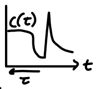
002 / CH1

- **NEURON STRUCTURE / ORDERS OF MAGNITUDE** (10^5 NEURONS/mm^3): DENDRITES (BRANCH-EXTENSIONS FROM SOMA, INPUTS) · SOMA (CELL BODY: NUCLEUS, ORGANELLES) · AXON (LONG EXTENSION, CARRIES KNOWLEDGE AWAY) → INFORMATION
- **ACTION POTENTIAL DYNAMICS**: NEURONS ARE CHARGED (-70 mV AT REST), EXP SHAPE, Na^+, K^+ CHANNELS, BRIEF SIGNAL IMPULSE (POLARIZATION PHASES $x+$), TRAVELS WITHOUT ATTENUATION OVER AXONS
  ↓
- **INFORMATION TRANSMISSION**: 1. SPIKE IN PRE-SYNAPTIC NEURON 2. VOLTAGE-GATED CALCIUM CHANNEL OPENS AT AXON TERMINAL 3. INFLUX OF CALCIUM IONS TRIGGERS NEUROTRANSM. RELEASE 4. POST-SYNAPTIC NEURON IS AFFECTED
- **RASTER PLOTS**: SPIKES OF NEURONS IN TIME, SYNCED TO STIMULUS

} NOISY REACTION TO STIMULUS → NEURAL · ENCODING: STUDY REACTION · DECODING: IDENTIFY THE STIMULUS
- **FIRING RATE PROPERTIES**: LOW, HIGH VARIABILITY BEFORE STIMULUS, HIGHER RATE ON AVERAGE AFTER STIMULUS, PLASTICITY ADDS NOISE FROM PAST STIMULI, POST-STIMULUS TIME HISTOGRAM: COUNT SPIKES IN TIME BIN AND DIVIDE BY # TRIALS } FIRING RATE PER ΔT → MUST BE TIME-ALIGNED
- **NEURONS AS DETECTORS (EX)** (VARIABLE SPARSITY): FIRING RATE IS MODELLED AS f(STIMULUS) FOR CONTIN. STIM. } TUNING CURVE BY STIMULUS:

ORIENTATION, DIRECTION, LOCATION | FREQUENCY, HEAD ANGULAR, VELOCITY | ≠ DISCRETE: – SPARSE CODING – CLUSTERS ACTIVATE } FACES, SMELLS
- **SPIKE-TRIGGERED AVERAGE**: AVERAGE STIMULUS PRECEDING A SPIKE



REVERSE CORRELATION METHOD: CROSS-CORR: $C(\tau) = \frac{1}{r} Q_{rs}(-\tau) = \frac{1}{r}\mathbb{E}[r(t)\cdot s(t-\tau)]$ (STA; r = FIRING RATE; NEURON ACTIVATION; STIMULUS VALUE) BY DEF, $C(\tau) = \frac{1}{\langle n \rangle}\sum_{i=1}^{n} s(t_i - \tau)$
- **INTERSPIKE INTERVAL VARIABILITY (CV_{ISI})**: IN POISSON ($CV_{ISI} = 1$), SAMPLE VARIANCE OF TIME BETWEEN SPIKES $= \sigma_{ISI}$ → NORM. COEFF. OF VARIATION $CV = \frac{\sigma_{ISI}}{\mu_{ISI}}$ } RELATIVE TO 1, IT QUANTIFIES REGULARITY
- **TRIAL-TO-TRIAL FLUCTUATIONS (FANO FACTOR)**: CV IN SPIKE COUNT: $\frac{V(\text{SPIKE COUNT})}{\mathbb{E}(\text{SPIKE COUNT})} = FF$, $FF = 1$ IN POISSON PROCESSES (ACTS AS REFERENCE), VALUE VARIES BY BIN SIZE, YOU TAKE MULTIPLE TRIALS (HIGH (>1) BEFORE STIM., LOWERS AFTER STIMULUS (≥1))

INTUITION ON CONVOLUTION

THE INTEGRAL OF THE PRODUCT OF FUNCTIONS IS AN INNER PRODUCT OVER FUNCTIONS.
$(f * g)(y) = \int_x f(x) g(y-x) dx$

$f(x)$ IS THE "KERNEL". IT GIVES A WEIGHING TO THE VALUE $g(y-x)$, AKA THE VALUE OF g AT x DIST FROM y.

