}{\partial x} + z[C] \frac{\partial V}{\partial x} \right)$$

The Nernst Equation

$$0 = \frac{RT}{F} \frac{\partial [C]}{\partial x} + z[C] \frac{\partial V}{\partial x}$$

$$\frac{RT}{F} \frac{\partial [C]}{\partial x} = -z[C] \frac{\partial V}{\partial x}$$

$$\frac{RT}{F} \partial [C] = -z[C] \partial V$$

$$\int_{[C]_i}^{[C]_o} \frac{1}{[C]} \partial [C] = \int_{Vi}^{V_o} -\frac{zF}{RT} \partial V$$

$$\ln[C]_o - \ln[C]_i = -\frac{zF}{RT} (V_o - Vi)$$

$$(V_i - V_o) = \frac{RT}{zF} \ln \frac{[C]_o}{[C]_i}$$

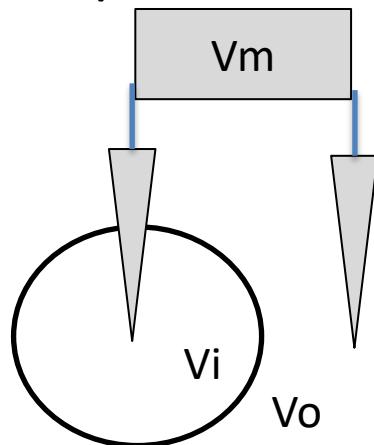
The Nernst Equation

At equilibrium, the potential at which $J = 0$ for a given ionic species (e.g. Na^+ , Cl^- , etc.), Nernst-Plank reduces to the Nernst Equation:

$$(V_i - V_o) = \frac{RT}{zF} \ln \frac{[C]_o}{[C]_i}$$

The Nernst Equation

At equilibrium, the potential at which $J = 0$ for a given ionic species (e.g. Na^+ , Cl^- , etc.), Nernst-Plank reduces to the Nernst Equation:



$$V_m = V_i - V_o$$

$$V_m = E_x = \frac{RT}{zF} \ln \frac{[x]_o}{[x]_i}$$

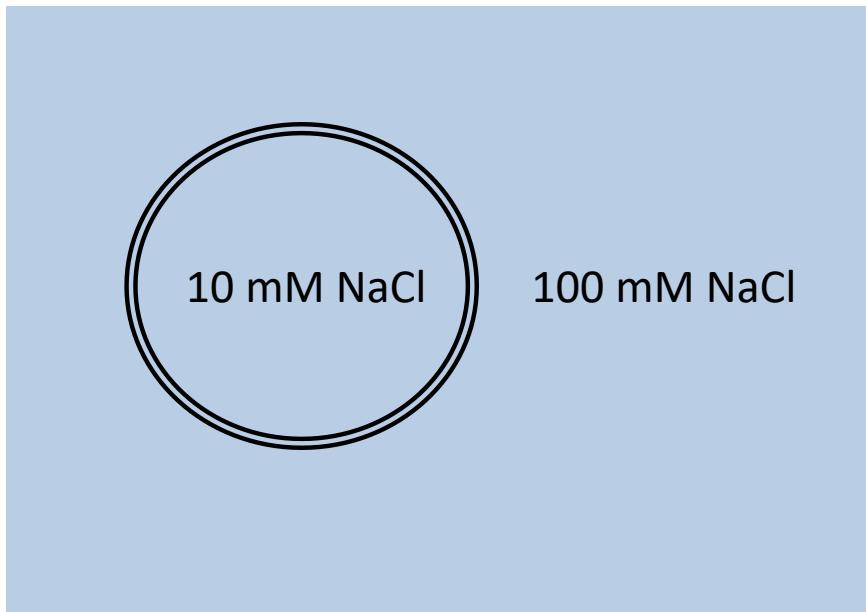
Iff permeable to that
one ionic species

- E_x is the equilibrium/Nernst/reversal potential for a given ionic species x
- $[x]_o$ is the external concentration, $[x]_i$ is the internal concentration

