Associazione Italiana Radioterapia Oncologica

SCHEDA D'ISCRIZIONE
COSENZA, 28 giugno 2013

LA RADIOTERAPIA PALLIATIVA CON TECNICHE SPECIALI DELLA MALATTIA METASTATICA

INFORMAZIONI GENERALI

SEDE
Italiana Hotels
Via Panebianco, 452
87100 Cosenza

ECM
Il Ministero della Salute ha assegnato all'evento n. 6 crediti formativi per la figura professionale di Medico Chirurgo (disciplina Radioterapia e Oncologia), di Fisico, di Infermiere e di Tecnico sanitario di radiologia medica.

COORDINATORE DEL CORSO
Dott. Luigi Marafioti
Direttore Radioterapia
Azienda Ospedaliera di Cosenza
Via S. Martino, 87100 Cosenza

Associazione Italiana Radioterapia Oncologica

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COSENZA
28 giugno 2013

SEGRETERIA ORGANIZZATIVA
Studio E.R. Congressi - Triumph Group
Via Marconi 36 - 40122 Bologna
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Novità nella terapia farmacologica

francesco amato

Treatment of chronic cancer pain
D. Ck , H D Wilson , A. Cahana

***Lancet* 2011; 377: 2226–35**

THE LANCET

Chronic pain is a pervasive problem that affects the patient,their significant others, and society in many ways. The past decade has seen advances in our understanding of the mechanisms under lying pain and in the availability of technically advanced diagnostic procedures; however, **the most notable therapeutic changes** have not been the development of novel evidenced based methods,but rather **changing trends in applications and practices** within the available clinical armamentarium.

We provide a **general overview** of **empirical evidence for the most commonly used interventions in the management of chronic non-cancer pain, including**

pharmacological,interventional,physical, psychological,rehabilitative,and alternative modalities.

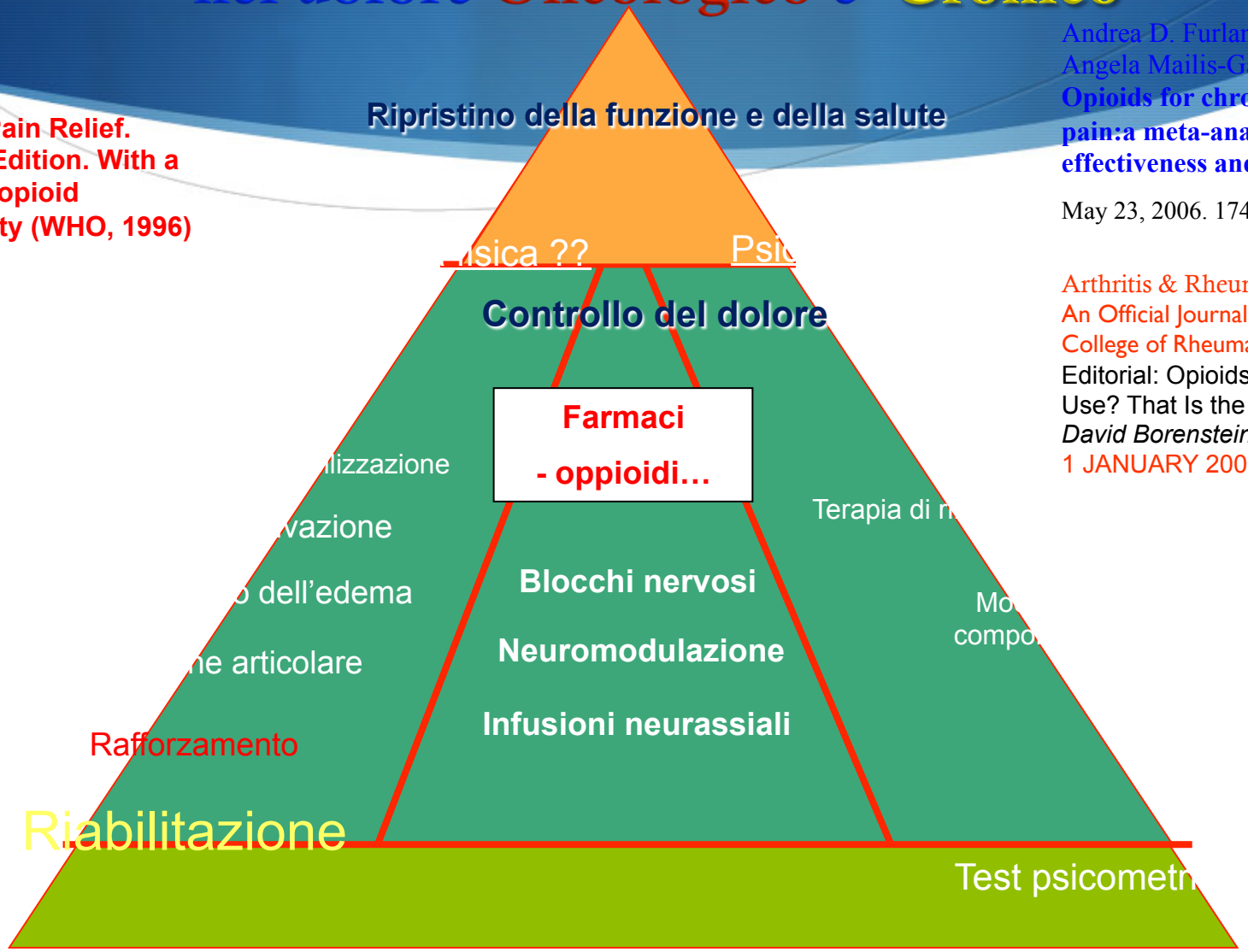
Overall,currently available treatments provide modest improvements in pain and minimum improvements in physical and emotional functioning.

