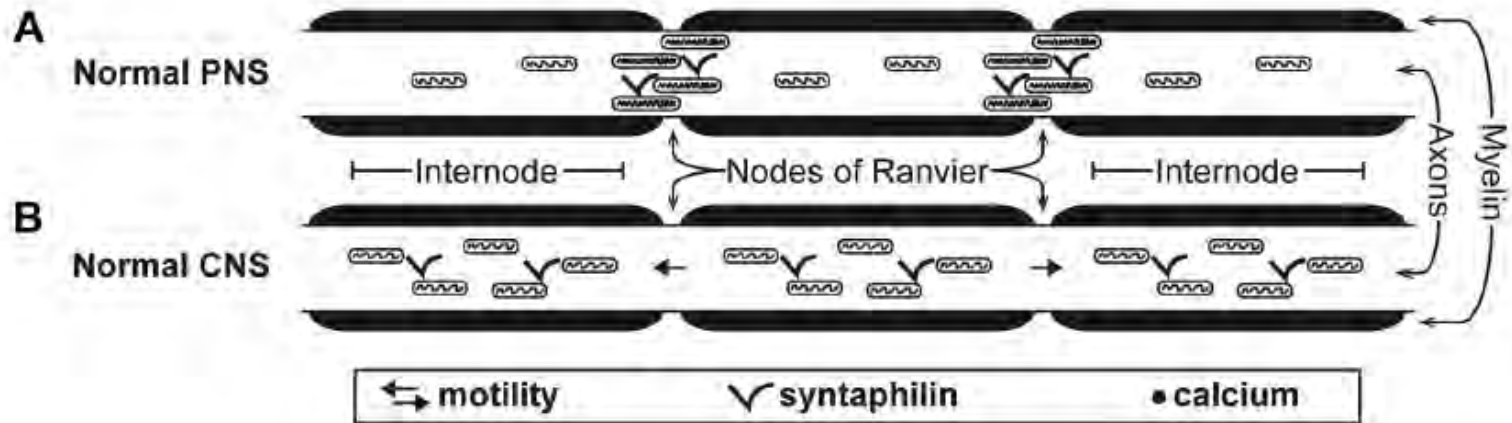


Y a-t-il accumulation de mitochondries au niveau des nœuds de Ranvier ?

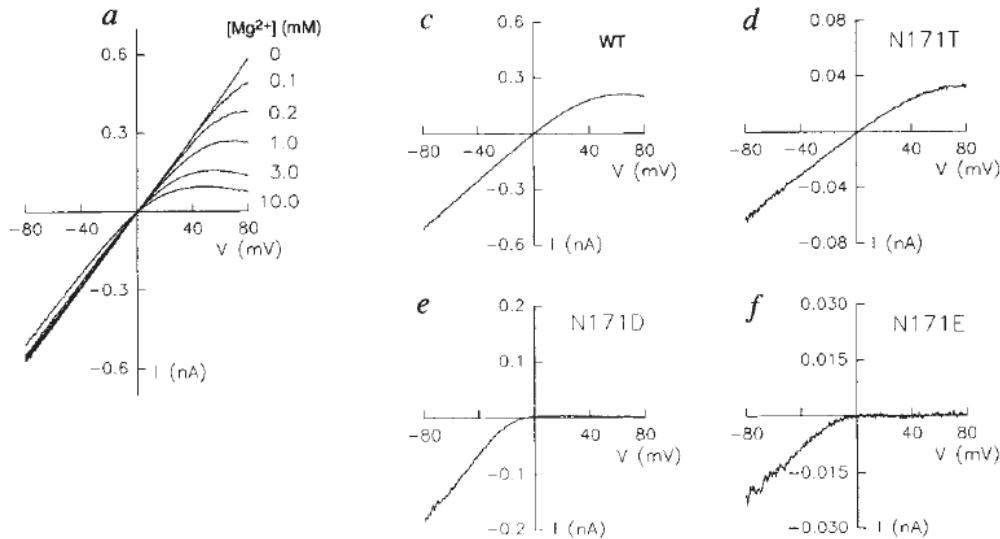
R.B. Griggs et al. / Neuroscience Research 116 (2017) 77–87



# Quel est le mécanisme sous-jacent à la rectification des canaux potassiques?

## • Rectification entrante

Rôle du Mg intracellulaire et d'un résidu polaire (D ou N) dans le domaine TM M2 (Kir 2.1 D171/GIRK1 D173/GIRK2 N184) : plus forte rectification pour les canaux avec résidu D plutôt que N



