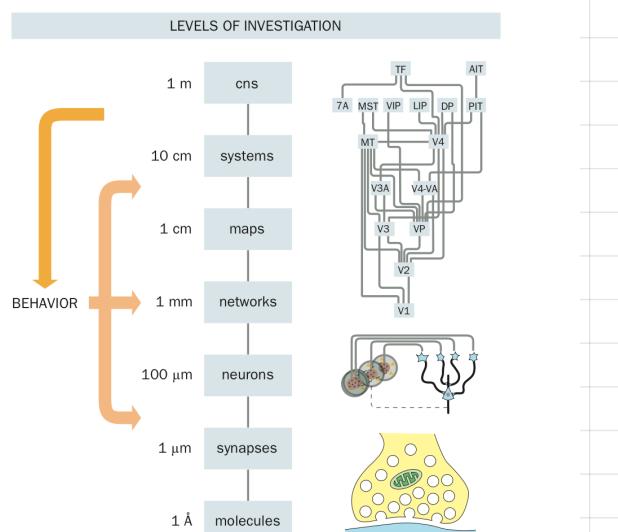


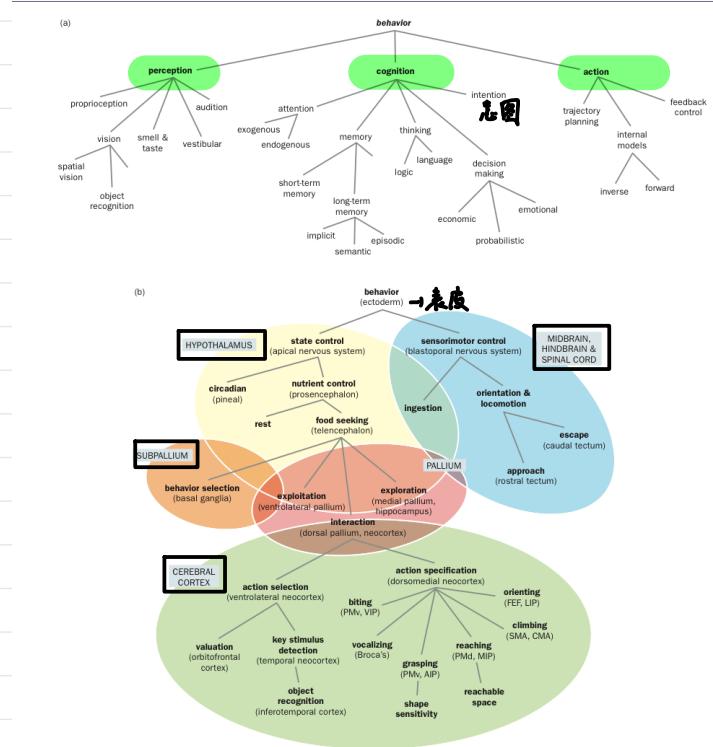
# Chapter I : Understanding the Cognitive Brain

1. Two kinds of (basic) philosophical questions:
  - ① one aims at providing insights from a neuroscientific perspective into translating puzzles such as **consciousness**
  - ② the other address the field's **epistemology question** like what constitutes an explanation in brain science
2. **Epistemology questions:** what does it mean to understand the brain and its functions  
how can we improve our philosophical methodologies in neuroscience.
3. A most important issue  $\Rightarrow$  the relationship between behaviour and neural circuit.
  - A popular guide to tackle the problem (by David Marr)**  $\Rightarrow$ 
    - (1) formulate the problem and identify its **normative solution**
    - (2) search for **computational algorithms** that accomplish the optimal solution
    - (3) elucidate **hardware implementations** of such algorithm in the brain

**Marrian Framework**
4. Unidirection function  $\rightarrow$  algorithm  $\rightarrow$  implementation
5. "From inside out approach"  $\rightarrow$  the brain is as active "at rest" in the absence of obvious external stimulation as during task
  - $\hookrightarrow$  conceptualization of brain merely as an input (stimulus) - output (behaviour) information processing system seems quite limited
6. The viewpoint of behaviour primacy is linked to the traditional metaphor of brains as computers.
  - $\hookrightarrow$  "multiple realizability"  $\rightarrow$  thoroughly criticized on epistemological grounds.
7. Limitation in "multiple realizability"  $\rightarrow$  we cannot limit ourselves to consider only one "target" function  $\rightarrow$  for PFC, we need to first identify a long list of various cognitive processes, then investigate how many ways they can actually all be realized in a single neural system
8. Brain functions are underpinned by neuroanatomy (connectome)
  - $\hookrightarrow$  we need a profound knowledge about the inner workings of its biological components.
9. Behavior - to - brain  $\Rightarrow$  "Top - down"
10. CNS on multiple spatial scales



## 12. Hierarchy of behaviour



13. Neither the mind nor the brain can be assigned to a single level in a linear Marrian hierarchy

↳ each is a rich world with its own multi-level structure

14. The connections between the two societies are defined through a web of complex interactions across many levels of the two hierarchies of brain and behaviour.

