



SYNDROMES DOULOUREUX CHRONIQUES : CONCEPT, DIAGNOSTIC ET APPROCHE THÉRAPEUTIQUE

Journées médicales havraises
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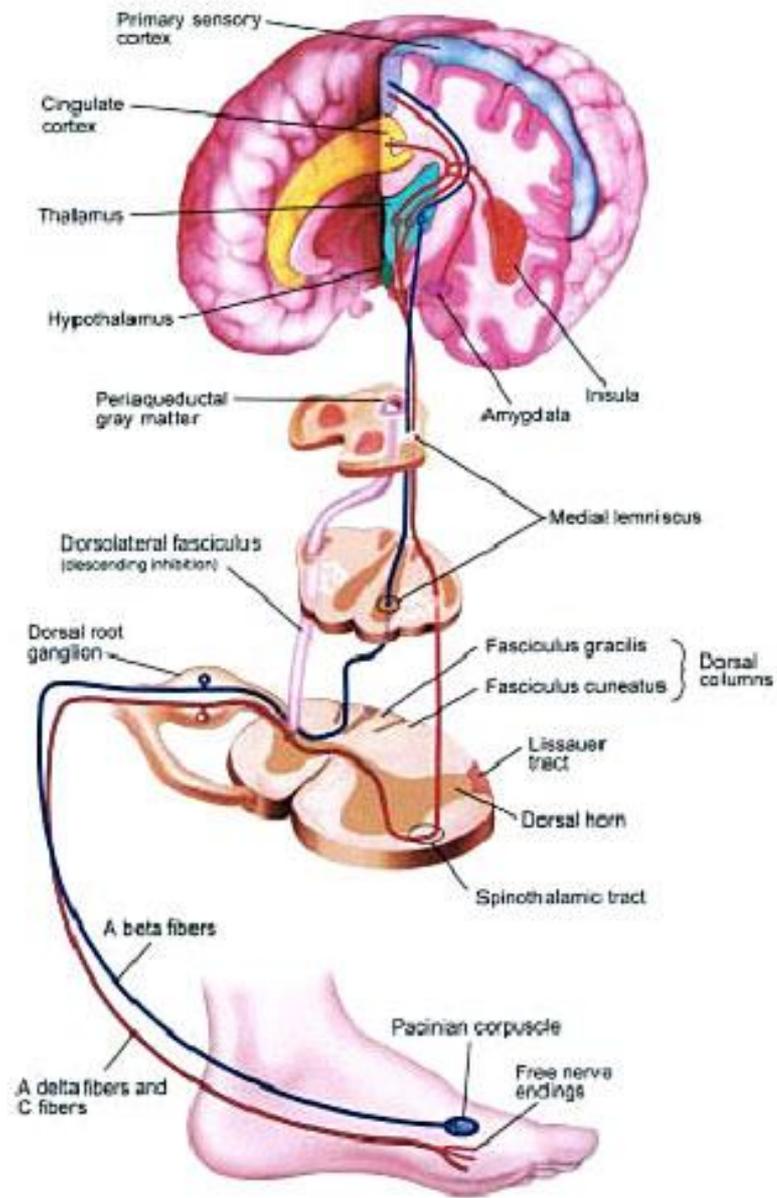
Introduction



Anatomie

	Diamètre	vitesse	rôle
Aα	13-30 μ m	80-120 m/s	Force musculaire
Aβ	6-12 μ m	35-75 m/s	Tact, proprioception
A δ myélinisées	1-5 μ m	5-35 m/s	Innervation de la peau (fibres somatiques) muscles cardiaques et lisses (fibres végétatives)
C Non myélinisées	0,2-1,5 m/s	0,5-2 m/s	Douleur, sensation thermique, fonctions végétatives

Axones sensoriels	A α	A β	A δ	C
Axones des fibres sensorielles musculaires	Groupe I	II	III	IV
				
Diamètre (μm)	13-20	6-12	1-5	0.2-1.5
Vitesse (m/s)	80-120	35-75	5-30	0.5-2
Récepteurs sensoriels	Propriocepteurs des muscles squelettiques	Mécanorécepteurs de la peau	Douleur, température	Température, douleur, démangeaison



Syndromes douloureux : des défis

- Mals définis
- Peu de signes objectifs
- Pas de traitement curatif
- Difficulté des médecins :
 - ▣ Reconnaissance/compréhension
 - ▣ Mise en échec
 - ▣ Chronophage



La Fibromyalgie

Epidémiologie

- Un syndrome fréquent : 2 à 4% de la population

(Wolfe F, Ross K, Anderson J, Russell IJ, Hebert L: The prevalence and characteristics of fibromyalgia in the general population. *Arthritis Rheum* 1995, 38:19–28.)

- Une majorité de femme
- Une entité discuté, des critères diagnostics peu spécifiques

Table 1 Prevalence of fibromyalgia in the general population

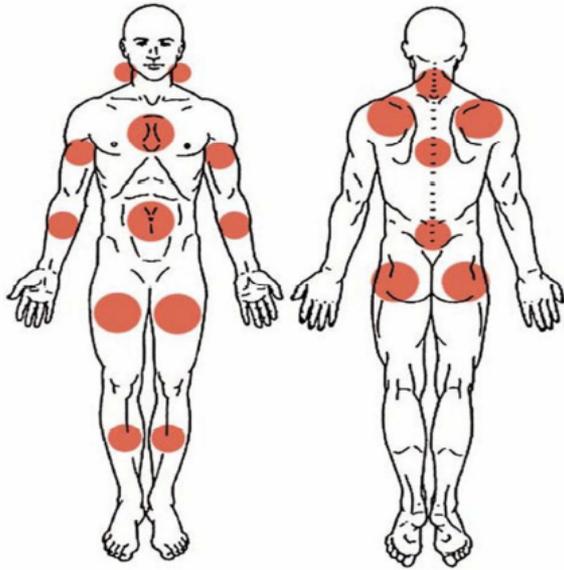
Country	Author	Case definition	N	Age range (y)	Prevalence (%)		
					Overall	Female	Male
Africa							
Tunisia	Guermazi [9]	LFESSQ	1,000	≥15	9.3	–	–
Americas							
Brazil	Senna [10]	COPCORD	3,038	≥16	2.5	3.9	0.1
Canada	White [11]	1990 ACR	3,395	≥18	3.3	4.9	1.6
Canada	McNally [12]	Self-reported	131,535	≥12	1.1	1.8	0.3
USA	Wolfe [13]	1990 ACR	3,006	≥18	2.2	3.4	0.5
USA	Vincent [14•]	2010 ACR	3,410	≥21	6.4	7.7	4.9
Asia							
Bangladesh	Haq [15]	COPCORD	5,211	≥15	3.6	6.2	0.9
China	Scudds [16]	1990 ACR	1,467	–	0.8	–	–
Israel	Ablin [3]	LFESSQ + 1990 ACR	1,019	≥18	2.0	2.8	1.1
Malaysia	Veerapen [17]	COPCORD	2,594	≥15	0.9	1.5	0.2
Pakistan	Farooqi [18]	COPCORD	1,997	≥15	2.1	–	–
Thailand	Prateepavanich [19]	2010 ACR	1,000	–	0.6	–	–
Europe							
Denmark	Prescott [20]	1990 ACR	1,219	18–79	0.7	–	–
France	Bannwarth [21]	LFESSQ + 1990 ACR	1,014	≥15	1.4	2.0	0.7
France	Perrot [22•]	LFESSQ + 1990 ACR	3,081	≥18	1.6	–	–
Finland	Mäkelä [23]	Yunus criteria	7,217	≥30	0.75	1.0	0.5
Germany	Branco [24•]	LFESSQ + 1990 ACR	1,002	≥15	3.2	3.9	2.5
Germany	Wolfe [25•]	2010 ACR	2,445	≥14	2.1	2.4	1.8
Greece	Andrianakos [26]	1990 ACR	8,740	≥19	0.4	–	–
Italy	Salaffi [27]	1990 ACR	2,155	≥18	2.2	–	–
Italy	Branco [24•]	LFESSQ + 1990 ACR	1,000	≥15	3.7	5.5	1.6
Portugal	Branco [24•]	LFESSQ + 1990 ACR	500	≥15	3.6	5.2	1.8
Spain	Branco [24•]	LFESSQ + 1990 ACR	1,001	≥15	2.3	3.3	1.3
Spain	Mas [28]	1990 ACR	2,192	≥20	2.4	4.2	0.2
Sweden	Lindell [29]	1990 ACR	2,425	20–74	1.3	2.4	0.0
Turkey	Turhanoglu [30]	1990 ACR	600	–	8.8	12.5	5.1
Mean					2.7	4.1	1.4

LFESSQ London Fibromyalgia Epidemiology Study Screening Questionnaire; COPCORD Community Oriented Program for the Control of Rheumatic Diseases; ACR American College of Rheumatology

Constatation :

1. Zones douloureuses(WPI): il faut comptabiliser le nombre de zones douloureuses présentes durant la semaine avant la consultation. Le score est de 0 à 19.