$(f*g)(y)$ IS OBTAINED BY WEIGHING SOME NEIGHBOURHOOD OF $g(y)$ BY f. IT IS THE DIFFERENTIAL LIMIT OF A WEIGHTED SUM

g CAN BE SEEN AS AN INFINITE VECTOR OF VALUES. $g(I(x,y))$ HAS $I(x,y) = y - x$ IF WHAT MATTERS ARE THE VALUES BEFORE y

"THEORETICAL NEUROSCIENCE: COMPUTATIONAL AND MATHEMATICAL MODELING OF NEURAL SYSTEMS".

003 / CH2

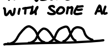
- **REVERSE CORRELATION METHOD TO ESTIMATE FIRING RATE**: $r_{est}(t) = r_0 + \int_0^\infty d\tau D(\tau) s(t-\tau)$ (VOLTERRA EXPANSION; KERNEL), $D^*(\tau) = \frac{\langle r \rangle}{\sigma_s^2} C(\tau)$ → STA (C: MEAN FIRING RATE; σ_s^2: VARIANCE OF STIMULUS)
- **MOST EFFECTIVE STIMULUS**: GIVEN $D(\tau)$, STIMULUS THAT MAXIMISES RESPONSE } $(S(t), r(t)) \to D(\tau) \to \lambda, S^*(t)$
- **NONLINEAR GENERALISATION**: ACTIVATION: $r(t) = r_0 + F\left(\int_0^\infty D(\tau) s(t-\tau) d\tau\right)$ } ReLu, Sigmoid, Fitted
- **LINEAR-NONLINEAR-POISSON MODEL (LNP)**: ANTICIPATE NEURON RESPONSE: $L = \int d\tau D \cdot S \to r = r_0 + F(L) \to$ SPIKE GENERATOR → RESPONSE, $\mathbb{P}(\text{SPIKE IN } \Delta t) = r_t \cdot \Delta t$
- **REVERSE-CORRELATION TIME + SPACE KERNELS**: YOU CAN EXTEND A KERNEL TO TAKE MORE PARAMETERS } CURSE OF DIMENSIONALITY
- **RECEPTIVE FIELD OF THALAMUS / RETINA (+ EVOLUTION IN TIME)**: START FROM WHITE NOISE AS STIMULUS → USE S, STA TO FIND $D(\tau)$ } A RECEPTIVE FIELD IS SEPARABLE IF $D(x,y) = D(x)D(y)$ } USUALLY GABOR FUNCTION

005 / CH4

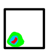
- **ENTROPY (JOINT, CONDITIONAL, ...)**: MEASURES UNCERTAINTY $H(X) = -\sum_i P_i \log P_i$; $H \geq 0$, $H = 0$ IFF $P(X = x_i) = 1$; MAXIMISED IN UNIFORM | $H(x,y) \leq H(x) + H(y)$, EQUALITY IF $P(x,y) = P(x)P(y)$ | $H(x|y) = H(x,y) - H(y)$, $I(x,y) = H(x) - H(x|y)$ MUTUAL INFO. $= H(y) - H(y|x)$
- **NOISY BINARY SYMMETRIC CHANNEL**: STIMULUS $\{0,1\}$, $P = 0.5$, $P(\text{SPIKE}|0) = p$, $P(\text{SPIKE}|1) = 1-p$ → $I(y, \vartheta) = 1 - H(P|y)$, $H(P|y) = -p\log_2 p - (1-p)\log_2(1-p)$ } ONE USES $\log_2$ ... FOR UNITS
- **NOISY GAUSSIAN CHANNEL**: $r = \vartheta + z$ | $\vartheta \sim N(\cdot, \sigma_\vartheta^2)$, $z \sim N(\cdot, \sigma_N^2)$; $I(r, \vartheta) = H(r) - H(r,\vartheta) = \frac{1}{2}\log_2\left(1 + \frac{\sigma_\vartheta^2}{\sigma_N^2}\right)$
- **EFFICIENT CODING HYPOTHESIS**: "SENSORY SYSTEMS ARE OPTIMISED FOR INFORMATION TRANSMISSION" } LIMITED # NEURONS, SPIKING RATE
- **OPTIMISING THE STATIC-NONLINEARITY**: $r = \phi(\vartheta) + z$ | ϕ NON-LINEARITY; $\max_\phi I(r,\vartheta) = H(r) - H(r|\vartheta)$ } MAXIMISE $H(r,\vartheta)$, USE THE "SCALE" OF MEASURE AS EFFICIENTLY AS POSSIBLE
- **HISTOGRAM EQUALISATION**: ENTROPY MAX PRODUCES A UNIFORM DISTR. } → $\vartheta \mapsto F(\vartheta)$ (CDF) } PRODUCES UNIFORM DISTRIBUTION