15. The goal of neuroscience is to understand the biological brain mechanisms that causally explain behaviour and cognition

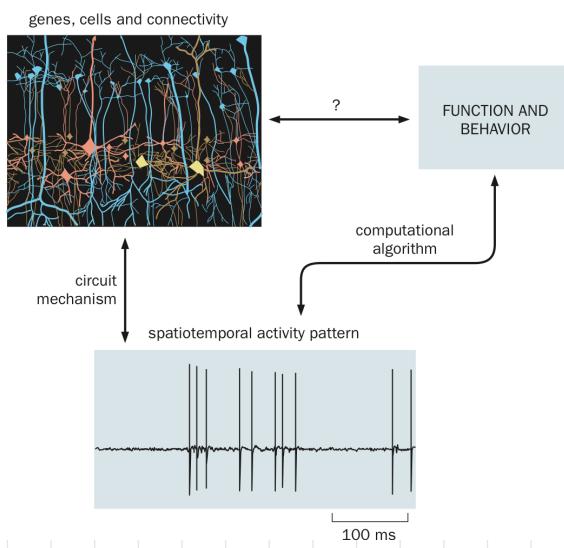
★ 全面

16. To understand the PFC, one would attempt an all-encompassing normative theory the must explain all PFC function



Theory and mathematical models in neuroscience should and eventually will play a similar role as theoretical physics does in physics

18. Theory is needed to: (a) identify computational algorithms carried out by a given empirically measured neural population activity and to assess whether it can explain behaviour;  
(b) elucidate the underlying circuit mechanism, i.e. how the observed neural signals arise from genes, cells and connectivity



19. Cross-level mechanistic understanding

20. Brain  $\Rightarrow$  collective phenomena of interacting neurons described as a large and complex nonlinear dynamical system

21. Brain is plastic  $\rightarrow$  behaviour generated by the brain in turn alters the nervous system on multiple spatial scales from synapses and neurons to circuits

↳ Challenge: achieve understanding across levels for complex systems.

22. To meet this challenge, eschew the computer as a metaphor for the brain.

yet to be  
metaphor

23. ★ Treating a neural circuit as a dynamic system (not just calculation)

The framework of dynamical systems provides a natural mathematical language for describing complex neural population spatiotemporal activity patterns.

24. A dynamic system describes  $\star$  how the current state of a system and interactions between constituent units predict the future evolution of the system over time.

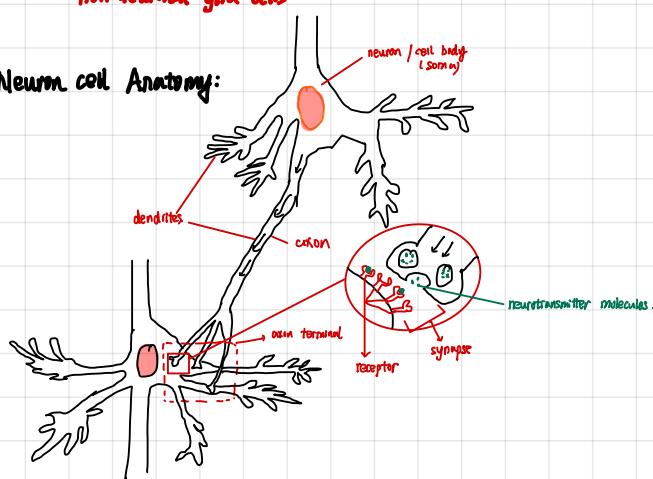
25. Dynamic system  $\Rightarrow$   emergent phenomena  
+  
capture properties of constituent units  $\Rightarrow$  "cross-level understanding"

# Chapter II: Neurons and Synapses

1. Brain

{ nerve cells  
non-neuronal glia cells

2. Neuron cell Anatomy:



3. Primate Prefrontal Cortex (PFC) → "the CEO of the brain" in primates

4. A good starting point ⇒ consider microcircuits within a brain region dedicated to a specific function \*

5. neocortex ⇒ plays a major role in higher brain functions.  
↓

{ excitatory neurons increase the activity of target neurons (majority ⇒ pyramidal cells (pyramid-shaped soma))  
inhibitory neurons ⇒ suppressing target neurons → interneurons → virtually all of them make connections locally within a cortical region