Les zones douloureuses sont configurées dans la **figure** et énumérées dans le **tableau**.



Nuque
Ceinture scapulaire droite et gauche
Haut et bas du dos
Thorax
Abdomen
Art temporo-mandibulaires droit et gauche
Bras droit et gauche
Avant-bras droit et gauche
Hanches droite et gauche (fesses, trochanter)
Cuisses droit et gauche
Jambes droit et gauche

2. L'échelle de sévérité des symptômes(SS) : Ces symptômes sont cotés de 0 à 3 :

Fatigue

Troubles du sommeil

Troubles cognitifs

} 0 : Pas de problème, 1 : très légers, 2 : modérés, 3 : sévères.

Symptômes somatiques : 0 = aucun symptôme, 1 = peu de symptômes, 2 = un nombre modéré de symptômes, 3 = de nombreux symptômes.

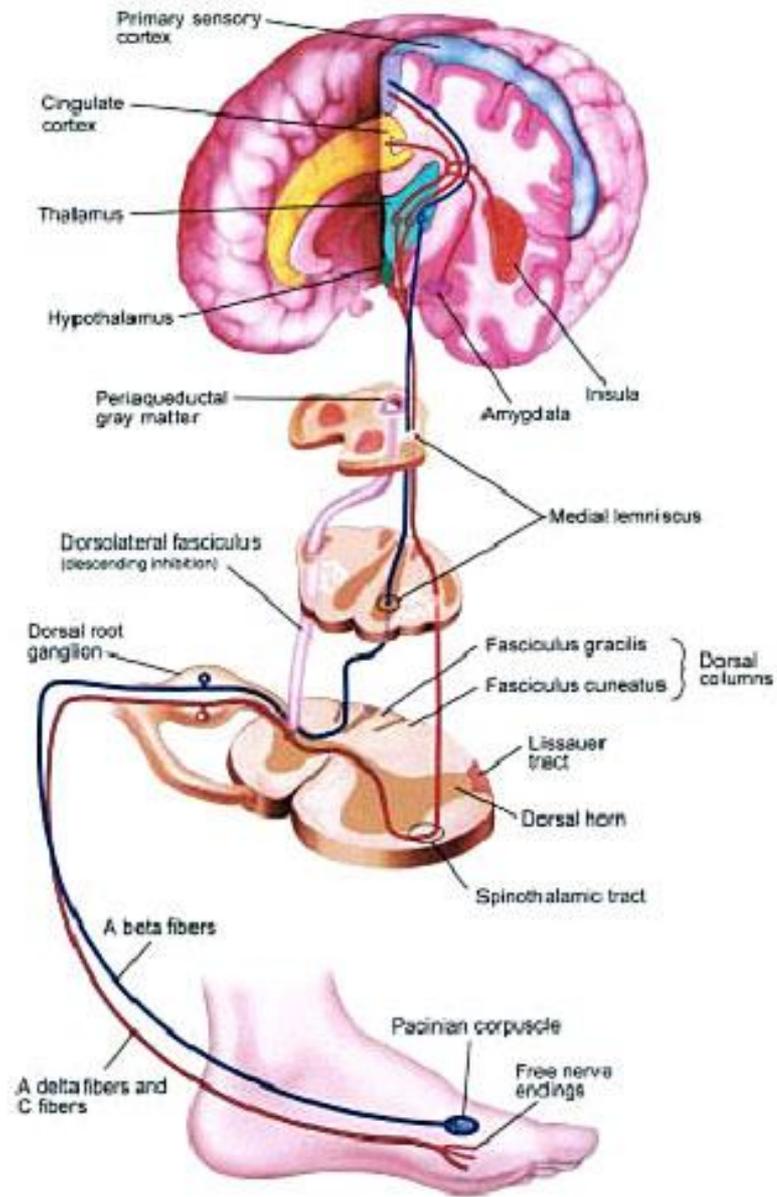
Critères ACR 2010

Selon ces nouveaux index (*WPI* de 0 à 19 et *Severity scale* de 0 à 12) (**tableau**), on arrive à un score total de 31.

Un score ≥ 13 a été retenu comme critère de base dans le diagnostic de la fibromyalgie.



Physiopathologie



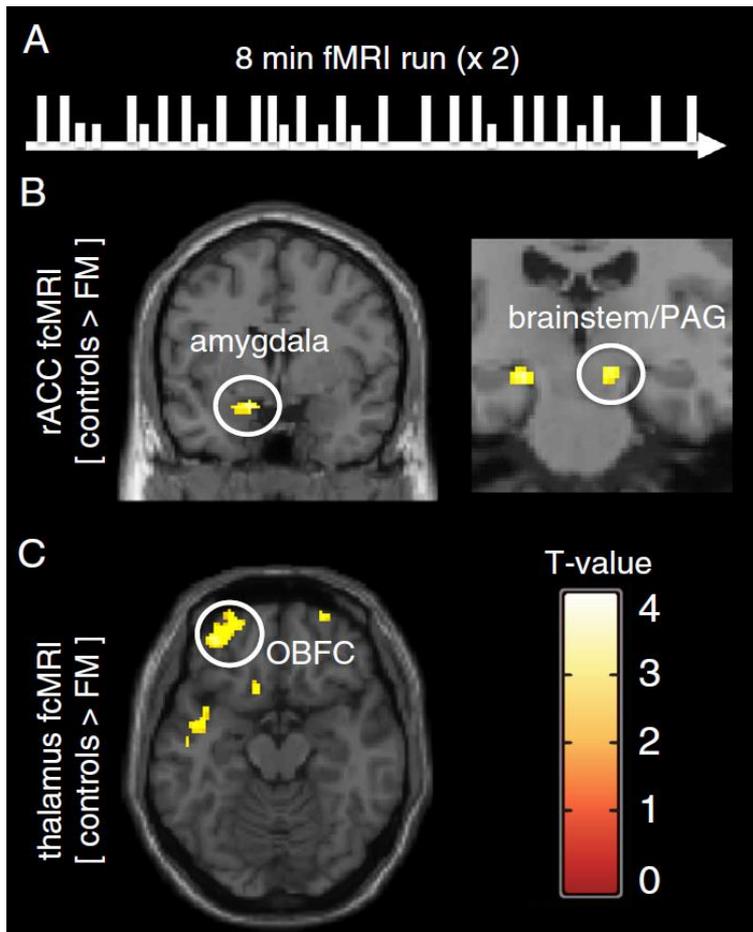


Figure 2 Experimental pain paradigm and functional connectivity results. The schematic illustration (A) represents the experimental pressure pain paradigm used during fMRI scanning. Calibrated painful pressures (long lines), representing 50 mm VAS, and non-painful pressures (short lines) were randomly delivered to the thumbnail during 2 adjacent 8 minute runs with 20 painful and 10 non-painful stimuli in each run. Functional connectivity results for the rACC seed (B) revealed increased connectivity to the amygdala (peak coordinate $x = -14, y = 0, z = -16$) and a cluster encompassing the brainstem/PAG/hippocampus (peak coordinate $x = 12, y = 24, z = -12$) in healthy controls, compared to FM patients. Functional connectivity results for the thalamus seed (C) revealed increased connectivity to the OBFC (peak coordinate $x = -34, y = 50, z = -18$) in healthy controls, compared to FM patients. All anatomical locations are given in Montreal Neurological Institute coordinates (MNI).

RESEARCH

Open Access

Patients with fibromyalgia display less functional connectivity in the brain's pain inhibitory network

Karin B Jensen^{1,2*}, Rita Loitole^{1,2}, Eva Kosek^{3,4}, Frank Petzke⁵, Serena Carville⁶, Peter Fransson³, Hanke Marcus⁷, Steven CR Williams⁸, Ernest Choy⁹, Yves Mainguy¹⁰, Olivier Vitton¹⁰, Richard H Gracely¹¹, Randy Gollub^{1,2}, Martin Ingvar^{3,4} and Jian Kong^{1,2}

Diagnostics différentiels

- Rhumatismes inflammatoires
- Connectivites (Gougerot, Lupus...)
- D'autres syndromes douloureux chroniques...!



