

物質機能科学**IIb**

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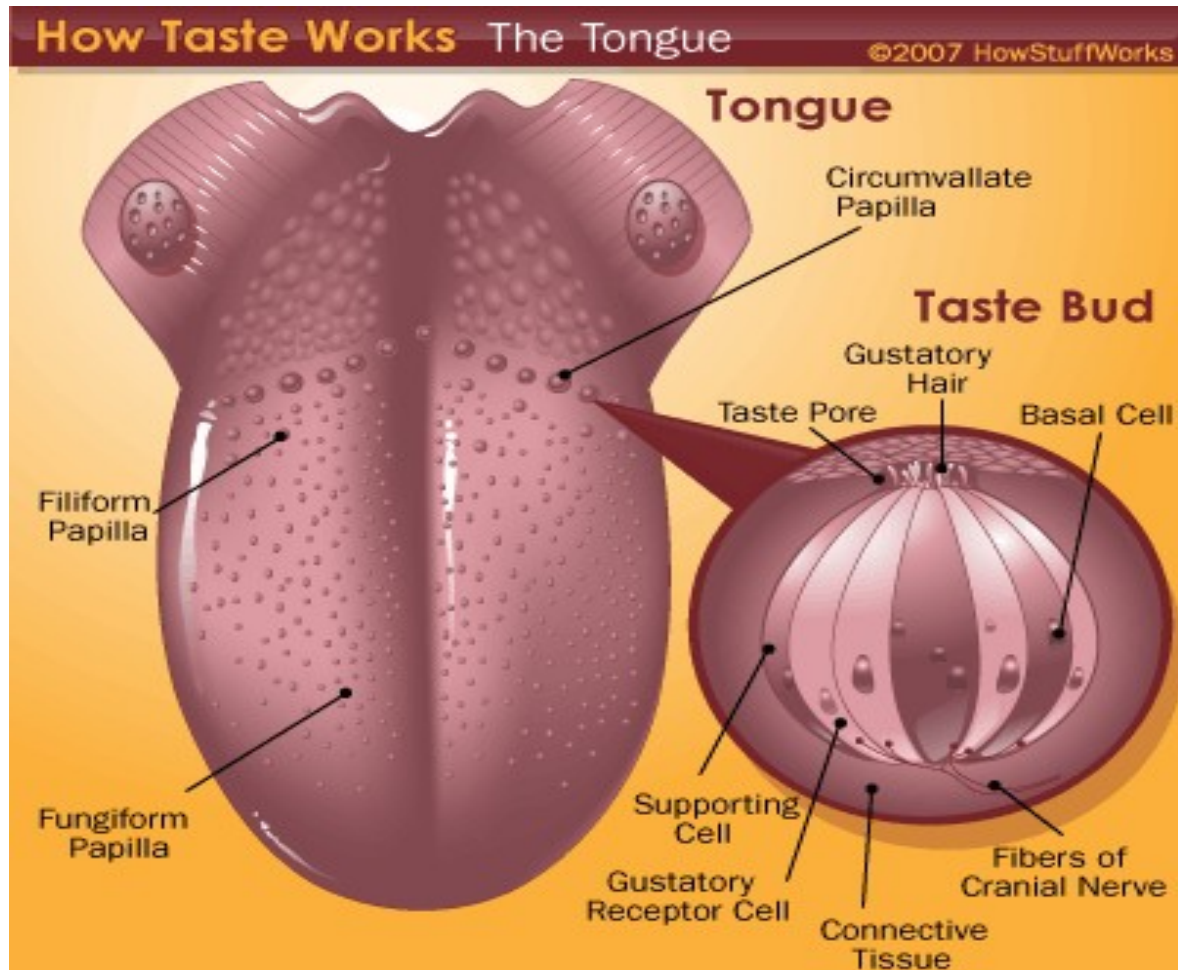
知覚情報科学

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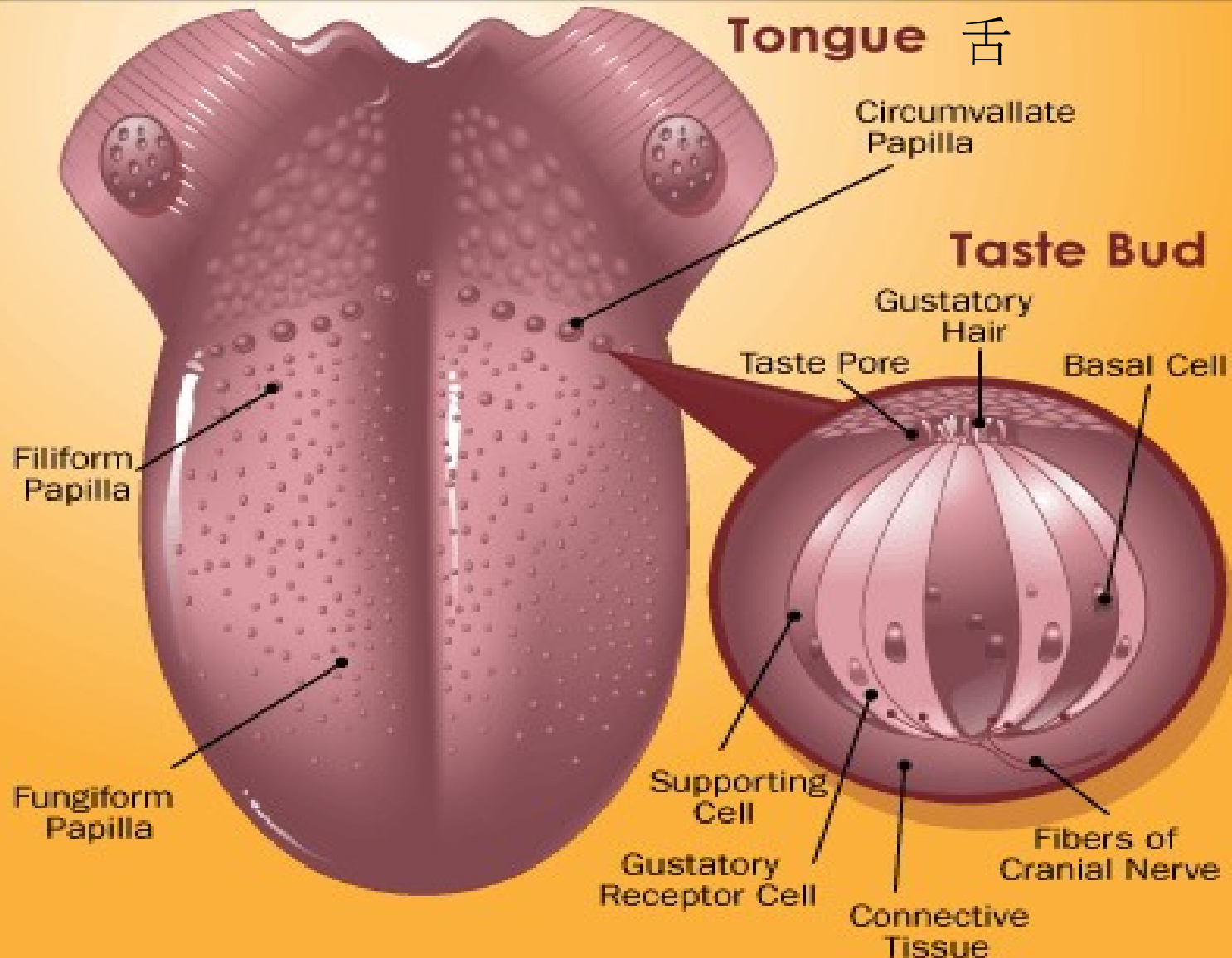
The Taste