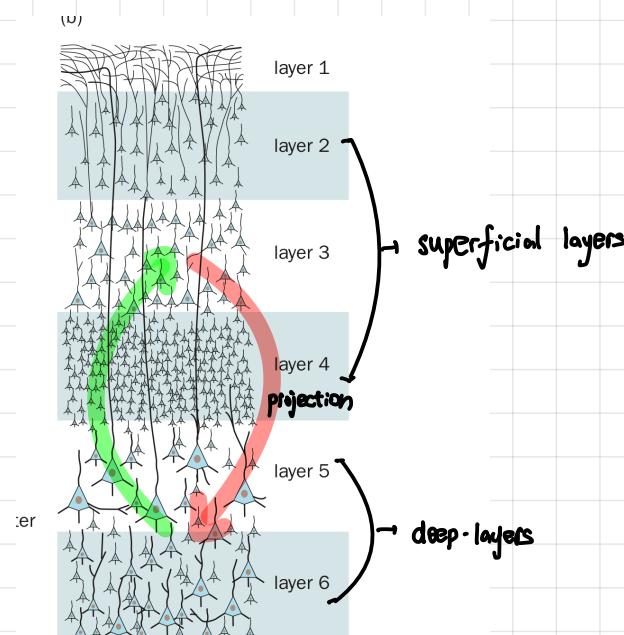
6. Excitatory : Inhibitory ratio varies significantly in different cortical regions.

7. In human, inhibitory neurons →

{ ~30% in PFC

{ ~15% in visual area (V1)

8. Cortical Microcircuit → Laminar Structure



9. LGN: lateral geniculate nucleus

10. Feedback loops within cortical microcircuit plays a role in explaining neural population dynamics and cognitive functions

11. Main anatomical properties of inhibitory interneurons. →

12. Three distinct inhibitory neuron cell:  
according to projections and protein expressions

**PV**

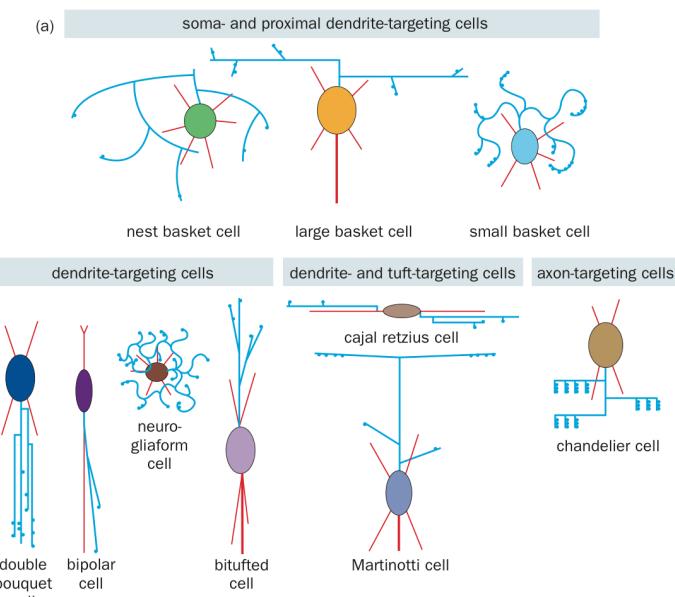
- target the perisomatic region of pyramidal cells and act to control spiking output of excitatory neurons
- Soma-targeting cells (STCs) express the calcium binding protein parvalbumin (PV)

**SST**  
(SST/CB)

- either express somatostatin (SST) or the calcium binding protein calbindin (CB)
- target pyramidal dendrites
- control inputs onto excitatory neurons

**VIP**

- express either vasoactive intestinal peptide (VIP) or the calcium-binding protein calcitonin gene-related peptide (CGRP)
- target SST/CB inhibitory neurons



Form a disinhibitory motif → represent a general feature of cortical microcircuits.

The fourth major type of inhibitory neurons

- not express PV, SST or VIP
- constitute 90% of interneurons in layer 1 with various firing patterns
- in other layers, either express neuropeptide Y and mediate GABA<sub>A</sub> receptor-dependent slow inhibition, or express peptide CCK and underline the cannabinoid action in the brain.

13. Neuronal operations are shaped by chemical substances.

14. [idea] chemical process → describe single-neuron adaption mediated by intracellular calcium ions.

15. Four types of major cells to be modelled: excitatory pyramidal cells and 3 inhibitory subtypes

16. The electrical signaling of a neuron is determined by voltage difference across its membrane:

$$V = V_{in} - V_{out}$$

where  $V_{in}$  and  $V_{out}$  are voltages inside and outside the neurons.