La neuropathie des petites fibres

Historique

- 1ere description de NPF en **1992** (John et *al.* *Muscle Nerve*)
- Concept de neuropathies douloureuses ou neuropathie des petites fibres émergent au début des années **2000**
- **Epidémiologie :**
 - ▣ Prévalence : 53/100 000
 - ▣ Incidence : 12/100 000

(Peters et al. Incidence and prevalence of SFN: a survey in the Netherland. Neurologie 2013)



Présentation clinique

Clinique



- Syndrome douloureux neuropathique, chronique
- Le plus souvent longeur dépendant
- Examen clinique est le plus souvent normal

Clinique

- Douleurs neuropathiques
 - ▣ Spontanées
 - ▣ Majorée la nuit
 - ▣ **Brûlures** (burning feet), sensation de froid/chaud, étaiu, engourdissement, prurit
 - ▣ Facteurs favorisants : efforts, changements de températures, stress...
- Symptômes dysautonomiques
 - ▣ Dysfonction érectile, syndrome sec, malaise orthostatique, incontinence urinaire, troubles sudoraux
- Examen clinique
 - ▣ **diminution de la sensibilité thermo-algique (50%)**
 - ▣ dysesthésie de contact
 - ▣ Hyperalgésie 10-20%
 - ▣ Allodynie
- Force motrice, réflexes et proprioception préservés

Tableau 1

Symptômes associés aux neuropathies des petites fibres [25,32,33,41].

Symptômes sensitifs

Brûlures

Piqûres

Engourdissement

Fourmillement

Sensation de chaud/de froid

Décharges électriques

Hyperesthésie, allodynie

Intolérance des draps

Prurit

Jambes sans repos

Symptômes végétatifs

Troubles érectiles

Syndrome sec

Sueurs, bouffées de chaleur

Vertige/malaise (hypotension orthostatique)

Troubles digestifs et/ou urinaires

Tachycardie de repos, palpitations

Symptômes vasculaires

Érythromélgie

Acrosyndrome

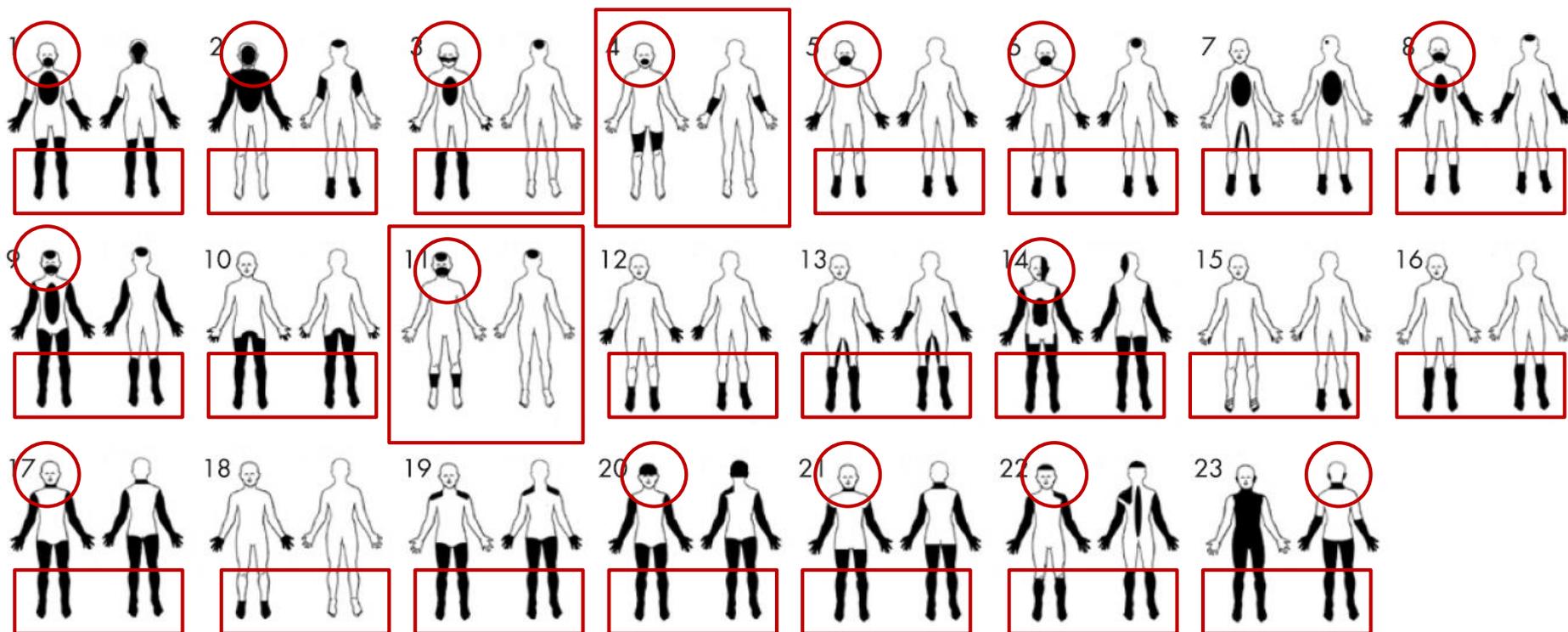
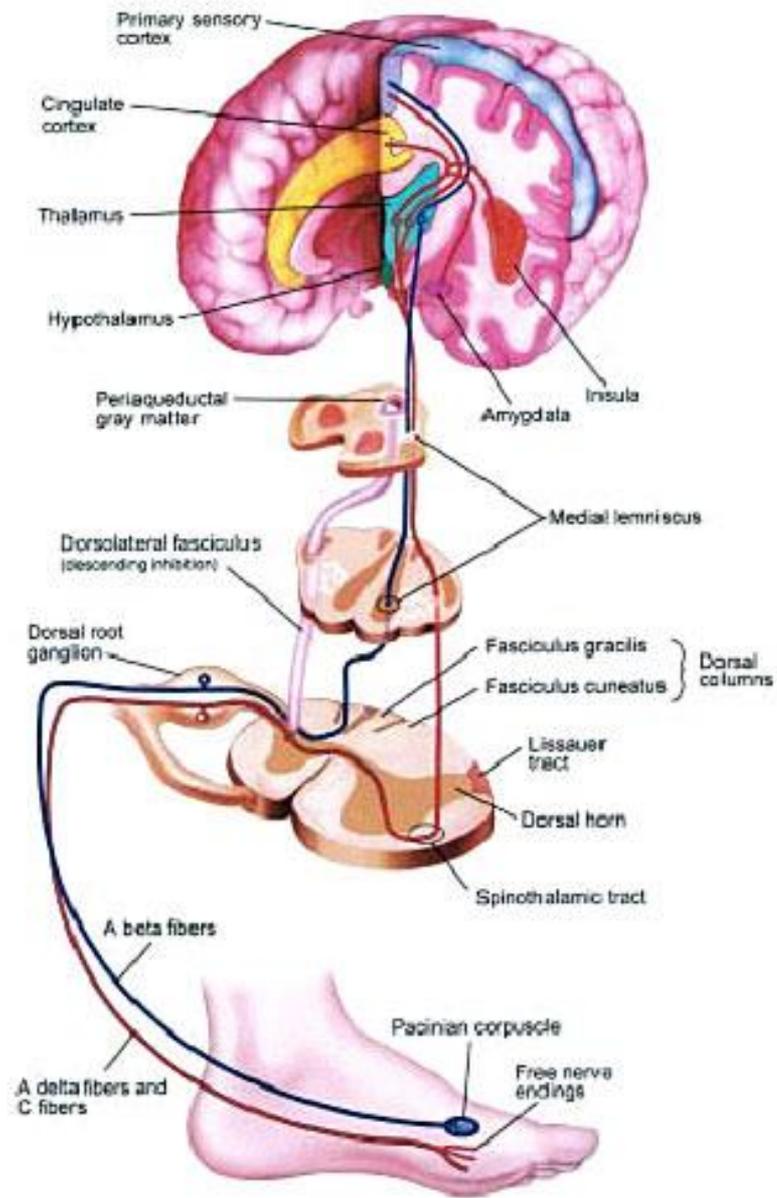


Figure 1 Body diagrams representing the regional distribution of neuropathic pain at the peak of symptoms in 23 patients with small fibre ganglionopathy. The number to the left of each figure corresponds to the patient number in tables 1 and 2.