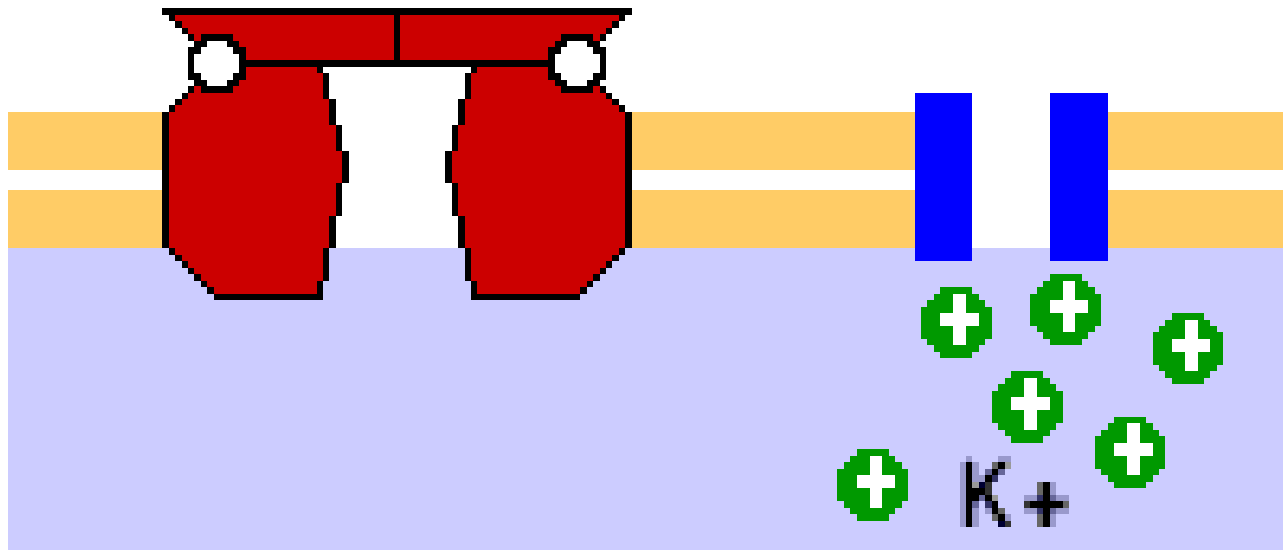
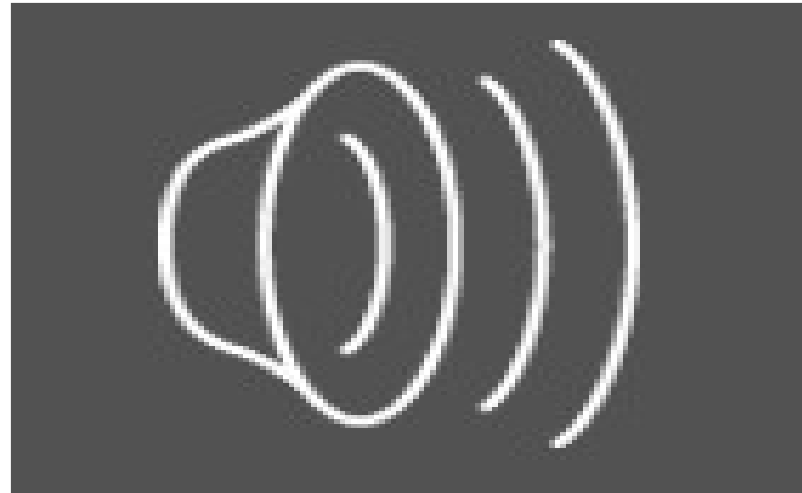