004 / CH3

- **STATISTICS AS DECODERS**: NEURAL RESPONSES TO STIMULUS ϑ } → USE ESTIMATOR $\hat{\vartheta}(\vec{r})$ } $r_i = \vartheta + z_i$ | $z_i \sim N(0,1)$; $\hat{\vartheta}(\vec{r}) = \frac{1}{N}\sum r_i \sim N(0, \frac{1}{N})$
- **POPULATION VECTOR DECODERS (2x EXC)**: 4x NEURONS TASKED WITH SOME ALIGNMENT



} HOW TO GO FROM $\vec{r}$ TO $\hat{\vartheta}$?
- **HIPPOCAMPAL CODE FOR SPACE**:



FOR A NEURON, COLOR BY MEAN FIRING WHEN NEURON IS THERE } r
- **ERROR QUANTIFICATION (BIAS, MSE)**: BIAS $b_\vartheta = \langle \hat{\vartheta}(\vec{r}) \rangle - \vartheta$ | MSE $\langle (\hat{\vartheta}(\vec{r}) - \vartheta)^2 \rangle$
- **SCORE, FISHER INFO., CRAMER-RAO**: SCORE $V(\vartheta, \vec{r})$: $\frac{\partial}{\partial \vartheta}\log P(\vec{r}|\vartheta)$ } HAS ZERO MEAN | FISHER INFO $\langle (V(\vartheta, \vec{r}))^2 \rangle$ [VAR. OF SCORE] | CRAMER-RAO: $MSE \geq \frac{1}{J(\vartheta)}$ } EQUALITY MAKES IT "EFFICIENT" (ASSUMING UNBIASED) → $b_\vartheta = 0$
- **MLE**: $\hat{\vartheta}_{ML} = \arg\max_\vartheta P(r, \vartheta)$ | $\hat{\vartheta}_{ML} \overset{N}{\sim} N(\vartheta, \frac{1}{J(\vartheta)})$
- **POPULATIONS OF NEURONS (INFORMATION)**: EACH NEURON HAS A TUNING CURVE (POISSON) $\vartheta = T \cdot \sum_{i=1}^{N} \frac{r_i'(\vartheta)}{r_i(\vartheta)}$ (T: BIN SIZE) } IF $r_i(\vartheta) = r(\vartheta - \phi_i)$, $J \sim NT$ WITH ERROR $\frac{1}{\sqrt{NT}}$

006/CH5

- **Neuronal membrane**: Lipid bilayer around neuron, neuron can store charge as capacitor, $V_{out}=0$ by convention
- **Ionic currents**:

• CHANNEL: open to allow flux of many specific ions	• PUMP: work to restore concentration of multiple ions