*Polaire, non chargé*

*Polaire, charge -*

Lu & McKinnon, Nature 1994

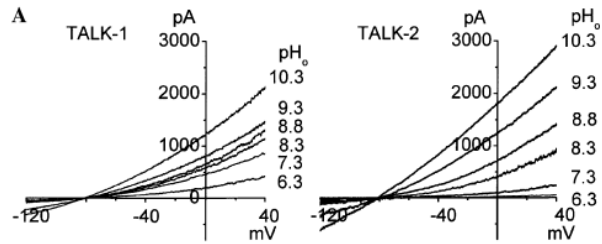
Rôle des polyamines intracellulaires

Autres résidus de l'extrémité C-terminale

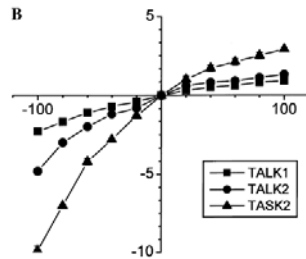
# Quel est le mécanisme sous-jacent à la rectification des canaux potassiques?

- Rectification sortante (e.g; K2P leak potassium channels)

Courant macroscopique: rectification sortante



Courant unitaire : rectification entrante ! > conclusion ?



## Calcul de la contribution de l'activité électrogénique de la Na/K ATPase au potentiel de membrane

$$58 \log \frac{(p_{\text{Na}}/p_{\text{K}}) [\text{Na}]_{\text{extra}} + r [\text{K}]_{\text{extra}}}{(p_{\text{Na}}/p_{\text{K}}) [\text{Na}]_{\text{intra}} + r [\text{K}]_{\text{intra}}} = V_m \quad r : \text{ratio de transport Na/K}$$

Courant par le transporteur :  $t_{\text{Na}}$  et  $t_{\text{K}}$

**Au repos, les conductances de fuite Na et K compensent exactement les flux liés au transporteur,**

Donc  $t_{\text{Na}} = -i_{\text{Na}}$  et  $t_{\text{K}} = -i_{\text{K}}$

Si  $t_{\text{Na}}/t_{\text{K}} = -r$  (négatif car le transport est en sens inverse)

Alors  $i_{\text{Na}} = -r i_{\text{K}}$

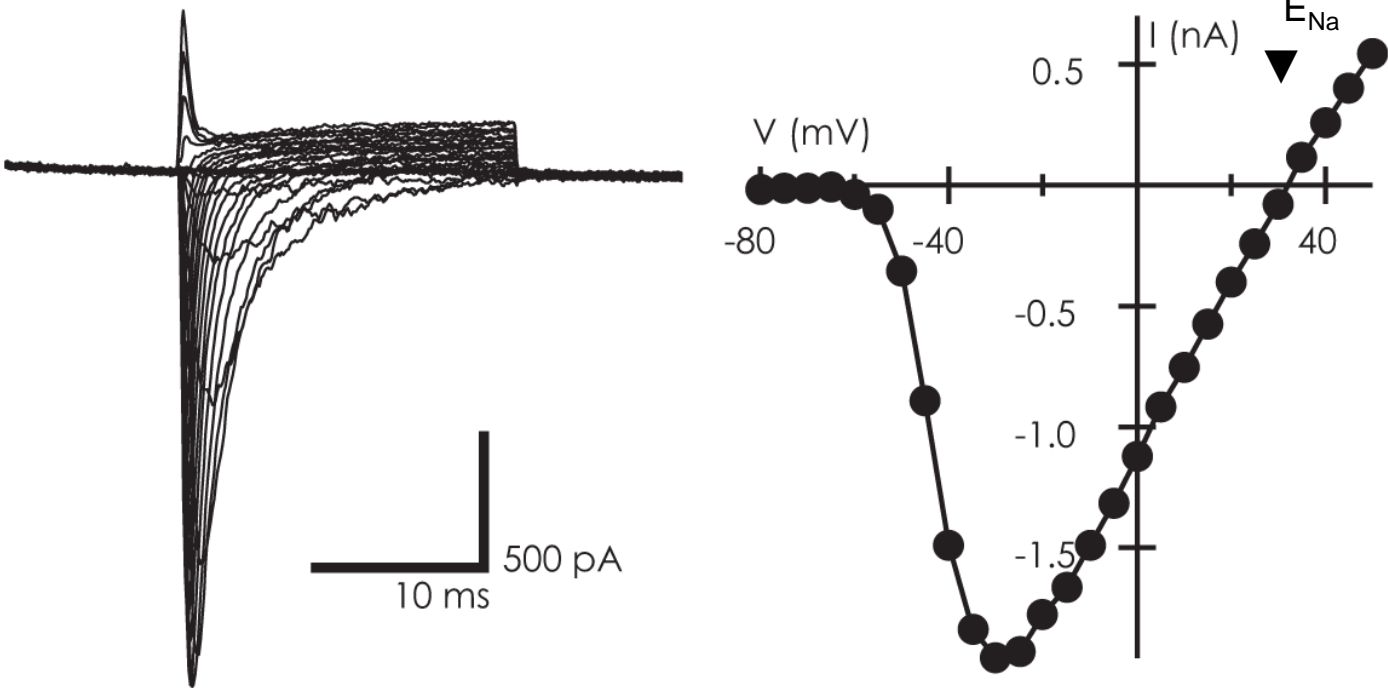
Donc  $p_{\text{Na}} ([\text{Na}]_{\text{extra}} - [\text{Na}]_{\text{intra}} e^{V'}) = -r p_{\text{K}} ([\text{K}]_{\text{extra}} - [\text{K}]_{\text{intra}} e^{V'})$  où  $V' = V_m F/RT$