The quality of evidence is mediocre and has not improved substantially during the past **decade.**

There is a crucial need for assessment of combination treatments,identification of indicators of treatment response,and assessment of the benefit of matching of treatments to patient characteristics.

Il nostro Goal Standard :
La gestione esperta dei farmaci oppioidi nel dolore Oncologico e Cronico

Cancer Pain Relief. Second Edition. With a guide to opioid availability (WHO, 1996)



Andrea D. Furlan, Juan A. Sandoval, Angela Mailis-Gagnon, Eldon Tunks
Opioids for chronic noncancer pain:a meta-analysis of effectiveness and side effects

May 23, 2006. 174(11) 1589-1594

Arthritis & Rheumatism
An Official Journal of the American College of Rheumatology
Editorial: Opioids: To Use or Not to Use? That Is the Question
David Borenstein VOLUME 52 NO. 1 JANUARY 2005

Professionisti e Procedure

La Qualità Professionale

Possibilità di più comportamenti professionali di fronte ad uno stesso paziente o ad una stessa patologia

Possibilità di più destini per uno stesso paziente

The development of guidelines for pain management by both national and international organizations validates the imperative for effective pain management

American Pain Society: Principles of Analgesic Use in the Treatment of Acute Pain and Cancer Pain (ed 4) Glenview, IL, American Pain Society, 1999, pp 1-64

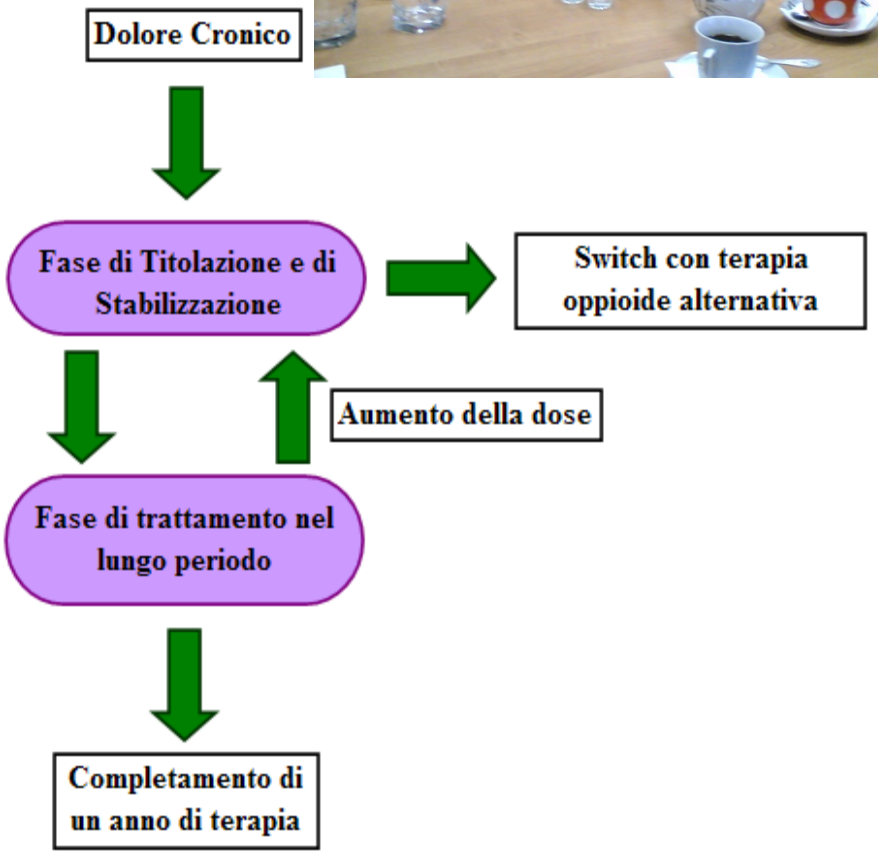
Rolfs RT, Johnson E, Williams NJ, Sundwall DN. J Pain Palliat Care Pharmacother. 2010 Sep;24(3):219-35.PMID: 20718642

Modello Decisionale Oppioidi

Il modello di Cura è diviso in due fasi principali:

1) la fase di titolazione e di stabilizzazione del trattamento (prima fase)

2) la fase di mantenimento del trattamento nel lungo periodo (seconda fase).



