Prévention de l'hyperalgésie et de la douleur chronique postopératoire: La nutrition a-t-elle un sens?

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Définition

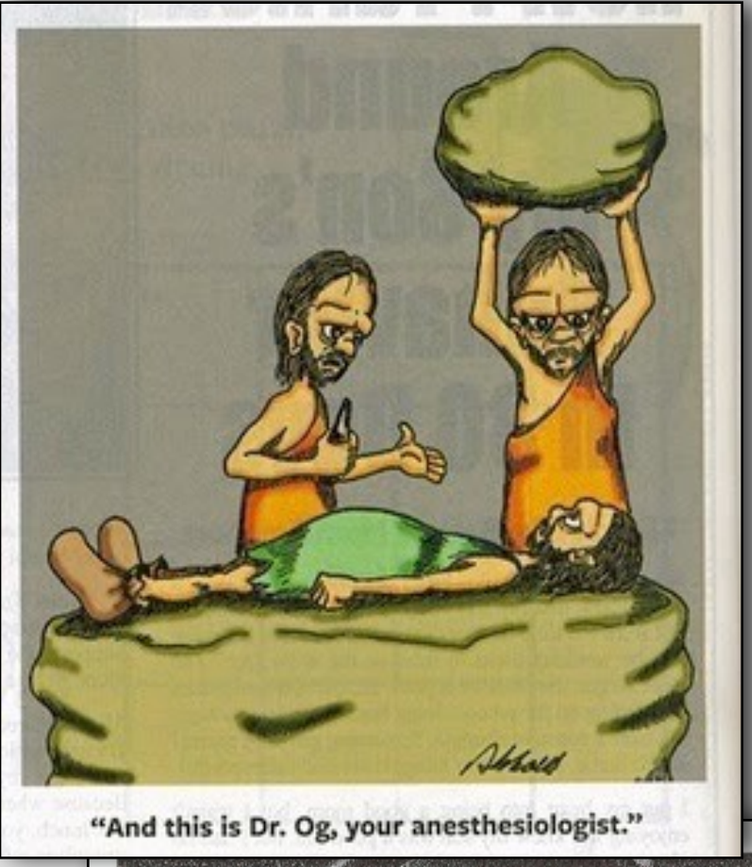
Douleur

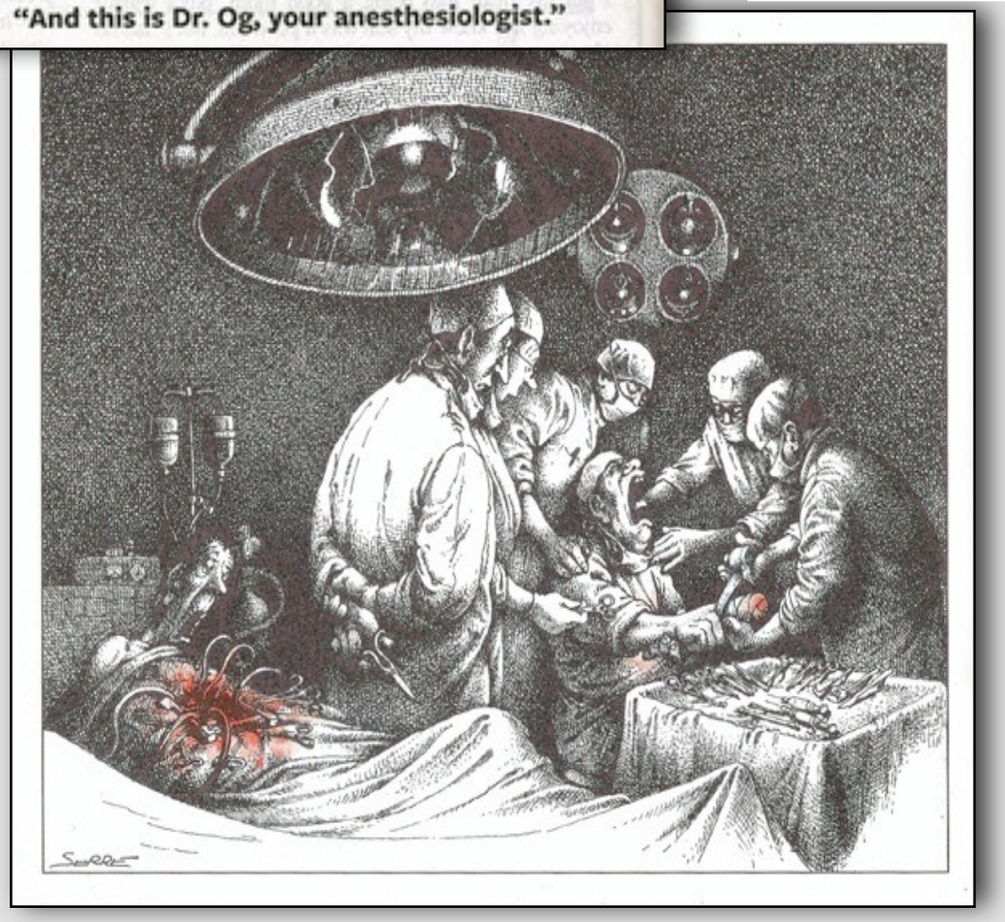
«Expérience sensorielle et émotionnelle désagréable liée à une lésion tissulaire réelle ou potentielle ou décrite dans des termes évoquant une telle lésion.» (IASP)

Merskey H et al: classification of Chronic Pain. IASP Taskforce on Taxonomy, 1994: 209-214

Douleur = Phénomène Subjectif

Prévention de la DPO et de la DCPO: rôle de chacun…





First Day After Surgery Indicates Chance for Chronic Pain Later

euroanesthesia 9/2015

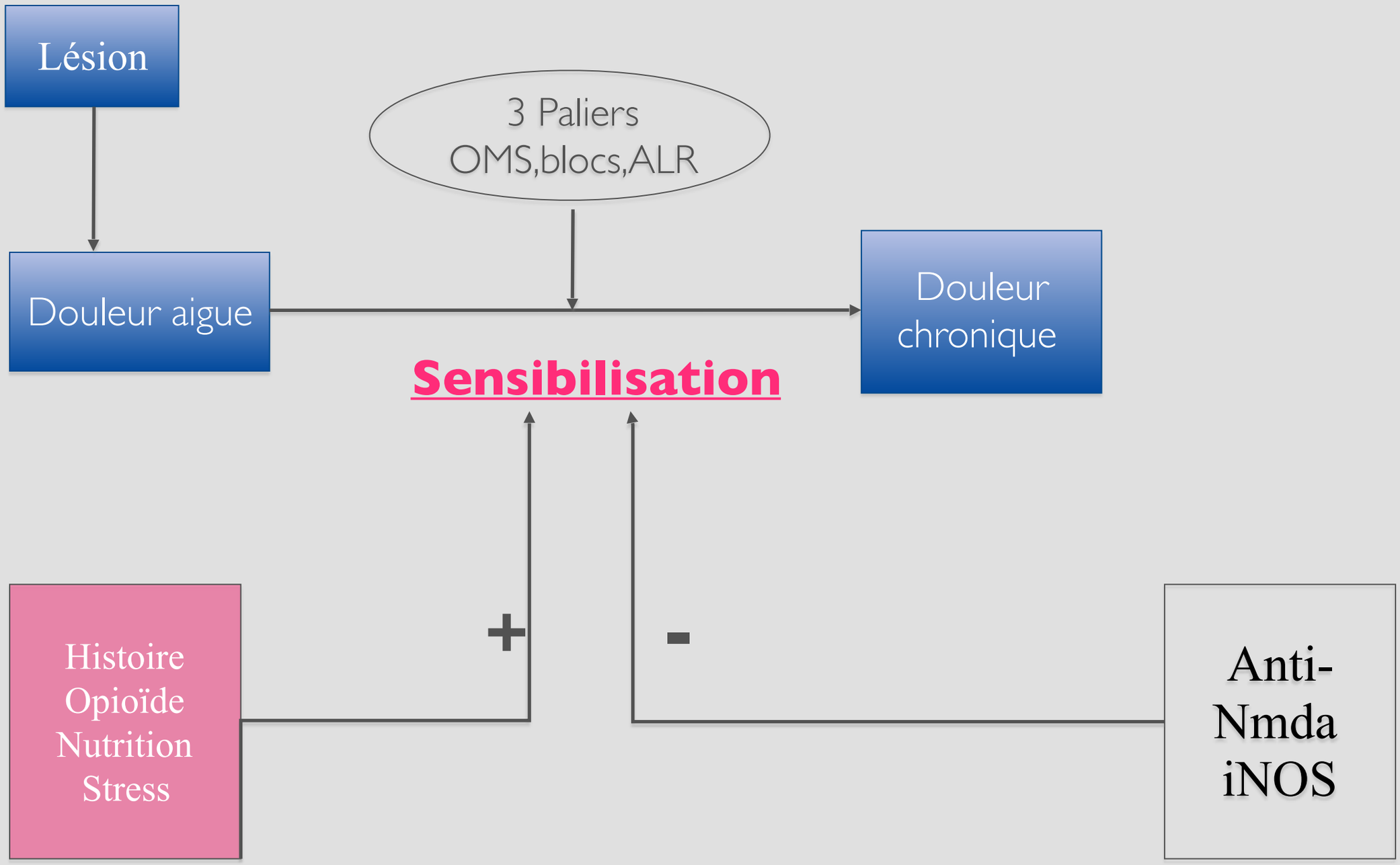
BERLIN—The longer a patient is in severe pain during the first 24 hours following surgery, the more likely he or she will still be experiencing some form of chronic postsurgical pain one year later, according to a study presented at Euroanaesthesia 2015.

This prospective, observational study (abstract ESAPC1-2), designed to investigate the incidence, characteristics and risk factors for chronic postsurgical pain in Europe, assessed patients undergoing elective procedures in 21 hospitals in 11 countries. Pain was measured during the first 24 hours following surgery using a standardized questionnaire, and follow-up evaluations were performed at six and 12 months postoperatively, using the Brief Pain Inventory (BPI) and DN4 (Douleur Neuropathique en 4 questions).

In all, more than 3,000 patients were assessed on day 1 following surgery, and six- and 12-month follow-up was performed on 889 of these patients. The authors found that the incidence of at least moderate chronic postsurgical pain (defined as Numeric Rating Scale score ≥3) was 16% on day 1 and 11.8% at six and 12 months. Severe pain (defined as Numeric Rating Scale score ≥6) was reported by 2.9% and 2.2% of patients at six and 12 months, respectively. In addition, at 12 months, signs of neuropathic pain were recorded in 39.2% of the patients with moderate pain, and in 57.1% of the patients with severe chronic postsurgical pain. Not surprisingly, the functional effect of pain on activities increased with severity of chronic postsurgical pain ($P<0.001$).

Multivariate analysis identified orthopedic surgery, preoperative chronic pain and percentage of time in severe pain 24 hours after surgery as predictive factors for chronic postsurgical pain. Furthermore, a 10% increase in the percentage of time in severe pain on day 1 was associated with a 30% increase in chronic postsurgical pain incidence at one year following surgery.

Chronicisation de la douleur



Introduction

- La douleur postopératoire (DPO)=**priorité** depuis dernière décennie.
- Elle est par définition **programmée** et donc doit être **anticipée**.
- Elle n'est pas toujours corrélée à l'importance de la chirurgie.
- Nouvelles **évolutions** techniques et pharmacologiques.
- Nouveaux **concepts** de prise en charge de la DPO et DCPO.

Pourquoi ?

Tout acte chirurgical doit faire l'objet d'un traitement antalgique.

- assurer un **confort** évident au patient.
- réduction de la **morbidité** et **mortalité** postopératoire.
- **réhabilitation** postopératoire rapide et efficace.

Kehlet H. Postoperative opiod sparing to hasten recovery. Anesthesiology 2005 :102 ;1083-5

- amélioration du **résultat fonctionnel** de la chirurgie.

Kehlet H, Holte K. Effect of postoperative analgesia on surgical outcome. Br J Anaesth 2001 : 87 ; 62-72

- **douleurs chroniques postopératoire (DCPO)** moindres.

Macrae WA. Chronic post-surgical pain : 10 years on. Br J Anaesth 2008 ; 101 : 77-86

Chronicisation de la douleur

Epidémiologie

- 10 à 50% des opérés souffrent de douleurs persistantes (30%pour les HI et >50%pour PTG)
- 61% en chirurgie thoracique et amputation de sein
- 22% de la population est handicapée par DP

Coût: **90 milliards de $ / an**

Peut-on prédire la DPO ?

et DCPO?

Facteurs de risques **pré-et per-opératoires** DCPO.

- **7 Facteurs prédictifs:** sexe féminin, jeune âge, **douleur préopératoire intense**, une grande incision chirurgicale, chirurgie invasive, **anxiété préopératoire importante**, faible besoin d'information… et nutrition ?

- Normogramme prédictif de DPO sévère.

Jannsen KJ, Kalmann CJ, Grobbee DE, et al. The risk of severe postoperative pain: modification and validation of clinical prediction rule. Anesth Analg 2008 ; 107 : 1330-31

Gestion du yellow flag

SDRC et inflammation neurogène

- **Facilitated neurogenic inflammation in unaffected limbs of patients with complex regional pain syndrome.**

Leis S, Weber M, Schmelz M, Birklein F.

Source

Department of Neurology, University of Erlangen-Nürnberg, Erlangen, Germany. stefan.leis@neuro.med.uni-erlangen.de

Abstract

Pain, edema, increased skin temperature, reddening and trophic changes characterize complex regional pain syndrome (CRPS). Recently, we have been able to show facilitated neurogenic inflammation on the affected limb. In the current study unaffected limbs were examined after resolution of the CRPS symptoms to assess possible generalized changes predisposing to CRPS. In 12 patients and in 12 healthy volunteers dermal microdialysis in combination with electrical C-fiber stimulation was employed to induce neuropeptide release. Dialysate protein concentration and axon reflex vasodilation were measured. Neither in patients nor in controls did electrical stimulation lead to protein extravasation, while axon reflex vasodilation was significantly enhanced even on the patients' unaffected limbs ($P < 0.05$). Our results support the hypothesis that facilitated neurogenic inflammation is a predisposing factor for CRPS. The lack of protein extravasation indicates that an initiating trauma is necessary to induce neuropeptide up-regulation in primary afferents

DCPO : hyperalgésie périphérique

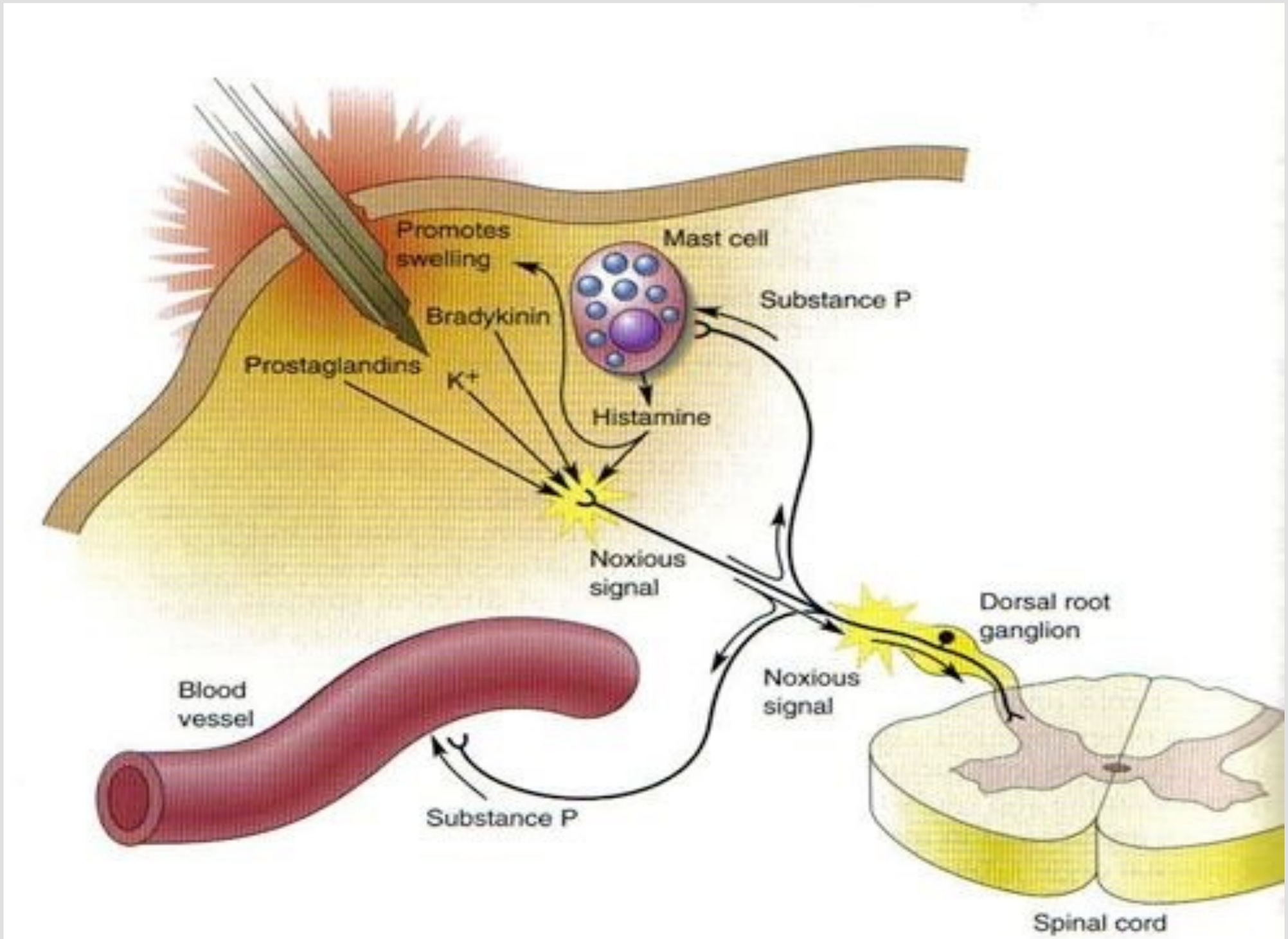


Figure 12.24
Peripheral chemical mediators of pain and hyperalgesia.