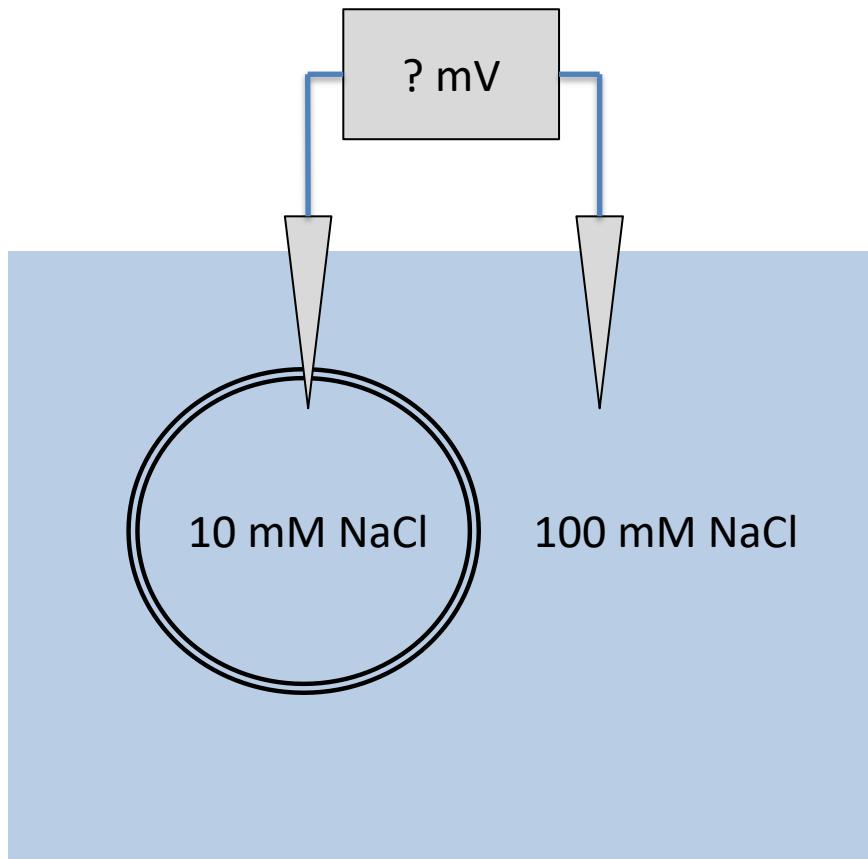
Common ion distributions within neurons

Ion	[C] _{out} mM	[C] _{in} mM	[Ex]/[In]	EQ mV
Na ⁺	145	5-15	14.5	+90/+61
K ⁺	5	140	0.036	-89.7
Ca ²⁺	2.5-5	1-2(10 ⁻⁴ free)	25000	+136/+145
Cl ⁻	110	4	27.5	-89