Dolore neuropatico

"Il dolore neuropatico è una **condizione morbosa determinata da una lesione o** da una **semplice disfunzione** (delle vie nervose deputate alla trasmissione dell'impulso dolorifico) **del sistema nervoso** centrale o periferico, che possono causare deficit parziali o completi della sensibilità dolorifica"

(*IASP, classification of chronic pain, 1994*)

"pain arising as a direct consequence of a lesion or disease affecting the somatosensory system'
"dolore che nasce quale diretta conseguenza di lesione o malattia del sistema somatosensoriale" (Treede et al. 2008)

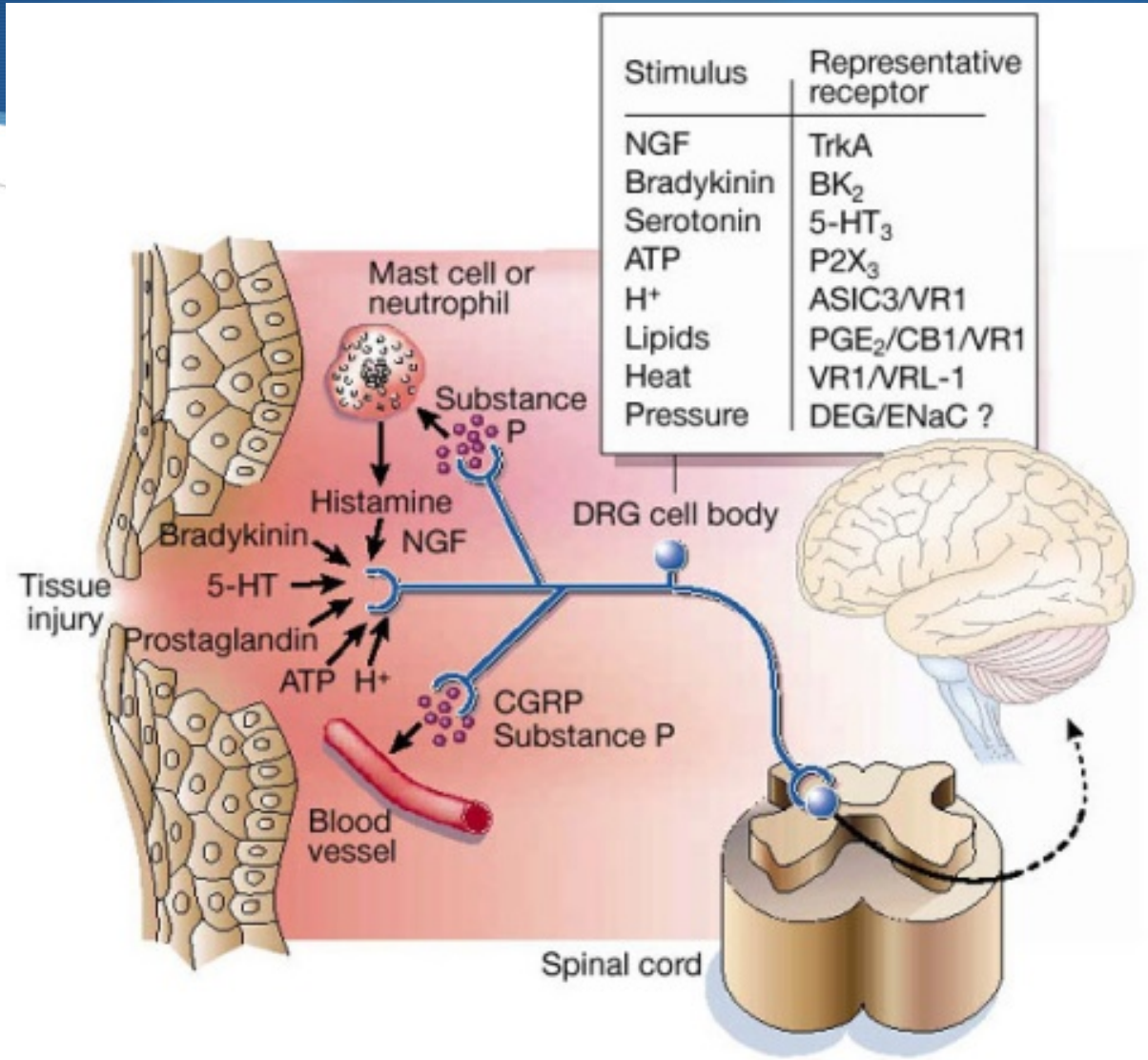
Neuropathic pain is a clinical description (and not a diagnosis) which requires a demonstrable lesion or a disease that satisfies established neurological diagnostic criteria. The term *lesion* is commonly used when diagnostic investigations (e.g. imaging, neurophysiology, biopsies, lab tests) reveal an abnormality or when there was obvious trauma. The term *disease* is commonly used when the underlying cause of the lesion is known (e.g. stroke, vasculitis, diabetes mellitus, genetic abnormality). *Somatosensory* refers to information about the body per se including visceral organs, rather than information about the external world (e.g., vision, hearing, or olfaction). The presence of symptoms or signs (e.g., touch-evoked pain) alone does not justify the use of the term *neuropathic*. Some disease entities, such as trigeminal neuralgia, are currently defined by their clinical presentation rather than by objective diagnostic testing. Other diagnoses such as postherpetic neuralgia are normally based upon the history. It is common when investigating neuropathic pain that diagnostic testing may yield inconclusive or even inconsistent data. In such instances, clinical judgment is required to reduce the totality of findings in a patient into one putative diagnosis or concise group of diagnoses.