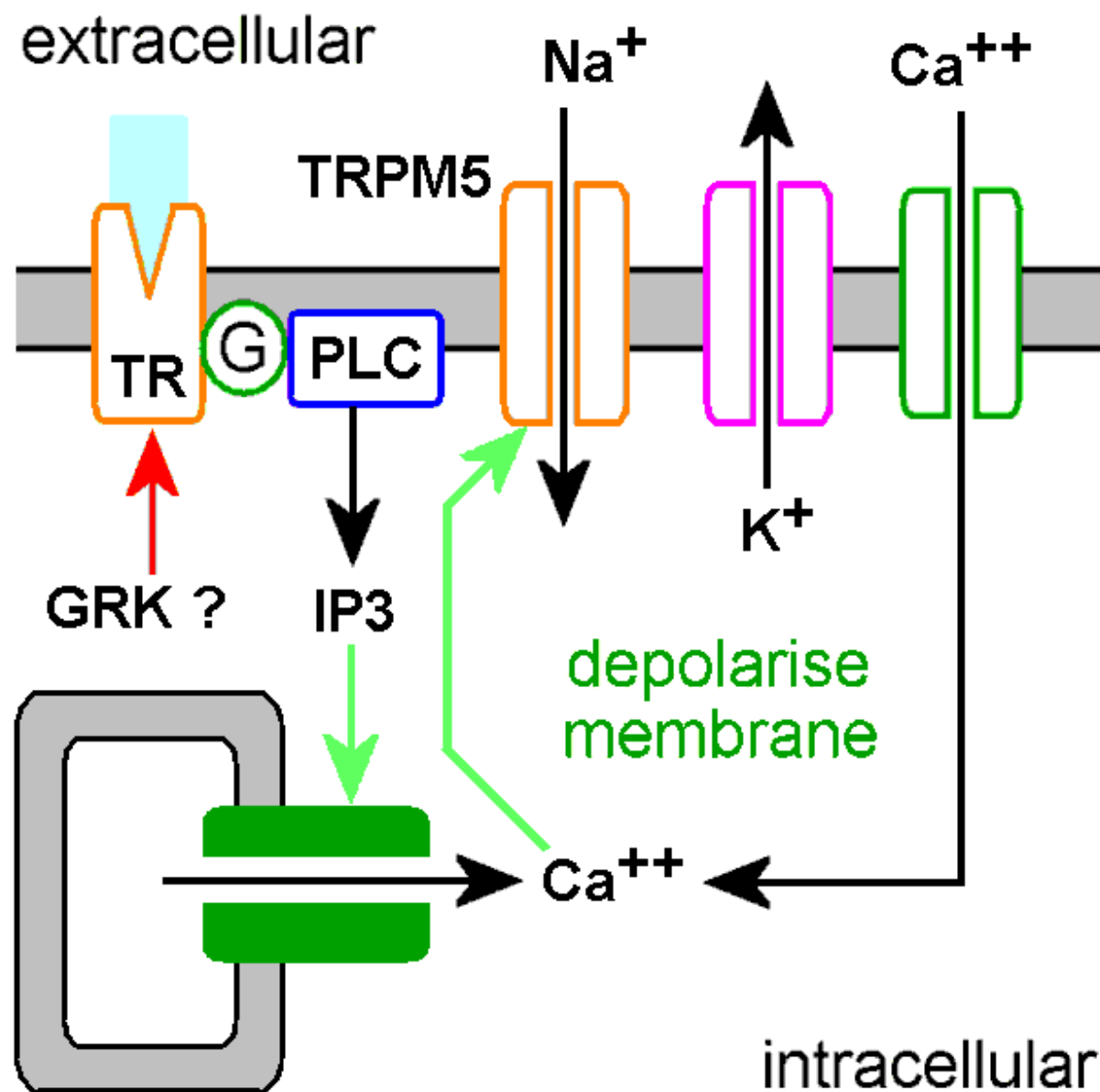
How Taste Works The Tongue

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Tongue 舌







- G - heterotrimeric G-protein
- GRK - G-protein receptor kinase
- IP3 - inositol triphosphate
- PLC - phospholipase
- TR - taste receptor

The Sweet Receptor function:

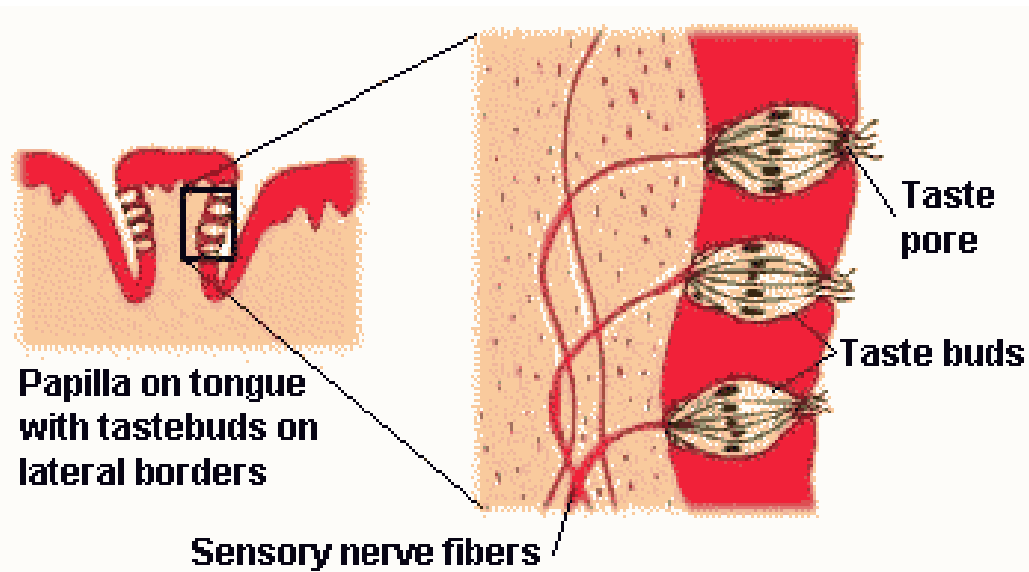
Each receptor contains 2 subunits designated **T1R2** and **T1R3** and is coupled to G proteins.

The complex of G proteins has been named **gustducin** because of its similarity in structure and action to the transducin that plays such an essential role in rod vision.

Activation of **gustducin** triggers a cascade of intracellular reactions:

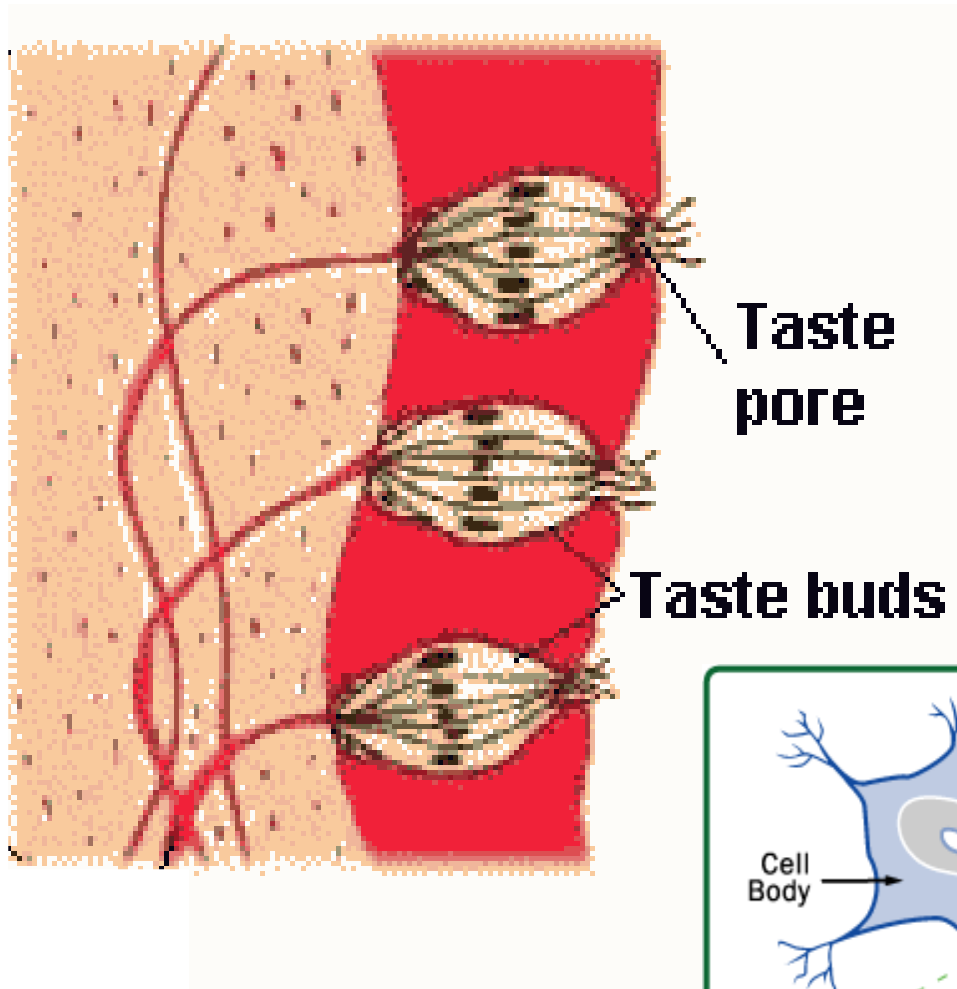
- activation of adenylyl cyclase
- formation of cyclic AMP (cAMP)
- the closing of K⁺ channels that leads to **depolarization** of the cell.