DCPO : hyperalgésie centrale

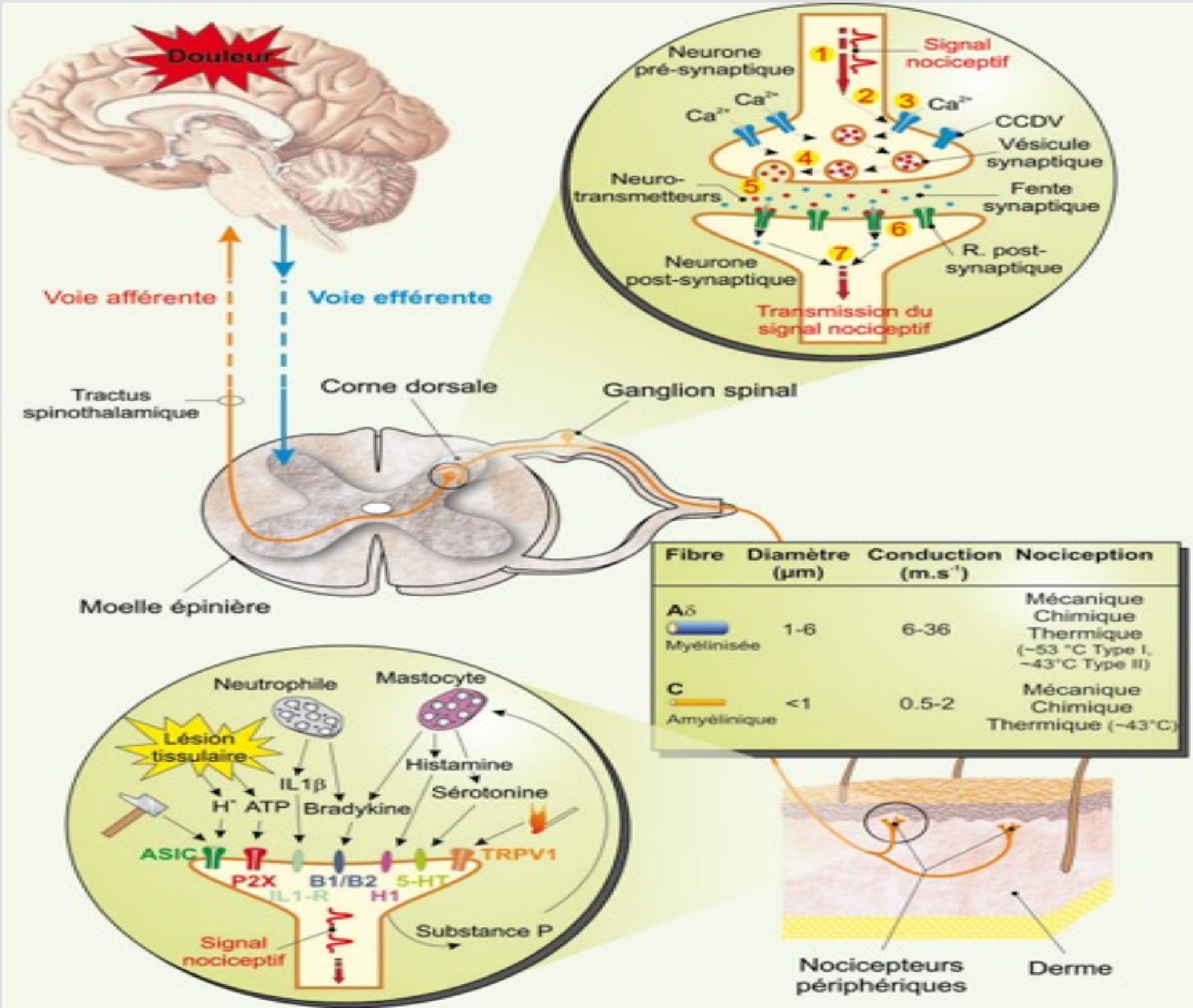
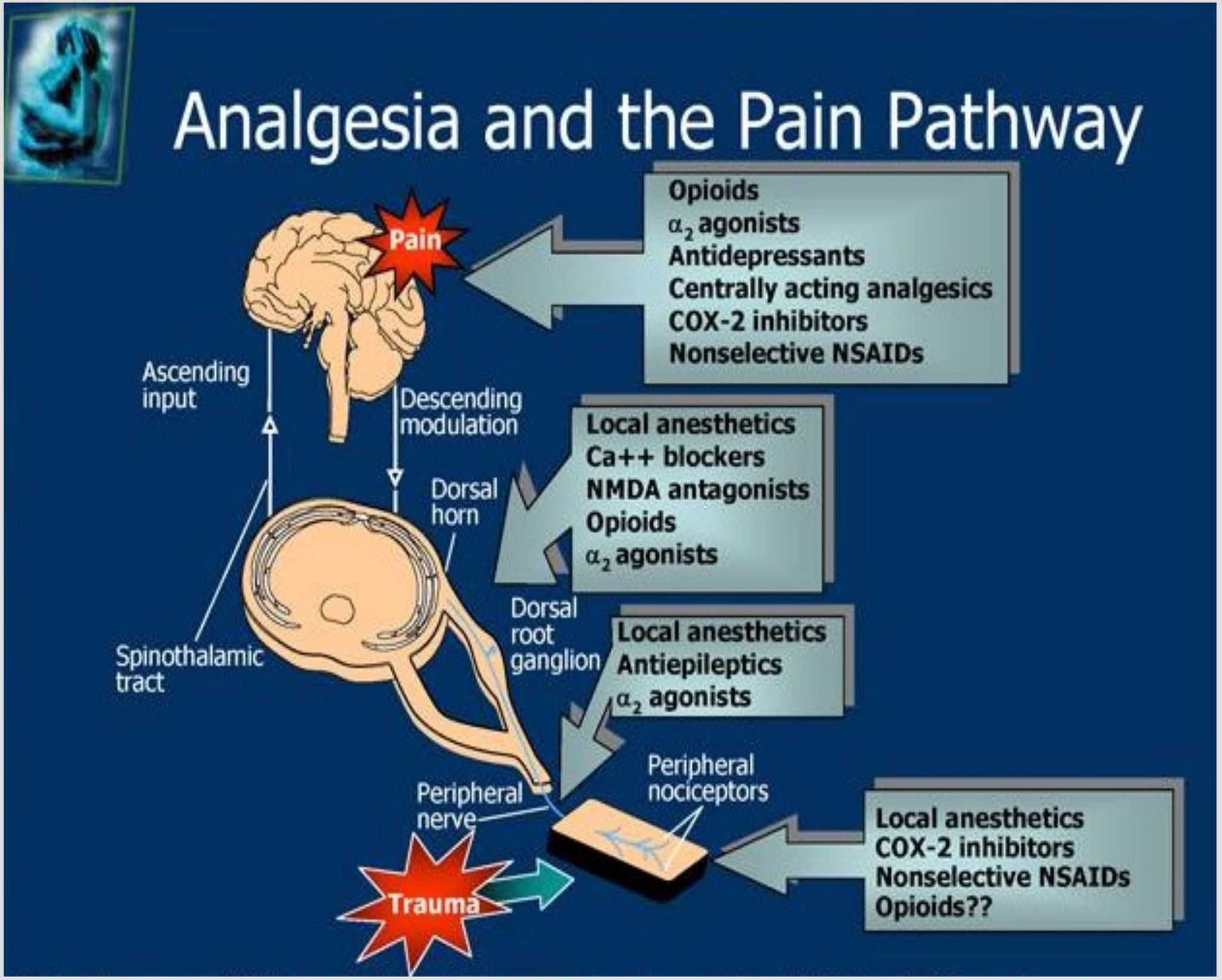


Tableau 1. Questionnaire Douleur Neuropathique

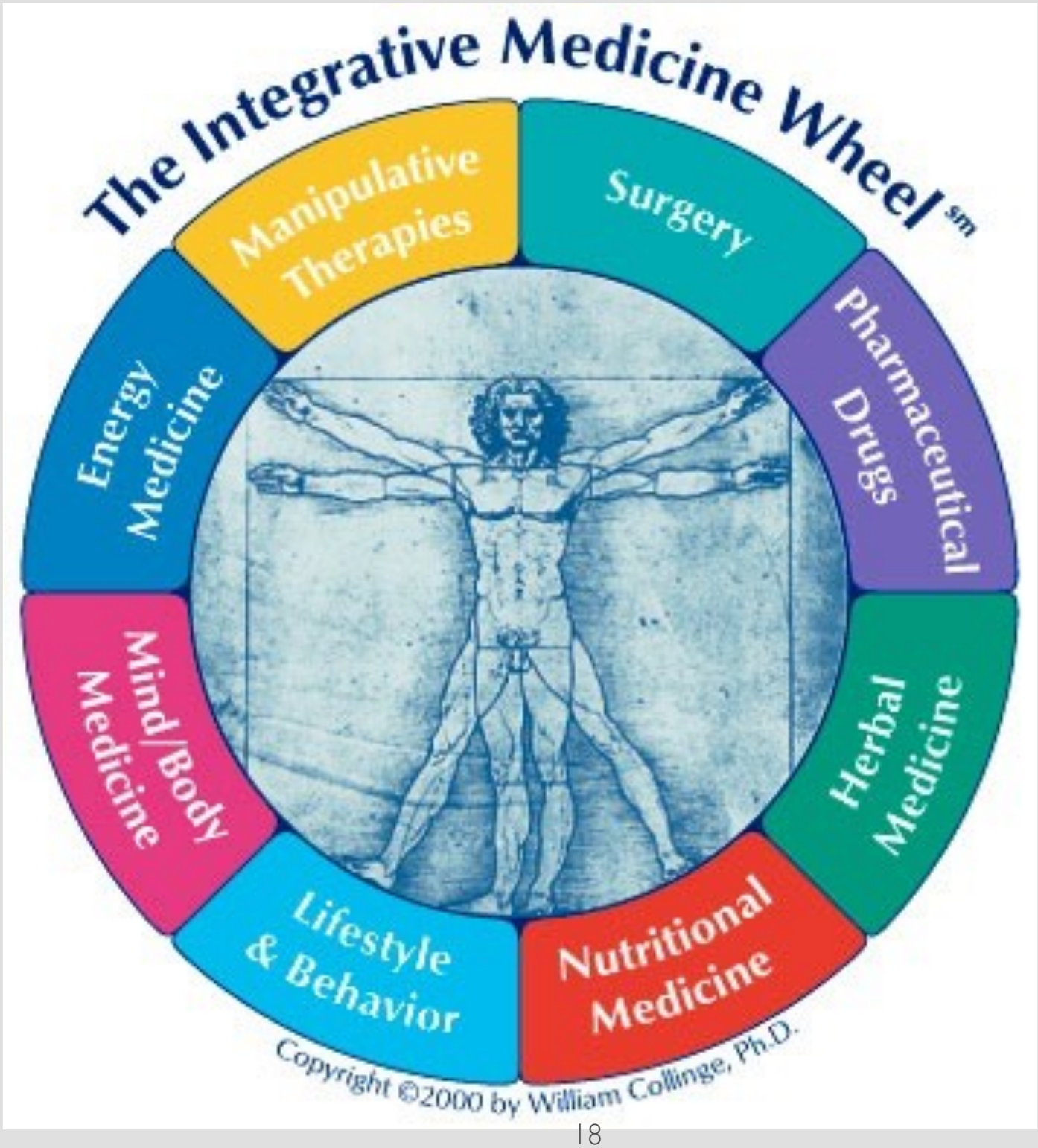
Répondez aux 4 questions ci-dessous en cochant une seule case pour chaque item.

Interrogatoire du patient	OUI : 1 Point	NON : 0 Point
Question 1 : La douleur présente-t-elle une ou plusieurs des caractéristiques suivantes ?		
1. Brûlure	◻	◻
2. Sensation de froid douloureux	◻	◻
3. Décharges électriques	◻	◻
Question 2 : La douleur est-elle associée dans la même région à un ou plusieurs des symptômes suivants ?		
4. Fourmillements	◻	◻
5. Picotements	◻	◻
6. Engourdissement	◻	◻
7. Démangeaisons	◻	◻
Examen du patient	OUI : 1 Point	NON : 0 Point
Question 3 : La douleur est-elle localisée dans un territoire où l'examen met en évidence :		
8. Hypoesthésie au tact	◻	◻
9. Hypoesthésie à la piqûre	◻	◻
Question 4 : La douleur est-elle provoquée ou augmentée par :		
10. Le frottement	◻	◻
	Score du patient : ...../10	


Analgesia and the Pain Pathway
Pain
Opioids
α_2 agonists
Antidepressants
Centrally acting analgesics
COX-2 inhibitors
Nonselective NSAIDs
Ascending input
Descending modulation
Local anesthetics
Ca++ blockers
NMDA antagonists
Opioids
α_2 agonists
Dorsal horn
Dorsal root ganglion
Local anesthetics
Antiepileptics
α_2 agonists
Spinothalamic tract
Peripheral nerve
Peripheral nociceptors
Local anesthetics
COX-2 inhibitors
Nonselective NSAIDs
Opioids??
Trauma

!!! Bien prescrire, c'est connaitre le mécanisme d'action adéquat mais aussi son mode d'administration!!!

Traitements non médicamenteux



CRPS: Phénomène de société ou nutrition inadaptée?

- Intolérance au lactose, allergies alimentaires (gluten, lait…), alimentation acide (lait, viande rouge…)
- Statut des acides gras: Ω6/Ω3 ↑↑↑
- Mauvaise hydratation, hypoglycémies réactionnelles...
- Surcharge du système de détoxication du foie (TNF,IL6…)

⇨ ***Réponses inflammatoires exagérées***

Terrain propice à la chronicisation!!!!