D'où  $e^{V'} = (rp_{\text{K}} [\text{K}]_{\text{extra}} + p_{\text{Na}} [\text{Na}]_{\text{extra}}) / (rp_{\text{K}} [\text{K}]_{\text{intra}} + p_{\text{Na}} [\text{Na}]_{\text{intra}})$

Donc  $V_m = RT/F \ln \frac{r [\text{K}]_{\text{extra}} + p_{\text{Na}}/p_{\text{K}} [\text{Na}]_{\text{extra}}}{r [\text{K}]_{\text{intra}} + p_{\text{Na}}/p_{\text{K}} [\text{Na}]_{\text{intra}}}$

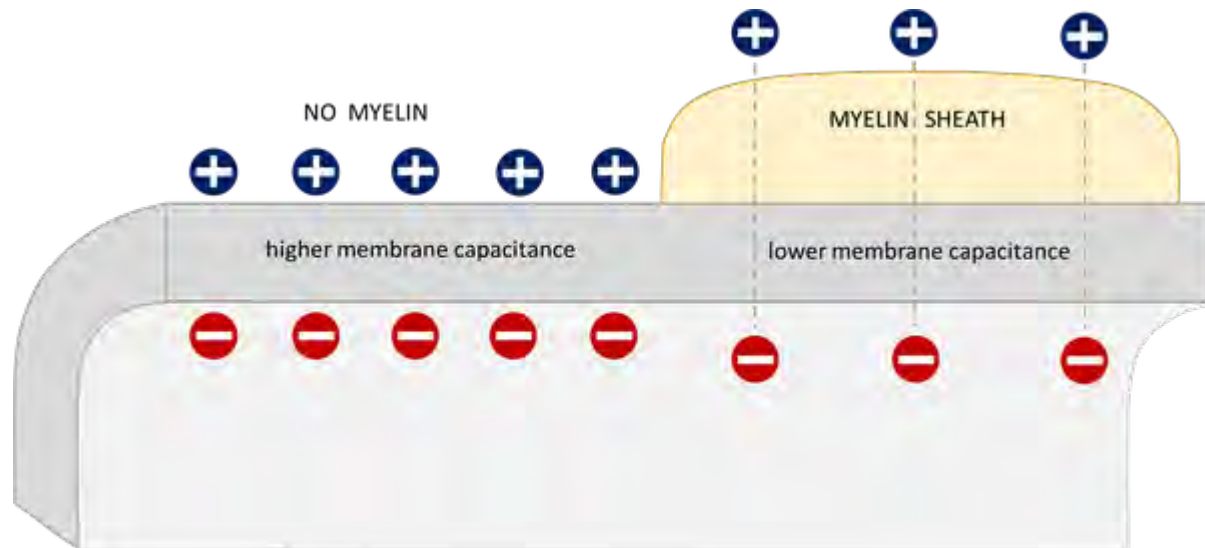
Les canaux sodiques voltage dépendants peuvent ils porter des courants sortants ?