Physiopathologie



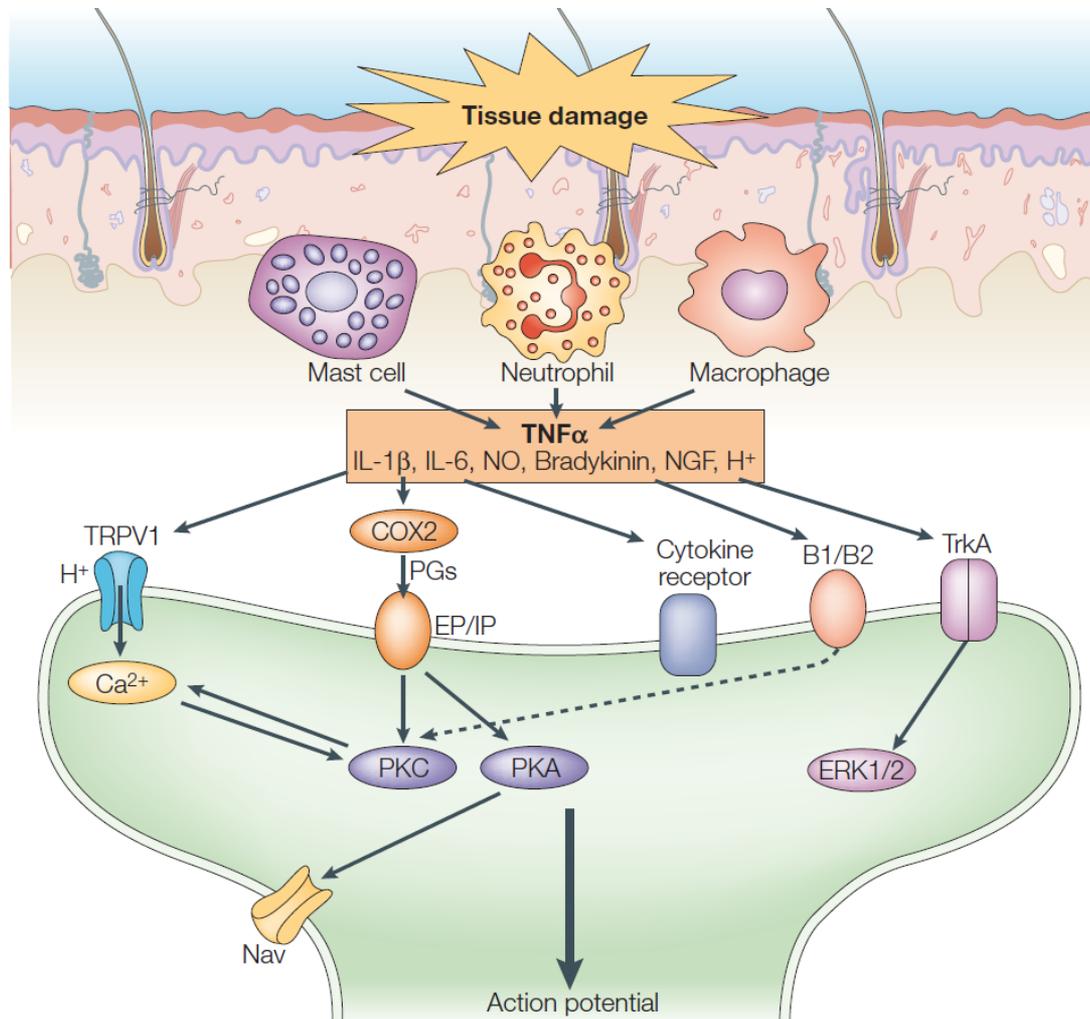


Figure 2 | **Inflammatory pain.** After tissue damage, mast cells and macrophages are activated and some blood-borne immune cells, including neutrophils, may be recruited. Various immune mediators are released (such as tumour necrosis factor- α (TNF α), interleukin-1 β (IL-1 β), interleukin-6 (IL-6), nitric oxide (NO), bradykinin, nerve growth factor (NGF) and protons), which exert their algescic effects by acting directly on nociceptors or indirectly through the release of other mediators, most notably prostanoids. There is increasing knowledge of the intracellular cascades that are activated in nociceptors by these mediators, which ultimately either activate or sensitize these neurons. COX2, cyclooxygenase 2; B1/B2, bradykinin receptor; EP/IP, prostanoid receptor; ERK1/2, extracellular signal-regulated kinase 1/2; Nav, voltage-activated sodium channel; PGs, prostaglandins; PKA/PKC, protein kinase A/C; TrkA, tyrosine receptor kinase A; TRPV1, transient receptor potential channel.

Physiopathologie

- Atteinte canaux sodiques (Nav1.7...)
 - Surtout présents au niveau des ganglions postérieurs de la moelle, du système végétatif, et des axones des petites fibres nerveuses
 - Forme héréditaire : Mutation gène SCN9A codant pour Nav1.7
 - Egalement Nav1.6, Nav1.8, Nav1.9, et NCX2

Physiopathologie

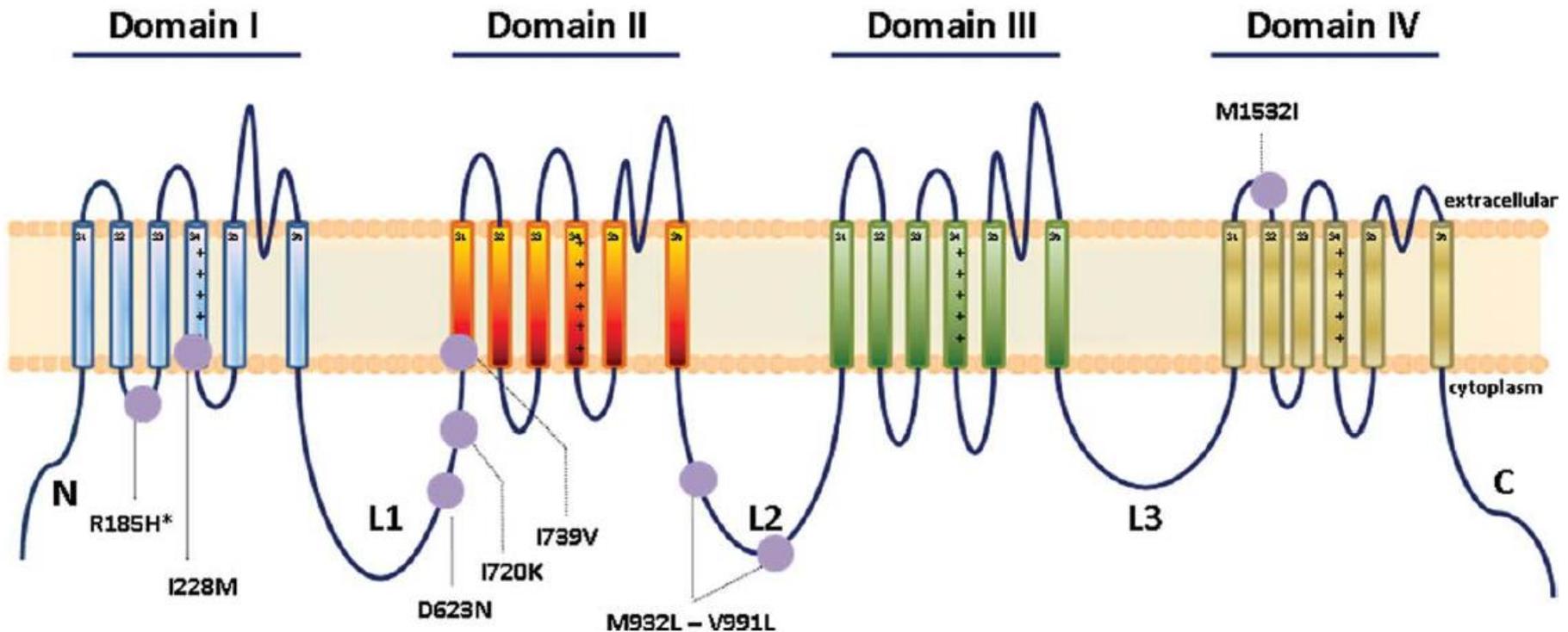


FIGURE 2: Schematic sodium channel showing the locations of the Nav1.7 mutations found in patients with idiopathic small nerve fiber neuropathy. Mutation R185H was found in 2 patients.