Example Problems

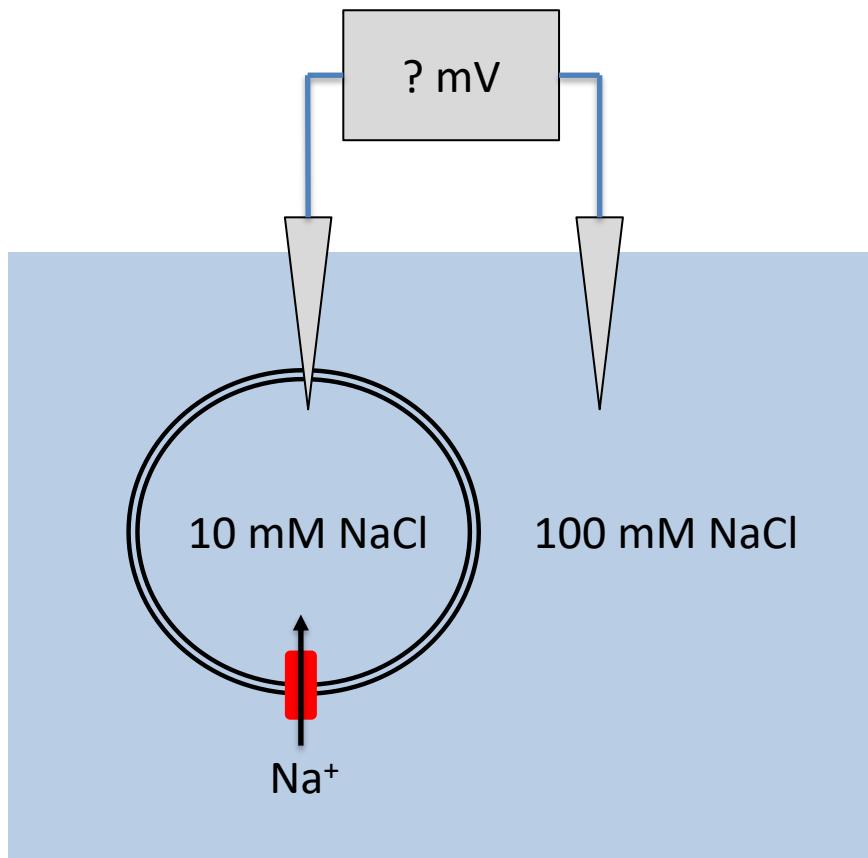


Example Problems



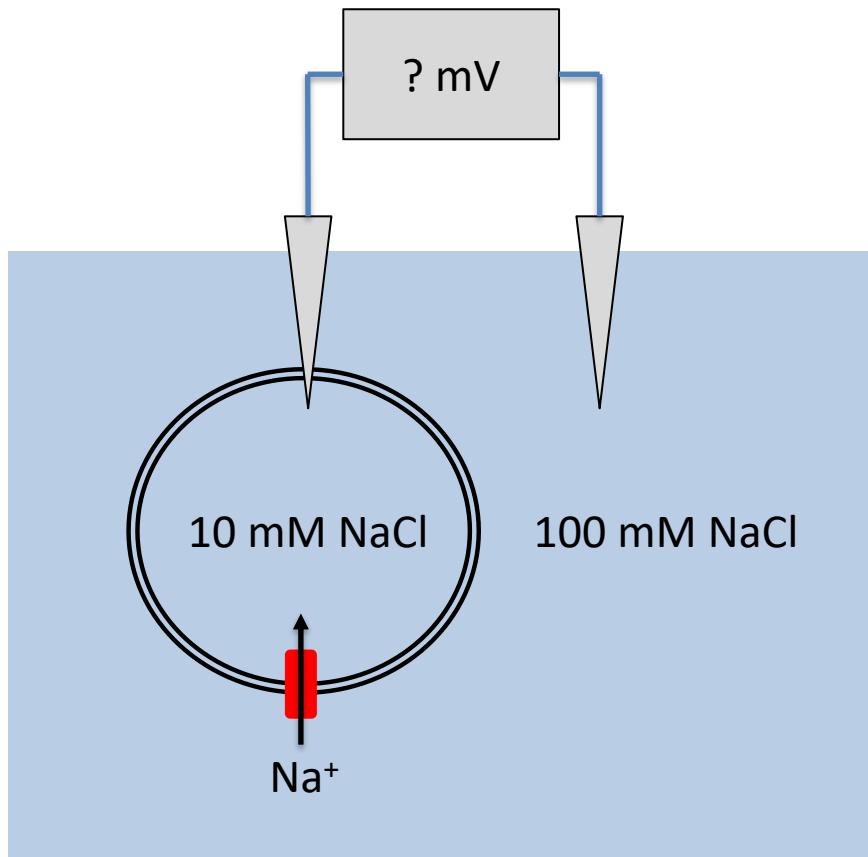
$$RT/F = 25.7$$

Example Problems



$$\text{RT/F} = 25.7$$

Example Problems

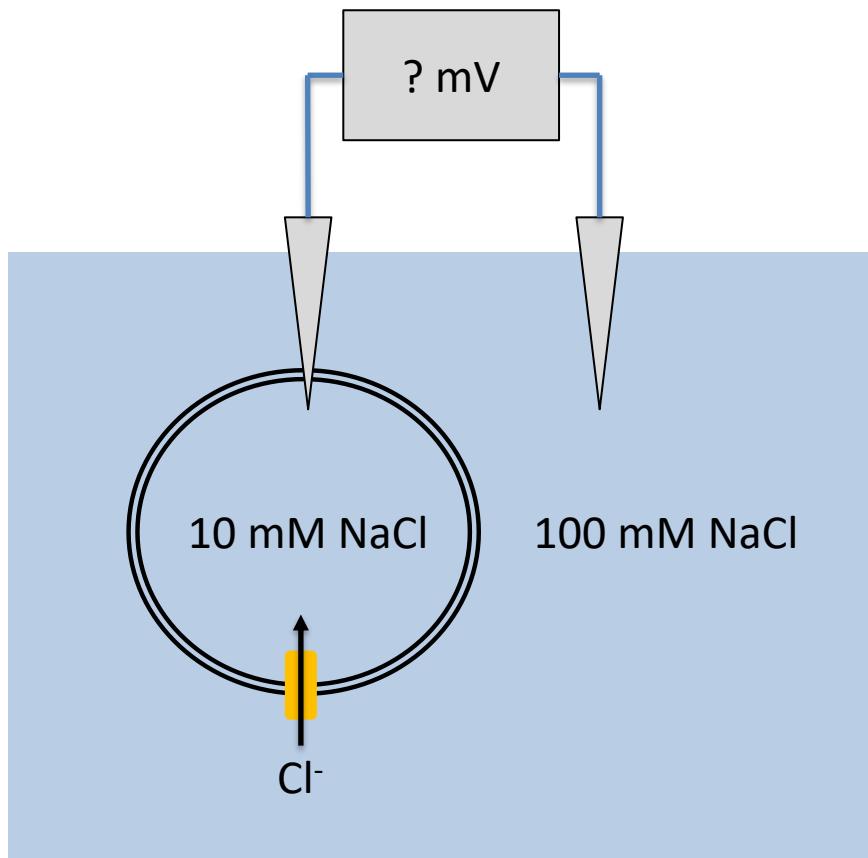


$$E_{Na} = \frac{RT}{zF} \ln \frac{[Na]_o}{[Na]_i}$$

$$V_m = E_{Na} = 59 \text{ mV}$$

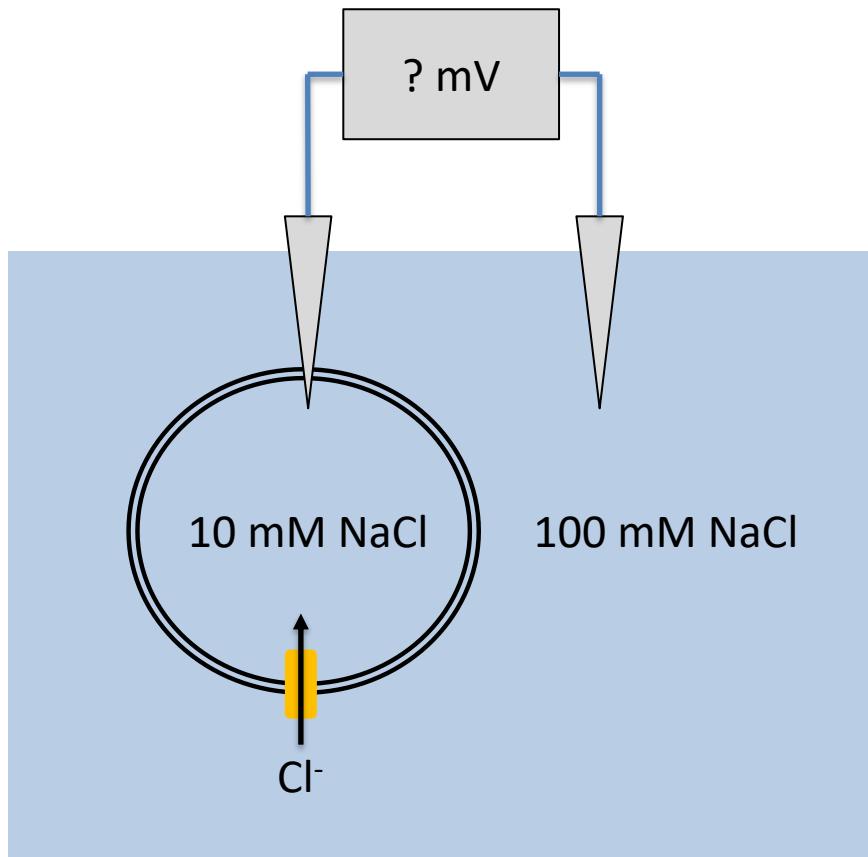
$$RT/F = 25.7$$

Example Problems



$$\text{RT/F} = 25.7$$

Example Problems

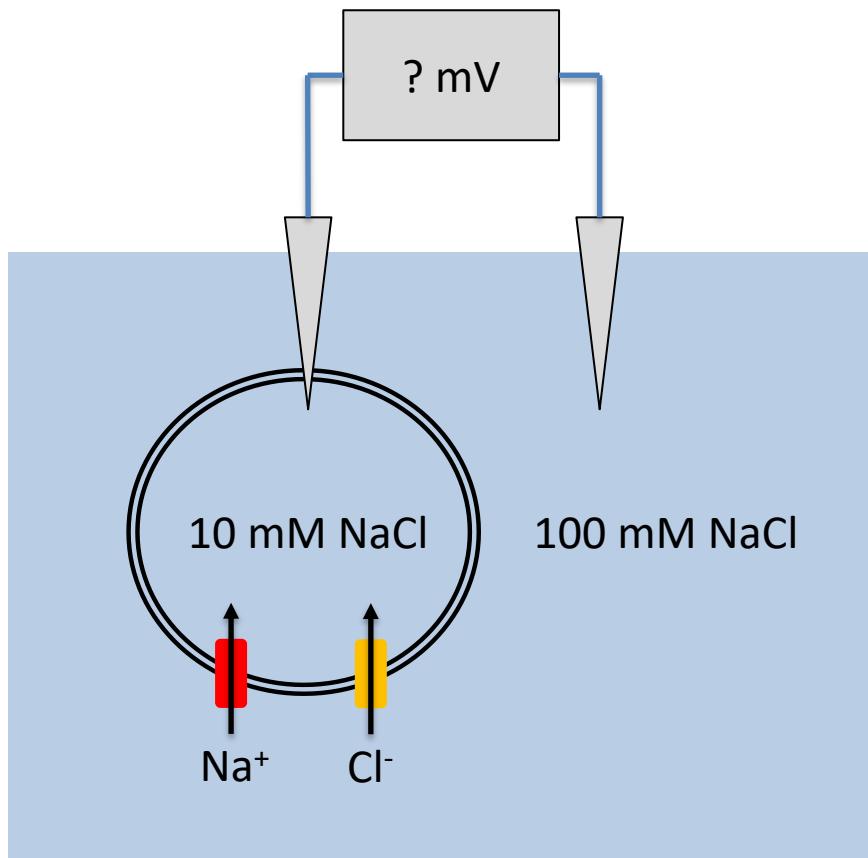