Nav1.5 in HEK cells



## Pourquoi la myélinisation accélère la conduction axonale ?

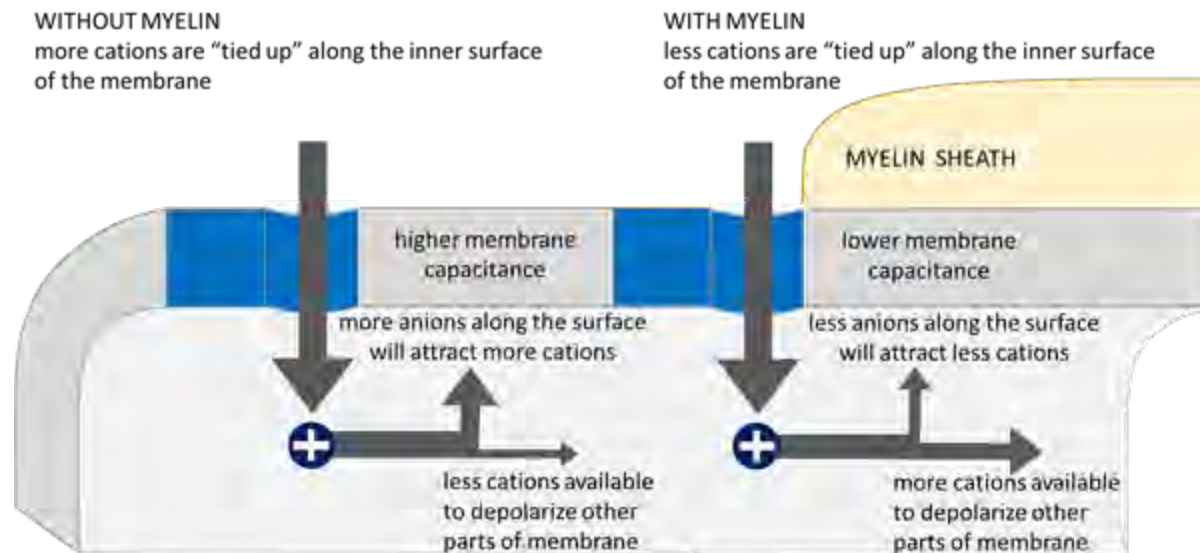
<https://www.quora.com/What-is-an-intuitive-way-of-explaining-how-myelin-speeds-up-nerve-conduction-by-reducing-the-capacitance-and-increasing-the-resistance-of-the-axonal-membrane>



**Fig 9. Myelin reduces membrane capacitance** by increasing the thickness of the membrane (increase in separation of cations and anions) and by decreasing the amount of charge stored on both sides of the membrane.

## Pourquoi la myélinisation accélère la conduction axonale ?

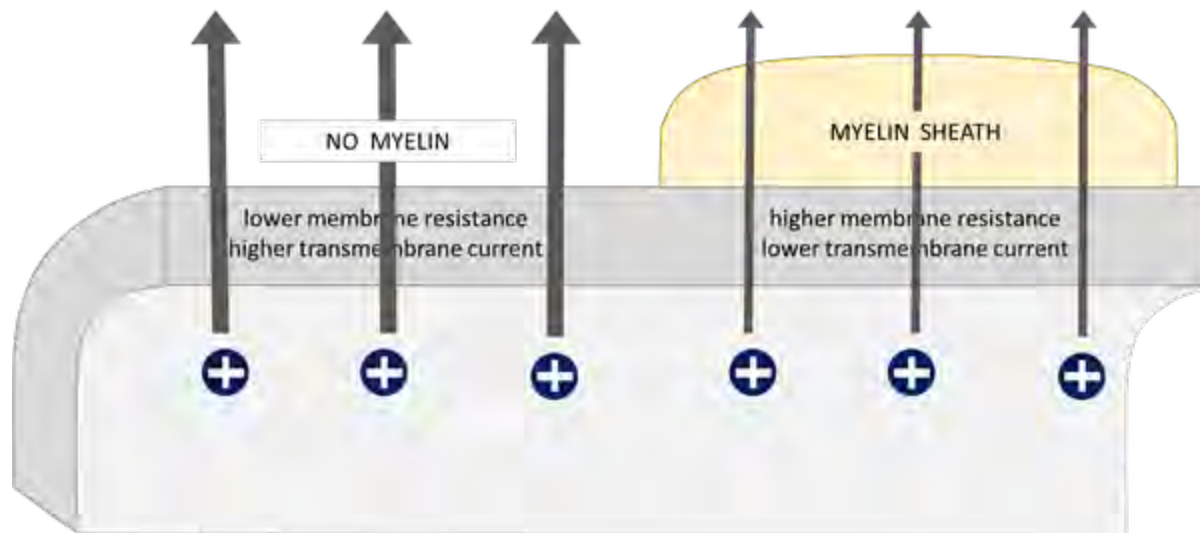
<https://www.quora.com/What-is-an-intuitive-way-of-explaining-how-myelin-speeds-up-nerve-conduction-by-reducing-the-capacitance-and-increasing-the-resistance-of-the-axonal-membrane>