The levels of  $V_{in}$  and  $V_{out}$  are determined by concentrations of ions ( $Cl^-, Na^+, K^+, Ca^{2+}$ )

7.  $V$  related to spatial and temporal location

$$V(x, t)$$

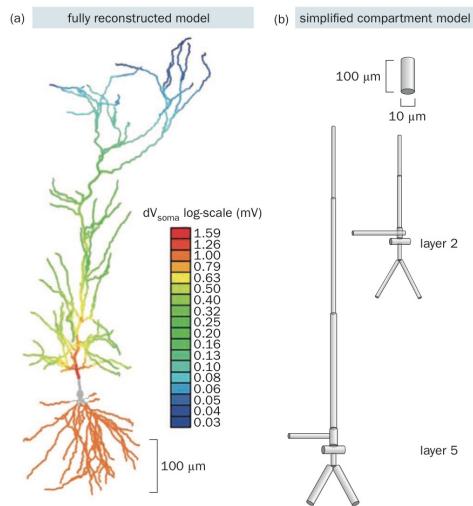
8. A morphologically fully reconstructed pyramidal neuron may be modeled with hundreds of compartments  $\rightarrow$  each assumed

to be **isopotential**  $\rightarrow$  simplified compartment model:

$\hookrightarrow$  capture the basic spatial structure of a neuron with apical and basal dendrites, down to just three or two compartments

 **my inspiration** An important idea in computational neuroscience is to **simplify**. We need

to find a simplified (or the most simplified) model that is biologically plausible to use math and other method to capture the basic principals.

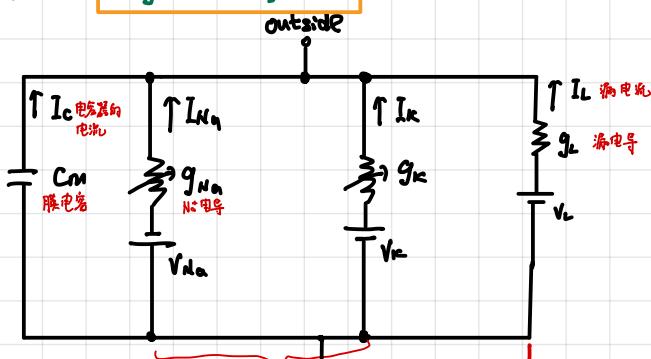


 When distinct dendritic versus somatic signaling is not the main focus

$\hookrightarrow$  "point neuron" with a single compartment

$\hookrightarrow$  the Hodgkin-Huxley conductance-based model (integrate-and-fire model)

Hodgkin-Huxley model



## 20. Neuronal Membrane as an RC circuit.

• Neuronal Membrane  $\rightarrow$  a fatty lipid bilayer  $\rightarrow$  not permeable to charged molecules  $\rightarrow$  capacitor with a capacitance  $C$

• Charge stored in capacitor  $Q = CV$

• Capacitive current:  $\frac{dQ}{dt} = C \frac{dV}{dt}$

• Opening an ion channel leads to a current:  $I = g(V - V_{\text{rev}})$  (Equation 2.1)  
(unit:  $\mu\text{A}/\text{cm}^2$ )

when  $g$  is a conductance ( $\frac{1}{\Omega}$ ) per unit area  $\text{cm}^2$  ( $\text{MS/cm}^2$ )

$V_{\text{rev}}$  is a reversal potential

"driving force"

• Given a membrane with area  $A \Rightarrow G = gA$ ,  $I = G(V - V_{\text{rev}})$  (unit:  $\text{nA}$ )

quantifies the strength of an ion current

•  $V_{\text{rev}}$  depends on the selective permeability of an ion channel and the intracellular and extracellular concentrations of ions that can pass through the channel.

• When a channel is permeable for a single type of ions  $\Rightarrow$  Nernst equation:  $V_{\text{rev}} = \left(\frac{RT}{F}\right) \ln([C_{\text{out}}]/[C_{\text{in}}])$  (Equation 2.2)

$\hookrightarrow R$ : the universal gas constant  $F$ : Faraday constant

$T$ : temperature

$i$ : the charge of an ion

- $K^+$ : inside  $\rightarrow$  outside  $\Rightarrow V_K < 0$  ( $\approx -80\text{mV}$ )

$Ca^{2+}, Na^+$ : inside  $<$  outside  $\Rightarrow V_{Na} > 0$   $\cdot V_{Ca} > 0$   
 ( $\approx +50\text{mV}$ ) ( $\approx +120\text{mV}$ )

- The combined effects of various ion types yield a net reversal potential given by:

Goldman-Hodgkin-Katz Equation:  $V_m = \frac{RT}{F} \ln \left[ \frac{\sum P_{ion} \cdot C_{ion,out}}{\sum P_{ion} \cdot C_{ion,in}} \right] \Rightarrow G \cdot \ln \frac{P_K \cdot C_{K^+,out} + P_{Na^+} \cdot C_{Na^+,out}}{P_K \cdot C_{K^+,in} + P_{Na^+} \cdot C_{Na^+,in}}$