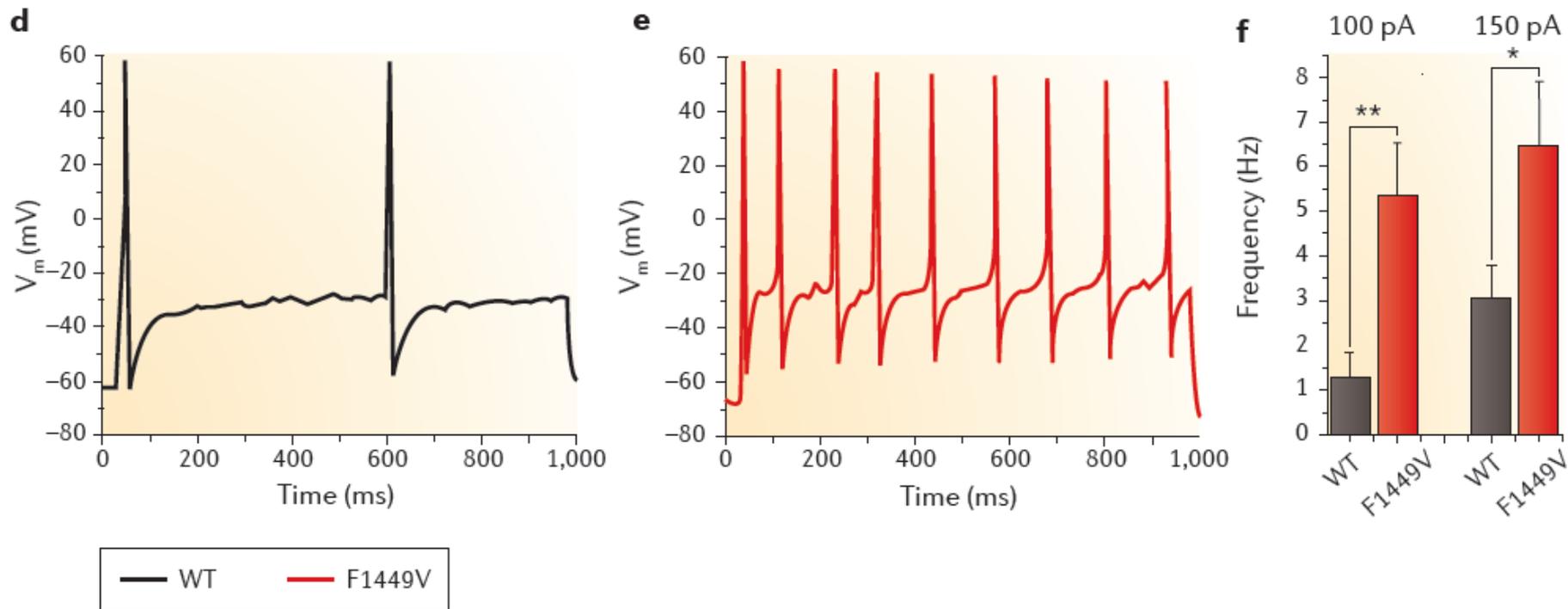


Figure 4 | **The F1449V mutation in Na_v1.7 makes DRG neurons hyperexcitable.** **a,b** | Representative traces from small (<30 μ m) dorsal root ganglion (DRG) neurons expressing wild-type (WT) Na_v1.7 or Na_v1.7 with the F1449V mutation (the variant linked to inherited erythromelalgia). These traces show that neurons expressing the mutant channel have a lower current threshold for action potential generation. **c** | The average current threshold is notably reduced in cells expressing F1449V compared with cells expressing WT channels (* $P < 0.05$). **d,e** | A neuron expressing WT Na_v1.7 responds to a 950 ms stimulation of 150 pA with a lower number of action potentials than does the neuron expressing the F1449V mutant (same cells as in panels **a** and **b**). **f** | There is a sizeable increase in the frequency of firing of action potentials in response to 100 pA and 150 pA stimuli (950 ms) with expression of F1449V versus expression of WT Na_v1.7 (* $P < 0.05$; ** $P < 0.01$). Figure is reproduced, with permission, from REF. 94 © (2005) Oxford University Press.

Physiopathologie

- Quid des formes associées au diabète, aux maladies auto immunes etc ...?
- Mécanisme :
 - ▣ Microangiopathie
 - ▣ Inflammation
 - ▣ Dysrégulation des canaux ioniques

... rien de démontré

Vous avez dit Nav1.7 et SCN9A ?



Erythromelalgie primitive : une neuropathie ?

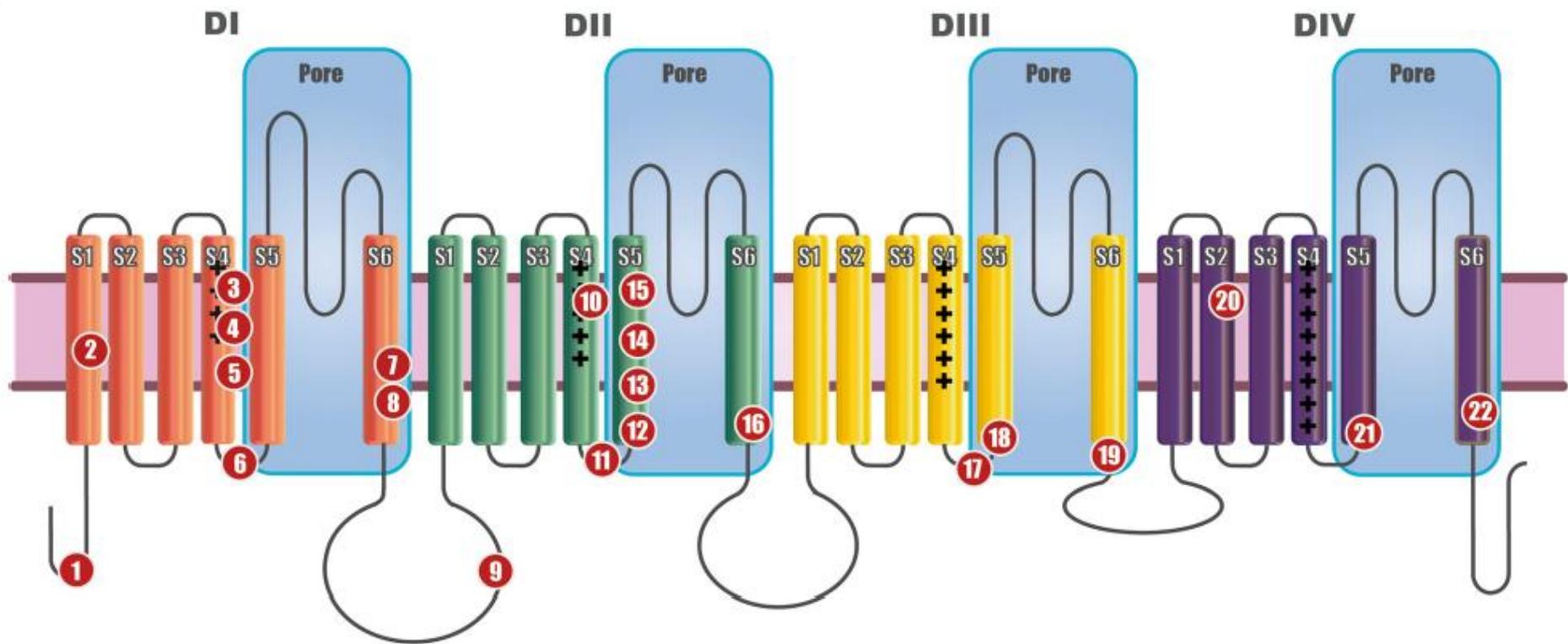
doi:10.1093/brain/aww007

BRAIN 2016; 139; 1052–1065 | 1052

BRAIN
A JOURNAL OF NEUROLOGY

Inherited erythromelalgia due to mutations in *SCN9A*: natural history, clinical phenotype and somatosensory profile

Aoibhinn McDonnell,¹ Betsy Schulman,² Zahid Ali,¹ Sulayman D. Dib-Hajj,² Fiona Brock,³ Sonia Cobain,³ Tina Mainka,^{4,5} Jan Vollert,^{4,6} Sanela Tarabar⁷ and Stephen G. Waxman²



1 Q10R/Q10K	6 I234T/S241T	11 I848T	16 Del-L955	21 A1632E/A1632T
2 I136V	7 N395K	12 L858F/L858H	17 P1308L	22 A1746G
3 F216S	8 V400M	13 A863P	18 V1316A	
4 S211P	9 G616R	14 V872G	19 F1449V	
5 I228M	10 L823R	15 Q875E	20 W1538R	

Fig. 1 Schematic of voltage-gated sodium channel alpha subunit and localization of reported PE mutations. The alpha subunit consists of four domains (I-IV). Each domain contains six helical transmembrane segments (S1-S6). The S4 segment of each domain contains positively charged amino acid residues

Et la Fibromyalgie ?