**Fig 10.** Membrane capacitance **without** (left) and **with myelin** (right).

## Pourquoi la myélinisation accélère la conduction axonale ?

<https://www.quora.com/What-is-an-intuitive-way-of-explaining-how-myelin-speeds-up-nerve-conduction-by-reducing-the-capacitance-and-increasing-the-resistance-of-the-axonal-membrane>

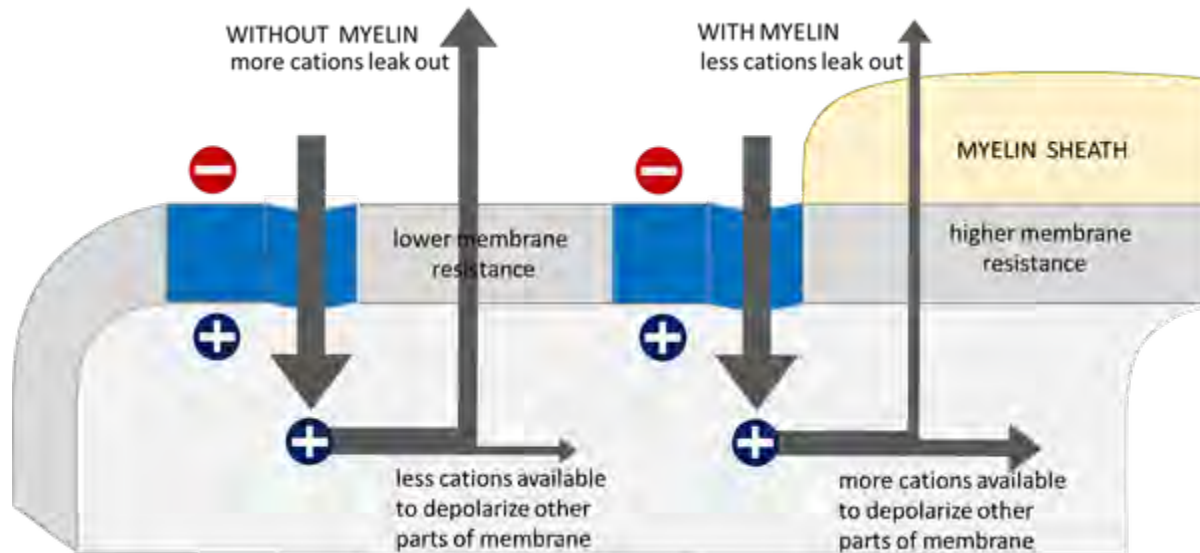


**Fig 11. Myelin increases membrane resistance** and this reduces the transmembrane ion current. The reduced cation "leak current" is shown as thinner arrows pointing outward (from axoplasm to ECF).



## Pourquoi la myélinisation accélère la conduction axonale ?

<https://www.quora.com/What-is-an-intuitive-way-of-explaining-how-myelin-speeds-up-nerve-conduction-by-reducing-the-capacitance-and-increasing-the-resistance-of-the-axonal-membrane>



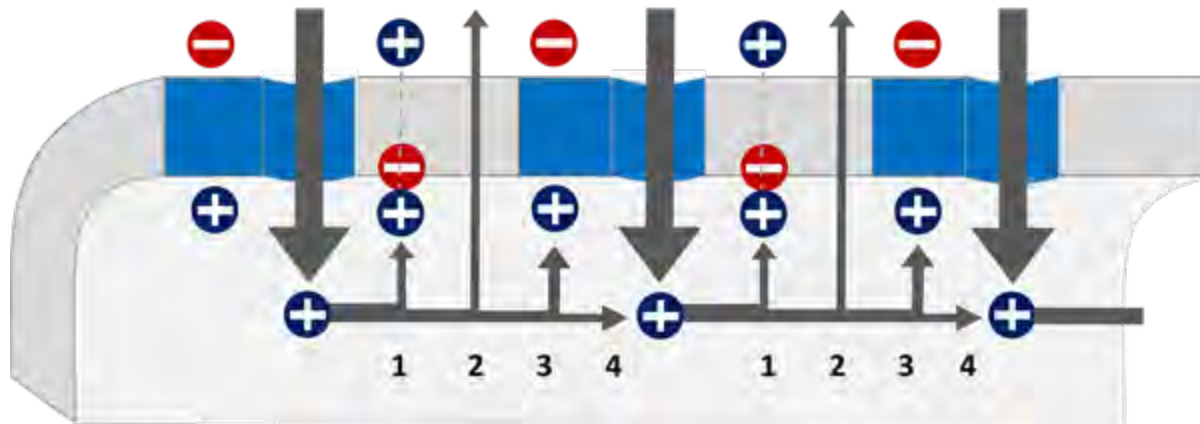
**Fig 12.** Membrane resistance **without** (left) and **with myelin** (right).