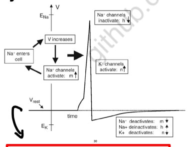
  - Na channel opens: Na^+ goes in, depolarising
  - K channel opens: K^+ goes out, hyperpolar.
- **Equivalent electric circuit**: V_m, Q internal charge, capacitor in parallel with current generator → neuron is a capacitor
- **Capacitance of a neuron**: C_m is capacitance of the membrane (ability to store charge) → $I_e = C_m \frac{dV_m}{dt}$
- **Response in the circuit**: A step current produces $V(t)=$  {exponential decay to} confirmed by (noisy) experiments
- **Movement of ions through the membrane**: Ion movement through an open gate:
  - $J_{diff} = -D\Delta[C]$ → Fick's law of diffusion → outgoing flux
  - $J_{drift} = -\mu z[C]\Delta V$ → Ohm's law → ingoing flux
- **Nernst equation**: At rest, $J_{diff}+J_{drift}=0$ → no net flux at equilibrium → $V_{eq}=\frac{V_T}{z}\cdot \ln\left(\frac{[C]_{in}}{[C]_{out}}\right)$
- **Goldman equation**: $V_{eq}=\frac{RT}{F}\cdot \log\left(\frac{\sum out}{\sum in}\right)$ permeability of all ions $\Rightarrow V_{REST}=-70\,mV$
- **Degrees of responses to step current**: Response to new current is an exp relaxation — explained by Goldman equation

007/CH5

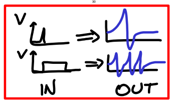
- **Equivalent circuit to neuron with ion channels**: Response to ext. I_e:

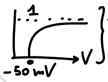
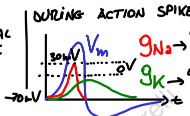
$$C_m\frac{dV}{dt}=-I_m+I_e$$
$$I_m=\sum g_i(V-E_i)$$

  g_i, E_i: conductance, g_i can depend on voltage; sum (i) over all channels



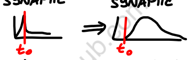
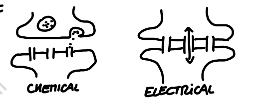
- **Sign conventions**: Membrane ion current: Na^+ outw → +, Na^+ inw → −; Injected (synapses, electrodes): outw → −, inw → +

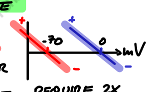


- **Conductance from voltage clamp**: 1. Fix membrane ΔV; 2. Measure $I(t)$ → $I_{ion}=g_{ion}(t)(V_m-E)$; $\frac{dI}{dV}=G_K$
- **Voltage-dependent channels**: Na, K conductance is voltage-dependant: $g_i=g_i(V)$ — typical shape 
  During action spike:  only at V spike; g_K lower than g_{Na}, delayed
- → **Persistent conductance** (channel stays open for $V\geq V_c$):
  Visualise as gate with sensor for opening: $\mathbb{P}(\text{gate opens})=f(V_m)$ → h^k → # events needed independently; $\mathbb{P}$ of one event
  $$I_K=\bar{g}_K\cdot n^k(V-E_K)$$ $\bar{g}_K$ = max conduct., E_K = eq. potential
  $\mathbb{P}$ should be seen as the % of open channels
  In K current: $I_K=\bar{g}_K(V-V_K)n^4$, $\frac{dn}{dt}=\alpha(V)(1-n)-\beta_n(V)n$
  ↳ Time dynamics: Dynamics are linear ODE: $\frac{dn}{dt}=\alpha_n(V)(1-n)-\beta_n(V)n$ (open rate, close rate), where n is $P(\text{open})$ for a gate → fit α_n,β_n to data (see voltage clamping)
  A K^+ gate look like: open / closed — there are $n=4$ parts, not 2 as drawn
- → **Transient conductance**: Activation gate + inactivation gate (≈ occluding ball) → $k\times$ activation variables m, inactivation var h
  - $m\in[0,1]$ → increases with V, like n
  - $h\in[0,1]$ → decreases with V

  $\mathbb{P}(\text{open})=P_{Na}$; $P_{Na}=m^k h$; $I_{Na}=\bar{g}_{Na}(V-V_{Na})m^k h$
  ↓ **Sodium current**:
  $$I_{Na}=\bar{g}_{Na}(V-V_{Na})m^k h$$
  $$\frac{dm}{dt}=\alpha_m(V)(1-m)-\beta_m(V)m$$
  $$\frac{dh}{dt}=\alpha_h(V)(1-h)-\beta_h(V)h$$
  → V increase: channels open then close → "transient current"
  M-current is another dynamic for K, resulting in spike frequency adaptation
- **HH model parts and roles**: $C_m\frac{dV}{dt}=-I_m+I_e$; I_m = membrane current $=g_L(V-E_L)+g_K(V-E_K)+g_{Na}(V-E_{Na})$; Leak current keeps cell at resting V; various ions moving; K and Na currents have conductance dependant on V, create action potential; voltage gated