SDRC et Inflammation

Objectif Nutrition

- **Prévenir l'inflammation (curcuma,oméga 3, LGS, surcharge foie, vit D...)**
- Rétablir la balance glutamate/sérotonine (sucre, alimentation acide…)
- Moduler l'hyperfonctionnement du NMDA

I: Inflammation: Statut des acides gras

Cytokines

Phospholipides
Ω6/Ω3
3/1

P kinases C

PLA2

Cortisone

NFkB

AA/EPA

Cox1/Cox2

Lox

AINS

PGE2/PGE3

LT4/LT5

ProINFLAM/AntiINFLAM

Inflammation

Cytokines

P kinases C

NFkB

Phospholipides

25/1

PLA2

Cortisone

AA/EPA

Cox1/Cox2

Lox

AINS

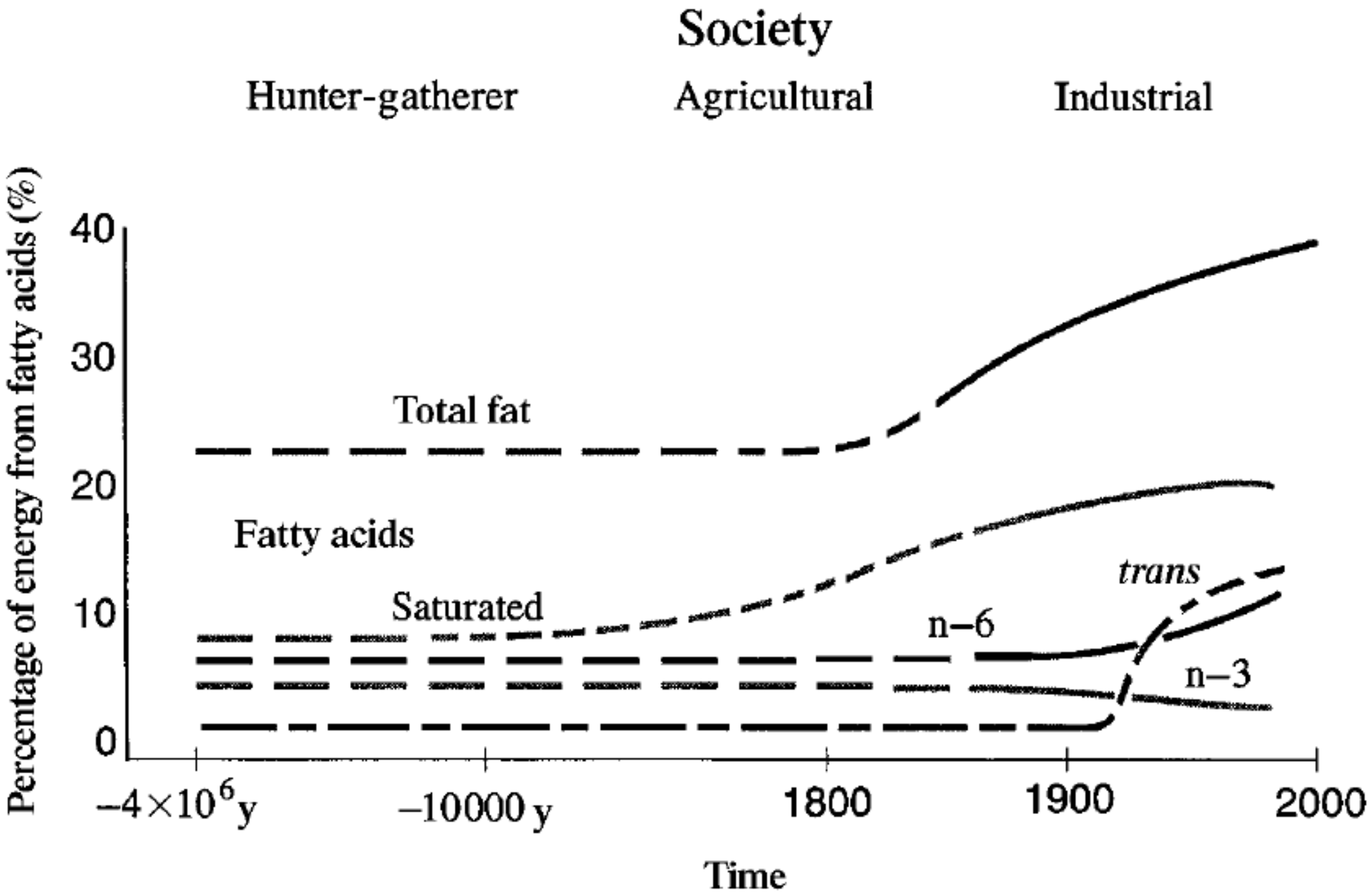
Réponse exagérée !!!

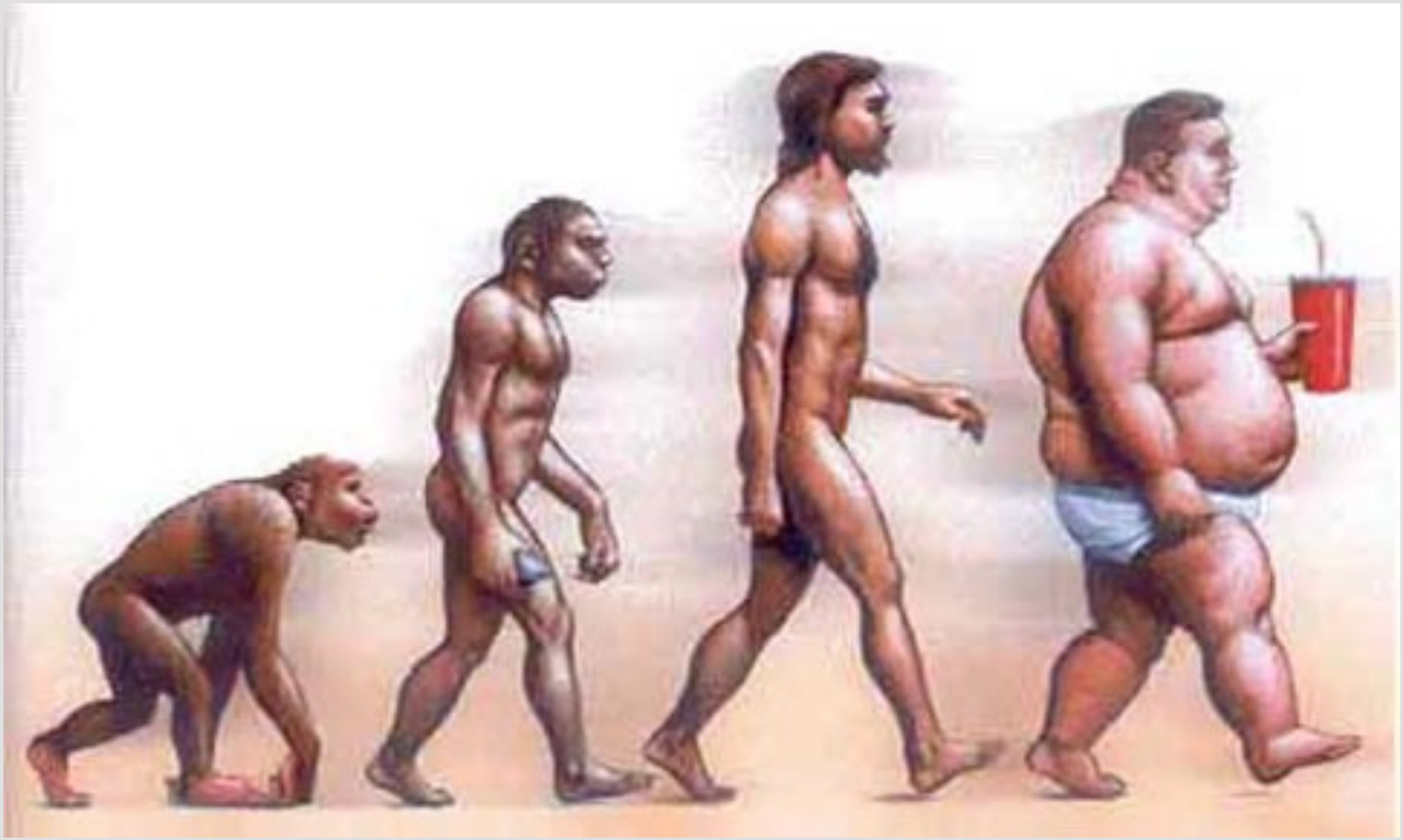
PGE2/

25/1

ProINFLAM/AntiINFLAM

LT4/
LT5

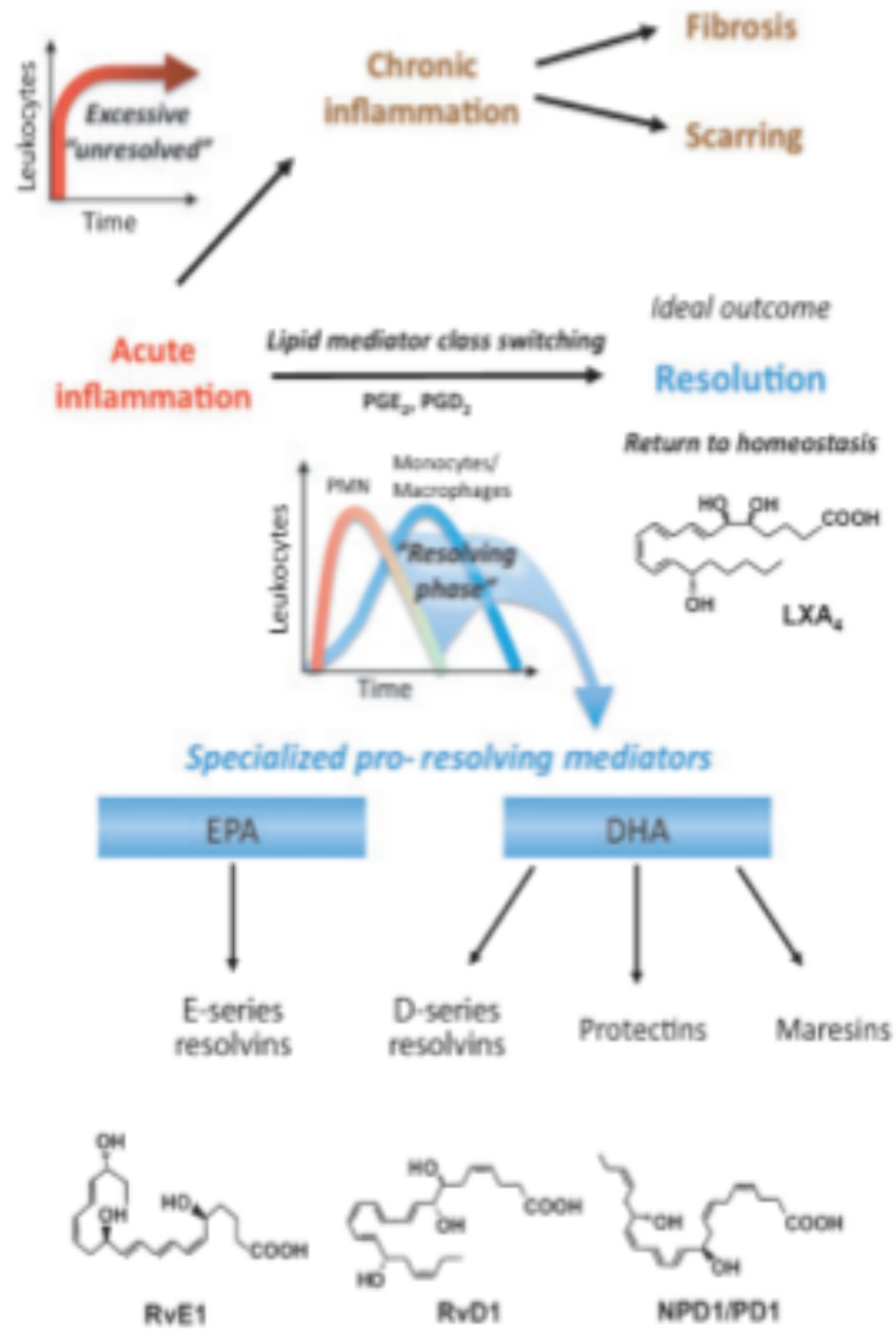

Society
Hunter-gatherer
Agricultural
Industrial
Percentage of energy from fatty acids (%)
40
30
20
10
0
Total fat
Fatty acids
Saturated
n–6
trans
n–3
–4×10^6 y
–10000 y
1800
1900
2000
Time



Homo
Sapiens
Sapiens

Homo Sapiens
Obesus
Diabeticus
Inflammaticus

Inflammation aigüe ou chronique ?



Norling LV & Serhan CN. J Intern Med 2010, 268 : 15-24

• Trends Neurosci. 2011 Nov;34(11):599-609. doi: 10.1016/j.tins.2011.08.005. Epub 2011 Sep 30.

Emerging roles of resolvins in the resolution of inflammation and pain.

Ji RR, Xu ZZ, Strichartz G, Serhan CN.

Source

Pain Research Center, Department of Anesthesiology, Perioperative and Pain Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, MA 02115, USA. rrji@zeus.bwh.harvard.edu

Abstract

Resolvins, including D and E series resolvins, are endogenous lipid mediators generated during the resolution phase of acute inflammation from the omega-3 polyunsaturated fatty acids docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA). Resolvins have potent anti-inflammatory and pro-resolution actions in several animal models of inflammation. Recent findings also demonstrate that resolvin E1 and resolvin D1 can each potently dampen inflammatory and postoperative pain. This review focuses on the mechanisms by which resolvins act on their receptors in immune cells and neurons to normalize exaggerated pain via regulation of inflammatory mediators, transient receptor potential (TRP) ion channels, and spinal cord synaptic transmission. Resolvins may offer novel therapeutic approaches for preventing and treating pain conditions associated with inflammation.

• J Neurosci. 2011 Dec 14;31(50):18433-8.

Resolvin D2 is a potent endogenous inhibitor for transient receptor potential subtype V1/A1, inflammatory pain, and spinal cord synaptic plasticity in mice: distinct roles of resolvin D1, D2, and E1.

Park CK, Xu ZZ, Liu T, Lü N, Serhan CN, Ji RR.

Source

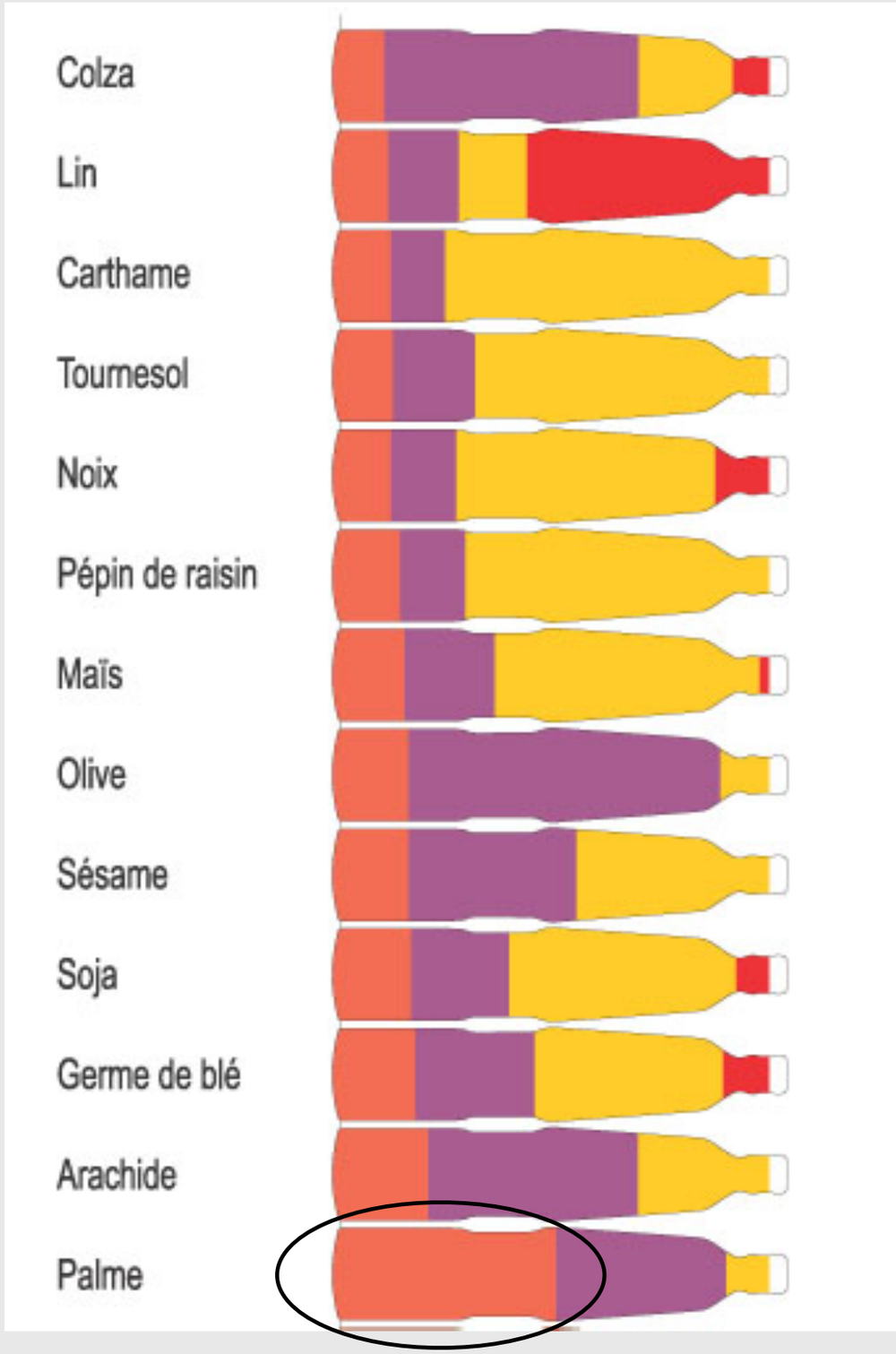
Sensory Plasticity Laboratory, Pain Research Center, Brigham and Women's Hospital, Harvard Medical School, Boston, Massachusetts 02115, USA.