$$V_x = V_o e^{-x/\lambda}$$

$$\lambda = (r_m/r_i)^{1/2} \text{ constante d'espace}$$

## Pourquoi la myélinisation accélère la conduction axonale ?

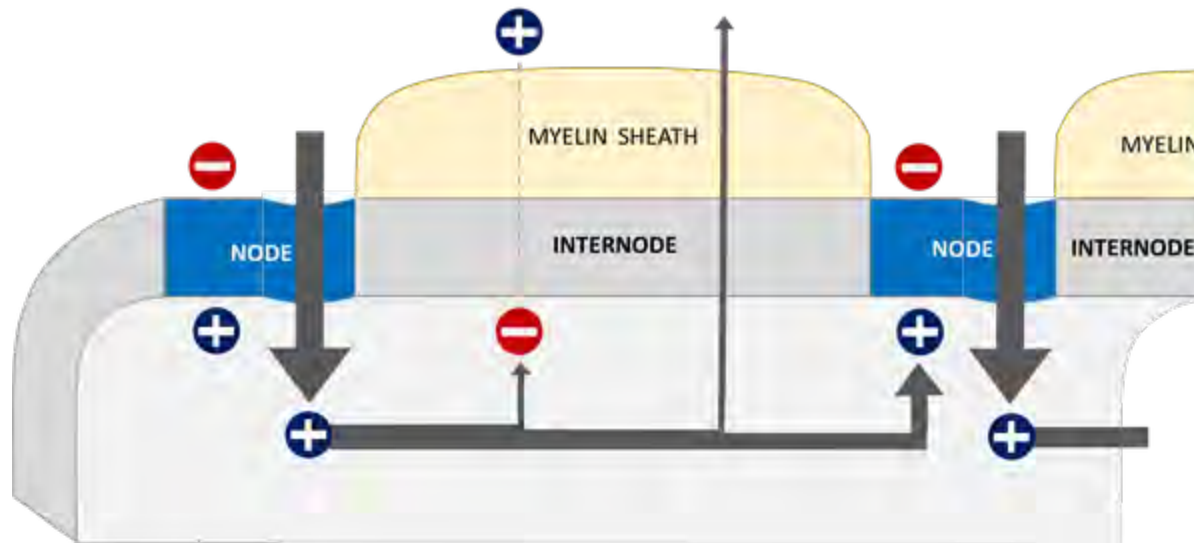
<https://www.quora.com/What-is-an-intuitive-way-of-explaining-how-myelin-speeds-up-nerve-conduction-by-reducing-the-capacitance-and-increasing-the-resistance-of-the-axonal-membrane>



**Fig 13.** In **unmyelinated axons**, paths 1 and 2 are non-trivial. More cations are "tied up" and more leak out, so less cations are available to depolarize the membrane some distance from the entry point. The next depolarization will therefore occur only a short distance from the current source. At some from the source, there are not enough cations to depolarize the membrane and trigger an action potential.

## Pourquoi la myélinisation accélère la conduction axonale ?

<https://www.quora.com/What-is-an-intuitive-way-of-explaining-how-myelin-speeds-up-nerve-conduction-by-reducing-the-capacitance-and-increasing-the-resistance-of-the-axonal-membrane>



**Fig 14.** In **myelinated axons**, paths 1 and 2 become less important. Because of *lower membrane capacitance*, less cations are "tied up" with the membrane. The *higher membrane resistance* also results in less cations leaking out of the membrane. Consequently, more cations (larger current) are available to depolarize the membrane at some distance from the entry point. Therefore, depolarization can effectively take place far from the current source