(Eqn 2.3)

p: permeability

- a neuron is "at rest"  $\Rightarrow$  voltage: inside  $<$  outside  $\Rightarrow V \approx -70\text{mV}$

$\hookrightarrow$  modeled by a "leak" current  $I_L = G_L(V - V_L)$   $\star$  (Eqn 2.4)

$G_L$ : constant  $V_L: -70\text{mV}$

21. A minimal model for a passive neuronal membrane  $\Rightarrow$  described by an RC circuit with  $R = \frac{1}{G_L}$ , and C associated

with the membrane's lipid bilayer. If I<sub>app</sub> represents an external current injected into the membrane, then:

$C_m \frac{dV}{dt} = -G_L(V - V_L) + I_{app}$  (Eqn 2.5)

can  
be  
rewritten as

$\frac{dV}{dt} = \frac{V_{ss} - V}{T_m}$  (Eqn 2.6)

$\hookrightarrow V_{ss} = V_L + \frac{I_{app}}{G_L}$   $\rightarrow$  steady-state

~~$T_m = \frac{C_m}{G_L} = R C_m$~~   $\rightarrow$  membrane time constant  $\star$   
 eg:  $C_m = 0.2\text{nF}$ ,  $G_L = 0.02\text{mS}$ ,  $T_m = 10\text{ms}$

$S: \text{membrane} : IS = \frac{1}{T_m}$

$F = \Omega \cdot S \quad S = FS = \Omega \cdot S \cdot \frac{1}{\Omega} = S \star$

In a given "state" (a particular V), the right-hand side determines the slope  $(dV/dt)$  of its change into future.

A "steady state" V<sub>ss</sub> is determined by V and not change with time  $\Rightarrow \frac{dV}{dt} = 0 \Rightarrow V = V_{ss}$ , which is in the absence of input I<sub>app</sub> = 0.

When given a I<sub>app</sub>, V<sub>ss</sub> increase linearly with I<sub>app</sub>, and inversely proportional to the G<sub>L</sub>.

This steady state is stable: if V is transiently depolarized or hyperpolarized by a current pulse, after the input offset it will evolve back to V<sub>ss</sub> exponentially with a time constant T<sub>m</sub>.

an example of:  
**Attractor State**

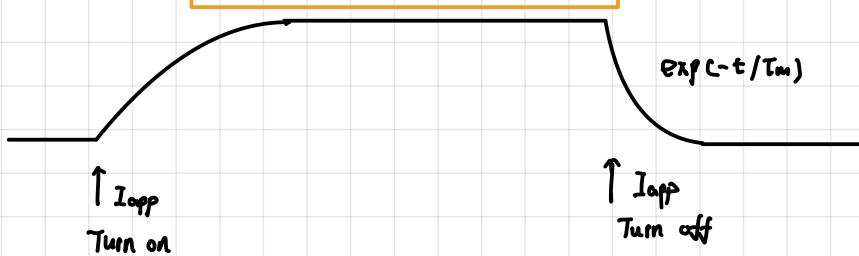
really important

$\hookrightarrow$  A mathematical term to describe a state of a dynamical system that is stable under perturbation if it

22. For an initial condition  $V(t=0) = V_0$ , the general solution for Eqn 2.5 is:

$$V(t) = V_{ss} + (V_0 - V_{ss}) \exp(-t/T_m)$$
Eqn 2.7

23. "Forget":



24. Neurons have many active channels  $\Rightarrow$  conductances are not fixed

a general version of Kirchhoff's circuit equation:

$$C_m \frac{dV}{dt} = -\sum_j I_{ion,j} + I_{opp}$$
Eqn 2.8

$$I_{ion,j} = g_j (V - V_{rev,j})$$
Eqn 2.9

Review:

$$V = V_{in} - V_{out}$$

25. Negative current  $\Rightarrow$  "inward"  $\rightarrow$  increase or depolarize  $V$

Positive current  $\Rightarrow$  "outward"  $\rightarrow$  decrease or hyperpolarize  $V$

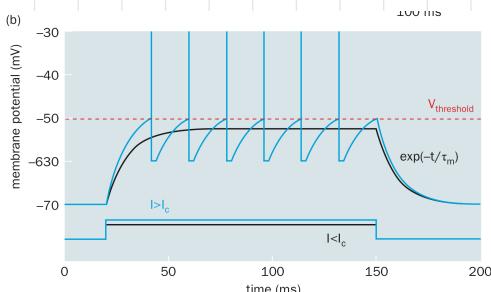
26. Neuronal communication is conveyed by action potentials, or spikes.  $\rightarrow$  brief pulses of electrical signals

27. The more spikes are emitted per unit time (s), the more active is a neuron.

28. Leaky integrate-and-fire (LIF) model  $\Rightarrow$  built on the simplification that spikes are stereotypical and brief events