008/CH5

- **Synapse kinds**: Synapses generate current which modulates membrane potential; between neurons synapses are uni- or bidirectional
- **Pre vs post behaviour**:  Reaction of the next neuron in the connection
- **Synaptic transmission phases**:
  - Initiation: • Action potential in presyn. terminal • Voltage dep. Ca^{2+} channels activate (more Ca^{2+})
  - Vesicle release: • Vesicles fuse with cell membrane • Release of transmitter molecules to syn. cleft
  - Transmission: • Diffusion + binding to receptors on post-syn. neuron
  - Response: • Opening of ion channels • Modification of post-syn. conductance
- **Ion channel activation (2x)**:
  - Direct activation (ionotropic): • Transmitter binds to synaptic channel • Rapid on/off responses • GABA, NDA
  - Indirect activation/ (metabotropic): • Specific pathways for signals • Slower onset, longer lasting • Amplify signal • Dopamine
  - "Cleft" is the empty volume between chemical synapses. • Neurotransmitters are GABA, dopamine
- **Synapse (2x)**: Chemical: • majority • slow, unidir. Electrical: • gap junctions • rapid, bidir. 

- **Synaptic current (Tsodsks-Markram)**: Ionic channel opening in postsynaptic terminal changes postsynaptic neuron conductance → $I_s=g_s(V-E_s)$; $g_s=\bar{g}_sP=\bar{g}_sP_sP_{rel}$; $P_s=\mathbb{P}(\text{postsyn. opens})$; $P_{rel}=\mathbb{P}(\text{neurotr. releases})$
- **Current after spike**: $\frac{dP_s}{dt}=\alpha_s(1-P_s)-\beta_sP_s$; α_s opening rate, β_s closing rate
- **Synapse classification**: 

- **NMDA transmitters**: Coincidence detectors: require 2x events to become active
- **Synaptic plasticity**:
  - Short-term: • P_{rel} altered (depression, facilitation)  changes in pre-synap. neuron
  - Long-term: • Affects both pre and post • Persistent
- **Short-term depression**: $P_{rel}\mapsto f_DP_{rel}$, f_D fraction of released vesicles, $0\leq f_D\leq 1$; $\tau_P\frac{dP_{rel}}{dt}=P_0-P_{rel}$ → returns to rest exponentially
- **Short-term facilitation**: $P_{rel}\mapsto f_F(1-P_{rel})+P_{rel}$, $0\leq f_F\leq 1$; aka. calcium influx increases neurotr. P_{rel}
- **Short-term plasticity**: Cortical neurons have short-term plasticity, depends on firing rate: low: facilitation; high: depression

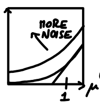
009/CH5

- **HH model evaluation**:

✓	✗
Captures qualitative diversity	Expensive to simulate, many vars, nonlinear

- **Leaky integrate-and-fire model**: Sub-threshold dynamics ($V<V_{th}$): capacitive + leak currents:
  $$C\frac{dV}{dt}=-g_L(V-E_L)+I_{syn}(t)$$
  $$\tau_m\frac{dV}{dt}=-(V-E_L)+\bar{I}_{syn}(t)$$
  When $V=V_{th}$: 1. Spike, $V=V_{rest}$; 2. (maybe) pause period τ_{rp} — adds realism
- **Adaptation LIF**: $r_{ISI}=\frac{1}{t_{ISI}}$ = rate for const. input → LIF firing captures the first spike; but successive spike are lower
  ↓ Interpretation in the model: Adaptation produces a g_a st. $g_a\mapsto g_a+\Delta g_a$ at each spike ⇒ larger neg. current that polarises neuron → it stops firing
  Analogous to M-current in HH, $\propto C_m\frac{dV}{dt}=\dots-I_a$; Spike rate adaptation: $I_a=R_mg_a(V-E_K)$, $\tau\frac{dg_a}{dt}=-g_a$
- **Pro/con LIF**: Can simulate large networks, but it's too simple to capture all dynamical features
- **LIF responses to currents**: Constant input ⇒ regular firing, $\#10^2-10^3$ presyn. neurons, 80% excitatory, 20% inhibitory, firing ~Poiss with 1–10 #/s
- **Synaptic current model**: Neuron driven by excitatory-inhibitory neurons, $I_s=I_{SI}+I_{SE}$, where each is $I_{S\dots}=\sum g_{\dots,i}(V-E_{\dots})$ → you divide neurons in two groups and sum both; $\tau_m\frac{dV}{dt}=E_L-V-I_s$
- → **Assumptions**: Presyn. fire at ~Poiss with r_{pre}, short-term plasticity is neglected (same current in all presyn. spikes), spikes happen instantaneously (as δ dirac), only takes current, no V depend. $I_{S\dots}=-\tau_m J_{\dots}\sum_i s_{\dots,i}$ (synaptic efficiency) (spike train)
- **LIF neuron, stochastic inputs**: For $N\gg 1$, use a Gaussian process
  → Regimes: 2x2 different contexts exist

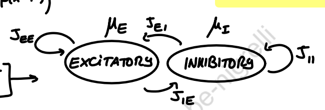
	No noise ($\sigma=0$)	Noise
Superthreshold ($\mu>V_{th}$)	Regular firing	Firing close to regular
Subthreshold	No firing	Irregular firing

- **Mean firing rate**: t_{ISI} can be computed directly as integral  Normalized st. $V_{th}-E_L=1$

011/CH2 CH7

- **2x layer NN (in, out)**: $\tau_r\frac{dv(t)}{dt}=-v(t)+F(Wu+Mv)$; Feedforw. have $M=0$ → RNN can be approx. by FF if recurr. is weak enough
  - FF. networks: Cerebellum: sensory info to movement
  - Recurr: Visual cortex, most areas of cortex
- **Coordinate transformation in premotor cortex**: Moving hand to grab an object is a coord transform taking gaze and body orientation → feedforw. architecture
- **Selectivity (LGN, V1)**:
  - LGN: • In thalamus, circular receptive field 
  - V1: • Simple: respond to edges, react to orientation, spatial frequency, phase • Complex: like simple but also phase invariant
  - Hubel, Wiesel model (how LGN produces V1): • Simple: LGN with offset "on" aligned in strips (on/off) • Complex: feedfw. inputs from more simple cells (similar orientation, different phase)
- **Recurrent NN 1D/homogeneous population**: $h \to v \circlearrowleft M$; $\tau\frac{dv}{dt}=-v+h+Mv$ ($M,v,h\in\mathbb{R}$); $M>1$ unstable, (<1 stable); $M=1$ perfect integrator $v(t)=\frac{1}{\tau}\int_0^t h(t')dt'$
- **Linear (N)**: $\tau_r\frac{d\mathbf{v}}{dt}=-\mathbf{v}+\mathbf{h}+M\mathbf{v}$; $\mathbf{v}(t)=\sum c_\lambda(t)\mathbf{e}_\lambda$ (basis of M eigen); Assuming M symmetric, λ_v real → $\exists\lambda_v>1$ → diverge; $\forall\lambda_v<1$ → converge (amplification of directions using λ); $\lambda_1=1$ → integration over e_λ