Emission of a electric **discharge signal**

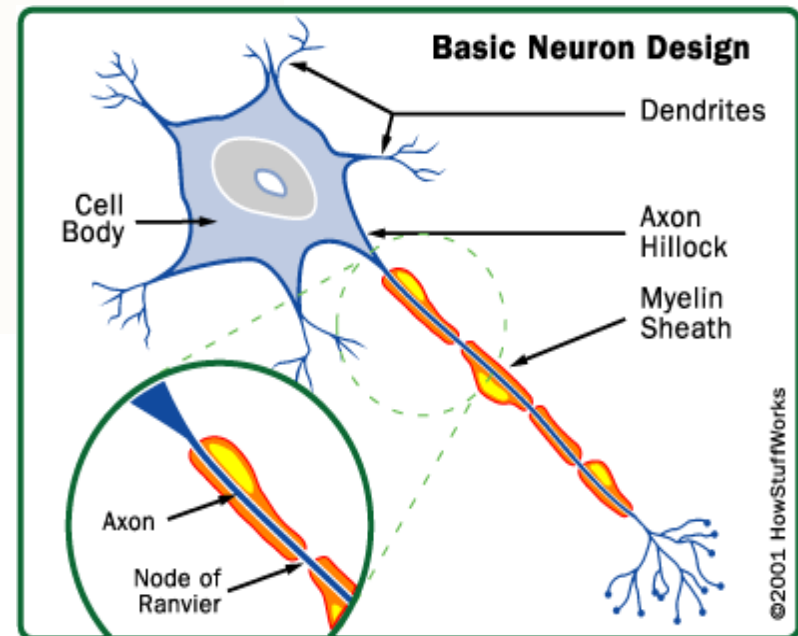


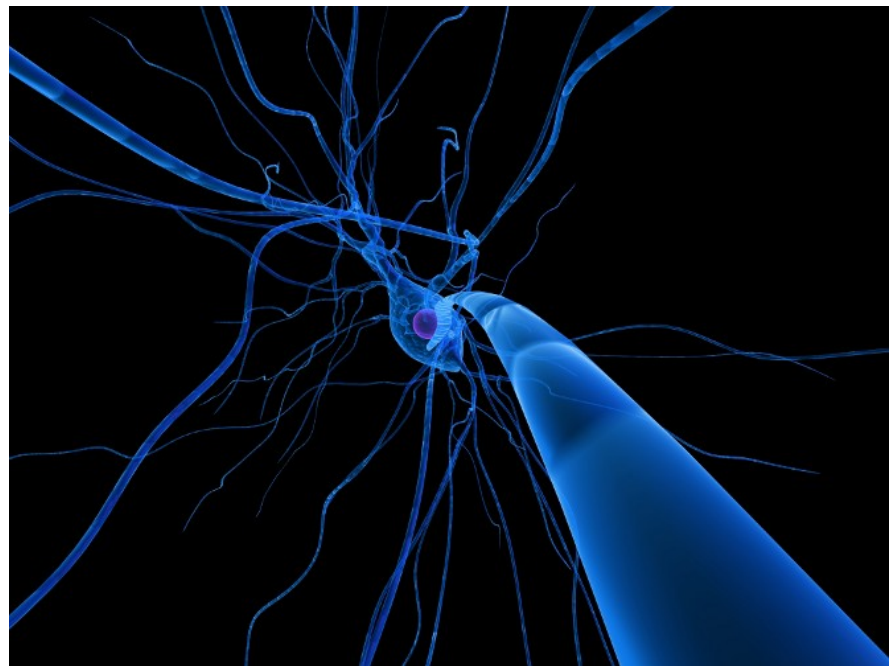
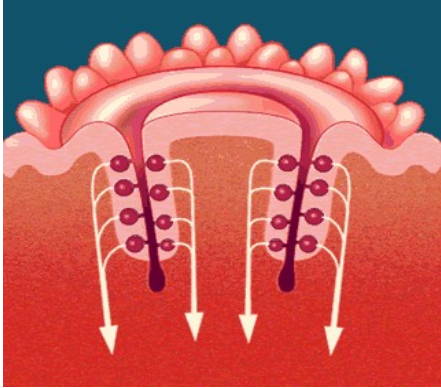
Examples of some human thresholds

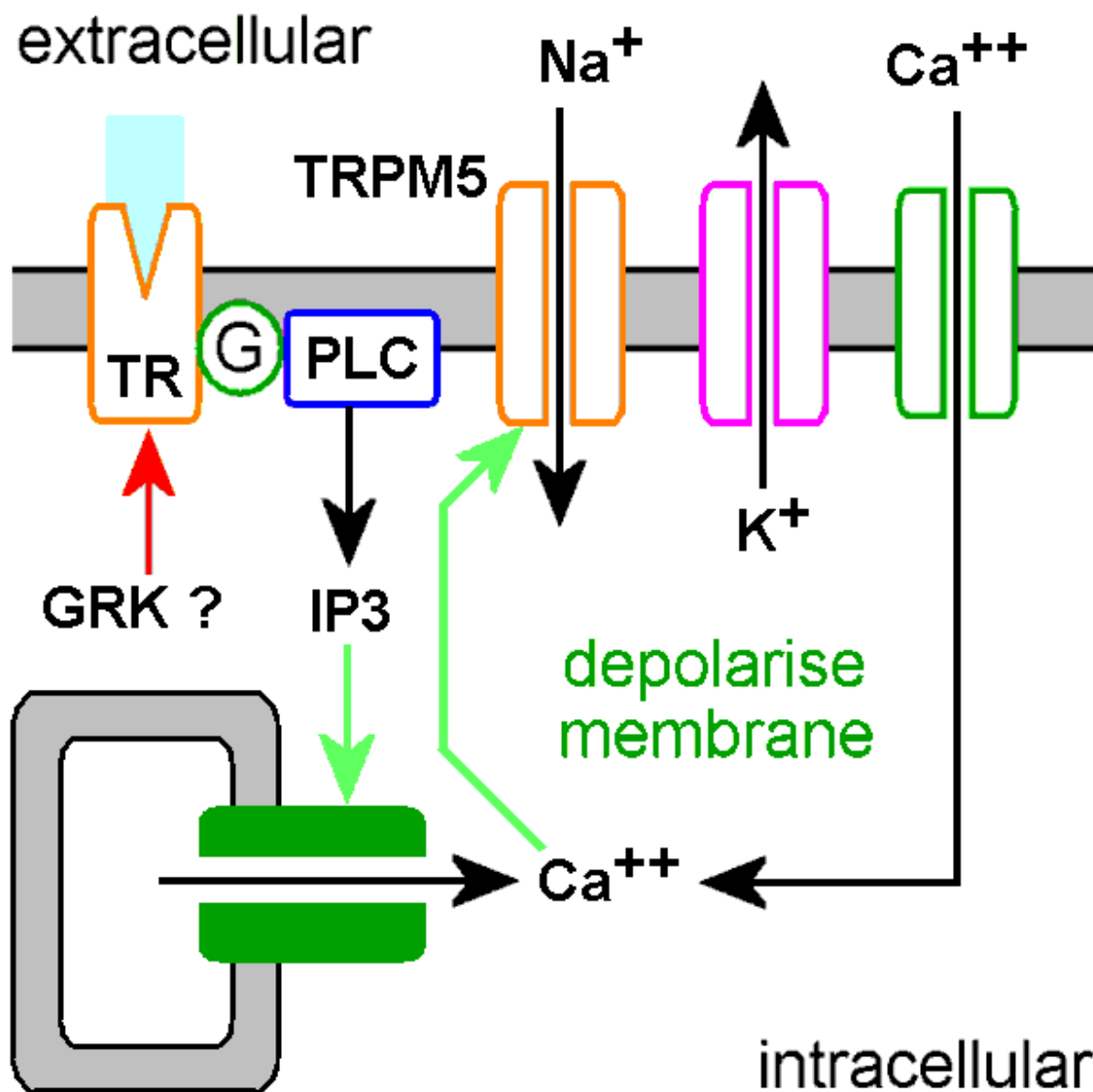
Taste	Substance	Threshold for tasting
Salty	NaCl	0.01 M
Sour	HCl	0.0009 M
Sweet	Sucrose	0.01 M
Bitter	Quinine	0.000008 M
Umami	Glutamate	0.0007 M