Abstract

Inflammatory pain such as arthritic pain is typically treated with opioids and cyclo-oxygenase-2 inhibitors with well known side effects. Transient receptor potential subtype vanilloid 1 (TRPV1) and TRP ankyryn 1 (TRPA1) contribute importantly to the genesis of inflammatory pain via both peripheral mechanisms (peripheral sensitization) and spinal cord mechanisms (central sensitization). Although these TRP channels have been intensively studied, little is known about their endogenous inhibitors. Recent studies have demonstrated that the endogenous lipid mediators resolvins (RvE1 and RvD1), derived from ω-3 unsaturated fatty acids, are potent inhibitors for inflammatory pain, without noticeable side effects. However, the molecular mechanisms underlying resolvins' distinct analgesic actions in mice are unclear. RvD2 is a novel family member of resolvins. Here we report that RvD2 is a remarkably potent inhibitor of TRPV1 (IC(50) = 0.1 nm) and TRPA1 (IC(50) = 2 nm) in primary sensory neurons, whereas RvE1 and RvD1 selectively inhibited TRPV1 (IC(50) = 1 nm) and TRPA1 (IC(50) = 9 nm), respectively. Accordingly, RvD2, RvE1, and RvD1 differentially regulated TRPV1 and TRPA1 agonist-elicited acute pain and spinal cord synaptic plasticity [spontaneous EPSC (sEPSC) frequency increase]. RvD2 also abolished inflammation-induced sEPSC increases (frequency and amplitude), without affecting basal synaptic transmission. Intrathecal administration of RvD2 at very low doses (0.01-1 ng) prevented formalin-induced spontaneous pain. Intrathecal RvD2 also reversed adjuvant-induced inflammatory pain without altering baseline pain and motor function. Finally, intrathecal RvD2 reversed C-fiber stimulation-evoked long-term potentiation in the spinal cord. Our findings suggest distinct roles of resolvins in regulating TRP channels and identify RvD2 as a potent endogenous inhibitor for TRPV1/TRPA1 and inflammatory pain.

Les sources végétales d'Oméga-3

Faire le bon choix



Saturés Mono Linoléïque Linolénique

!

Les sources animales d'0méga-3

Faire le bon choix

→ **Poissons gras**

- Thon
- Saumon
- Hareng
- Maquereau
- Sardine …

→ **Coquillages et crustacés**

→ **Œufs Oméga 3** (à la coque)

→ **Autres dérivés…**

→ **Complément alimentaire d'huile de poisson STABLE**

Inflammation

Cytokines

P kinases C

NFkB

Phospholipides
Ω6/Ω3
3/1

PLA2

Cortisone

AA/EPA

Cox1/Cox2

Lox

AINS

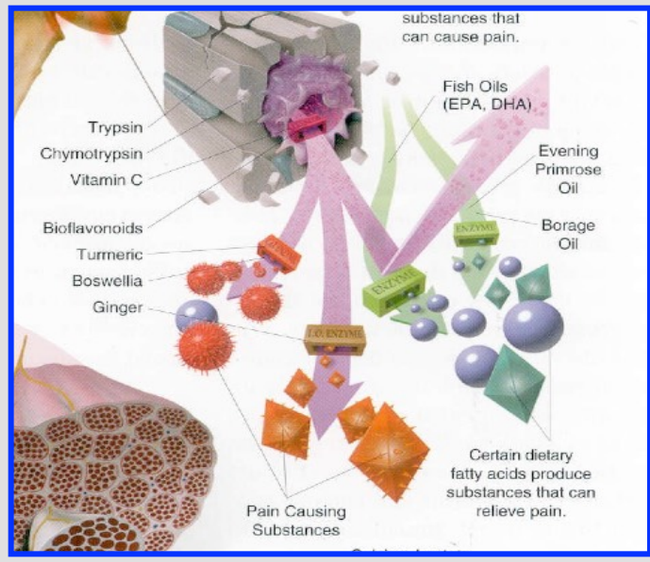
AI Naturels

PGE2/PGE3

LT4/LT5

ProINFLAM/AntiINFLAM

Modulation of Inflammation



Cell Membrane

Quercetin

Phospholipase A2

Curcuma longa

Arachidonic Acid

Zingiber officinale
Curcuma longa
Quercetin
Rosmarinus officinalis

cyclooxygenase **lipooxygenase**

Quercetin
Curcuma longa
Boswellia serrata

Prostaglandins Series 2

Thromboxane A2

Leukotrienes

Inflammation

Cytokines

P kinases C

NFkB

Inh. du NFkB

Phospholipides
Ω6/Ω3
3/1

PLA2

Cortisone

AA/EPA

Cox1/Cox2

Lox

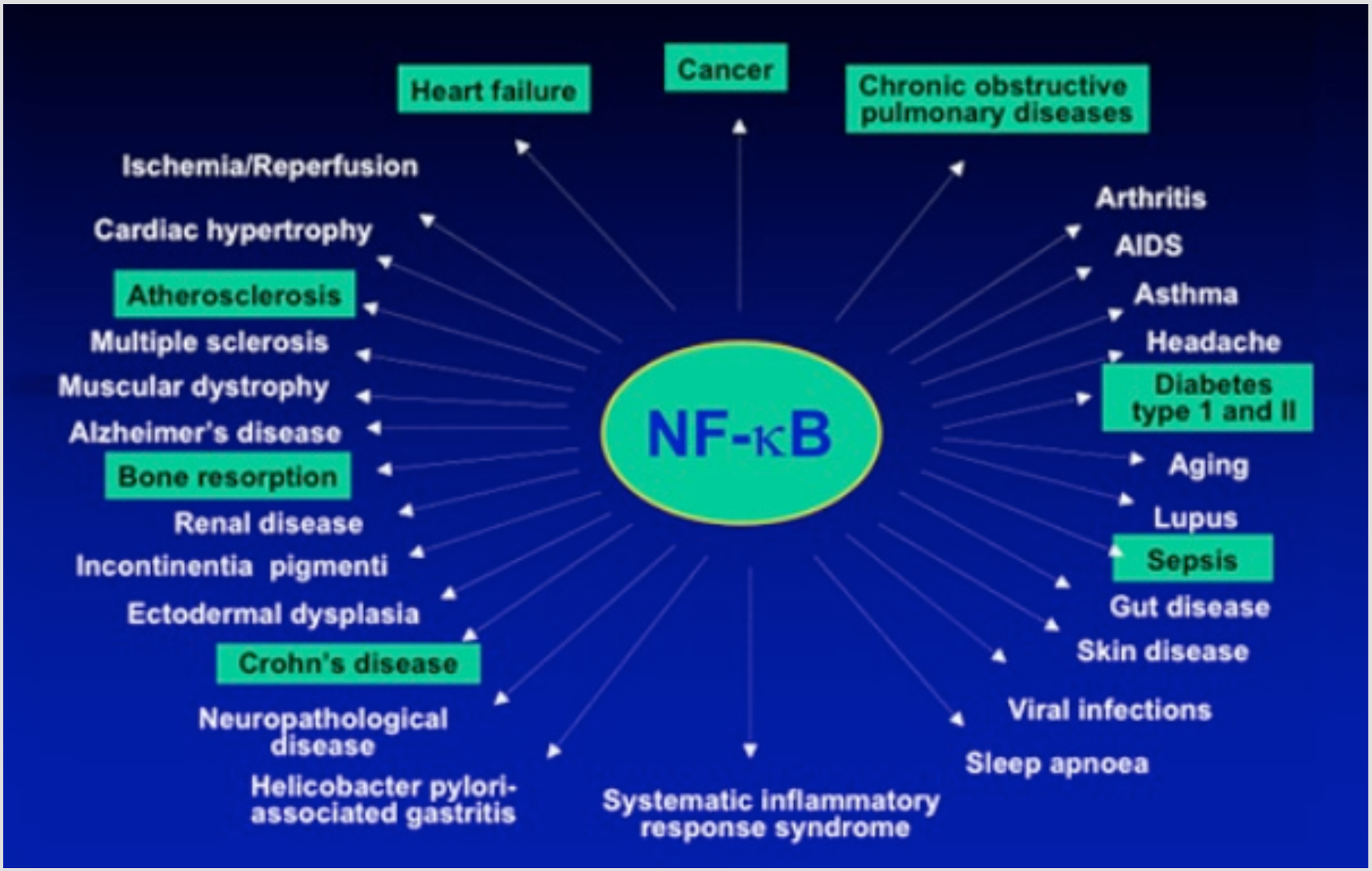
AINS

PGE2/PGE3

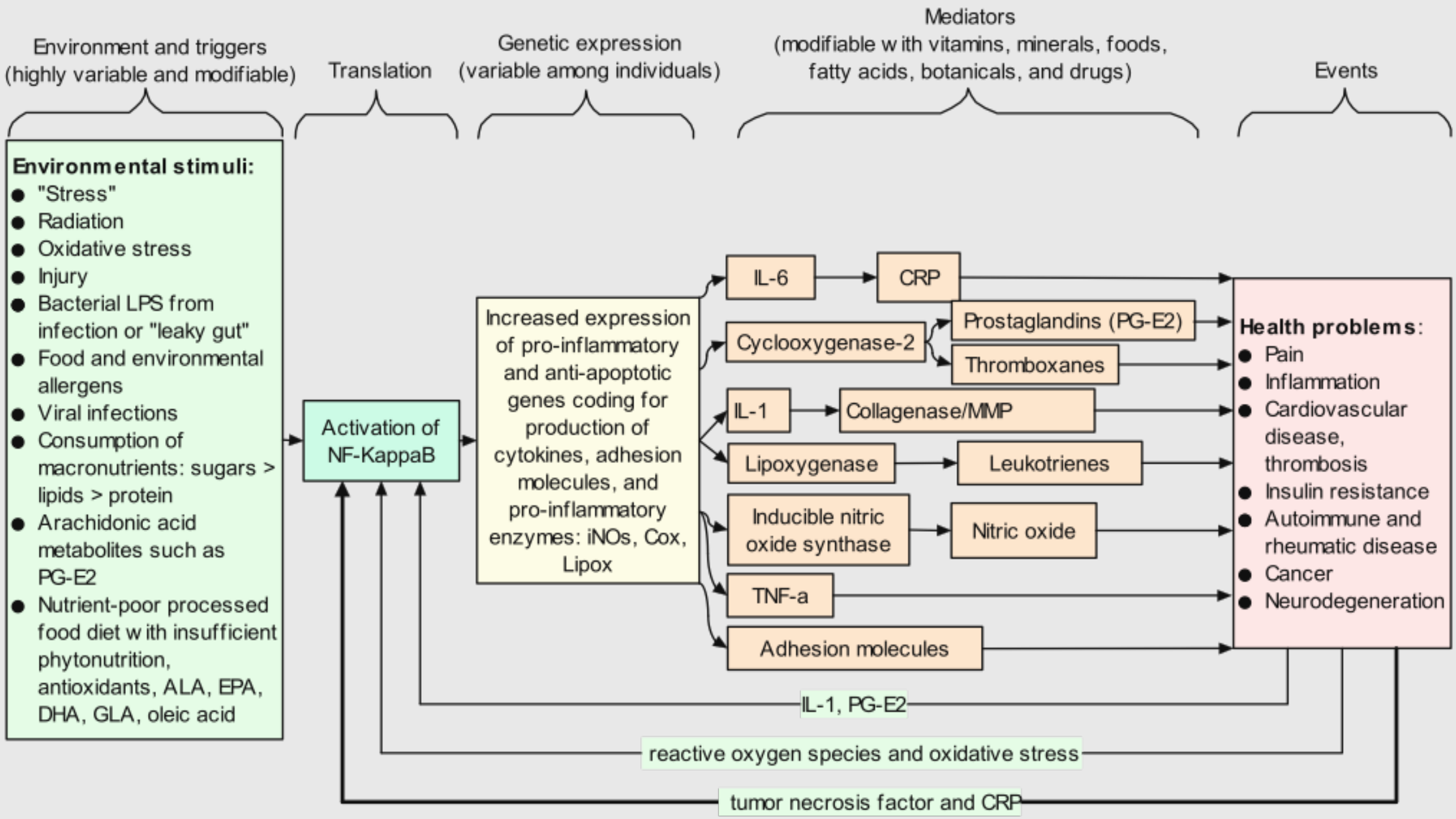
LT4/LT5

ProINFLAM/AntiINFLAM

NF-kB inducteur du déclin?



Les anti-NF-KappaB stoppent l'inflammation avant qu'elle ne débute…



Des inhibiteurs du NF-KappaB sains et efficaces…

Curcuminoides (en association avec le poivre noir)

Boswellia

Thé vert

Gingembre

Romarin

Acide alipoïque

Vitamine D

RIAA Houblon

Jus de grenade fermenté

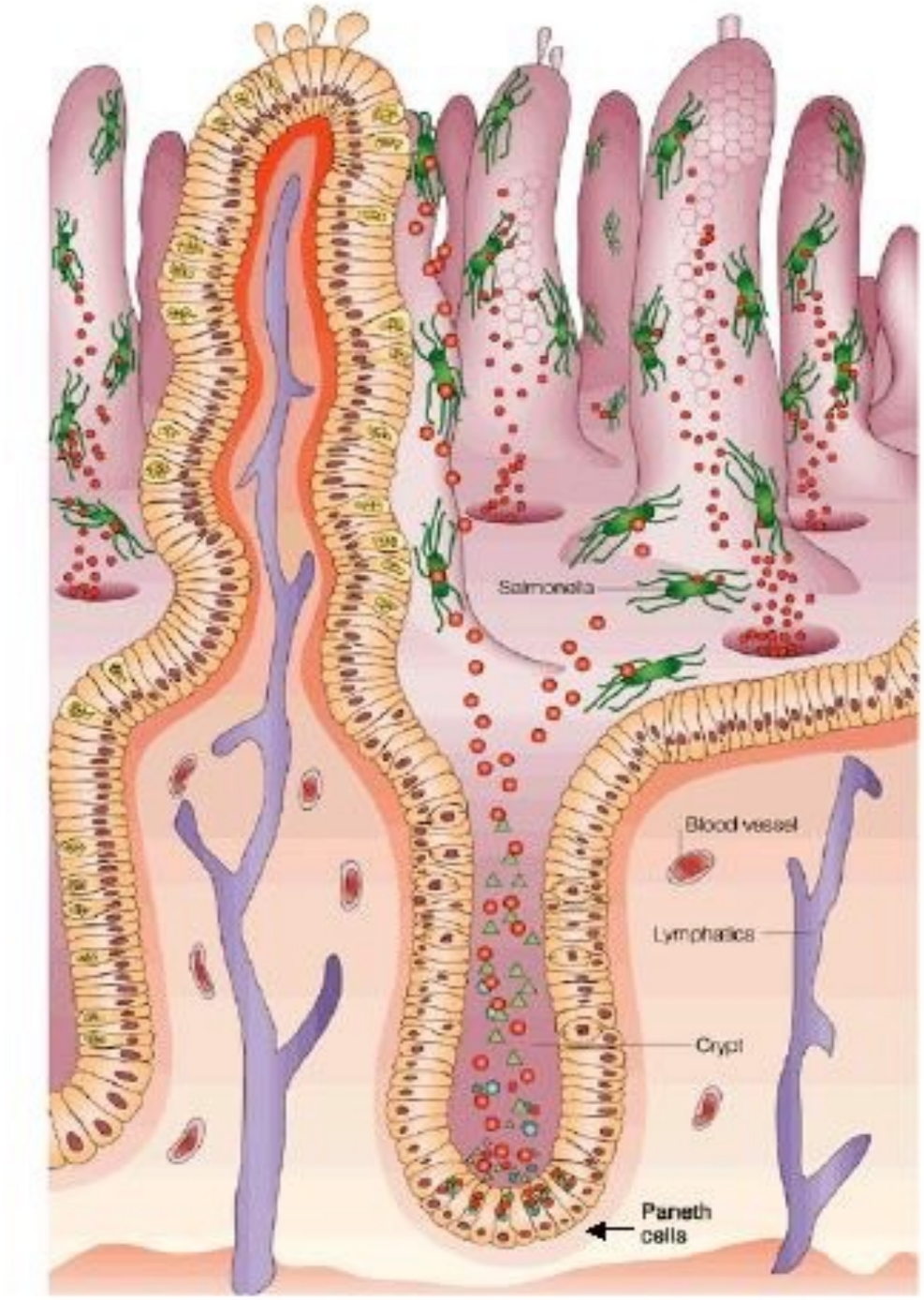
- **Ecosystème intestinal et inflammation?**

▪ Ecosystème Intestinal

Prise en charge et optimisation de l'activité du tube digestif:

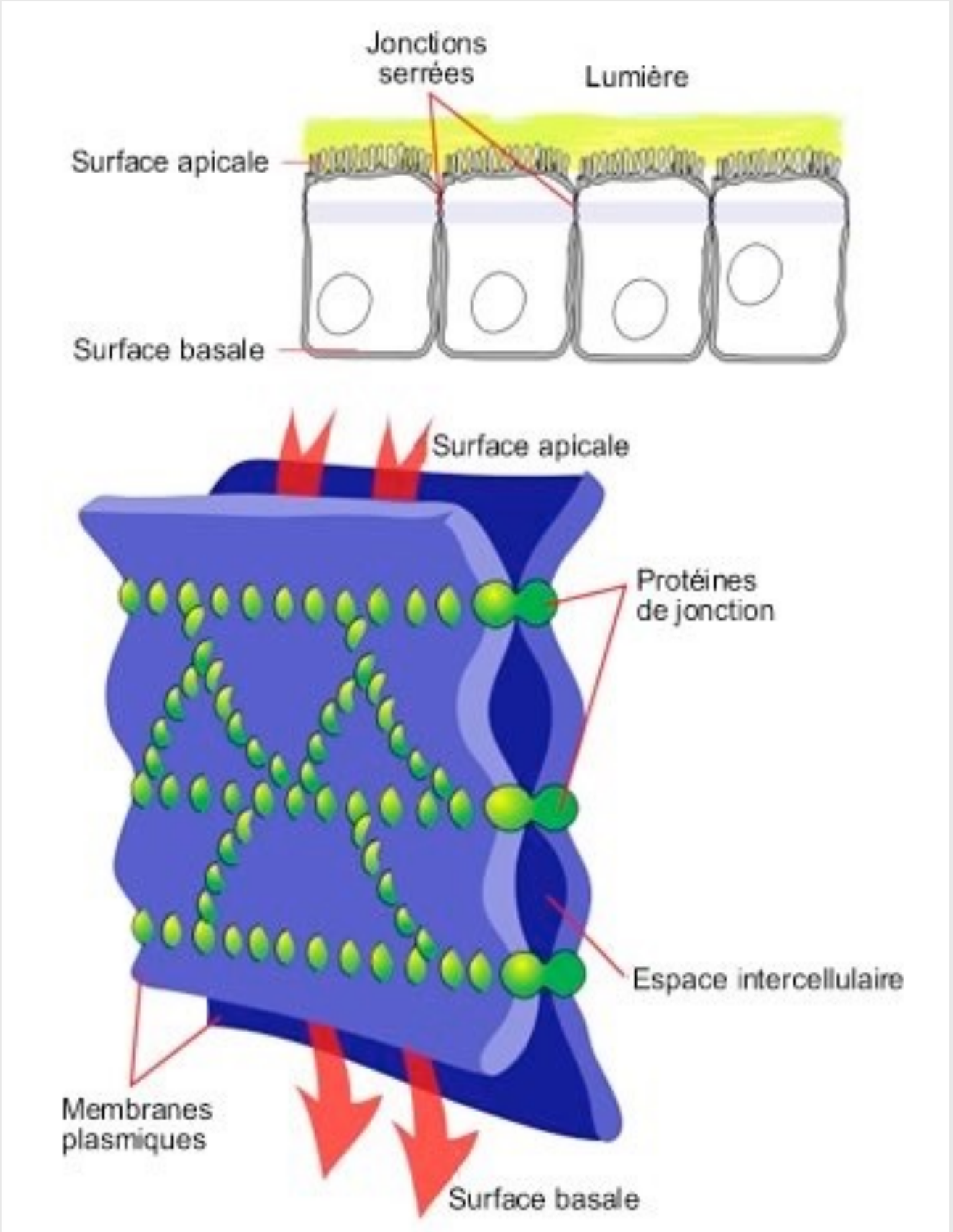
- Digestion
- Absorption intestinale
- Barrière intestinale
- Flore intestinale

PAS DE SANTE SANS BONNE DIGESTION



Les cellules de Paneth, protectrices de l'épithélium se trouvent au fond des cryptes, à proximité immédiate des cellules souches intestinales

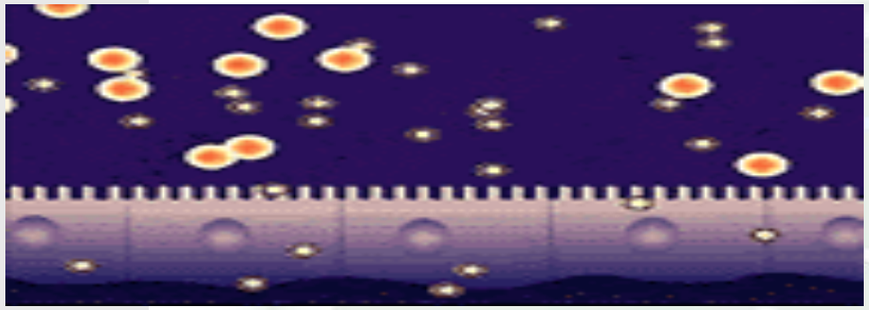
Figure N° 14



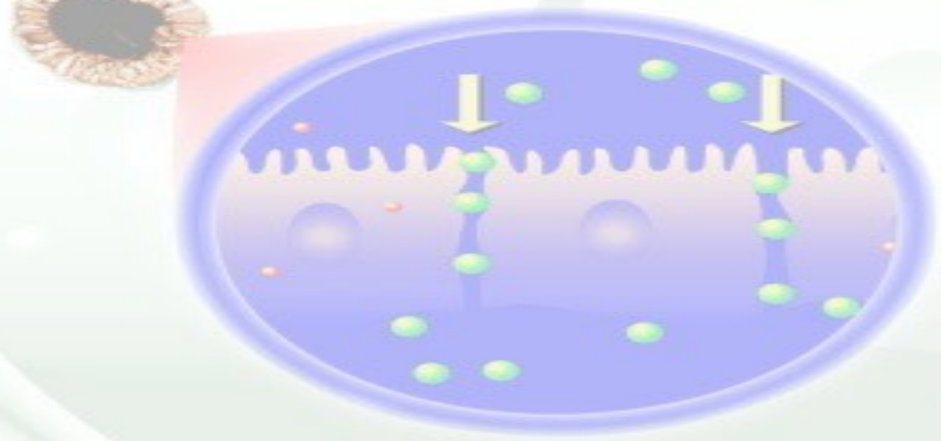
Homéostasie intestinale nécessite :

- PGE2
- Glutamine
- Ac Butyrique (équilibre entre ferments et germes pathogènes)

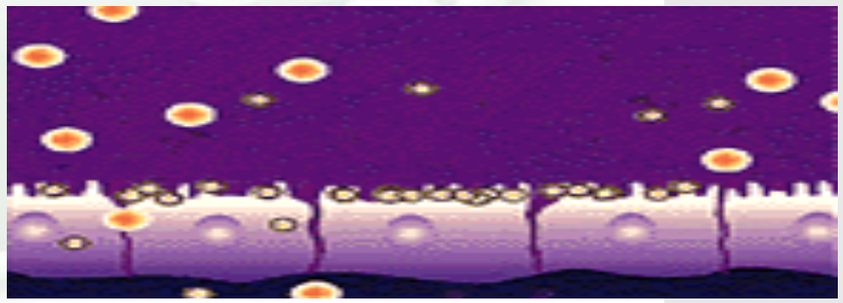
HYPER-PERMEABILITE INTESTINALE ou LGS



<u>Paroi intestinale normale</u>:
- passage des micronutriments
- blocage des grosses molécules



<u>Hyper-perméabilité de la paroi intestinale:</u>
- blocage des micronutriments
- passage des grosses molécules



CAUSES ET CIRCONSTANCES

➢ Physiologiques

- nouveau-nés
- ischémie intestinale
- *activité physique intensive*
- *jeûne prolongé*

➢ Pathologiques

- *maladie de crohn*
- recto-colite ulcéro-hémorragique
- spondylarthrite ankylosante
- maladie de Behçet
- asthme
- eczéma
- *maladie cœliaque*
- *allergie au lait de vache*
- *allergies alimentaires (gluten)*
- malnutrition
- déficits nutritionnels
- déficit en glutamine
- déficit en IgA
- insuffisance en glycocalyx (mucus)
- claudication intermittente
- leucémie myéloïde aiguë
- pathologies graves, soins intensifs

➢ Infectieuses

- *dysbiose intestinale* (levures, protozoaires, bactéries, virus)
- prolifération bactérienne du grêle = ***SIBO!!!***
- sprue tropicale
- diarrhée à Clostridium difficile
- syndrome immunodéficitaire acquis

➢ Iatrogènes

- *anti-inflammatoires non stéroïdiens*
- *antibiothérapie prolongée*
- *Inhibiteur de la pompe à protons!!!*
- stress hyperosmolaire
- alimentation parentérale
- chirurgie digestive
- Chimiothérapie/radiothérapie

➢ Toxiques

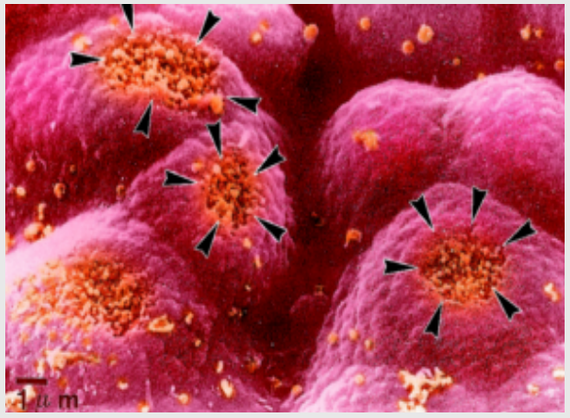
- alcool
- huile de ricin

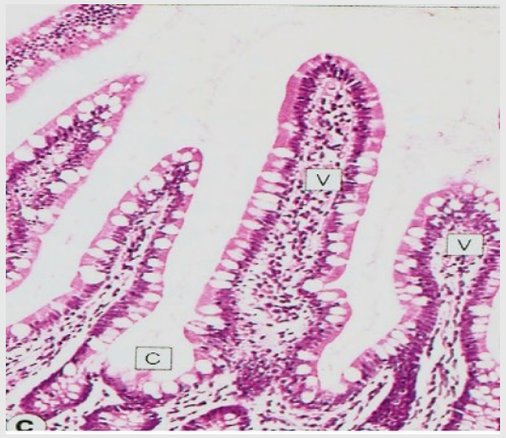
➢ Accidentelles

- brûlures
- traumatismes

CONSEQUENCES DE L'HYPER-PERMEABILITE INTESTINALE

➢ Entrée massive d'antigènes





→ ***Maladies inflammatoires*** : contact Ag/Ac , dépôt intraarticulaire et stimulation du complément

→ ***Maladies auto-immunes :*** réaction Ag/Ac et Ac contre le « soi » par **mimétisme moléculaire**

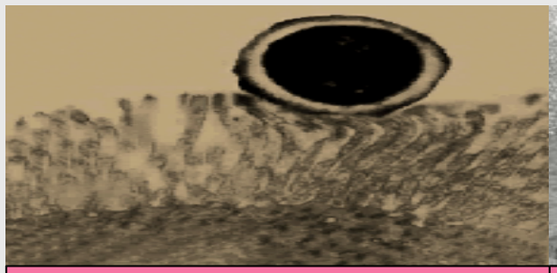
→ **Phénomènes allergiques**

➢ Entrée de pathogènes

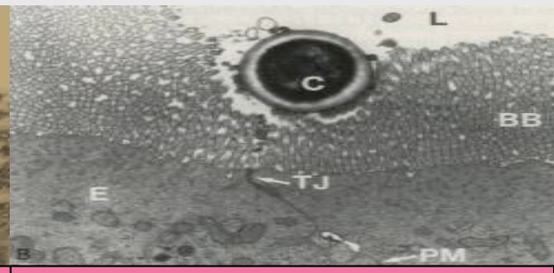
→ **Translocation bactérienne**

→ **Candidose invasive**

→ **Infections opportunistes (fatigue chronique?)**



Candida à l'origine d'une distorsion des microvillosités



Levure pénétrant dans la bordure en brosse

➢ Entrée de toxines

→ Mycotoxines altérant le fonctionnement cérébral et induisant les pulsions sucrées
Endotoxines (**LPS**) aux multiples conséquences
Toxines bactériennes et du clostridium **(GAP Syndrome)**

• Autoimmunity against the β2 adrenergic receptor and muscarinic-2 receptor in complex regional pain syndrome.

Kohr D, Singh P, Tschernatsch M, Kaps M, Pouokam E, Diener M, Kummer W, Birklein F, Vincent A, Goebel A, Wallukat G, Blaes F.

Source

Department of Neurology, Justus-Liebig-University, Giessen, Germany.

Abstract

Complex regional pain syndrome (CRPS) is a painful condition affecting one or more extremities of the body, marked by a wide variety of symptoms and signs that are often difficult to manage because the pathophysiology is incompletely understood. Thus, diverse treatments might be ineffective. A recent report revealed the presence of autoantibodies against differentiated autonomic neurons in CRPS patients. However, it remained unclear how the antibodies act in the development of CRPS. We therefore aimed to characterize these antibodies and identify target antigens. Functional properties of affinity-purified immunoglobulin G of control subjects or CRPS patients were assessed using a cardiomyocyte bioassay. Putative corresponding receptors were identified using antagonistic drugs, and synthesized peptide sequences corresponding to segments of these receptors were used to identify the target epitopes. Chinese hamster ovary cells were transfected with putative receptors to ensure observed binding. Further, changes in the intracellular Ca(2+) concentration induced by agonistic immunoglobulin G were measured using the Ca(2+)-sensitive fluorescent dye fura-2 assay. Herein, we demonstrate the presence of autoantibodies in a subset of CRPS patients with agonistic-like properties on the β(2) adrenergic receptor and/or the muscarinic-2 receptor. We identified these autoantibodies as immunoglobulin G directed against peptide sequences from the second extracellular loop of these receptors. The identification of functionally active autoantibodies in serum samples from CRPS patients supports an autoimmune pathogenesis of CRPS. Thus, our findings contribute to the further understanding of this disease, could help in the diagnosis in future, and encourage new treatment strategies focusing on the immune system.

Anamnèse nutritionnelle et digestive avant chirurgie orthopédique lourde?

▪Ecosystème Intestinal

LGS ou syndrome de l'intestin poreux:

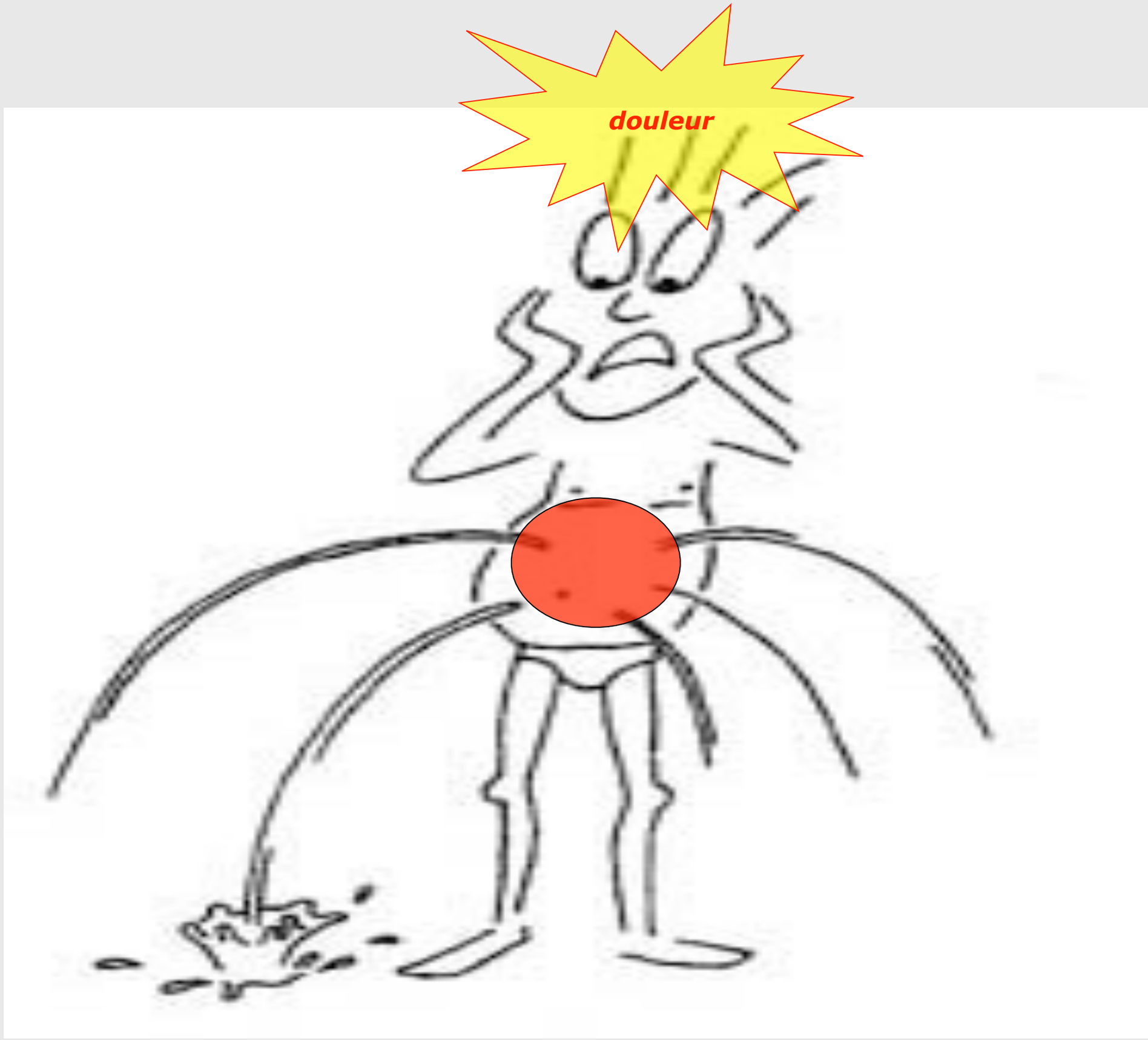
- entrées massives de toxines et de pathogènes en particulier de l'endotoxine (LPS) mais aussi mycotoxine (*Candida*), formaldéhyde (*Clostridium)* …

- réactions inflammatoires locales par mimétisme Ag (lait, gluten)
- surcharge du foie

Etat Inflammatoire général accru:

- allergies

- colon irritable, complications postopératoires digestives
- céphalées et migraines
- douleurs musculaires et DCPO sur réponse inflammatoire exagérée
- fatigue…



hhh

- Entrées massives d’endotoxines LPS

- Réduites si:
    - alimentation pauvre en graisses »trans »
    - alimentation pauvre en sucres raffinés
    - alimentation riche en fibres, omégas 3 et curcuma
    - apport de bifidobactéries
    - apport de prébiotiques

Cani et Delzenne Diabetes 2007

Surcharge du système de détoxication du foie

▪ Détoxication Hépatique

L'organisme est constamment agressé par des substances nocives compromettant le bon fonctionnement des cellules:

- Intestin poreux ou LGS

- Métaux lourds

- Endotoxines (bactéries)

- Hormones et pesticides

- Pollution, solvants

- Médicaments (ains,atb,et surtout polymédication…)

- Allergies alimentaires

L'intoxication de nos systèmes et la surcharge du foie crée un terrain propice pour le développement de maladies diverses dont les maladies douloureuses chroniques (arthrites,fibromyalgie,céphalées,…) … et DPCO?