Small fibre pathology in patients with fibromyalgia syndrome

Nurcan Üçeyler,¹ Daniel Zeller,¹ Ann-Kathrin Kahn,¹ Susanne Kewenig,¹ Sarah Kittel-Schneider,² Annina Schmid,¹ Jordi Casanova-Molla,¹ Karlheinz Reiners¹ and Claudia Sommer¹

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Evidence of Abnormal Epidermal Nerve Fiber Density in Fibromyalgia

Clinical and Immunologic Implications

Xavier J. Caro¹ and Earl F. Winter²

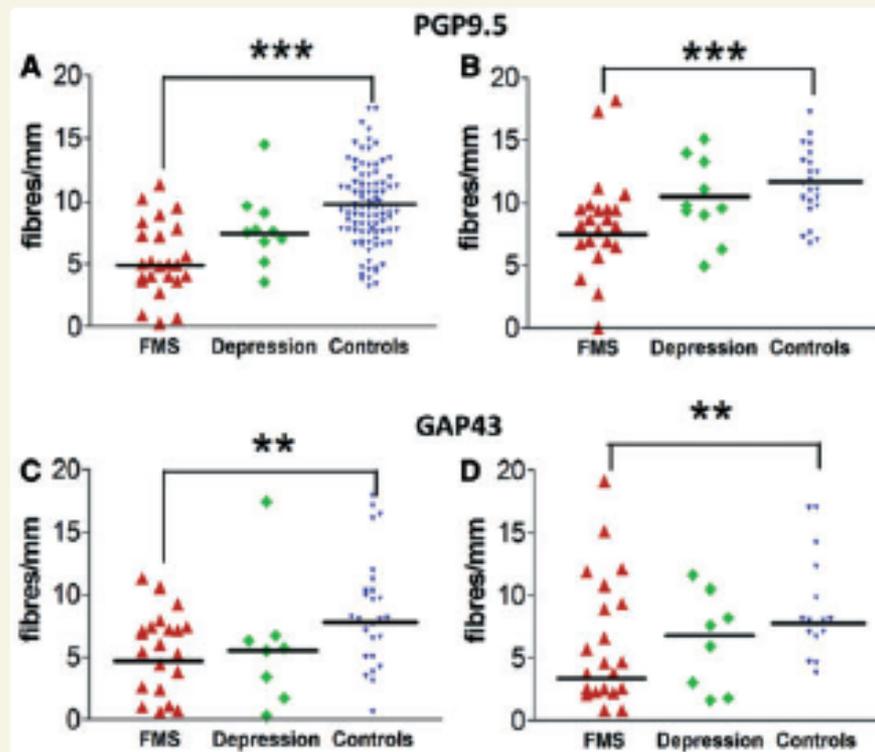


Figure 4 IENFD at the lower leg (A) and the proximal thigh (B) of patients with fibromyalgia syndrome, depression, and of healthy control subjects investigated with the pan-axonal marker PGP9.5. Patients with fibromyalgia syndrome have lower PGP9.5-positive IENFD compared with healthy control subjects at both biopsy sites. Density of regenerative intraepidermal nerve fibres immunoreacted with GAP43 at the lower leg (C) and the proximal thigh (D) of patients with fibromyalgia syndrome, depression, and of healthy control subjects. Patients with fibromyalgia syndrome have lower immunoreactivity for GAP43 compared with healthy control subjects at both biopsy sites. ** $P < 0.01$; *** $P < 0.001$. FMS = fibromyalgia syndrome. GAP43 = growth-associated protein 43. PGP9.5 = protein-gene product 9.5.

Explorations et critères diagnostiques

Références :

- The diagnostic criteria for small fibre neuropathy: from symptoms to neuropathology, Devigili G, Brain 2008
- Diabetic neuropathies: update on definitions, diagnostic criteria, estimation of severity, and treatments. Tesfaye S et al, Diabetes Care 2010;33:2285–93
- Khoshnoodi MA, Truelove S, Burakgazi A, Hoke A, Mammen AL, Polydefkis M. Longitudinal assessment of small fiber neuropathy: evidence of a non-length-dependent distal axonopathy. JAMA Neurol 2016;73:684

Examens complémentaires

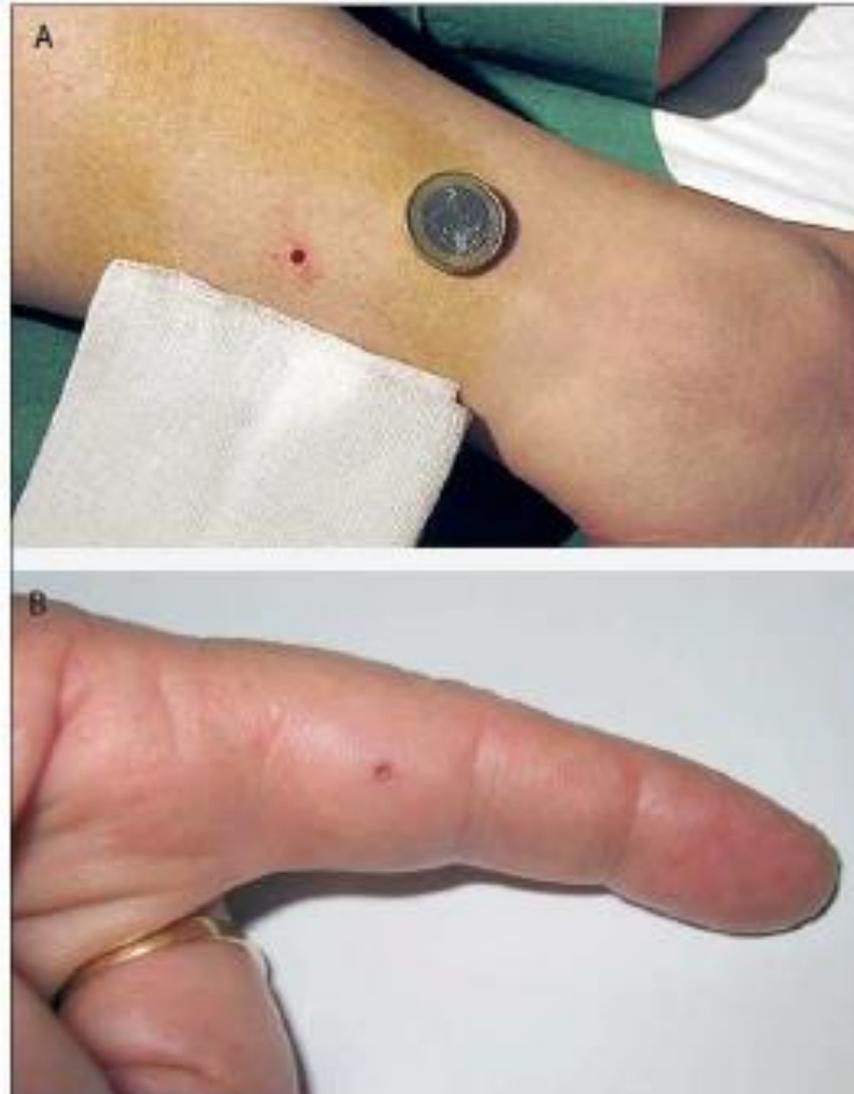
- ENMG : normal
 - ▣ RCS
 - ▣ Intervalle R-R
- Potentiel évoqué laser
- Sudoscan®
- QSART : Quantitative Sudomotor Axon Reflex Test
- QST : Quantitative sensory test



Examens complémentaires

- La biopsie cutanée
 - Sensibilité : 88%, Spécificité 89%
 - Immunofluorescence indirecte avec anticorps anti-PGP9.5
 - Peu de centres peuvent analyser les prélèvements en France