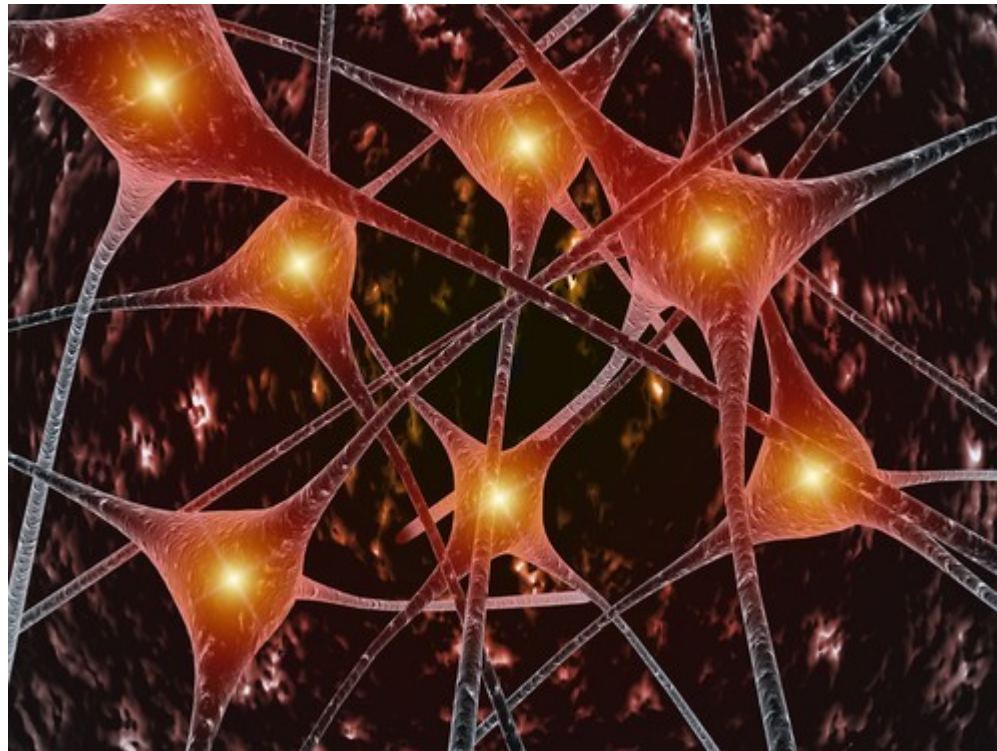
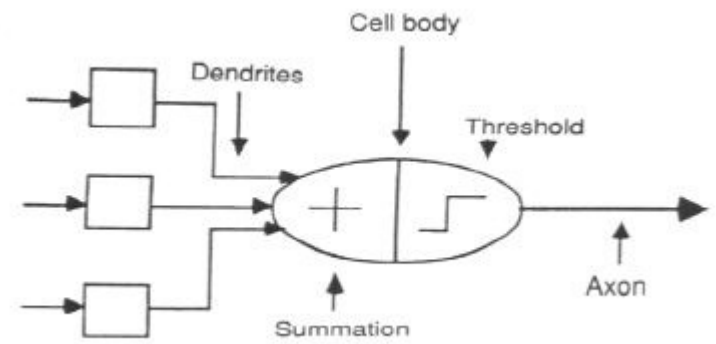
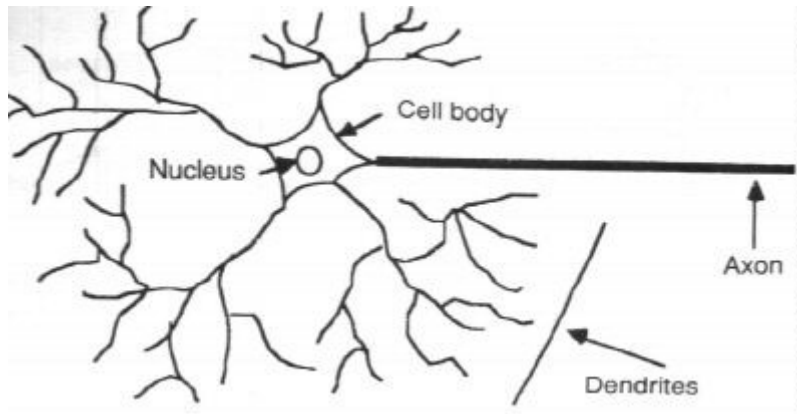
SPIKE signal is
transmitted
directly to the
BRAIN