$$E_{Cl} = \frac{RT}{zF} \ln \frac{[Cl]_o}{[Cl]_i}$$

$$V_m = E_{Cl} = -59 \text{ mV}$$

$$RT/F = 25.7$$

Example Problems



$$\text{RT/F} = 25.7$$

Adding complexity to our model to deal with multiple ions

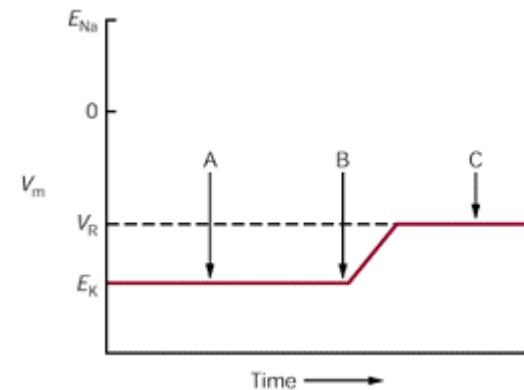
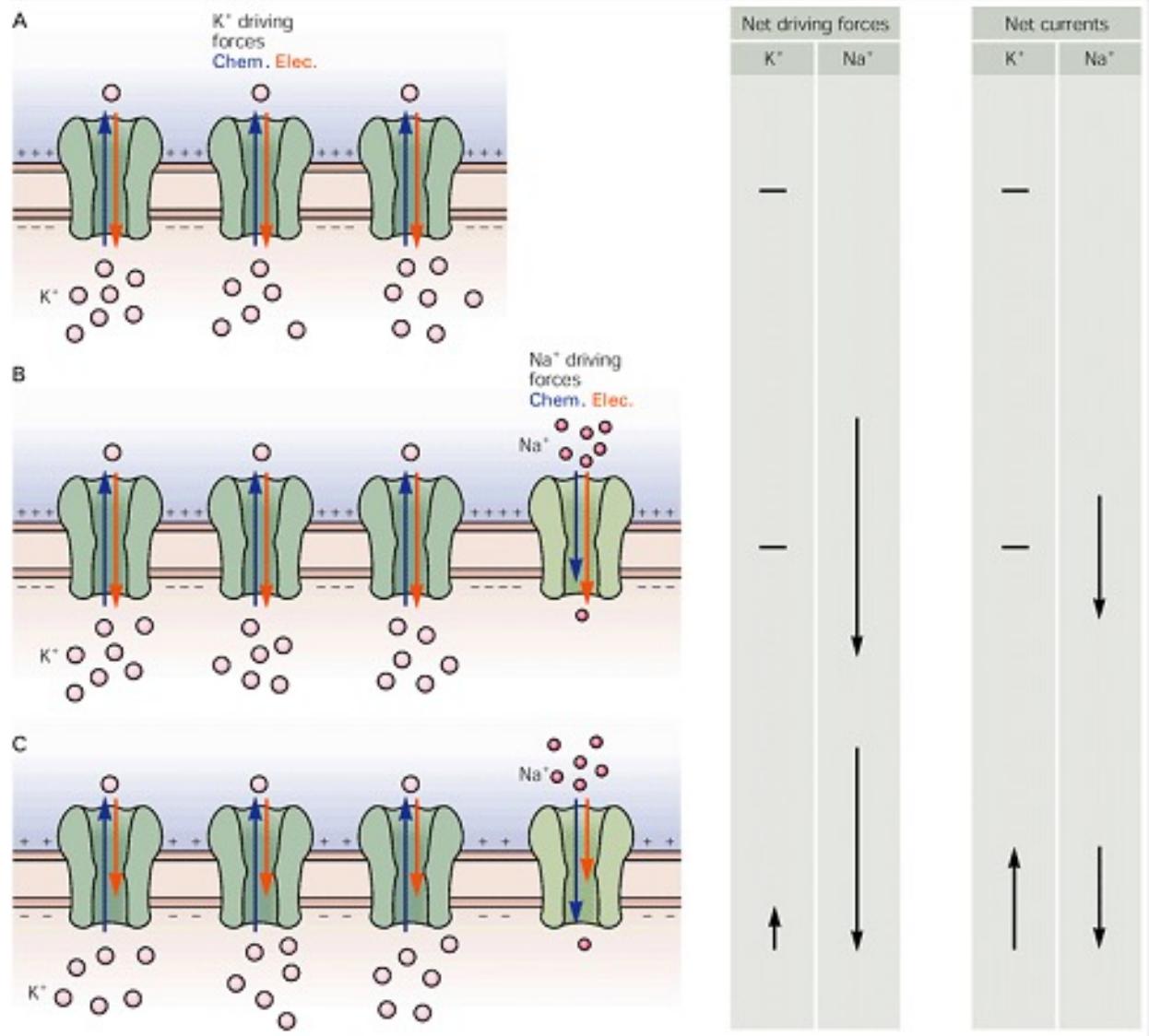
- Isopotential cell
 - Multiple cations and anions at different intracellular and extracellular concentrations.
 - Selectively permeable membrane where the permeabilities for each ion can change in time.