Les phases de détoxications

PHASE I
(cytochrome P450)

PHASE II
(Conjugation)

Toxines

(liposolubles)

REACTIONS

Oxidation
Reduction
Hydrolyse
Hydratation
Dehalogenation

Métabolites intermédiaires (réactifs)

(toxines oxydées)

REACTIONS

Sulfation
Glucuronidation
Acetylation
Glutathione conjugation
Methylation

Dérivés excrétables

(hydrosolubles)

bile

feces

serum

rein

urine

Radicaux libres (ex, superoxide)

Dommages tissulaires (DNA, RNA, proteines)

Les cellules de Kupffer augmentent l'inflammation par une sur-régulation de l'activité immunitaire

Cytokines:
IL-1
IL-6
IL:-18
IFN-gamma
TNF-alpha

PGE2

LT4

Radicaux libres

NO

INFLAMMATION

Blatties CM, Li S, Perlik V, Feleder C. Signaling the brain in systemic inflammation:the role of complement. ***Front Biosci*** 2004;9:915-31.

DETOXICATION HEPATIQUE

Phase I

⊕

- Caféïne, **alcool**, nicotine,…
- Dioxine, organophosphorés, formaldéhyde.
- **Barbituriques, Halogénés, corticoïdes**.
- **Jus d'agrumes (!!! pamplemousse), vin blanc,…**
- Diète Protéinée!!!
- Vitamines B3, B1
- Carvi, cumin, aneth

Substances naturelles

<u>Cofacteurs</u> :
-Magnésium et Potassium
-Vit B2,B3,B6,B9,B12

- Carences en cofacteurs
- Variabilité GENETIQUE
- Benzodiazépines, antihistaminique, cimétidine
- Anti-acides, kétoconazoles
- Calendula,
- Age, flore de putréfaction
- **!!! La curcumine inhibe la bioactivation des carcinogènes en phase 1 et stimule la phase 2 !!!**

⊖

DETOXICATION HEPATIQUE

Phase II

⊕

- **glutamine**, glycine.
- cystéine, **N-acétyl-cystéine**,méthionine.
- Thé vert
- choux, choux de bruxelles, brocolis.
- sésame
- Curcumine, ail, oignons
- limonène :
  → pelures de citrus (**Citrus vulgaris**).
  → huile de graines de fenouil.

Substances naturelles

- Carences en cofacteurs
- Surconsommation, **stress**
- Maladies telles que Sida, **néoplasies**…
- **Chimiothérapie !!!**
- Aspirine **et AINS**

⊖

Cofacteurs :

- vit.B_2, B_3, B_6, B_9, B_{12}, Vit C
  - Se - Zn - **Mg** - Cu-**S**
- **glutathion réduit**,**méthionine**

Symptômes de surcharge:

- fatigue et troubles du sommeil

- douleurs musculaires,céphalées,fibromyalgies

- faible résistance au stress et à l'effort

- sensibilité à la caféïne,à l'alcool et au parfum

- neuropathies et paresthésies

- langue pâteuse et chargée, parfois teint jaune

- Seuil de la douleur et **Hyperalgésie**

- troubles digestifs (selles grasses, nausées, vo+, dysbiose intestinale…)

- Intolérance à l'anesthésie

Effect of vitamin C on prevention of complex regional pain syndrome type I in foot and ankle surgery.

Besse JL, Gadeyne S, Galand-Desmé S, Lerat JL, Moyen B.

Source

Université de Lyon, Lyon, France. jean-luc.besse@chu-lyon.fr

Abstract

BACKGROUND:

The public health cost impact of complex regional pain syndrome type I (CRPS I) is considerable in both emergency and scheduled orthopaedic surgery. We proposed to assess the effectiveness of vitamin C in prevention of CRPS I in foot and ankle surgery.

METHODS:

We carried out a "before-after" quasi-experimental study comparing two chronologically successive groups without (Group I: July 2002-June 2003) and with (Group II: July 2003-June 2004) preventive 1g daily vitamin C treatment. All patients having surgery on the foot or ankle were enrolled, with the exception of diabetic foot cases. Several factors were analysed: sex, age, type of pathology, history of CRPS I, psychological context, tourniquet time, and cast immobilisation time.

RESULTS:

420 feet (392 patients) were included in the study: 185 in Group I, 235 in Group II. CRPS I occurred in 18 cases in Group I (9.6%) and 4 cases in Group II (1.7%) ($p<10(-4)$), with history of CRPS I as a significantly correlated factor (relative risk=10.4). The psychological context (anxio-depressive state) showed a (sub-significant) tendency to increase the risk of CRPS I (relative risk=2.6).

CONCLUSION:

Vitamin C has been shown to be effective in preventing CRPS I secondary to wrist fracture, but few data are available with respect to foot and ankle cases. The present study demonstrates the effectiveness of vitamin C in preventing CRPS I of the foot and ankle-a frequent complication in our control group (9.6%). The authors recommend preventive management by vitamin C.

. **Can vitamin C prevent complex regional pain syndrome in patients with wrist fractures? A randomized, controlled, multicenter dose-response study.**

Zollinger PE, Tuinebreijer WE, Breederveld RS, Kreis RW.

Source

Department of Surgery, Red Cross Hospital, Beverwijk, The Netherlands. PE.Zollinger@tiscali.nl

Abstract

BACKGROUND:

Complex regional pain syndrome type I is treated symptomatically. A protective effect of vitamin C (ascorbic acid) has been reported previously. A dose-response study was designed to evaluate its effect in patients with wrist fractures.

METHODS:

In a double-blind, prospective, multicenter trial, 416 patients with 427 wrist fractures were randomly allocated to treatment with placebo or treatment with 200, 500, or 1500 mg of vitamin C daily for fifty days. The effect of gender, age, fracture type, and cast-related complaints on the occurrence of complex regional pain syndrome was analyzed.

CONCLUSIONS:

Vitamin C reduces the prevalence of complex regional pain syndrome after wrist fractures. A daily dose of 500 mg for fifty days is recommended.

The treatment of complex regional pain syndrome type I with free radical scavengers: a randomized controlled study.

Perez RS, Zuurmond WW, Bezemer PD, Kuik DJ, van Loenen AC, de Lange JJ, Zuidhof AJ.

Source

Department of Anesthesiology, Vrije Universiteit Medical Center, P.O. Box 7057, 1007 MB Amsterdam, The Netherlands. rsgm.perez@azvu.nl

Abstract

To compare the effects of two free radical scavengers, dimethylsulfoxide 50% (DMSO) and N-acetylcysteine (NAC), for treatment of complex regional pain syndrome I (CRPS I), a randomized, double-dummy controlled, double-blind trial was conducted. Two outpatient clinics of two university hospitals in The Netherlands participated in the study and 146 patients, were included over a period of 24 months. Patients were randomized into two treatment groups, one was instructed to apply DMSO 50% five times daily to the affected extremity, the second was treated with NAC 600mg effervescent tablets three times daily, both combined with placebo. Interventions were accompanied by pain medication, occupational therapy for upper extremity CRPS I and physical therapy for lower extremity CRPS I in specific circumstances. Treatment was given for 17 weeks, with a possibility to continue or switch medication after this period, up to 1 year following the onset of treatment. An impairment level sum score was the primary outcome measure. Upper and lower extremity skills and functions, and general health status were also evaluated. Overall, no significant differences were found between NAC and DMSO after 17 and 52 weeks on impairment level and general health status. Significant differences were found for subscores of lower extremity function, in favor of DMSO-treatment. Subgroup analysis showed more favorable results for DMSO for warm CRPS I and significantly better performance of NAC for patients with a cold CRPS I. Results tended to be negatively influenced if the duration of the complaint was longer. Treatment with DMSO and **NAC** are generally equally effective in treatment of CRPS I. Strong indications exist for differences in effects for subgroups of patients with warm or cold CRPS I: for warm CRPS I, DMSO-treatment appears more favorable, while for cold CRPS I, **NAC-treatment appears to be more effective**.

Evidence based guidelines for complex regional pain syndrome type 1.

Perez RS, Zollinger PE, Dijkstra PU, Thomassen-Hilgersom IL, Zuurmond WW, Rosenbrand KC, Geertzen JH; CRPS I task force..

VU University Medical Center, Department of Anaesthesiology, Amsterdam, the Netherlands. rsgm.perez@vumc.nl

Abstract

BACKGROUND:

Treatment of complex regional pain syndrome type I (CRPS-I) is subject to discussion. The purpose of this study was to develop multidisciplinary guidelines for treatment of CRPS-I.

METHOD:

A multidisciplinary task force graded literature evaluating treatment effects for CRPS-I according to their strength of evidence, published between 1980 to June 2005. Treatment recommendations based on the literature findings were formulated and formally approved by all Dutch professional associations involved in CRPS-I treatment.

RESULTS:

For pain treatment, the WHO analgesic ladder is advised with the exception of strong opioids. For neuropathic pain, anticonvulsants and tricyclic antidepressants may be considered. For inflammatory symptoms, free-radical scavengers (dimethylsulphoxide or **acetylcysteine**) are advised. To promote peripheral blood flow, vasodilatory medication may be considered. Percutaneous sympathetic blockades may be used to increase blood flow in case vasodilatory medication has insufficient effect. To decrease functional limitations, standardised physiotherapy and occupational therapy are advised. To prevent the occurrence of CRPS-I after wrist fractures, **vitamin C is recommended.** Adequate perioperative analgesia, limitation of operating time, limited use of tourniquet, and use of regional anaesthetic techniques are recommended for secondary prevention of CRPS-I.

CONCLUSIONS:

Based on the literature identified and the extent of evidence found for therapeutic interventions for CRPS-I, we conclude that further research is needed into each of the therapeutic modalities discussed in the guidelines.

Homo Sapiens
Sapiens

Homo Sapiens
Obesus
Diabeticus
Inflammaticus

Gérer les AGPI,la Vit D,la dysbiose et le foie pour prévenir l'inflammation

Chronicisation de la douleur

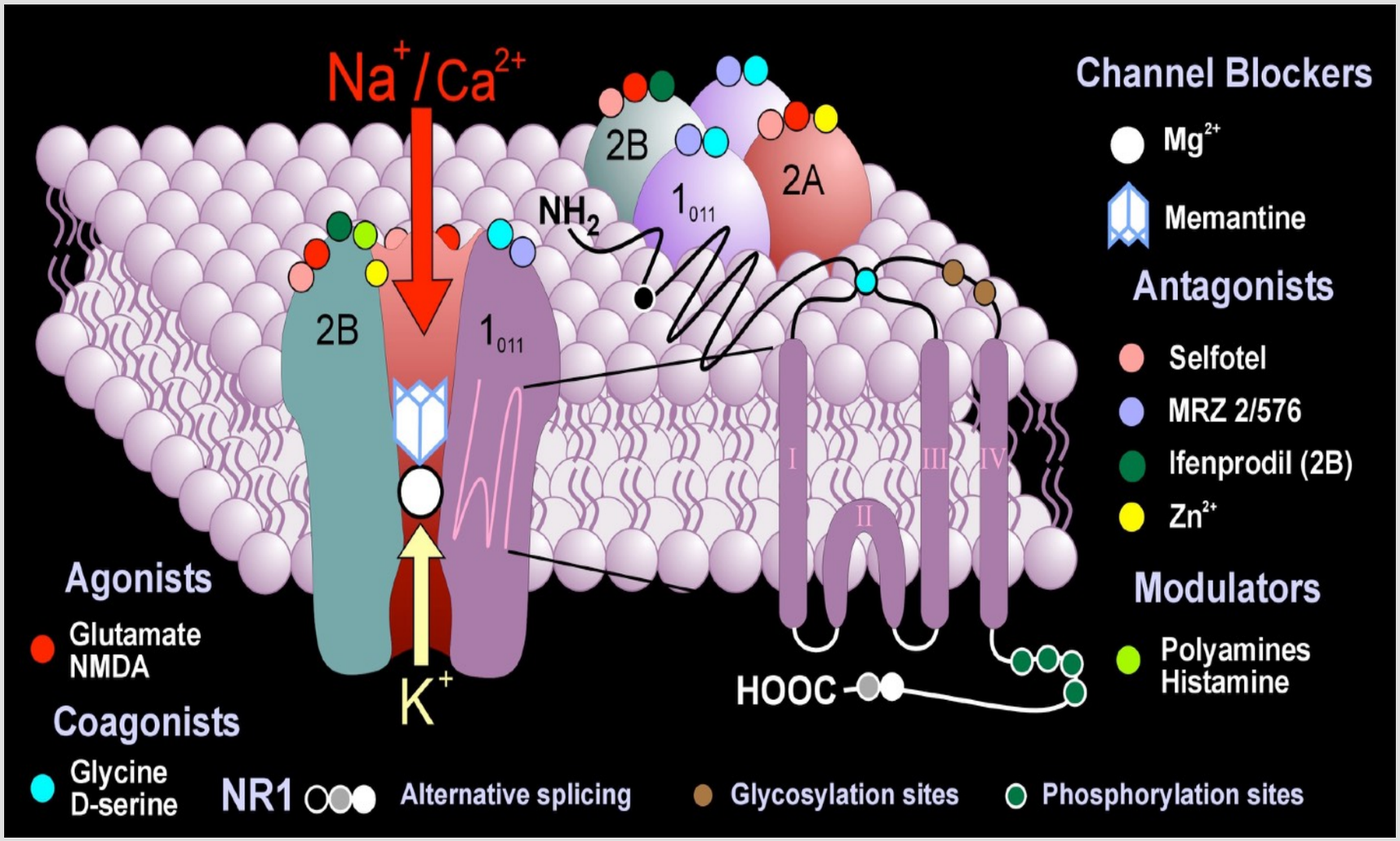
Objectif nutrition

- Prévenir l'inflammation
- Rétablir la balance glutamate/sérotonine
- **Moduler l'hyperfonctionnement du NMDA**