- it describes a passive neuron that incorporates spikes into an RC circuit in a simplified way
- assume the generation of a spike as a "point event" in time whenever  $V$  exceeds a preset voltage threshold  $V_{th}$ .
- if  $V$  reaches  $V_{th}$  at time  $t \Rightarrow$  a spike is discharged with the spike time  $t$
- $V$  is instantaneously reset to  $V_{rest}$  and stays there for a refractory period of time  $T_{ref}$ . ★
- $V_{th}, V_{rest}, T_{ref}$  can be determined by physiological measurements for a particular type of neurons

- the behaviour of LIF depends on the intensity of  $I_{opp}$   $\rightarrow$ 
  - small  $I_{opp} \rightarrow V_{ss}$  below  $V_{th}$
  - large  $I_{opp} \rightarrow V_{ss}$  exceeds  $V_{th}$   $\rightarrow$  (exceed a threshold  $I_c$ )



when  $V$  crosses  $V_{th}$ ,  $V$  will be reset to  $V_{rest}$

$I_c = G_L (V_{th} - V_L)$

where,  $V_{ss} = V_{th}$

$V_{rise}$  and reaches  $V_{th}$  again

repetitive firing of spikes

Eqn 2.10

With a fixed  $I_{app}$  the firing is **regular** in time, characterized by a constant period  $T$  or inter-spike interval (ISI) and firing rate  $r = \frac{1}{T}$

What and why is "firing rate"

29. ISI:  $V$ : from  $V_{reset}$  to  $V_{th}$

According to eqn 2.7,  $V$  will reach  $V_{th}$  at  $t = T - T_{ref}$

Solve the eqn 2.7

$$r = \begin{cases} \frac{1}{T_{ref} + \frac{C_m}{G_L} \ln \left[ 1 + \frac{G_L \Delta V}{I_{app} - I_c} \right]} & \text{if } I_{app} > I_c \\ 0 & \text{otherwise} \end{cases}$$

Specially  
→ When  $V_L = V_{reset}$   $r = \frac{1}{T_{ref} + \frac{C_m}{G_L} \ln \left[ 1 + \frac{I_c}{I_{app} - I_c} \right]}$

Eqn 2.11

$$\Delta V = V_{th} - V_{reset}$$

Try to solve the eqn 2.7

$$V(t) = V_{ss} + (V_{reset} - V_{ss}) \exp(-t/T_m)$$

When  $V = V_{reset}$   $t=0$

When  $V = V_{th}$   $t = T - T_{ref}$

$$V_{th} = V_{ss} + (V_{reset} - V_{ss}) \exp\left(-\frac{T - T_{ref}}{T_m}\right)$$

$$\ln \frac{V_{th} - V_{ss}}{V_{reset} - V_{ss}} = -\frac{T - T_{ref}}{T_m}$$

$$T = -T_m \ln \frac{V_{th} - V_{ss}}{V_{reset} - V_{ss}} + T_{ref}$$

$$= -T_m \ln \frac{V_{th} - V_L - \frac{I_{app}}{G_L}}{V_{reset} - V_L - \frac{I_{app}}{G_L}} + T_{ref}$$

$$= -\frac{C_m}{G_L} \ln \frac{G_L(V_{th} - V_L) - I_{app}}{G_L(V_{reset} - V_L) - I_{app}} + T_{ref}$$

$$= \frac{C_m}{G_L} \ln \left[ 1 + \frac{G_L(V_{th} - V_{reset})}{I_{app} - I_c} \right] + T_{ref}$$

$$r = \frac{1}{T} = \frac{1}{T_{ref} + \frac{C_m}{G_L} \ln \left[ 1 + \frac{G_L \Delta V}{I_{app} - I_c} \right]}$$

30: Neuronal input-output transfer function  $\Rightarrow$  the frequency-current relationship ( $f$ - $I$  curve)  $\Rightarrow$  fundamental concept

describing a neuron's mapping from an input current into an output firing rate

$$\xrightarrow{\frac{\text{Input}}{I_{app}}} r = \dots \xrightarrow{\frac{\text{output}}{\text{firing rate } r}}$$

31. Well above  $I_c$  ( $I_{app} \gg I_c$ )  $\Rightarrow r$  is approximately linear with  $I_{app}$ , with a slope given by  $\frac{1}{G_{mV}}$

When  $I_{app}$  is extremely large  $I_{app} \rightarrow \infty$ .  $r \rightarrow \frac{1}{I_{app}}$   $\Rightarrow$  plateaus  $\frac{1}{I_{app}}$