Driving Force

$$Driving\ Force = V_m - E_x$$

- The further V_m is away from the Nernst Potential for an ionic species, the larger the driving force for that ion.
- This current that will flow through a channel that is selective to that ionic species (if it is open) is proportional to the driving force.
- If the resting membrane potential is far away from the Nernst Potential for an ionic species, opening channels for that ionic species will have a large effect on the membrane potential.

Example: K⁺ and Na⁺



Goldman-Hodgkin-Katz equation

- For multiple ionic species, the resting membrane potential depends on the permeability of the channels present in the membrane and the concentration gradients for their selected ions.

$$V_m = \frac{RT}{F} \ln \frac{P_K[K]_o + P_{Na}[Na]_o + P_{Cl}[Cl]_i}{P_K[K]_i + P_{Na}[Na]_i + P_{Cl}[Cl]_o}$$

- Notice that V_m is not equal to any reversal (Nernst) potential except when?
- Remember that this equation only applies when the net ion flux (net current) across the membrane is equal to zero (when it is at steady state).

Example Problem

During rest, permeability ratios are as follows:

$$P_K:P_{Na}:P_{Cl} = 1:0.03:0.1$$

And ion distributions are as below:

Concentration (mM)	K ⁺	Na ⁺	Cl ⁻
Inside	400	50	40
Outside	10	460	540

Calculate the resting potential. How does this compare to the equilibrium potentials for each ion independently?

$$RT/F = 25.7$$

$$V_{rest} = \frac{RT}{F} \ln \frac{P_K[K]_o + P_{Na}[Na]_o + P_{Cl}[Cl]_i}{P_K[K]_i + P_{Na}[Na]_i + P_{Cl}[Cl]_o}$$