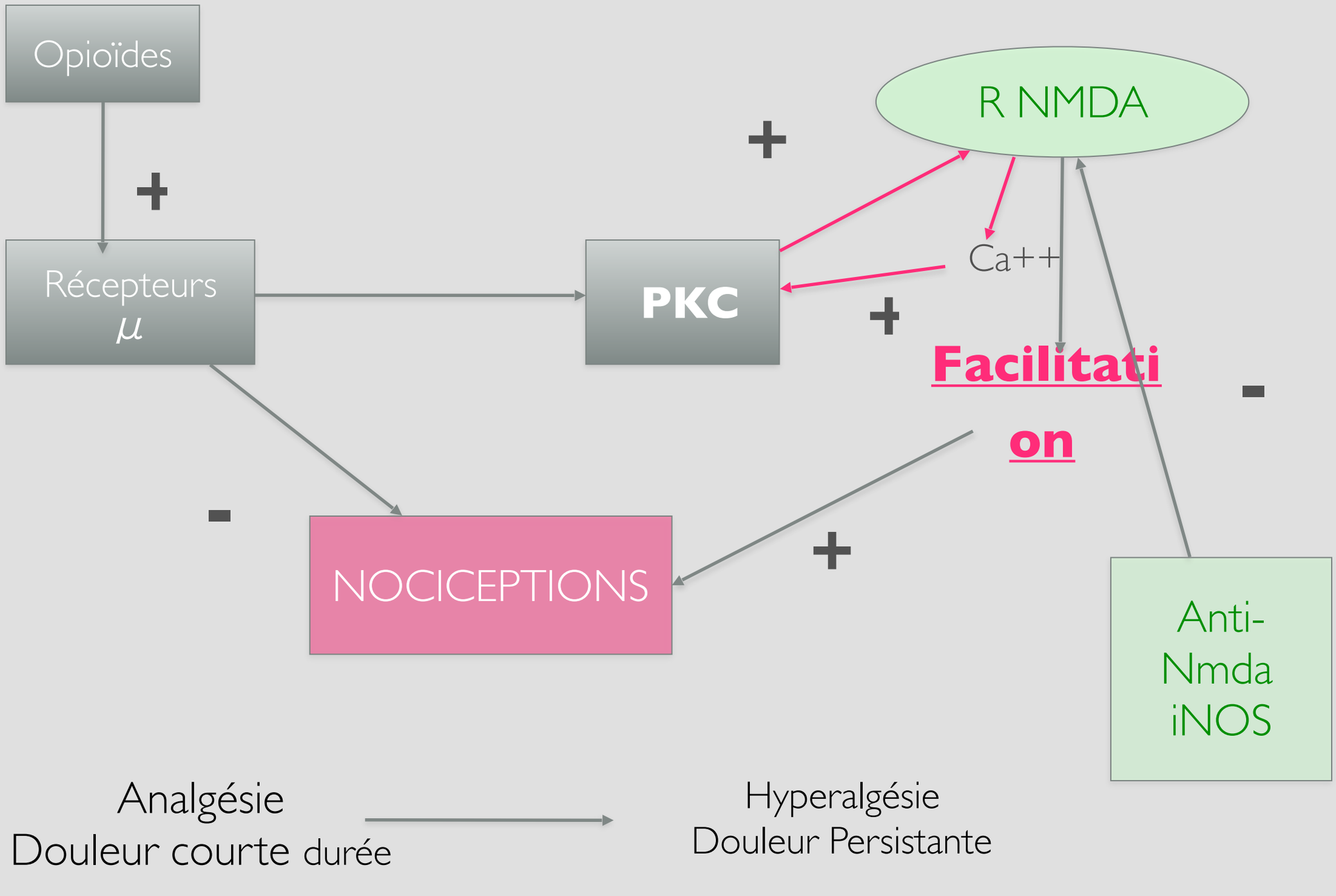
(Théorie du Pr Guy Simonnet)

La théorie de Guy Simonnet

Prévenir la chronicisation de la douleur en évitant l'hyperalgésie postopératoire via le récepteur NMDA



Chronicisation de la douleur



Role of peripheral polyamines in the development of inflammatory pain

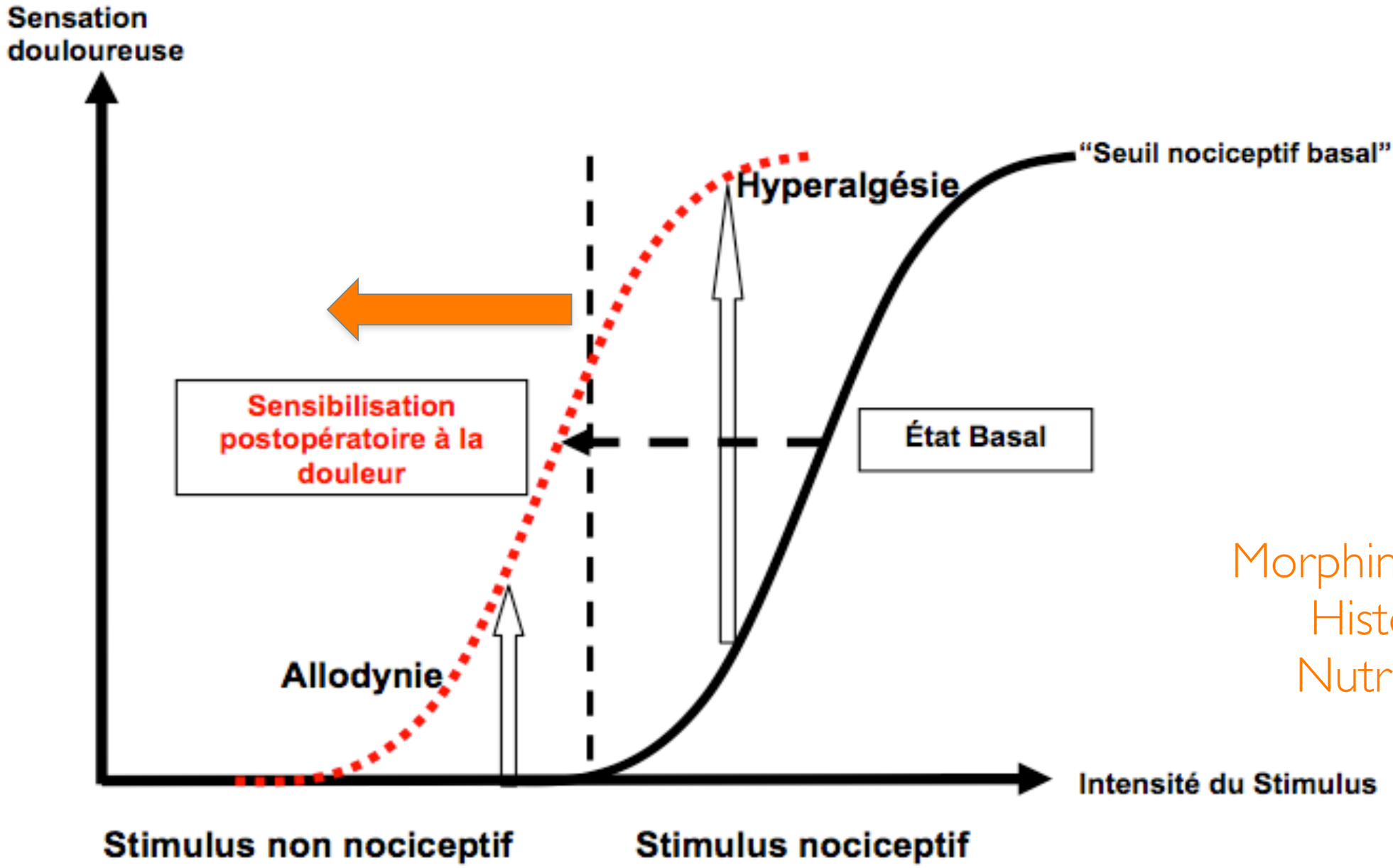
Mariane A. Silva[a], Jonatas Z. Klafke[a], Mateus F. Rossato[a], Camila Gewehr[b], Gustavo P. Guerra[a], Maribel A. Rubin[a, b], Juliano Ferreira[a, b, ,] VALIDHTMLVALIDHTMLVALIDHTML

Abstract

Polyamines (putrescine, spermidine and spermine) are aliphatic amines that are produced by the action of ornithine decarboxylase (ODC) in a rate-limiting and protein kinase C (PKC)-regulated step. Because high levels of polyamines are found in the synovial fluid of arthritic patients, the aim of the present study was to identify the role of peripherally produced polyamines in a model of inflammatory pain induced by adjuvant. The subcutaneous injection of Complete Freund's adjuvant (CFA, 50 μL/paw) caused the development of mechanical allodynia and edema. Moreover, it increased ODC expression and activity and PKC activation. Administration of the selective ODC inhibitor DFMO (10 μmol/paw) attenuated the development of allodynia and edema and decreased ODC activity in both control and CFA-treated animals. Furthermore, administration of the PKC inhibitor GF109203X (1 nmol/paw) reduced allodynia and ODC activity in animals injected with CFA. A subcutaneous injection of putrescine (10 μmol/paw), spermidine (3–10 μmol/paw) or spermine (0.3–3 μmol/paw) into the rat paw also caused mechanical allodynia and edema. The present results suggest that endogenously synthesized polyamines are involved in the development of nociception and edema caused by an adjuvant. Moreover, polyamine production in inflammatory sites seems to be related to an increase in ODC activity stimulated by PKC activation. Thus, controlling polyamine synthesis and action could be a method of controlling inflammatory pain.

La théorie de Guy Simonnet

Figure 2 : Évolution de la sensation douloureuse évoquée ou seuil nociceptif en fonction de l'intensité du stimulus douloureux appliqué au sujet. La courbe pleine représente les réponses préopératoires d'un sujet non sensibilisé, la courbe en pointillés celles d'un sujet sensibilisé, par exemple en période postopératoire



Morphine,stress
Histoire
Nutrition

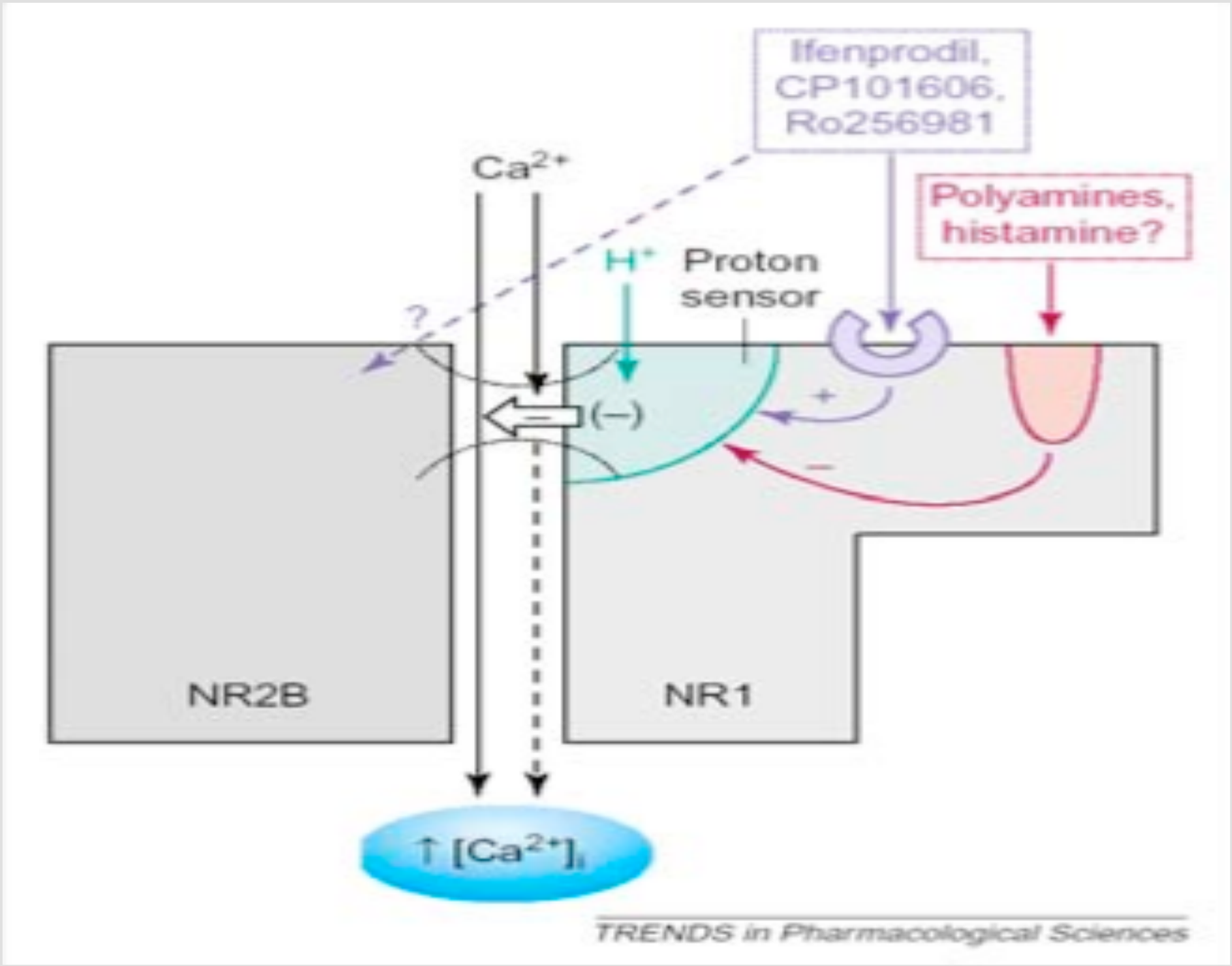
La théorie de Guy Simonnet

- ***Concept***: « Réduire l'hyperfonctionnement du récepteur NMDA par un mécanisme de modulation négative plutôt que de le bloquer sur une longue durée »

- Il existe, associés aux récepteurs NMDA, des sites-récepteurs modulateurs

- Il existe des récepteurs aux polyamines qui les inhibent (effet de double inhibition=excitateur)

La théorie de Guy Simonnet



La théorie de Guy Simonnet

- **Les polyamines**: Spermine, spermidine, putrescine… via le cycle de l'ornithine

- Facteurs de croissance libérés par l'intestin, les cellules tumorales et retrouvés dans l'alimentation…

- ***Aliments***: choux, champignons, orange

  produits fermentés, peau de poissons, poulet

  légumes surgelés non verts

  conserves

La théorie de Guy Simonnet

Ce Guide nutritionnel est complémentaire à la consommation de solutés Castase ou Polydol.
Si votre médecin vous a prescrit une cure à très faible teneur en polyamines vous pourrez alterner sa consommation avec celle d'aliments et de boissons figurant dans ce guide.

Pour toute information complémentaire vous pouvez contacter nos services par e-mail **info@nutrialys.fr** ou par téléphone (numéro vert, coût d'un appel local).

0 811 46 28 56

NUTRIALYS®
MEDICAL NUTRITION

Parc d'Affaires Edonia - rue de la Terre Victoria
35760 Saint Grégoire - France

Produits laitiers, Fromages

- 0 Beurre
- 0 Beurre salé
- 0 Bleu d'Auvergne
- 0 0 0 Brie pasteurisé avec croûte
- 0 Brie pasteurisé sans croûte
- 0 Camembert pasteurisé
- 0 0 0 Cantal doux sans croûte
- 0 0 0 Cantal doux avec croûte
- 0 0 0 Comté
- 0 Crème fraîche
- 0 Emmental
- 0 Féta mariné ou barquette
- 0 0 Fromage de chèvre avec croûte
- 0 Fromage de chèvre sans croûte
- 0 Fromage fondu à tartiner
- 0 Fromage frais aux herbes
- 0 Lait
- 0 Lait demi-écrémé
- 0 Lait en poudre
- 0 Lait ribot (caillé)
- 0 Lait en poudre
- 0 Mozzarella
- 0 0 0 Roquefort
- 0 0 0 Saint Nectaire avec croûte
- 0 0 0 Saint Nectaire sans croûte
- 0 Yaourt fraise
- 0 Yaourt nature

Œufs

- 0 Blanc d'œuf frais
- 0 Jaune d'œuf frais
- 0 Œuf frais

Pains

- 0 Biscotte (froment)
- 0 0 0 Brioche
- 0 Pain au son
- 0 0 0 Pain blanc (froment)
- 0 0 0 Pain complet
- 0 Pain de mie (froment)
- 0 Pain de mie tendre
- 0 0 0 Pain de seigle
- 0 Pain flocon d'avoine

Desserts

- 0 Chocolat au lait
- 0 Chocolat noir (80% cacao)
- 0 Confiture d'abricot
- 0 Confiture de fraise
- 0 Confiture de framboise
- 0 0 Confiture de prune
- 0 Corn Flakes
- 0 Crêpe de froment
- 0 Crêpe dentelle
- 0 Eclair au chocolat
- 0 Gâteau breton
- 0 0 Noix de coco poudre
- 0 Miel toutes fleurs
- 0 Petit beurre
- 0 Pop Corn sachet
- 0 Quatre quarts
- 0 0 Salade de fruits
- 0 Sucre
- 0 Tarte aux fraises (pâtisserie)
- 0 Tartelette à la fraise
- 0 Tartelette au citron