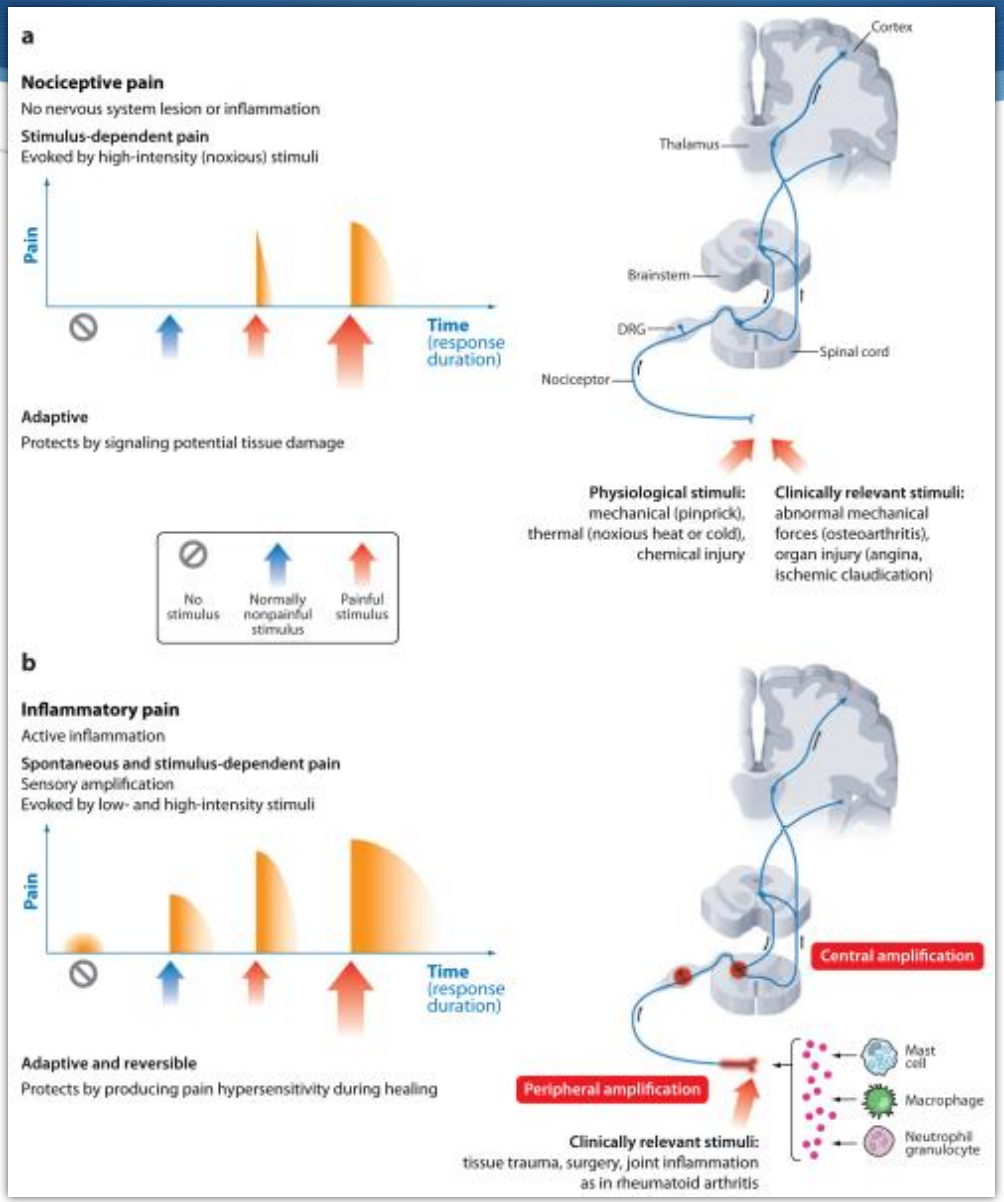
Molecular mechanisms of nociception

Nature 2010-08-14 22:56:57

David Julius1 & Allan I. Basbaum2

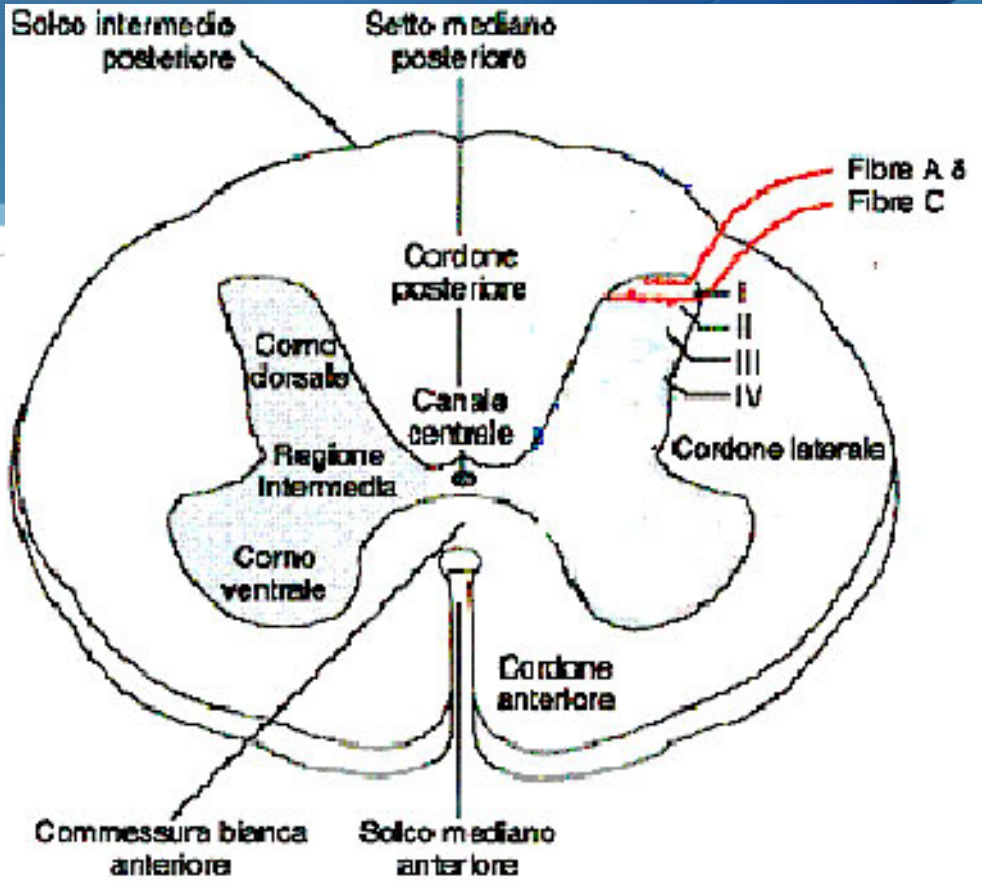
1 Department of Cellular and Molecular Pharmacology, University of California San Francisco, San Francisco, California 94143, USA
2 Departments of Anatomy and Physiology and W. M. Keck Foundation Center for Integrative Neuroscience, University of California San Francisco, San Francisco, California 94143, USA