$$V_{rest} = -72 \text{ mV}$$

Maintaining gradients

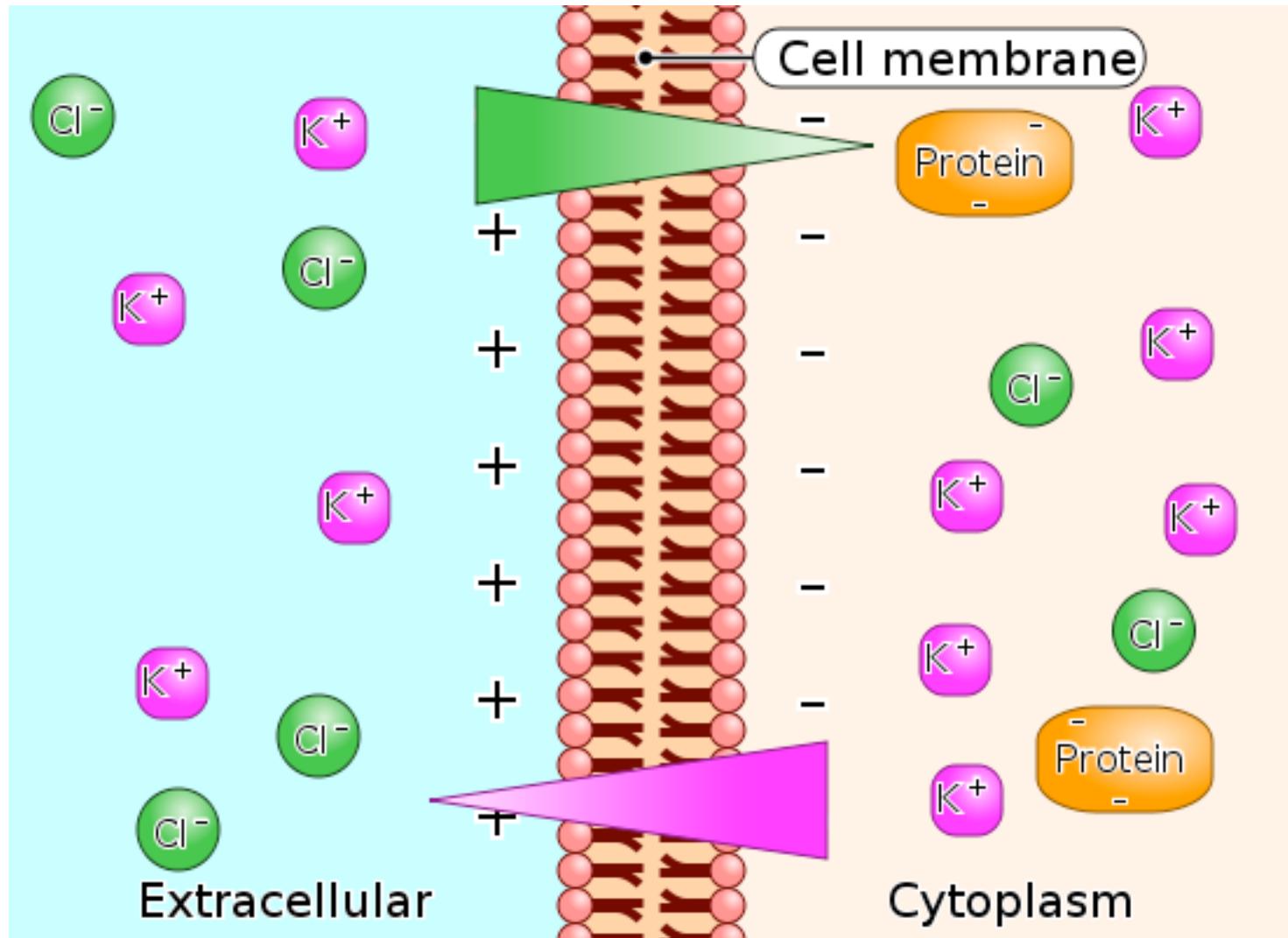
- To effectively communicate, both intra- and extracellular gradients must be maintained.
- However, both diffusion and drift are gradient-neutralizing...
- ...and Nernst and GHK assume that gradients are maintained. Are they valid models?

In neurons, gradients are maintained..

- **Passively:**
 - Through impermeant charged molecules and proteins (**Donnan Equilibrium**)
- **with Active Transport (require ATP)**
 - Sodium/potassium or calcium pumps
- **with Exchangers**
 - Sodium/calcium ion exchange
 - Ion co-transporters

Creating gradients with impermeable ionic species

- Isopotential cell
 - A semi-permeable membrane
 - Multiple cations and anions at different intracellular and extracellular concentrations that are membrane permeant.
 - Intracellular charged proteins or large molecules that are membrane impermeant.



https://en.wikipedia.org/wiki/Gibbs-Donnan_effect

Donnan Equilibrium rule

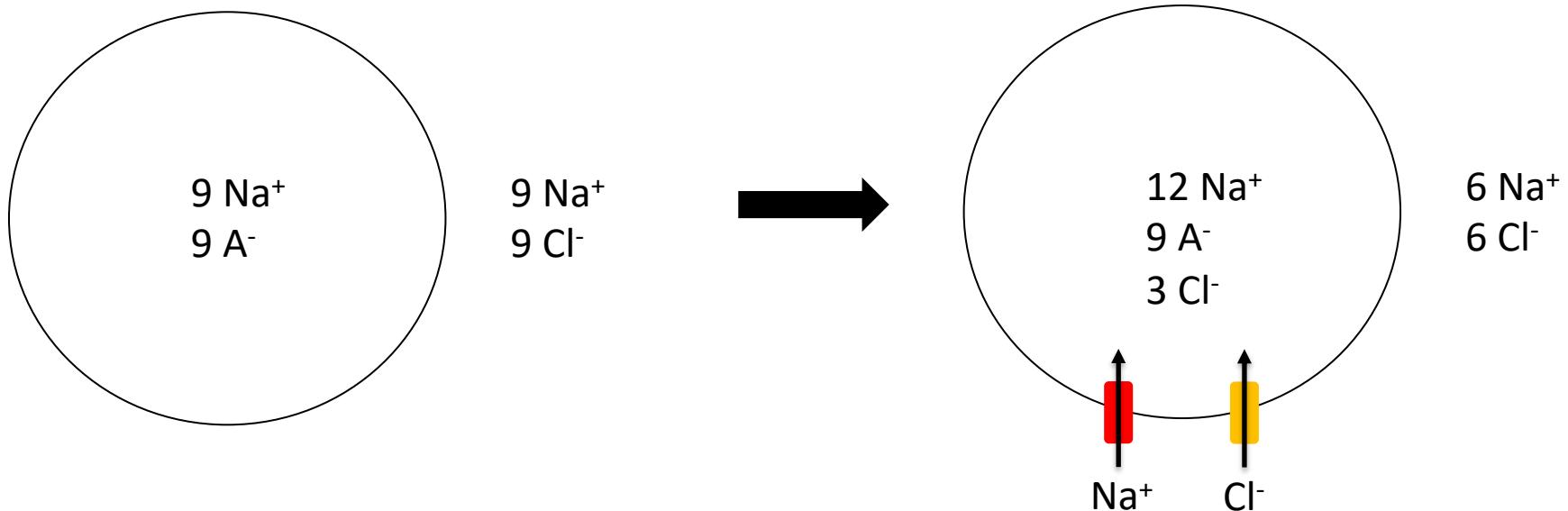
- At equilibrium, cations and anions have to balance:

$$\left(\frac{C_{in}^{+m}}{C_{out}^{+m}} \right)^{1/m} = \left(\frac{C_{out}^{-n}}{C_{in}^{-n}} \right)^{1/n}$$

Simple Donnan Equilibrium example

$$\left(\frac{C_{in}^{+m}}{C_{out}^{+m}}\right)^{1/m} = \left(\frac{C_{out}^{-n}}{C_{in}^{-n}}\right)^{1/n}$$

$$\frac{[Na]_{in}}{[Na]_{out}} = \frac{[Cl]_{out}}{[Cl]_{in}}$$



Pumps (active transport)

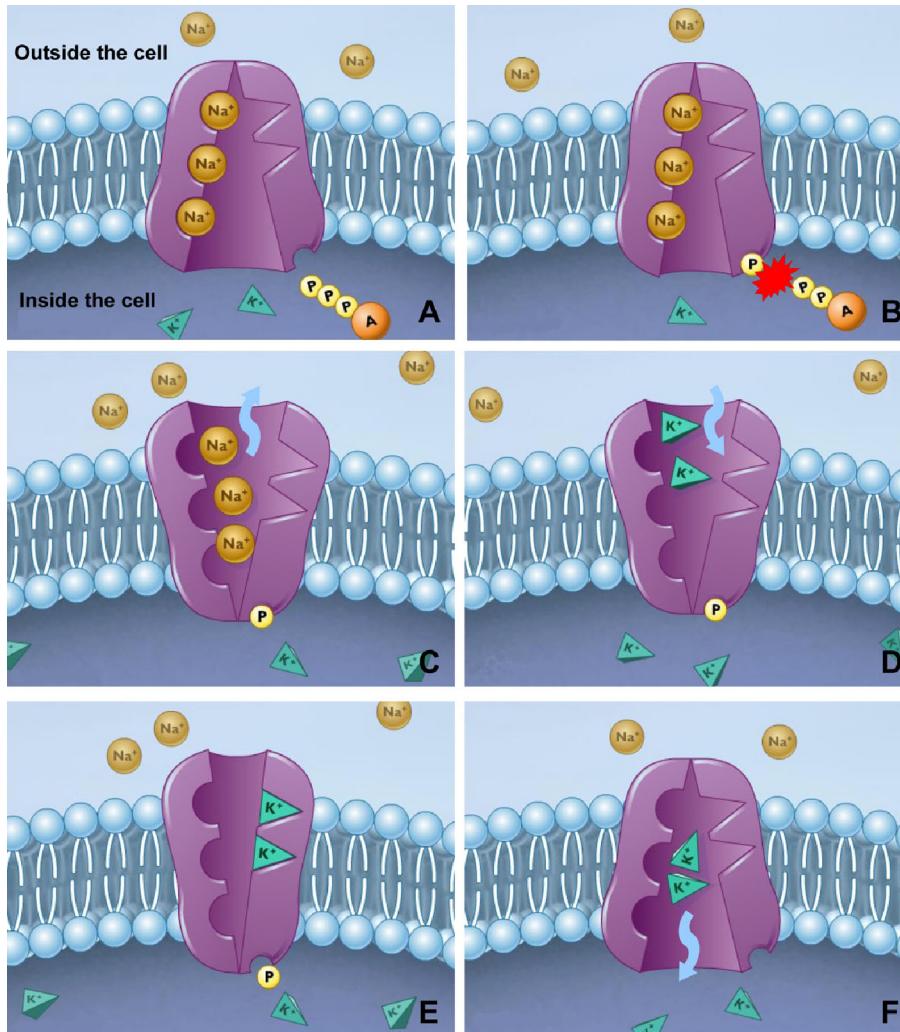


Figure 1 The sodium-potassium exchange pump mechanism. The Na,K-pump that moves two potassium ions from outside the cell to inside and three sodium ions from inside the cell to outside by the breakdown of ATP molecules.