Fruits

- 0 0 0 Abricot
- 0 Abricot sec
- 0 Amande
- 0 0 Ananas
- 0 Avocat
- 0 0 Amande grillée
- 0 0 Cœur de palmier
- 0 0 0 Banane
- 0 0 0 Cacahuète grillée
- 0 0 Citron
- 0 Châtaigne
- 0 0 Datte (sèche)
- 0 Figue de Barbarie
- 0 Figue noire
- 0 0 0 Fraise
- 0 Grenade
- 0 0 Kiwi
- 0 Mangue
- 0 0 0 Melon
- 0 0 0 Nectarine
- 0 0 Noisette
- 0 Noisette sèche
- 0 0 Noix
- 0 0 0 Orange
- 0 Pamplemousse
- 0 Papaye morceaux
- 0 Pêche
- 0 Pêche blanche
- 0 0 0 Pistache grillée
- 0 Poire
- 0 0 0 Pomelo
- 0 Pomme
- 0 Prune
- 0 0 0 Prune jaune
- 0 0 Raisin
- 0 Raisin sec
- 0 0 0 Reine Claude
- 0 0 Tomate branche
- 0 0 0 Tomate cerise
- 0 0 0 Tomate mûre épluchée

Boissons

- 0 Apéritif à base de vin cuit
- 0 Bière blonde
- 0 Bière brune
- 0 Boisson au cola
- 0 Café
- 0 Champagne
- 0 Chicorée
- 0 Cidre
- 0 Cognac
- 0 Eau minérale plate et gazeuse
- 0 Indian tonic
- 0 Jus d'abricot
- 0 Jus de pomme
- 0 Jus de raisin
- 0 Jus de pamplemousse
- 0 0 Jus de tomate
- 0 0 0 Jus d'ananas
- 0 0 0 Jus d'orange
- 0 Limonade
- 0 0 0 Mélange multifruits
- 0 Pastis
- 0 Porto
- 0 Thé (Ceylan)
- 0 Tilleul menthe
- 0 Vin blanc (Bourgogne)
- 0 Vin blanc (Loire)
- 0 Vin rouge de table (Bordeaux)
- 0 Vin rouge de table (Côtes-du-Rhône)
- 0 0 Vin rouge de Bordeaux (supérieur)
- 0 0 Vin rouge de Touraine (supérieur)
- 0 0 Vin rouge du Beaujolais (supérieur)
- 0 Whisky

Edition avril 2008

GUIDE NUTRITIONNEL POUR UN REGIME ALIMENTAIRE PAUVRE EN POLYAMINES

NUTRIALYS®
MEDICAL NUTRITION

Régime « vert » avec ou sans Polydol®

La théorie de Guy Simonnet

Guide nutritionnel pour un régime alimentaire pauvre en polyamines

Le Guide nutritionnel Nutrialys permet, en complément de la consommation de solutés Castase ou Polydol, de réduire la consommation de polyamines d'origine alimentaire.
Ces molécules sont présentes en plus ou moins grande quantité dans l'alimentation et leur consommation excessive en cas de maladie chronique n'est pas souhaitable.
Les nutriments sont répartis en 3 classes suivant leur teneur en polyamines.

0 Vert **consommer de préférence**
0 Jaune **consommer avec modération**
0 Rouge **s'abstenir**

Plus le nombre de points est élevé, plus cet aliment est déconseillé.

Si votre médecin vous a prescrit une cure Castase ou Polydol, vous pourrez alterner sa consommation avec celle d'aliments et de boissons figurant dans ce guide.

Pour une efficacité optimale du traitement prescrit, la consommation des aliments et boissons présentés dans ce guide doit impérativement être associée aux solutés développés par Nutrialys.

Condiments

- 0 Ail
- 0 0 0 Aneth
- 0 Cerfeuil
- 0 0 0 Ciboulette
- 0 0 0 Estragon
- 0 0 0 Oseille
- 0 0 0 Persil
- 0 Poivre blanc
- 0 Sel

Sauces

- 0 0 0 Concentré de tomate
- 0 0 Ketchup
- 0 Mayonnaise
- 0 0 Moutarde
- 0 Sauce à la rouille
- 0 Sauce aïoli
- 0 Sauce béarnaise
- 0 Sauce beurre blanc
- 0 0 Sauce bourguignonne
- 0 Sauce moutarde
- 0 Sauce tartare
- 0 Vinaigre de vin

Huiles

- 0 Huile d'olive
- 0 Huiles de table
- 0 Margarine

Poissons, Coquillages, Crustacés

- 0 0 Bar
- 0 Cabillaud
- 0 Calamar
- 0 0 0 Caviar
- 0 0 0 Crabe (pince)
- 0 Colin
- 0 Crevette
- 0 0 0 Dorade royale
- 0 Ecrevisse
- 0 Filet de hareng fumé
- 0 0 0 Filet de julienne
- 0 Filet de loup de mer
- 0 Filet de maquereau fumé
- 0 0 Filet de lotte
- 0 0 Filet de saumon
- 0 Homard
- 0 0 0 Huître
- 0 Langoustine
- 0 0 0 Maquereau
- 0 0 0 Merlan
- 0 0 0 Moule
- 0 0 0 Noix St Jacques (blanc)
- 0 0 0 Noix St Jacques (corail)
- 0 0 0 Praire farcie
- 0 Rouget
- 0 Sardine
- 0 0 0 Saumon frais
- 0 0 0 Saumon fumé
- 0 0 0 Sole
- 0 Thon
- 0 0 Truite rosée

Riz, Pâtes, Céréales

- 0 0 Blé tendre
- 0 0 Pâtes
- 0 0 Riz blanc
- 0 0 Semoule

Viandes, Volailles

- 0 0 Aiguillette de canard
- 0 0 0 Bœuf
- 0 0 0 Chipolata
- 0 0 Escalope de dinde
- 0 0 0 Gésiers de canard confit
- 0 0 0 Langue de bœuf
- 0 Lapin
- 0 Lardon
- 0 Merguez
- 0 Mouton
- 0 0 0 Porc
- 0 0 0 Porcelet
- 0 Poulet
- 0 0 0 Rognons (veau)
- 0 Steak de cheval
- 0 0 0 Sanglier
- 0 0 0 Saucisse de Strasbourg
- 0 Saucisse de Toulouse
- 0 0 0 Saucisse sèche mezenc
- 0 Veau (escalope)

Charcuteries

- 0 0 Andouille
- 0 0 Cervelas
- 0 0 Chorizo
- 0 0 Jambon blanc
- 0 0 Jambon fumé
- 0 Mousse de foie de porc
- 0 0 0 Pâté de foie de canard
- 0 Poitrine de lard
- 0 0 Rillettes de porc
- 0 0 0 Rosette
- 0 0 Rôti de porc (tranche)
- 0 0 Salami
- 0 0 Saucisson de porc
- 0 0 0 Terrine de foie de porc

Légumes

- 0 0 0 Artichaut (cœur)
- 0 0 Asperge
- 0 0 0 Aubergine
- 0 0 0 Bette
- 0 0 Betterave rouge
- 0 0 0 Brocolis
- 0 0 Carotte
- 0 0 Céleri
- 0 0 0 Champignon de Paris, girolles, morilles, trompettes, cèpes
- 0 Chicorée
- 0 0 0 Choux de Bruxelles
- 0 0 0 Choux fleur
- 0 0 Choux rouge
- 0 0 Choux vert
- 0 0 Concombre
- 0 0 0 Courgette
- 0 Echalote
- 0 Endive
- 0 0 0 Fécule de pomme de terre
- 0 Fenouil
- 0 Feuille de chêne (salade)
- 0 0 Gingembre
- 0 0 Haricot gros plat sec
- 0 0 0 Haricot rouge
- 0 0 0 Haricot vert
- 0 0 0 Laitue
- 0 0 0 Lentille verte
- 0 Mogette
- 0 Navet
- 0 0 Oignon
- 0 Olive noire à la grecque
- 0 0 0 Patate douce
- 0 0 0 Petit pois
- 0 0 0 Poivron rouge
- 0 0 0 Pomme de terre épluchée
- 0 0 Pomme de terre avec peau
- 0 0 0 Pomme de terre nouvelle

Légumes (suite)

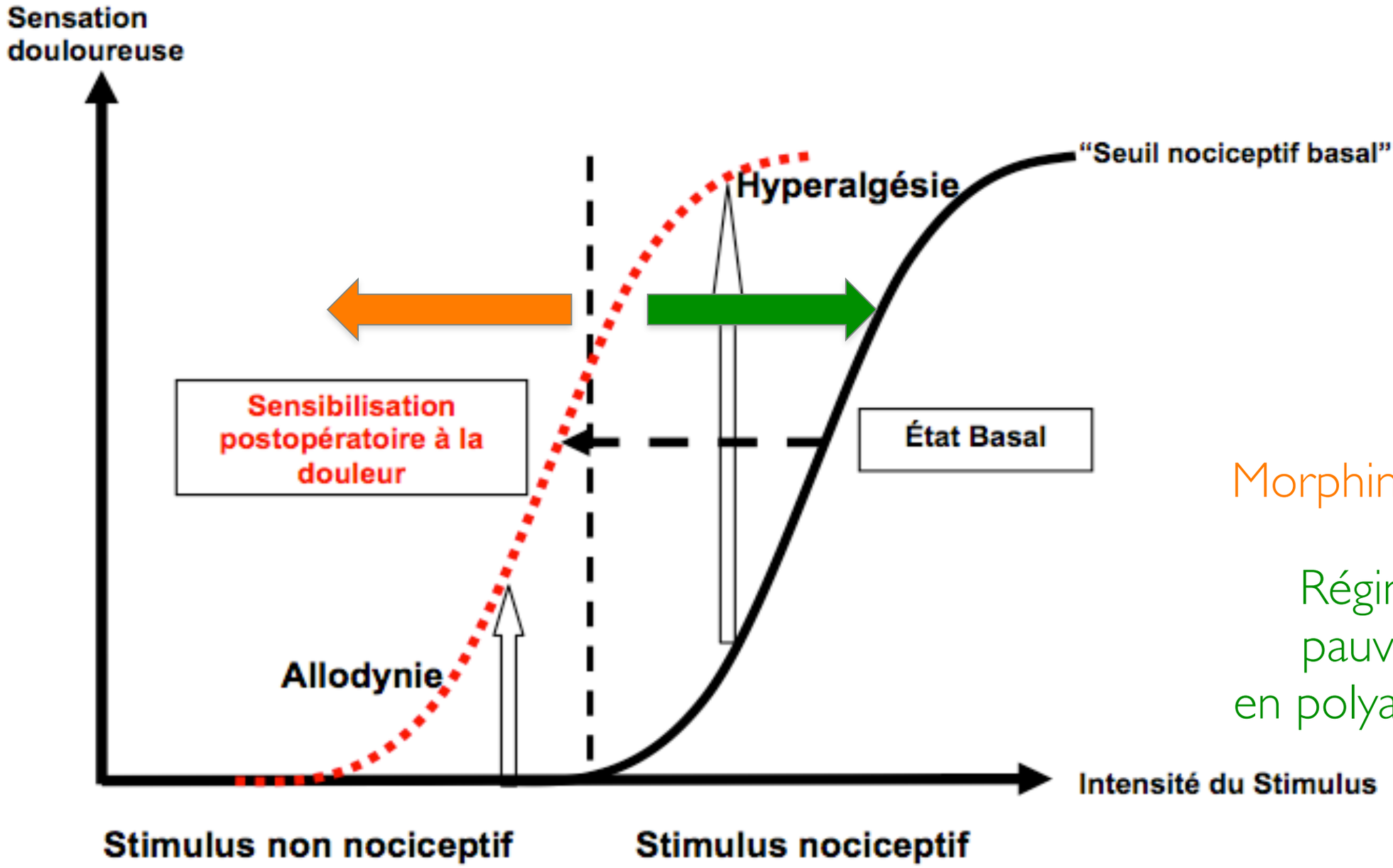
- 0 Poireau
- 0 0 Poivron vert
- 0 0 0 Pois cassé vert
- 0 0 0 Pois chiche
- 0 Potiron
- 0 Radis
- 0 Radis noir
- 0 0 0 Rutabaga
- 0 Salsifis
- 0 Scarole
- 0 0 0 Truffe

Aliments en conserve

- 0 Blé tendre
- 0 Compote de pomme
- 0 0 Carotte
- 0 0 Cassoulet
- 0 0 0 Choucroute
- 0 0 0 Cornichon en conserve
- 0 Epinards hachés
- 0 0 0 Filets d'anchois
- 0 0 Haricot rouge
- 0 0 Haricot vert
- 0 Julienne de carotte
- 0 Langue de bœuf
- 0 Lentille
- 0 0 Macédoine de légumes
- 0 0 0 Maïs boite
- 0 Marron entier
- 0 Paella
- 0 0 0 Petit pois
- 0 0 0 Petit pois / Carotte
- 0 0 Pois chiche
- 0 Purée (flocons)
- 0 0 0 Ratatouille
- 0 Saumon
- 0 0 Soupe de légumes
- 0 Thon au naturel
- 0 Tomate entière épluchée

La théorie de Guy Simonnet

Figure 2 : Évolution de la sensation douloureuse évoquée ou seuil nociceptif en fonction de l'intensité du stimulus douloureux appliqué au sujet. La courbe pleine représente les réponses préopératoires d'un sujet non sensibilisé, la courbe en pointillés celles d'un sujet sensibilisé, par exemple en période postopératoire



Morphine,stress

Régime pauvre en polyamines

Polyamine deficient diet to relieve pain hypersensitivity

Cyril Rivat[a], Philippe Richebé[a, b], Emilie Laboureyras[a], Jean-Paul Laulin[a], René Havouis[c], Florence Noble[d], Jacques-Philippe Moulinoux[c], Guy Simonnet[a, ,] VALIDHTMLVALIDHTMLVALIDHTML

Abstract

There is a compelling body of evidence that *N*-methyl-d-aspartate receptors (NMDA-R) play a critical role in the development and maintenance of pain hypersensitivity. However, long-term treatments with NMDA-R antagonists are limited by unacceptable side effects. Since polyamines modulate the functioning of NMDA-R and mainly originate from normal dietary intake and bacterial metabolism in the gut, we developed a nutritional therapy based on dietary polyamine deficiency. Here, we reported that a polyamine deficient diet (PD diet) for 7 days prevented the enhancement of tyrosine phosphorylation of the spinal NR2B subunit-containing NMDA-R associated with inflammation in rats. Based on these data, we studied the ability of PD diet to prevent long-lasting pain hypersensitivity associated with tissue injury on one hind paw by evaluating long-lasting changes in both mechanical nociceptive threshold and weight bearing. A PD diet strongly reduced long-lasting hyperalgesia induced by inflammation or incision, especially in fentanyl-treated rats. Moreover a PD diet also prevented the exaggerated hyperalgesia induced by a second inflammation performed 7 days after the first one. A PD diet also opposed paradoxical hyperalgesia induced by non-nociceptive environmental stress in rats with pain and opioid experiences. A PD diet reversed pain hypersensitivity associated with monoarthritis or neuropathy and restored the analgesic effect of morphine. Since PD diet was devoid of any noticeable side effects, this nutritional therapy could be part of an effective and safe strategy for pre-emptive analgesia and for reducing the transition from acute to chronic pain and its outcomes in various pain syndromes.

La théorie de Guy Simonnet

Chirurgie ambulatoire

Régime pauvre en polyamines
8j avant et 8j postop

	Sans	Avec
Douleur moyenne	Vas = 4,43	Vas = 1,91
Prise médicamenteuse complémentaire	14% = 0 médicament	27%= 0 médicament
		13% Vas=0; 80% Vas de 1 à 3

Pr Tandonnet Fr.
CH. Argenteuil

La théorie de Guy Simonnet

Colectomies en 2010

Régime pauvre en polyamines 8j pré et 8j postop
Même rapport bénin/malin; gauche/droit
Même chirurgie; même anesthésie

	Sans R	**Avec R**
Reprise transit	3 jours	1,7 jour
Alimentation Orale	4,37 jours	2,7 jours
Alim. Liquide/Solide	4/6	2/3
Cons. Antalgiques	Paracétamol/Ains/ Tramadol	=/-/-
Cons. Antalgiques	PCA Morphine	1/10
Fin d'hospitalisation	8,7 jours	5,5 jours

ê Coût ê charge de travail

Pr Dantrenay
Hop Argenteuil

Concrètement

- **Prévention:**

  - 2 semaine préop: régime "oméga 3", index glycémique bas et vert, vitamine C, curcuma et N'acétylcystéïne, vitamine D, probiotiques et glutamine…

  - Préop: Gabapentine ou prégabaline, aspirine ou cox2 en prénarcose

  - Perop: ALR si possible et infiltrations préincisionnelles du site opératoire, Kétamine, Magnésium et N2O (Meopa) ...

- **Traitement du CRPS:** "analgésie balancée" , NAC, oméga 3, curcuma, magnésium et acide alpha lipoique.

- **Gestion de la DCPO et dl neuropathique (DN)**

Il est une épargne aussi poignante que des sous dans la bourse d´une mère pauvre; de l´eau dans le désert de Gobi; de foin dans la mangeoire d´un âne…C´est l´épargne de la douleur."

Francis Jammes, XX^{e} siècle

Merci…