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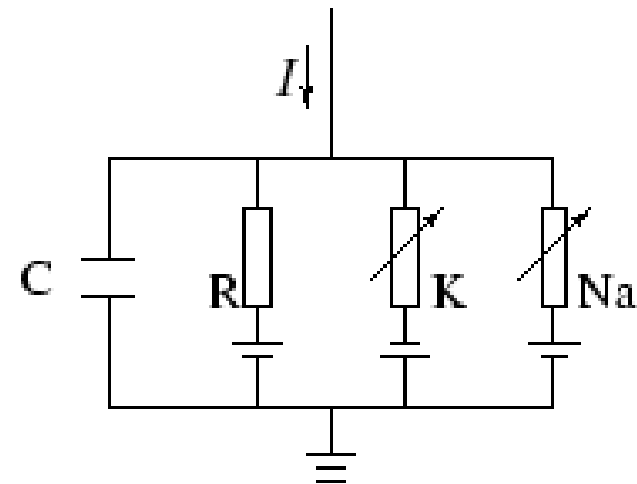
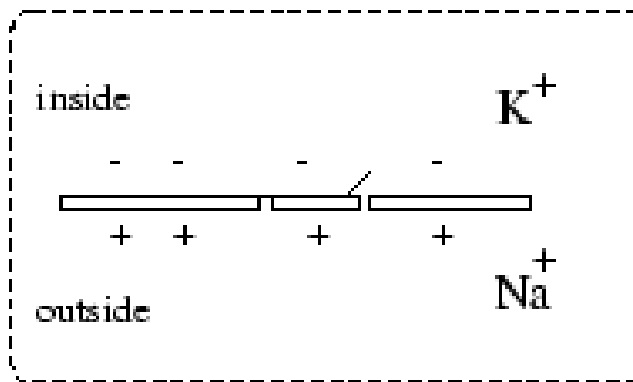
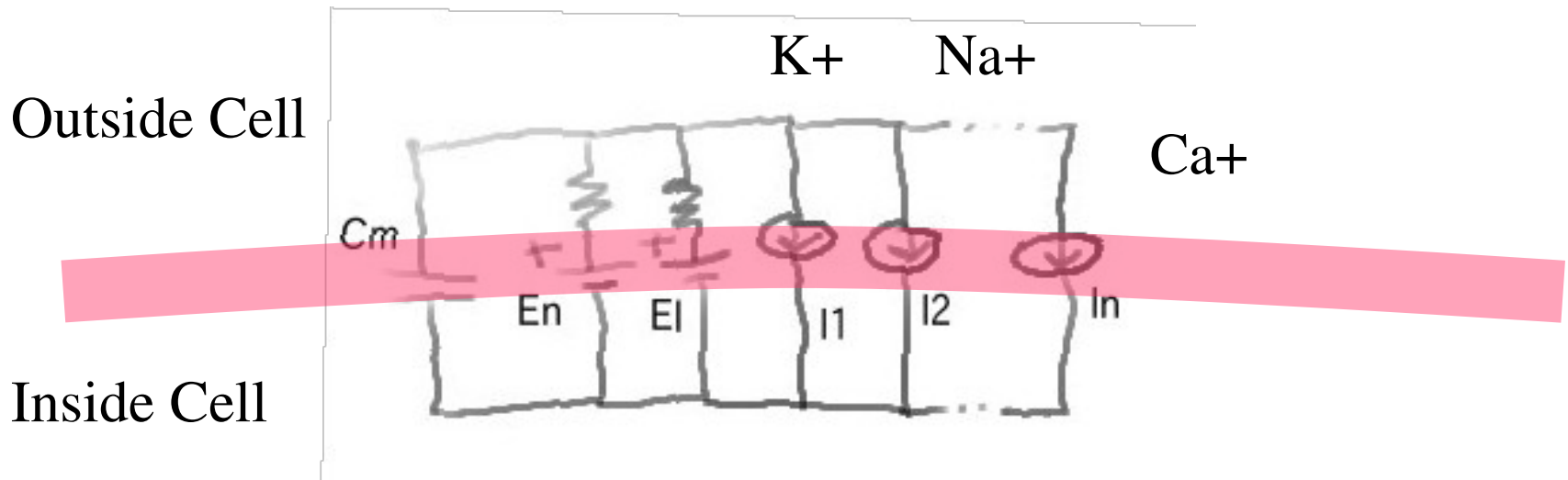
First Neuron Model

1907 by Lapicque

$$I(t) = C_m \frac{dV_m}{dt}$$

from $Q=CV$

Hodgkin-Huxley Model (1952)



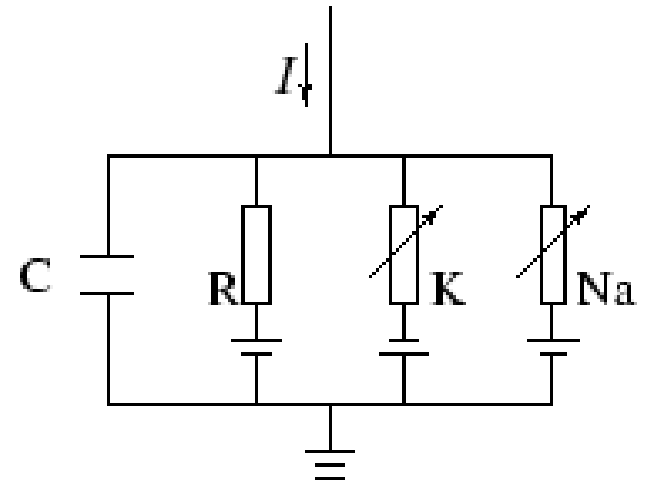
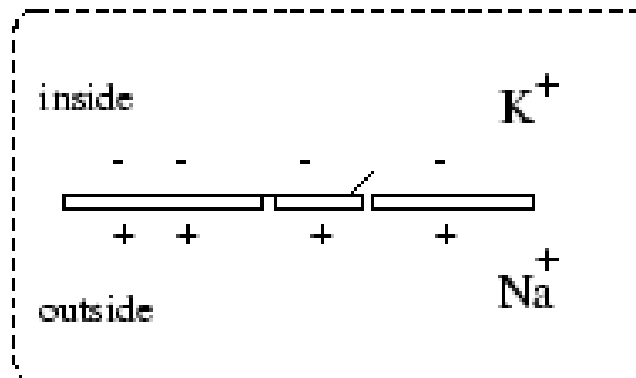


Hodgkin-Huxley Model (1952)

J. Physiol. Vol 117 500-540, 1952 by A.L. Hodgkin and A.F. Huxley.

Based on Giant Squid experiments

$$C \frac{du}{dt} = - \sum_k I_k(t) + I(t) .$$



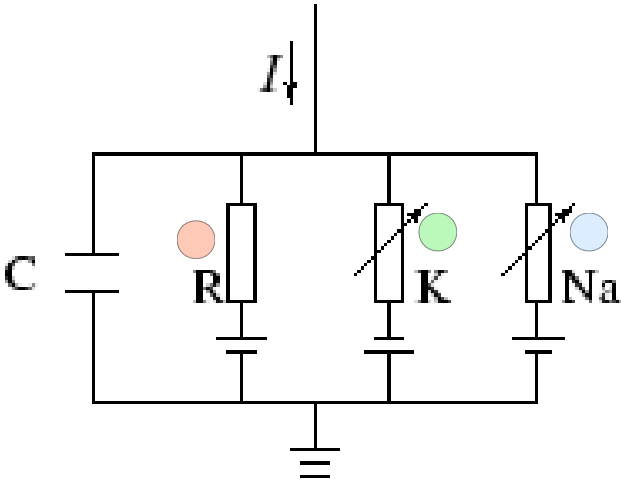
$$C \frac{du}{dt} = - \sum_k I_k(t) + I(t) .$$

$$\sum_k I_k = g_{\text{Na}}^{\text{blue}} m^3 h (u - E_{\text{Na}}) + g_{\text{K}}^{\text{green}} n^4 (u - E_{\text{K}}) + g_{\text{L}}^{\text{orange}} (u - E_{\text{L}}).$$