Try to prove

$$1. \text{ When } I_{app} \gg I_c, r = \frac{1}{T_{ref} + \frac{C_m}{G_m} \ln\left(1 + \frac{G_m V}{I_{app}}\right)}$$

$$\text{* Taylor: } \ln(1+x) = x - \frac{x^2}{2} + \frac{x^3}{3} - \dots$$

$$\text{For } x \ll 1 \quad \ln(1+x) \approx x$$

$$\text{For } \frac{G_m V}{I_{app} - I_c} \ll 1, \quad \ln\left(1 + \frac{G_m V}{I_{app} - I_c}\right) \approx \frac{G_m V}{I_{app} - I_c}$$

$$r = \frac{1}{T_{ref} + \frac{C_m \cdot G_m V}{I_{app}}} \xrightarrow{\substack{\text{approx} \\ \text{linear to}}} \frac{1}{C_m \cdot G_m} \cdot I_{app}$$

$$2. \text{ When } I_{app} \rightarrow \infty \quad r \rightarrow \frac{1}{T_{ref} + T_m \ln(1+0)} = \frac{1}{T_{ref}}$$

$$\text{for } T_{ref} = 2 \text{ ms} \quad f_{max} = \frac{1}{T_{ref}} = 500 \text{ Hz}$$

波动

↑

32. In an intact brain *in vivo*, synaptic inputs to a neuron fluctuate considerably in time  
 ↓ 突触的

neuronal firing of a spike train is **highly irregular**

33. For a periodic spike train  $\rightarrow ISI$  is constant  $\Rightarrow$  review: ISI: Interspike interval  $\Rightarrow T$

For a stochastic point process  $\rightarrow ISI$  is a random variable

34. The **stochasticity** of a spike train can qualified in two ways:

1. the Coefficient of Variation (CV) of ISIs

$$CV = \frac{\text{Std}(ISI)}{\text{Mean}(ISI)} = \frac{\langle (ISI - \langle ISI \rangle)^2 \rangle^{1/2}}{\langle ISI \rangle} \quad \text{Eqn 2.12}$$

where  $\langle x \rangle = \int p(x) dx \Rightarrow$  average of  $x$

2. Fano factor of spike count  $N(T)$  (number of spikes in a time window  $T$ ):

$$F(T) = \frac{\text{Variance}(N(T))}{\text{Mean}(N(T))} \quad \text{Eqn 2.13}$$

35. For a regular periodic spike train  $\Rightarrow \begin{cases} CV=0 \\ F(T)=0 \end{cases}$

$$\text{Std} = N \cdot \sigma = 0$$

$$P(ISI) = \lambda e^{-\lambda ISI}$$

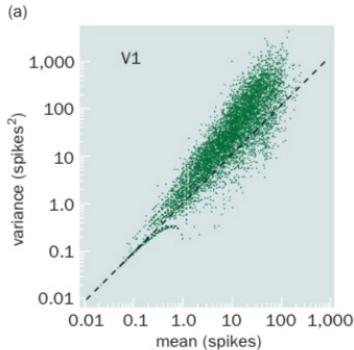
If a spike train is Poisson  $\Rightarrow \begin{cases} CV=1 \\ F(T)=1 \end{cases}$

36. High variability represents a salient characteristic of spike train  $\rightarrow$  typically the spike-count variance exceeds the mean.  
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### Inspiration

Draw variance-mean plot to find the characteristic of spike train

e.g.:



37. CV is typically larger than 1 for neural activity in the prefrontal cortex of a behaving macaque monkey

38. To capture stochastic spike firing in an integrate-and-fire model



Introduce a noise  $\eta(t)$  into  $I_{app}$ :

$$C \frac{dV}{dT} = -G_L(V - V_L) + I_{app} + \delta \eta(t) \quad \text{Equ 2-14}$$

$I_{app}$  is the mean of  $I_{app}$ ,  $\delta$  is the noise level

39. The simplest kind of noise  $\rightarrow$  no temporal correlation and a Gaussian probability density of  $\eta$  of zero mean and unit variance:

$$P(\eta) = \frac{1}{\sqrt{2\pi}} e^{-\frac{\eta^2}{2}} \quad \text{Equ 2-15}$$

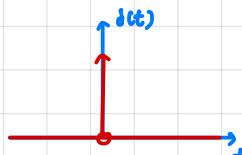


"White Noise"  $\rightarrow$  because its power spectrum is constant and therefore completely devoid of dependence on frequencies

40. The absence of temporal correlation in math is described by:

$$\langle \eta(t) \eta(t') \rangle = \delta(t-t') \quad \text{Equ 2-16}$$

where the delta function  $\delta(t)$  is infinity when  $t=0$  and zero otherwise



41: Equ 2-14

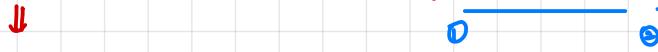
$$C \frac{dV}{dT} = -G_L(V - V_L) + I_{app} + \delta \eta(t) \quad \text{Equ 2-14}$$

with a leak and white noise  $\Rightarrow$  Ornstein-Uhlenbeck process \*

42. When  $\mu$  is below  $I_c \Rightarrow$  the deterministic steady-state  $V_{ss}$  is below  $V_{th} \Rightarrow$  but  $V$  is driven by noise and may still reach  $V_{th}$  through fluctuations