Biopsie cutanée



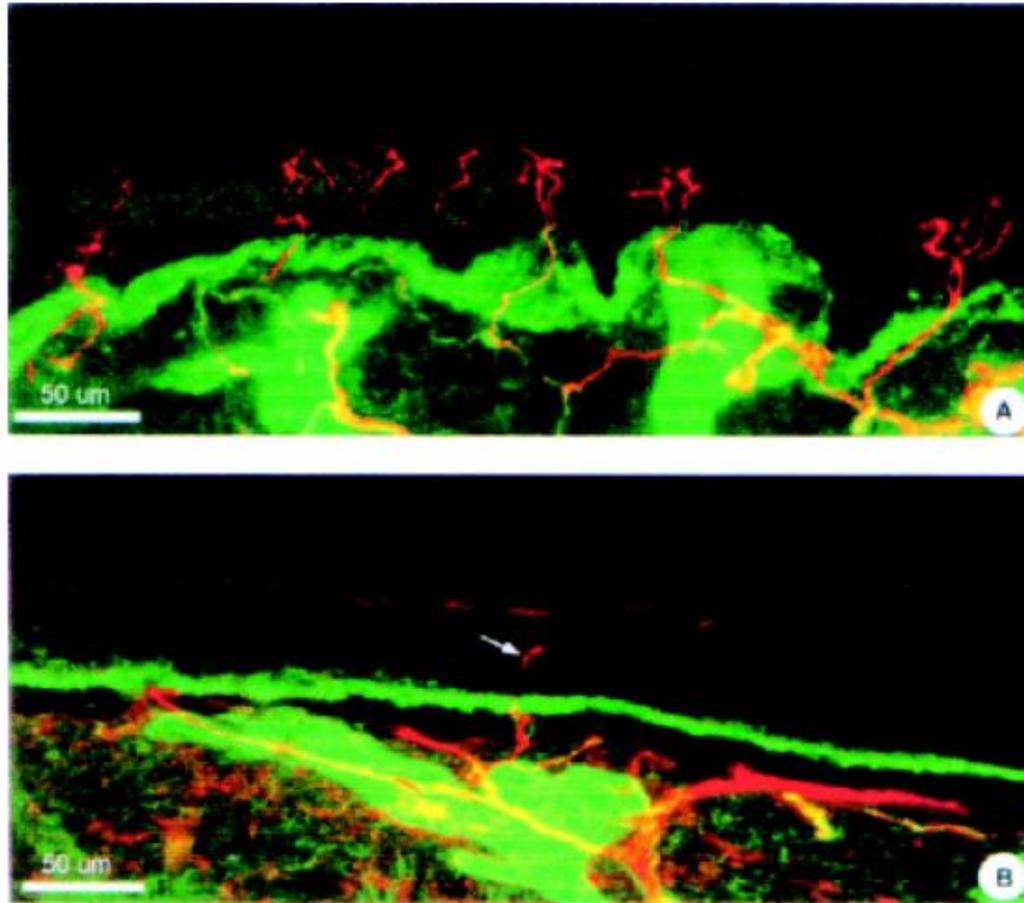


FIGURE 2. Skin biopsies from a normal patient (**A**) and from a patient with a small-fiber neuropathy (**B**) viewed by confocal microscopy. The epidermal nerve fibers appear red and are reacted with an antibody against protein gene product 9.5. The basal lamina appears green due to its immunoreactivity against a collagen IV antibody. The epidermal nerve fiber density and branching pattern are normal in (**A**), whereas only a single nerve (arrow) extends beyond the basal lamina in the neuropathy patient's specimen (**B**). (Reproduced from reference 73 with permission from Lippincott Williams & Wilkins, Inc.)

Critères diagnostiques

Les premiers critères diagnostiques ont été définis par Devigili et al. en 2008 [25]. Le diagnostic positif de NPF nécessite au moins 2 des 3 critères suivants :

- la présence de signes cliniques compatibles avec une altération des petites fibres (trouble de la perception du chaud/froid et/ou allodynie et/ou hyperesthésie) dont la topographie évoque une neuropathie périphérique (longueur ou non-longueur dépendante) ;
- l'anomalie du seuil de perception du chaud et/ou du froid aux pieds mesurée par le *Quantitative sensory test* (QST) ;
- la réduction de la densité intra-épidermique en petites fibres nerveuses en distalité.

Avant de retenir le diagnostic, il convient d'éliminer la présence d'une altération clinique des grosses fibres nerveuses (hypoesthésie et/ou anomalie de la pallesthésie et/ou atteinte proprioceptive et/ou absence des réflexes ostéo-tendineux), d'une altération clinique des fibres motrices (déficit moteur, etc.) ou d'une altération de l'électromyogramme.

Quels explorations pour quels patients ?

- ENMG : sera normal. Eliminer un diagnostic différentiel
- Biopsie cutanée...si doute ou si besoin d'une certitude diagnostique...mais accessibilité limitée pour l'instant
- Potentiel laser, sudoscan, QSART, QST difficilement accessibles.



Bilan étiologique

Etiologies

Table 2. Acquired and genetic diseases associated with pure and predominantly somatic small fibre neuropathy, and most common distribution of symptoms and signs (length and non-length-dependent)

	Cause	Length-dependent	Non-length-dependent	References
Metabolic	Diabetes	+		[5,6,9,17–19]
	Impaired glucose tolerance	+		[5,6,9,17–19]
	Rapid glycaemia control in diabetes	+		[20]
	Hypothyroidism	+	+	[6,21–23]
	Vitamin B ₁₂ deficiency		+	[22]
Infectious disease	HIV	+		[24,25]
	Influenza		+	[26]
	Hepatitis C	+	+	[27]
Drugs and toxics	Antiretroviral drugs	+		[25]
	Metronidazole	+		[28]
	Bortezomib	+		[29,30]
	Statin	+		[31]
	Nitrofurantoin		+	[32]
	Flecainide	+		[33]
	Linezolid	+		[34,35]
Chronic alcohol abuse	+		[36,37]	

Immune-mediated	Celiac disease		+	[38]
	Sarcoidosis		+	[15,39]
	Other rare associations		+	[6,11,40–42]
	• Parkinson disease ¹²⁹			[43,44]
Paraneoplastic	• Pompe disease ¹³⁰		+	[10,42]
• Multiple myeloma	• Fragile X–associated tremor ataxia syndrome ¹³¹		+	[10,45]
• Lung cancer	• Benign fasciculation syndrome ¹³²		+	[46 [■]]
	• Critical illness ¹³³		+	[6,11,47–49]
	• Celiac disease ¹³⁴		+	[50]
Genetic	• Wilson disease ^{135,136}	Neuropilin-1 ⁹¹	+	[51]
	• Amyotrophic lateral sclerosis ^{137,138}		+	[52]
	• X-linked adrenoleukodystrophy ¹³⁹			[53 [■]]
	Idiopathic ⁴²			[54]
	Sodium channel mutations (SCN9A)		+	[55,56]
Idiopathic	Unknown		+	[16 [■] ,57 [■]]

HSAN, hereditary sensory neuropathy.

Treatment-induced neuropathy of diabetes: an acute, iatrogenic complication of diabetes

Christopher H. Gibbons and Roy Freeman

Le bilan étiologique

- Interrogatoire :
 - Souvent un facteur déclenchant à rechercher
 - Analyse des traitements
 - Alcool, autres toxiques
- Bilan biologique :
 - Glycémie à jeun +++
 - Bilan lipidique
 - Recherche d'une intolérance au glucose
 - TSH
 - VIH, VHB, VHC
 - Folate, B12
 - FAN, anti SSA, anti SSB, Anti transglutaminase IgA
 - Immunoélectrophorèse des protéines sériques
 - Angiotensine convertase
- Imagerie
 - Radiographie de thorax
- Autres examens en fonction du contexte :
 - Discuter BGSA
 - Recherche d'amylose
 - Recherche génétique...

En deuxième intention

- Ne pas oublier les étiologies plus rares si le contexte est évocateur :
 - ▣ Amylose (douleur + signes dysautonomiques)
 - ▣ Fabry (douleur + angiokératome)
 - ▣ Tangier...



Prise en charge thérapeutique

Prise en charge

- Médicaments
- Soutien psychologique
- Méthodes alternatives (Hypnose, acuponcture, Yoga, sophro, Tai shi...)
- Activité sportive régulière d'endurance
- Maintien d'une vie sociale (travail, associatif...)