a
Nociceptive pain
No nervous system lesion or inflammation
Stimulus-dependent pain
Evoked by high-intensity (noxious) stimuli
Pain
Time
(response
duration)
Adaptive
Protects by signaling potential tissue damage
No stimulus
Normally nonpainful stimulus
Painful stimulus
Cortex
Thalamus
Brainstem
DRG
Spinal cord
Nociceptor
Physiological stimuli:
mechanical (pinprick),
thermal (noxious heat or cold),
chemical injury
Clinically relevant stimuli:
abnormal mechanical
forces (osteoarthritis),
organ injury (angina,
ischemic claudication)
b
Inflammatory pain
Active inflammation
Spontaneous and stimulus-dependent pain
Sensory amplification
Evoked by low- and high-intensity stimuli
Pain
Time
(response
duration)
Adaptive and reversible
Protects by producing pain hypersensitivity during healing
Central amplification
Peripheral amplification
Mast cell
Macrophage
Neutrophil granulocyte
Clinically relevant stimuli:
tissue trauma, surgery, joint inflammation
as in rheumatoid arthritis

Midollo spinale

- In sezione trasversale, la sostanza grigia del midollo spinale ha un aspetto “a farfalla”.



- Ciascuna delle due “ali” presenta una zona anteriore (corno ventrale) e una posteriore (corno dorsale).

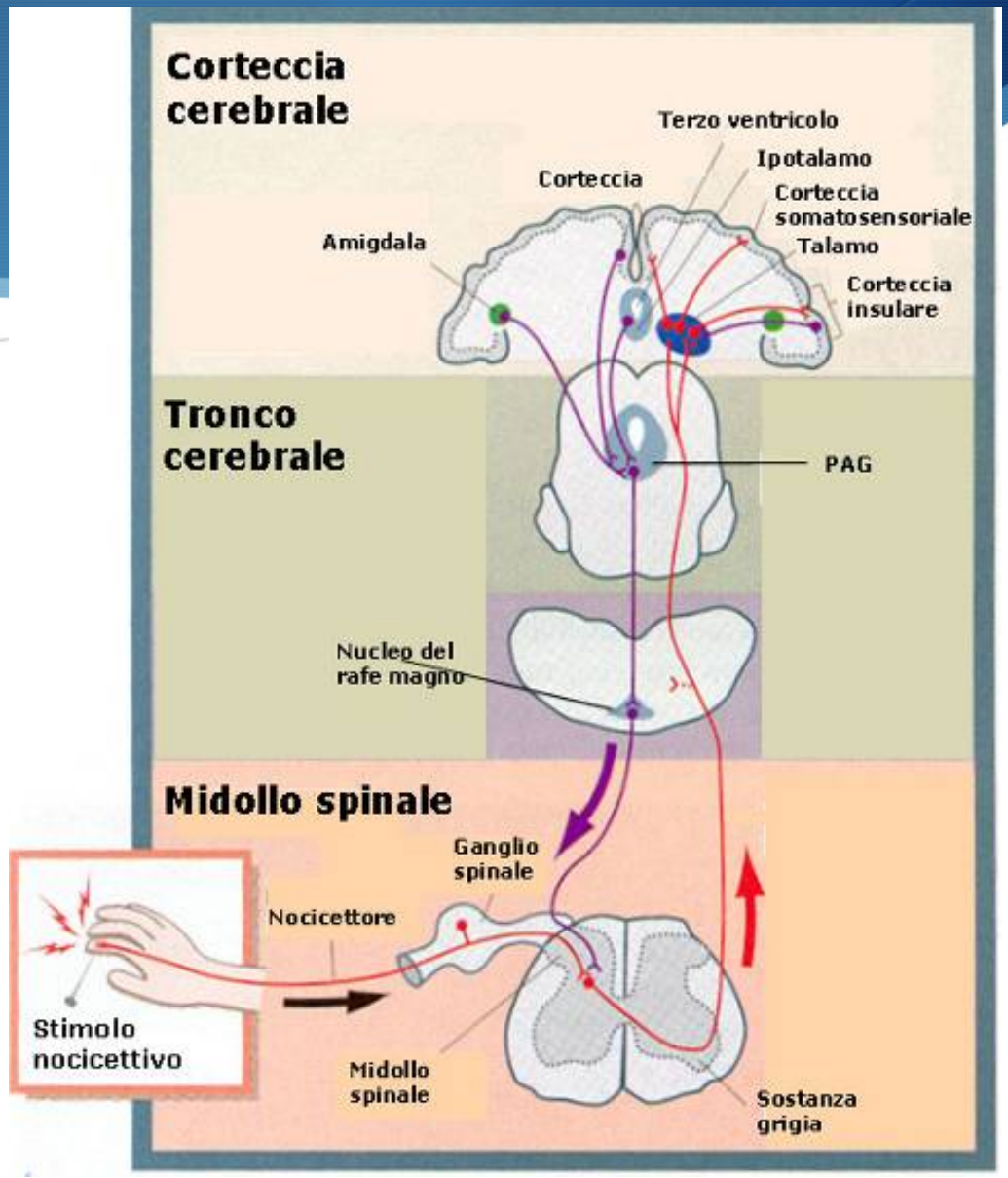
- Le fibre C terminano soprattutto in corrispondenza delle lamine I e II del corno dorsale.