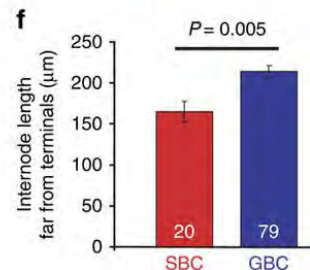
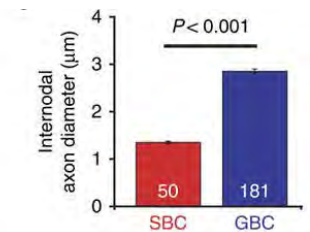
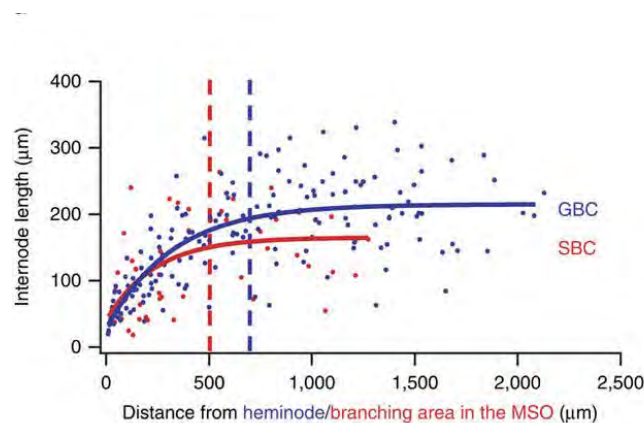
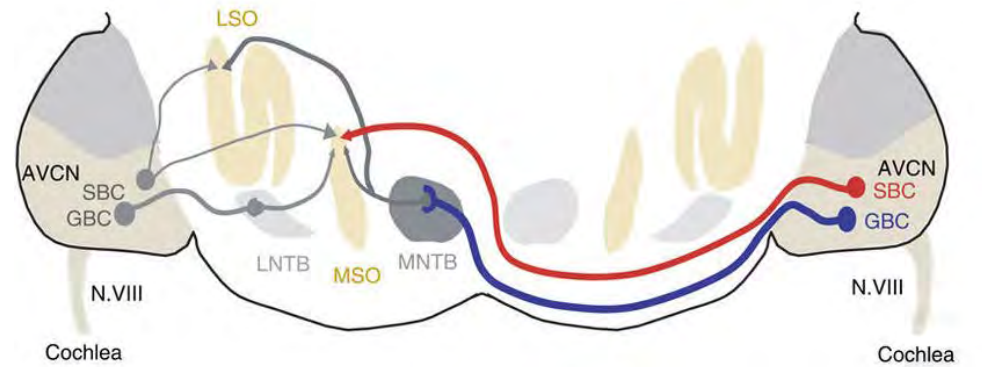
# La distance internodale est-elle « optimale » ?

Modélisation : Young et al. J Comput Neurosci. 2013

Biologie : Tuning of Ranvier node and internode properties in myelinated axons to adjust action potential timing  
Ford et al. Nature Communications 2015

In the bird pathway, processing interaural time differences (ITD), variation in axon morphology is used for adjusting AP arrival times : thicker axons with longer internodes compensate for different axonal lengths.

The analogous mammalian auditory brainstem circuits that process ITDs and interaural level differences (ILDs) have similar exceptional needs for temporal precision in AP propagation



A quoi sert un canal sodique voltage-dépendant chez la bactérie ?

REPORTS

## A Prokaryotic Voltage-Gated Sodium Channel

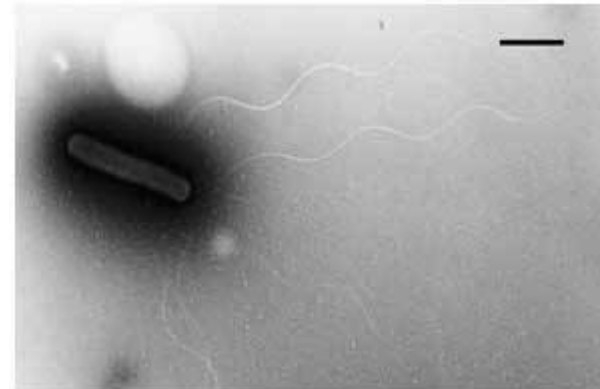
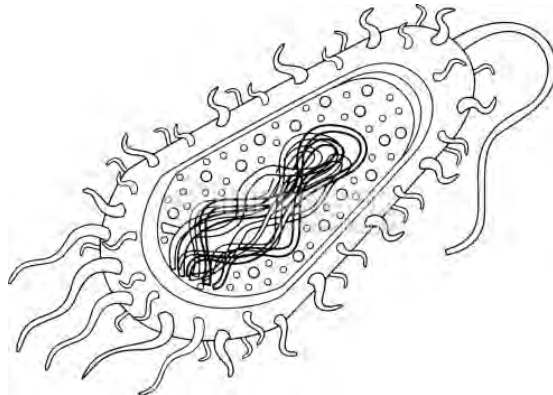
Dejian Ren,<sup>1\*</sup> Betsy Navarro,<sup>1,2\*</sup> Haoxing Xu,<sup>1\*</sup> Lixia Yue,<sup>1\*</sup>  
Qing Shi,<sup>1</sup> David E. Clapham<sup>1,†</sup>