$$\dot{m} = \alpha_m(u) (1 - m) - \beta_m(u) m$$

$$\dot{n} = \alpha_n(u) (1 - n) - \beta_n(u) n$$

$$\dot{h} = \alpha_h(u) (1 - h) - \beta_h(u) h$$

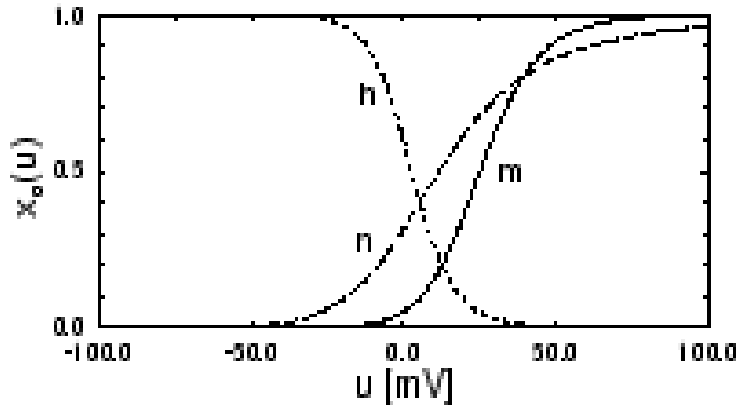


From Experiments

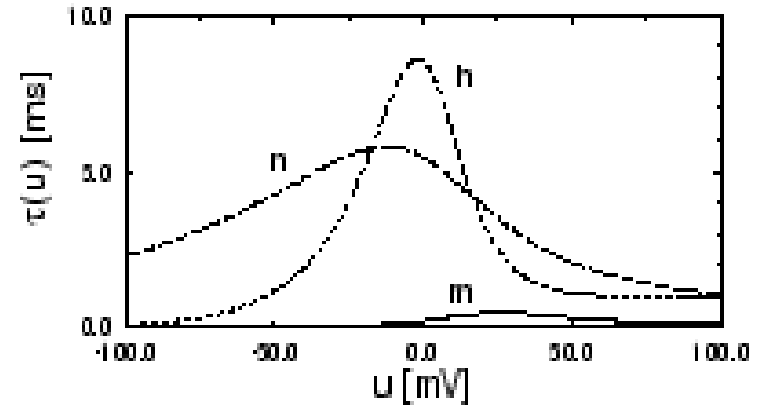
x	$\alpha_x(u / \text{mV})$	$\beta_x(u / \text{mV})$
n	$(0.1 - 0.01 u) / [\exp(1 - 0.1 u) - 1]$	$0.125 \exp(-u / 80)$
m	$(2.5 - 0.1 u) / [\exp(2.5 - 0.1 u) - 1]$	$4 \exp(-u / 18)$
h	$0.07 \exp(-u / 20)$	$1 / [\exp(3 - 0.1 u) + 1]$

x	E_x	g_x
Na	115 mV	120 mS/cm ²
K	-12 mV	36 mS/cm ²
L	10.6mV	0.3mS/cm ²

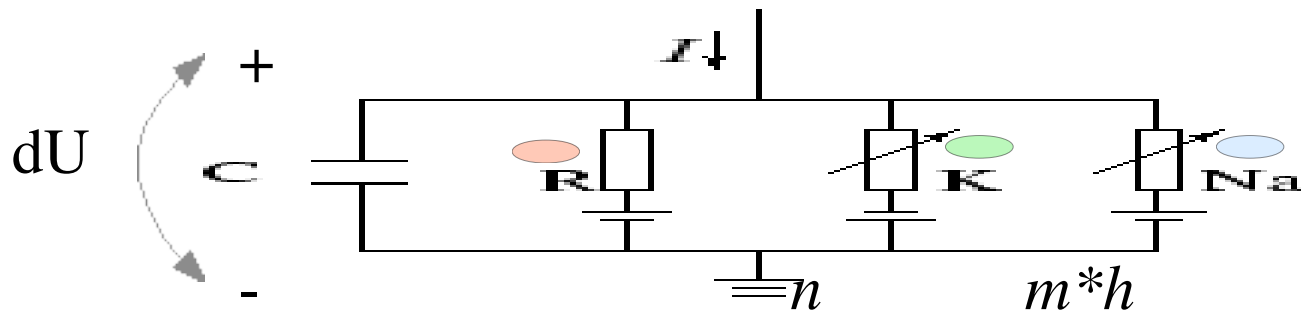
A

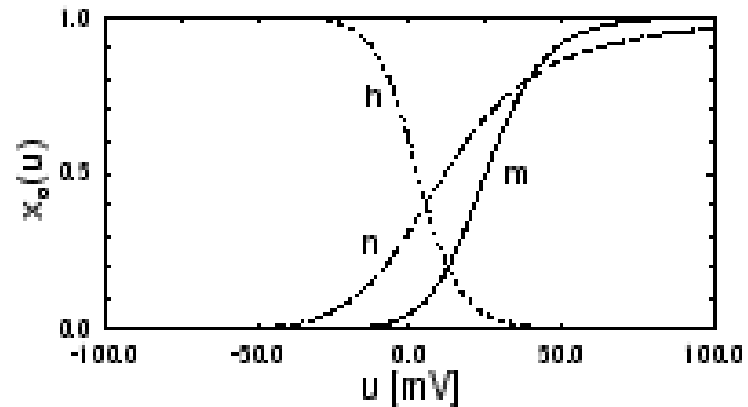
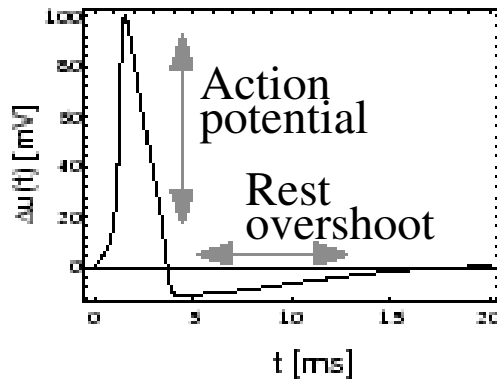


B



We see from that m and n increase with u whereas h decreases. Thus, if some external input causes the membrane voltage to rise, the conductance of sodium channels increases due to increasing m . As a result, positive sodium ions flow into the cell and raise the membrane potential even further. If this **positive feedback** is large enough, an **action potential** is initiated.