43: ISIs are statistically independent of each other, since each time a spike is fired,  $V$  is reset to  $V_{reset}$  and memory about the past is lost

44. Starting at  $V_{reset} \Rightarrow$  the time it takes for  $V$  to reach  $V_{th}$  for the first time is mathematically called "first passage time"



an ISI is the first passage time +  $T_{ref}$

$$\text{ISI: } T = T_{\text{first-passage}} + T_{\text{ref}}$$

Eqn 2.17

The statistics of ISIs can be analysed by the theory of first passage times of an Ornstein-Uhlenbeck process

In particular, the average  $\langle \text{ISI} \rangle \propto \frac{1}{f}$ , the input-output function is:

$$\frac{1}{f} = T_{\text{ref}} + T_m \sqrt{\pi} \int_{\frac{C_m(V_{th}-V_{ss})}{\sqrt{T_m}\delta}}^{\frac{C_m(V_{th}-V_{ss})}{\sqrt{T_m}\delta}} e^{x^2} (1 + \text{erf}(x)) dx$$

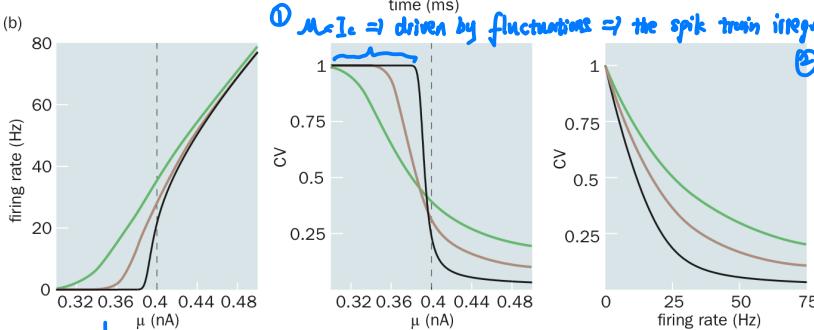
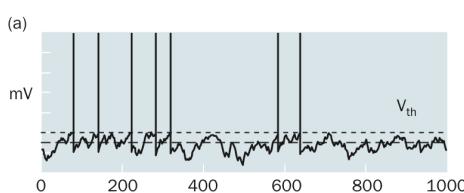
Eqn 2.18

where.  $\text{erf}(x) = \frac{2}{\sqrt{\pi}} \int_0^x \exp(-x'^2) dx' \Rightarrow$  error function

$CV$  is given by:

$$CV^2 = 2\pi f^2 \int_{\frac{C_m(V_{th}-V_{ss})}{\sqrt{T_m}\delta}}^{\frac{C_m(V_{th}-V_{ss})}{\sqrt{T_m}\delta}} e^{x^2} \left[ \int_{-\infty}^x e^{y^2} (1 + \text{erf}(y)) dy \right] dx$$

Eqn 2.19



$\Rightarrow$  line  $\Rightarrow$  different  $\delta$

(Unit:  $\delta: nA\sqrt{mA}$ ;  $\delta: nN$ )

①  $\mu = I_c \Rightarrow$  driven by fluctuations  $\Rightarrow$  the spiketrain irregular  $\Rightarrow$   $CV$  close to 1

② when  $(\mu - I_c)$  is much larger than  $\delta = \frac{\delta}{\sqrt{T_m}}$  the firing becomes increasingly regular

③ With large  $\delta$ ,  $CV$  decays to zero and spiking becomes regular

↳ sharpness of this transition depends on  $\delta$

↳ when the fluctuations are small, { the transition is very sharp the large value of  $\delta$ , the transition is smooth

45. A more basic physiological question: how a high variability of neural spike firing can be explained mechanistically  
↓  
the total input to a single cortical cell must operate near the firing threshold, regardless of the firing rate level

### Conductance-based Models of Single Neurons

46. Hodgkin-Huxley model, an action potential is produced by an interplay of voltage-gated  $\text{Na}^+$  and  $\text{K}^+$  ion currents ( $I_{\text{Na}}$  and  $I_{\text{K}}$ )

{  $I_{\text{Na}}$ : inward = depolarize  
 $I_{\text{K}}$ : outward = hyperpolarized

$$I_{\text{Na}} = g_{\text{Na}}(V - V_{\text{Na}})$$

Eqn 2.20

$$I_{\text{K}} = g_{\text{K}}(V - V_{\text{K}})$$

with reversal potentials  $V_{\text{Na}}$  and  $V_{\text{K}}$