14 DECEMBER 2001 VOL 294 SCIENCE www.sciencemag.org

We have not tested the biological role of NaChBac in the extremophile *Bacillus halodurans*.

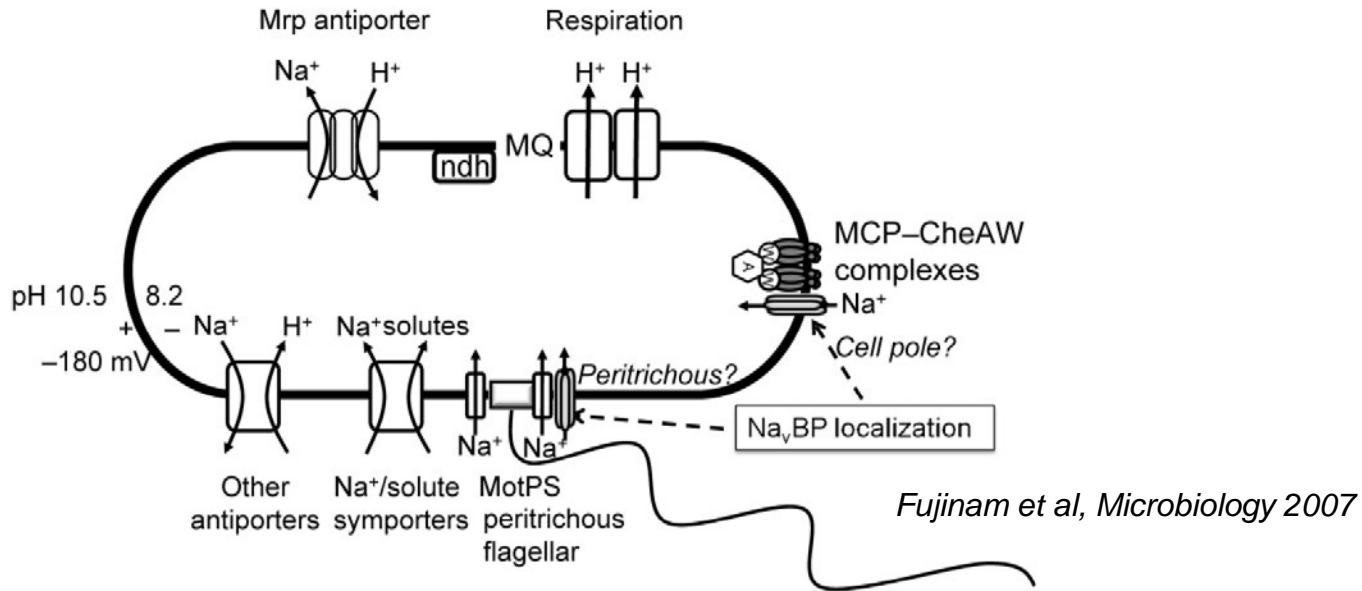
This bacterium lives in extremely high salt (up to 1 M), highly alkaline (up to pH 11) conditions, and thus Na<sup>+</sup> influx through the open channel should be large.

Na<sup>+</sup> drives the flagellar motor used by alkaphilic *Bacillus*, and NaChBac is a good candidate for control of flagellar activity.



# A quoi sert un canal sodique voltage-dépendant chez la bactérie ?

Le canal NavBP s'accumule à proximité du pôle chemotactile et du flagelle de la bactérie *Bacillus pseudofirmus*



The voltage-gated Na<sup>+</sup> channel NavBP has a role in motility, chemotaxis, and pH homeostasis of an alkaliphilic *Bacillus*

PNAS

Masahiro Ito<sup>1\*</sup>, Haoxing Xu<sup>1,2</sup>, Arthur A. Guffanti<sup>3</sup>, Yi Wei<sup>3</sup>, Lior Zvi<sup>3</sup>, David E. Clapham<sup>4</sup>, and Terry A. Krulwich<sup>5†</sup>