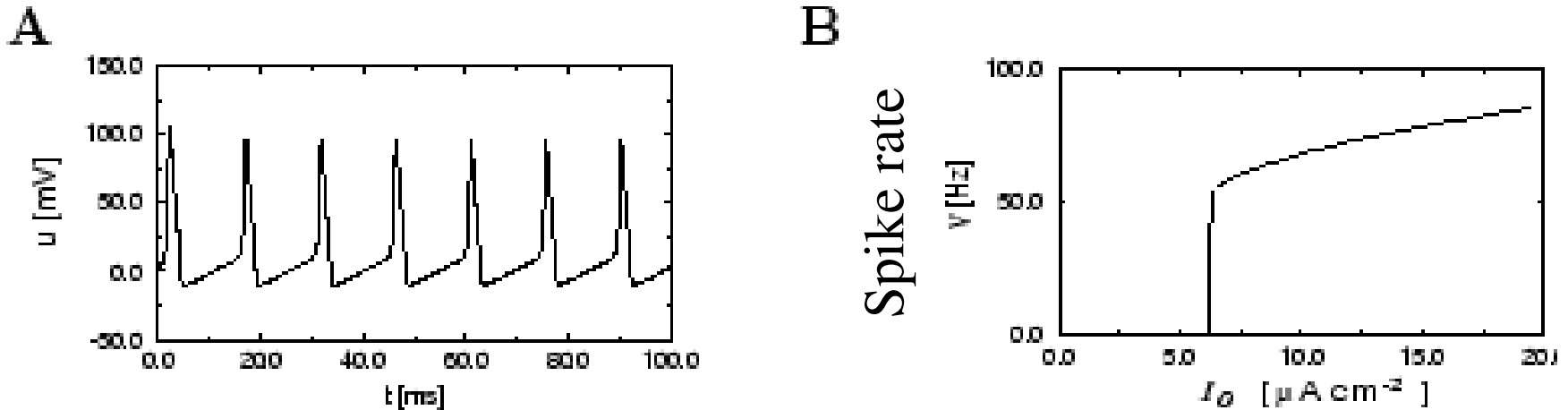




At high values of u the sodium conductance is shut off due to the factor h . As indicated the 'time constant' is always larger than τ_n . Thus the variable h which closes the channels reacts more slowly to the voltage increase than the variable m which opens the channel. On a similar slow time scale, the potassium (K^+) current sets in. Since it is a current in outward direction, it lowers the potential. The overall effect of the sodium and potassium currents is a short **action potential** followed by a **negative overshoot**; The amplitude of the spike is about 100 mV

$$\sum_k I_k = \overset{\text{blue}}{g_{Na}} m^3 \overset{\text{red}}{h} (u - E_{Na}) + \overset{\text{green}}{g_K} n^4 (u - E_K) + \overset{\text{orange}}{g_L} (u - E_L).$$

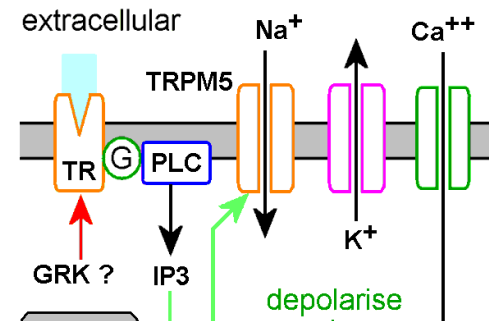
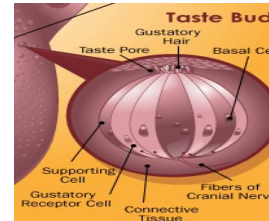
Auto-oscillation is possible



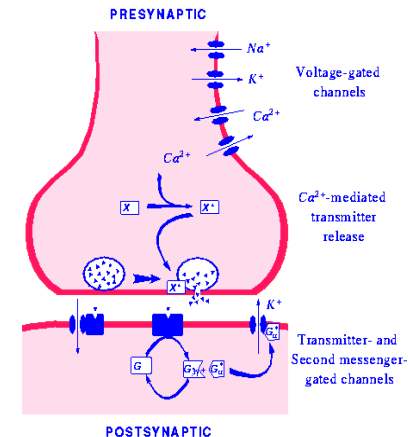
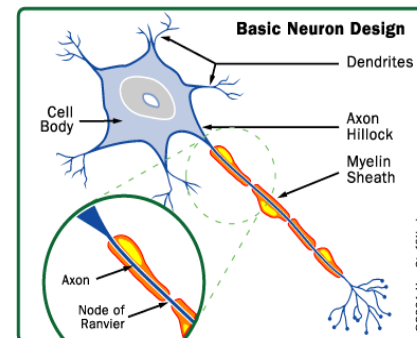
The Hodgkin-Huxley equations may also be studied for **constant** input $I(t) = I_0$ for $t > 0$. (The input is zero for $t < 0$). If the value I_0 is larger than a **critical value** $I_0 \approx 6 \mu\text{A/cm}^2$, we observe **regular spiking**; We may define a firing rate $= 1/T$ where T is the inter-spike interval. The firing rate as a function of the constant input I_0 defines the gain function plotted.

TASTE-TONGUE PROCESS - Conclusions

- food is dissolved in mouth
- molecules enter the “Taste Bud”
- contact with cell membrane



- “Taste Receptor” activate (sweet, salty, bitter, sour...)
- “SPIKE” electrical signal is generated
- transported to brain by neurons



Synthetic pores with reactive signal amplifiers as artificial tongues

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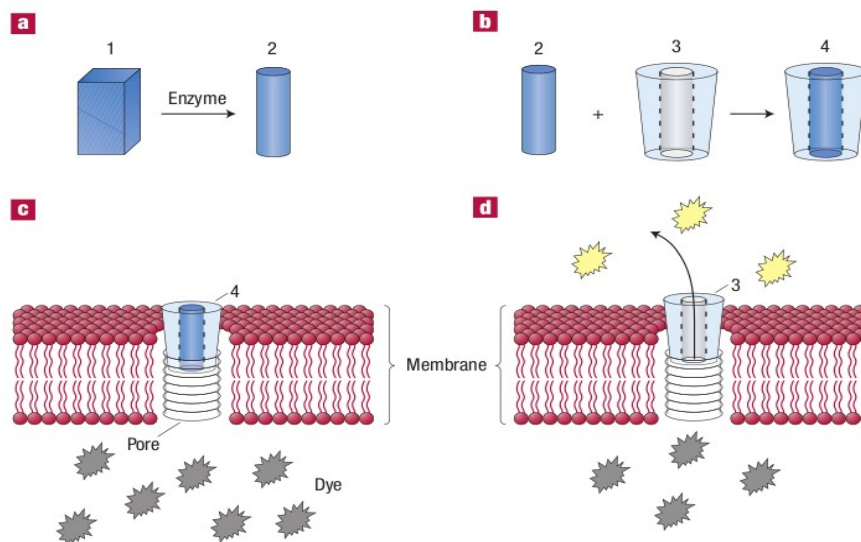


Figure 1 The processes involved in the artificial tongue of Matile and colleagues⁹. **a**, Signal generation. An enzyme converts 1 to 2. **b**, Reactive amplification. The product of the enzyme reaction, 2, reacts with the reactive amplifier 3, to form 4. **c**, Pore blockage. If analyte 1 is present, 4 is formed, which can block the pore and stop release of the dye. **d**, Dye release. If no analyte 1 is present, then 4 is not formed, and only 3 is present. Even though 3 can bind to the pore to some extent, it does not block release of the dye, and 2 cannot block the pore on its own.

What do we need to make an Artificial Tongue ?

- food is something that must be **TOUCHED**
- sensing **CHEMICAL** properties at **CONTACT**

**We need different chemical RECEPTORS
for different TASTES (sweet, salty, bitter, sour...)**