010/CH7

- **Connectivity features**: Feedforward, recurrent, feedback → higher-to-lower information flow for context/error corr.; Neurons are classified as excitatory/inhibitory; connection strength depends on neuron responses, has high variability; Dale's law: neuron transmits the same set of neurotransmitters at all post-syn. connections
- **Modelling brain network**: Depends on scope, may neglect Dale's law; Networks of HH: rarely used, expensive and analytically hard; LIF networks: less expensive, more used but still hard to make interpretable models
- **Firing rate models**: Manageable analytically but cannot account for timing/correlations; compute trial averaged response from a neuron: "response" = spike train, FF ≥ 1, noisy; input
  $$\tau_s\frac{dI_s(t)}{dt}=-I_s(t)+\sum_i w_i u_i(t)$$
  $$\tau_r\frac{dv(t)}{dt}=-v(t)+F(I_s(t))$$ output
- **Activation function**: $ReLu(x)=[x-T]_+$, $\text{sigmoid}=(1+e^{-\beta(x-T)})^{-1}$
- **Modeling populations**: Mean field approximation that groups neurons by behaviour 

012 / CH7

• ANALYSIS OF 1D RNN

$\tau_r \frac{dv(t)}{dt} = -v(t) + F(h + Mv)$

WITH $h = Wu$ | FIXED POINTS $\{v \mid v'(t) = 0\}$

YOU CAN STUDY A FIXED POINT LOCALLY WITH A LINEAR SYSTEM. FROM THE TAYLOR EXPANSION $\tau_r \frac{dv}{dt} = \lambda v \;\Big|\; \lambda = -1 + M \frac{dF(I)}{dI}\Big|_{v=v_0}$

$\lambda > 0 \to$ UNSTABLE
$\lambda < 0 \to$ ATTRACTOR
$\lambda = 0 \to$ FIXED VALUE

• 2, N DIM CASES

BUT $\underline{v}(t)$. LINEARISED: $\tau_r \frac{d\underline{v}}{dt} = J\underline{v}$

| J DOES NOT HAVE TO BE SYMMETRIC
$\lambda_c \in \mathbb{C}$, e_μ NOT ALWAYS $\perp$ $\longrightarrow$

(ASSUMING λ_ν DISTINCT $\Rightarrow e_\mu \perp$)

$\mathrm{Im}(\lambda_\mu) = 0$ HAS 1D BEHAVIOUR OVER e_μ

ELSE, OSCILLATION WITH β_μ FREQ:

BEHAVIOUR IS EXP $\{$ $\mathrm{Re}(\lambda_\mu) < 0$ HAS DAMPING, ELSE GROWTH

• HETERO VS. HOM. NETWORKS

2x GROUPS OF NEURONS $\{E, I\}$ $\to$

HOM	INHOM
• 2x NEURONS TOT • USED FOR STABILITY ANALYSIS	$N \gg 1$, TESTING OF FIRING RATES

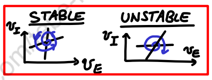
• HOMOGENOUS EXC/INH. POPULATIONS

$$M_{\dots} > 0 \quad \begin{cases} \tau \frac{dv_E}{dt} = -v_E + [h_E + M_{EE} v_E - M_{EI} v_I]_+ \\ \tau \frac{dv_I}{dt} = -v_I + [h_I + M_{IE} v_E - M_{II} v_I]_+ \end{cases}$$

$\to$ IN BOTH CASES THERE IS A SINGLE EFFECTIVE NEURON

• ANALYSIS OF NETWORK MODEL]

1. FOR FIXED POINT P FIND NULLCLINES $[\dots : \frac{dv}{dt}\big|_{P_{\dots}} = 0]$
2. PLOT NULLCLINES WITH P AT INTERSECTION
3. STABLE IF $\mathrm{Re}(\lambda_{\text{LARGEST EIG. VALUE}}) < 0$



• INHIBITORY STABILISATION PARADOXES

TWO POPULATIONS MODEL HAS $M = \begin{bmatrix} M_{EE} & M_{EI} \\ M_{IE} & M_{II} \end{bmatrix}$,

A FIXED POINT IS STABLE IF $\mathrm{Re}(\lambda) > 0$ FOR λ EIGEN IN THE LINEARISED SYSTEM,

IT'S POSSIBLE THAT A HIGHER STIMULUS TO INHIBITORY NEURONS LOWERS OVERALL FIRING RATE $\longrightarrow$ BY FIRING MORE OFTEN, INHIBITORY NEURONS REDUCE THE RATE OF THE NEURONS THAT WERE STIMULATING THEM