- Le fibre Aδ terminano soprattutto in corrispondenza delle lamine I e V del corno dorsale.

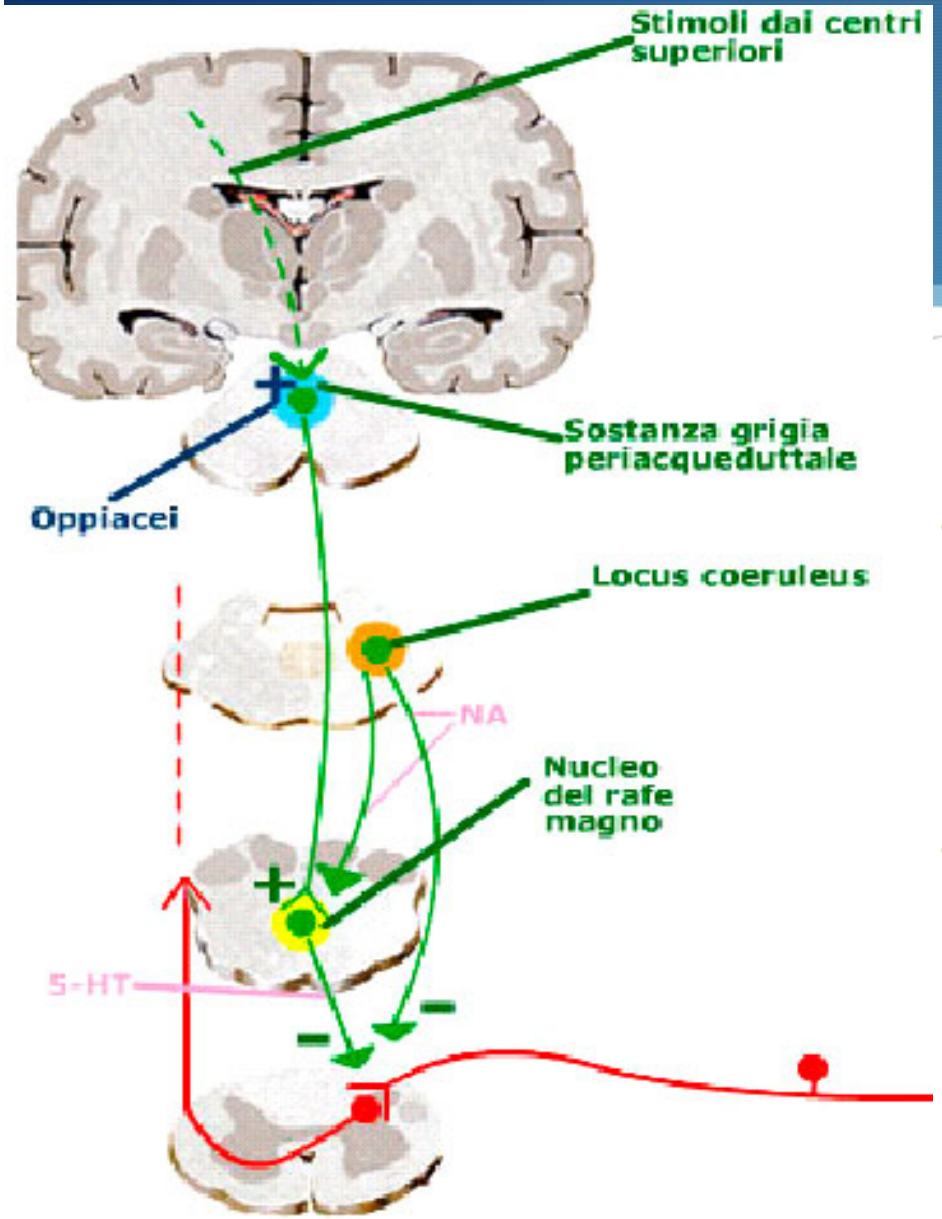
Una visione d'insieme

Lungo il percorso dalla periferia alla corteccia cerebrale (dove avviene la percezione del dolore) il messaggio nocicettivo attraversa tre aree fondamentali:

- il midollo spinale
- il tronco cerebrale
- il talamo



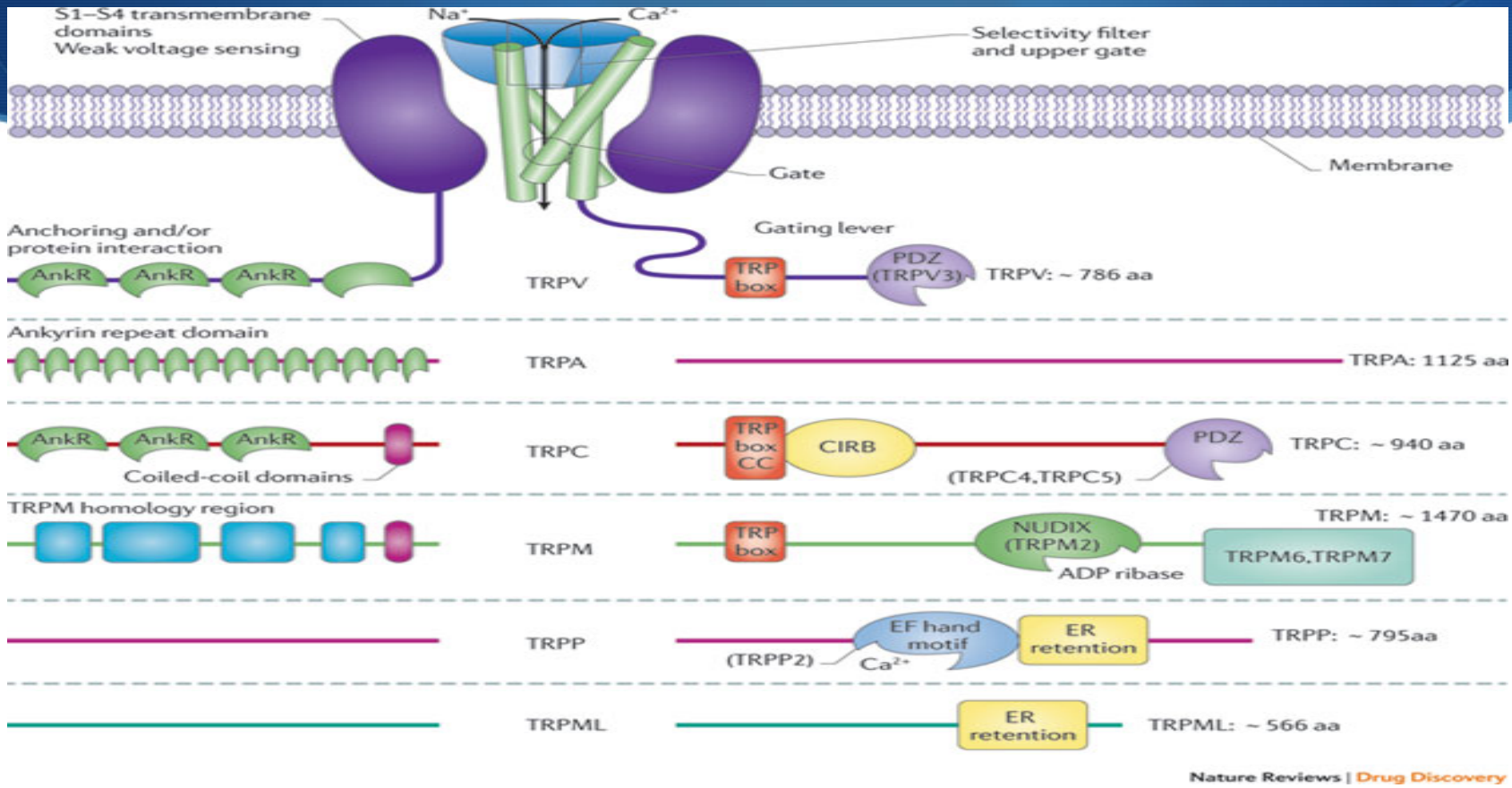
Modulazione discendente



- Le vie inibitorie discendenti partono da alcuni nuclei del tronco cerebrale (locus coeruleus e nucleo del rafe magno) e raggiungono i neuroni spinali.
- I neurotrasmettitori coinvolti sono la noradrenalina (NA) e, soprattutto, la serotonina (5-HT).
- I neuroni serotoninergici del nucleo del rafe magno vengono attivati anche da fibre provenienti dal grigio periacqueduttale (PAG), un'area mesencefalica particolarmente ricca di recettori per gli oppiacei.