Traitements

Table 3. Recommended agents for the treatment of neuropathic pain

Mechanism	Medication	Efficacy studies	Evidence
Antidepressant			
<ul style="list-style-type: none"> • Tricyclic antidepressants • Serotonin–norepinephrine reuptake inhibitor 	Nortriptyline, despramine, amitriptyline, imipramine Duloxetine, venlafaxine	Neuropathic pain Neuropathic pain	First line (NeuPSIG, MER EFNS, LA, CPS) Level B (AAN), first line (NeuPSIG, EFNS), second line (MER, CPS), third line (LA)
Anticonvulsant			
<ul style="list-style-type: none"> • Calcium channel $\alpha 2\text{-}\delta$ ligand 	Gabapentin, pregabalin	Neuropathic pain	Level A (AAN), first line (NeuPSIG, EFNS, MER, CPS), second line (LA)
Opioid	Oxycodone, morphine, tramadol	Neuropathic pain	Level A (EFNS), level B (AAN), second line (NeuPSIG, EFNS, MER, LA, CPS)
Topical therapy	Capsaicin, lidocaine	HIV neuropathy, post-herpetic neuralgia	First line (NeuPSIG, EFNS, MER, LA), second line (CPS)

None of the listed agents were tested in SFN alone. All were tested in large fiber neuropathies accompanied by pain and are summarized from earlier studies.^{10,110–116,140–142} Duloxetine and venlafaxine NNT and NNH are calculated together. NeuPSIG, Neuropathic Pain Special Interest Group (2010); MER, Expert Panel Recommendations for the Middle East Region (2010); EFNS, European Federation of Neurological Societies (2010); LA, Guidelines for the Diagnosis and Management of Neuropathic Pain: Consensus of a Group of Latin American Experts (2009); CPS, Canadian Pain Society (2014).

Traitements

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Differential Block of Sensory Neuronal Voltage-Gated Sodium Channels by Lacosamide [(2*R*)-2-(Acetylamino)-*N*-benzyl-3-methoxypropanamide], Lidocaine, and Carbamazepine[□]

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L'avenir : thérapie ciblée en fonction de la génétique ?



Efficacy, safety, and tolerability of lacosamide in patients with gain-of-function $\text{Na}_v1.7$ mutation-related small fiber neuropathy: study protocol of a randomized controlled trial—the LENSS study

Bianca T. A. de Greef^{1*}, Ingemar S. J. Merkies^{1,2}, Margot and Janneke G. J. Hoeijmakers¹



Intravenous immunoglobulin therapy for small fiber neuropathy: study protocol for a randomized controlled trial

Respir Med. 2017 May;126:135-138. doi: 10.1016/j.rmed.2017.03.011. Epub 2017 Mar 9.

Sarcoidosis-associated small fiber neuropathy in a large cohort: Clinical aspects and response to IVIG and anti-TNF alpha treatment.

Tavee JO¹, Karwa K², Ahmed Z³, Thompson N⁴, Parambil J⁵, Culver DA⁵.

Special Issue

Cibinetide Improves Corneal Nerve Fiber Abundance in Patients With Sarcoidosis-Associated Small Nerve Fiber Loss and Neuropathic Pain

Daniel A. Culver,¹ Albert Dahan,² Daiva Bajorunas,³ Maria Jeziorska,⁴ Monique van Velzen,² Leon P. H. J. Aarts,² Jinny Tavee,⁵ Martijn R. Tannemaat,⁶ Ann N. Dunne,³ Rita I. Kirk,³ Ioannis N. Petropoulos,⁷ Anthony Cerami,³ Rayaz A. Malik,⁷ and Michael Brines³

ORIGINAL ARTICLE

A Randomized Trial of Tai Chi for Fibromyalgia

Chenchen Wang, M.D., M.P.H., Christopher H. Schmid, Ph.D., Ramel Roncs, B.S., Robert Kalish, M.D., Janeth Yin, M.D., Don L. Goldenberg, M.D., Yoojin Lee, M.S., and Timothy McAlindon, M.D., M.P.H.

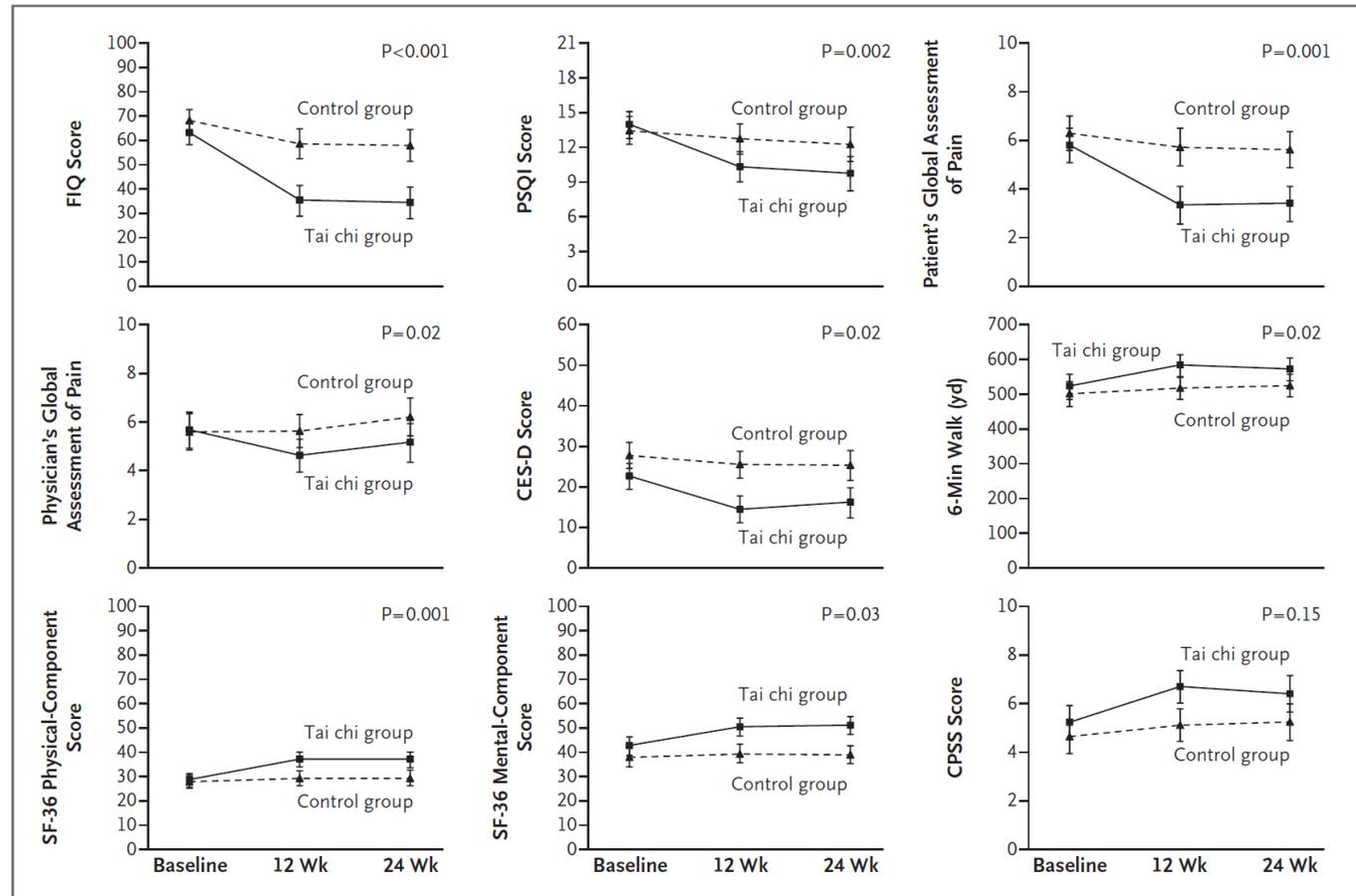


Figure 2. Mean Changes in Nine Secondary Outcomes at 12 and 24 Weeks, According to Treatment Group.

Le rôle du médecin

- Besoin du patient :
 - ▣ Reconnaissance de la douleur/symptôme
 - ▣ Comprendre pourquoi
 - ▣ Aller mieux
- Comment y répondre :
 - ▣ Ne pas méconnaître une autre pathologie
 - ▣ Optimisme mesuré : ne pas promettre l'impossible !!!
 - ▣ **Eviter le nomadisme médical et la surmédicalisation**
 - ▣ Prise en charge globale
 - ▣ Donner les cartes aux patients : à lui de les saisir !

Conclusion

- Syndromes douloureux chroniques : un domaine où la médecine à du travail
- Un bilan étiologique à ne pas méconnaître
- Des techniques diagnostics qui avancent
- Traitement symptomatique nécessitant une prise en charge globale



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Mise au point

La neuropathie des petites fibres

Small fiber neuropathy

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