Exchangers and cotransporters

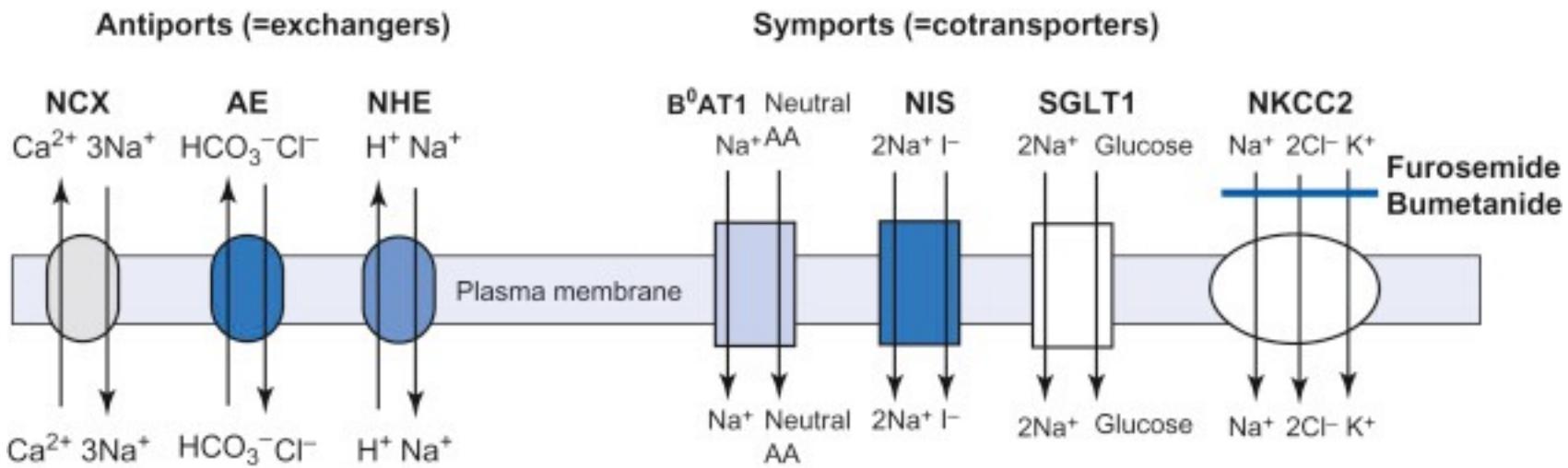


Table 1. Summary of the diseases caused by mutation in two Na^+/K^+ pump genes

	Na ⁺ /K ⁺ Pump gene	Somatic vs Germline	Mutation	Na ⁺ /K ⁺ Pump Function Loss	Inward Current	Dominant Negative
Primary aldosteronism (PA)	ATP1A1	Somatic de novo recurrent	L104R, V332G delF100_L104 delM102_L103 delM102_L106 delL103_L104 del delI322_I325 delF956_E961 delF959_E961 delE960_L964 EETA963S G99R I327S	Yes	Yes	Unlikely
Charcot-Marie Tooth (CMT) 2	ATP1A1	Germline familial	L48R P600A P600T A597T D601F D811A	Yes	No	Unknown
Intermediate CMT	ATP1A1	Germline familial	F207S	Yes	Unknown	Likely
Hypomagnesemia accompanied by seizures and cognitive delay (HASCD)	ATP1A1	Germline de novo	G877S L302R G303R M857R	Yes	Likely	Unknown
Complex spastic paraparesis (CSP) Borderline learning impairment/ sleep disorder/poor emotional control Severe developmental delay/ focal seizures Isolated dominant hypomagnesemia (IDH)	ATP1A1	Germline de novo	L337P	Yes	Unclear	Unknown
	ATP1A1	Germline de novo	G864R	Not tested	Unknown	Unknown
	ATP1A1	Germline de novo	D933N	Yes	No	Unknown
	FXYD2	Germline familial	G41R	Yes	Unclear	Likely

Additional reading

Paul Miller

Ch 1.1-1.3 & 2.1

An Introductory Course in
**COMPUTATIONAL
NEUROSCIENCE**

Chapter 1 The Hodgkin–Huxley Equations

G.B. Ermentrout and D.H. Terman, *Mathematical Foundations of Neuroscience*,
Interdisciplinary Applied Mathematics 35, DOI 10.1007/978-0-387-87708-2_1,
© Springer Science+Business Media, LLC 2010

What is missing from our models?

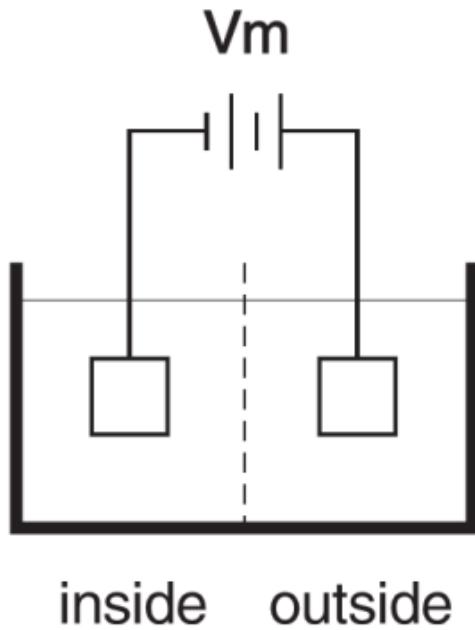
- We can use Nernst, GHK, and Donnan to predict a neuron's membrane potential when the net flux (net current) is zero.
- What happens in the dynamic state, when the net current is not zero? For example, if we inject current into our isopotential neuron model?

Next time

- Short quiz (Nernst, GHK, driving force)
 - Bring your calculator
- Introduction equivalent circuits

Lab 1 is due Monday at 11:59 PM

Lab 1



You have a container with two compartments of equal volume: “inside” (intracellular) and “outside” (extracellular). Each compartment contains an ionic solution in water. They are separated by a semi-permeable membrane that allows one of the ionic species to pass through it. Electrodes from an external voltage source (a **“voltage clamp”**) are inserted into the compartments. The voltage clamp lets you control the transmembrane potential V_m .

Lab Assignments

For all Lab Assignments, make sure to

1. Paste all figures that you generated into a word document.
2. For each question, describe what you observed and answer the question in the word doc.
3. Upload code (for labs where you have generated your own code) as a .m file. We grade your word document, but will run your code to make sure it is working.