Transient receptor potential channels as therapeutic targets

Magdalene M. Moran, Michael Allen McAlexander, Tamás Bíró & Arpad Szallasi
Nature Reviews Drug Discovery **10**, 601-620 August 2011



The six transient receptor potential (TRP) cation families contain very different motifs in their amino and carboxyl termini. The TRP cation channel subfamily V (TRPV), TRP cation channel subfamily A (TRPA) and TRP cation channel subfamily C (TRPC) families have amino terminal ankyrin repeat (AnkR) domains that are not present in other TRP channel subfamilies

Molecular mechanisms of nociception

David Julius1 & Allan I. Basbaum2

Nature 2010-08-14 22:56:57

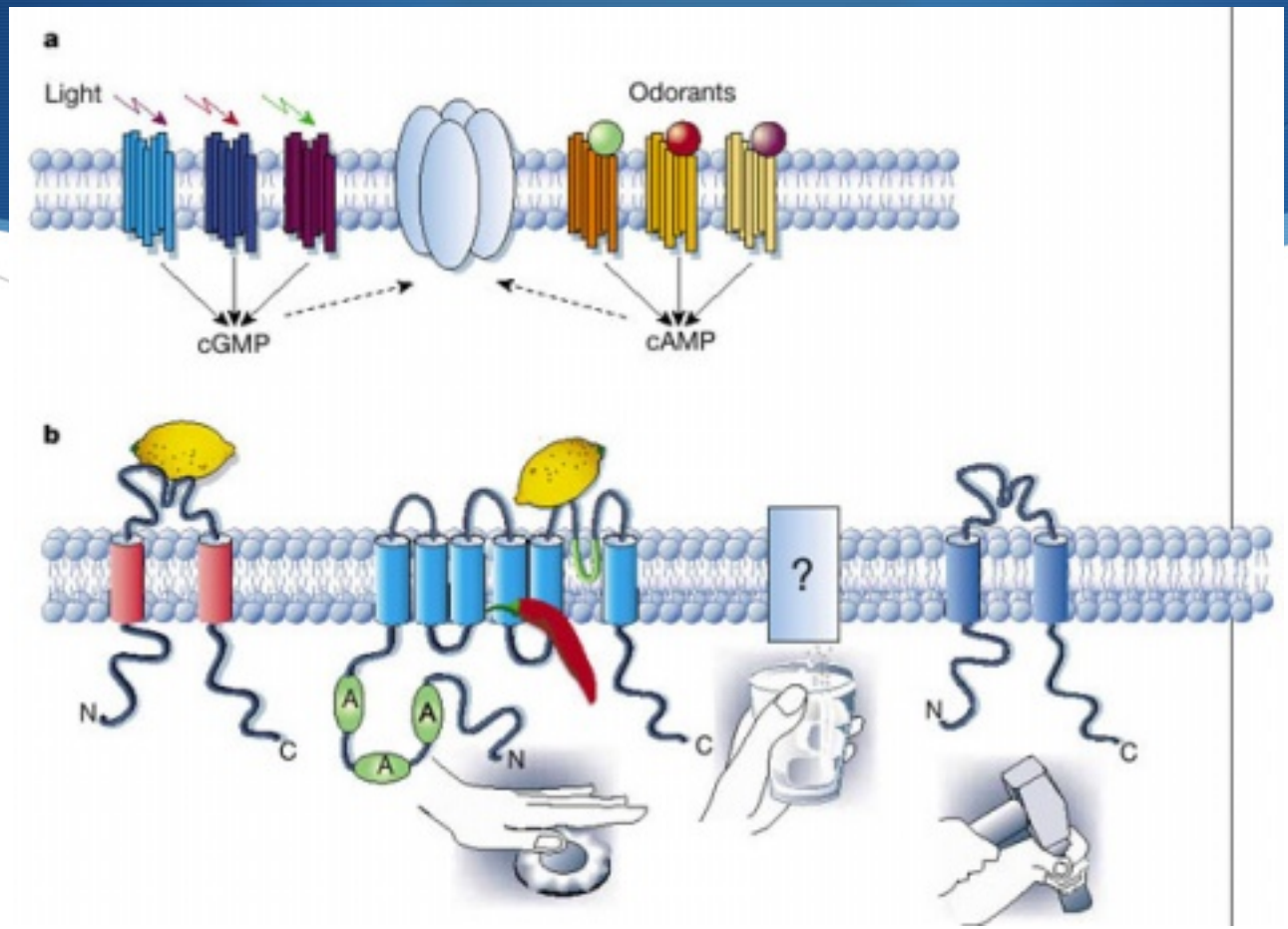
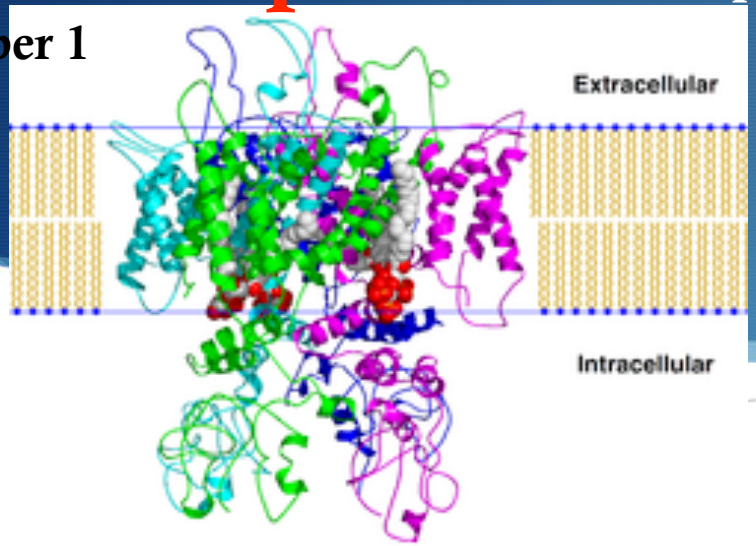
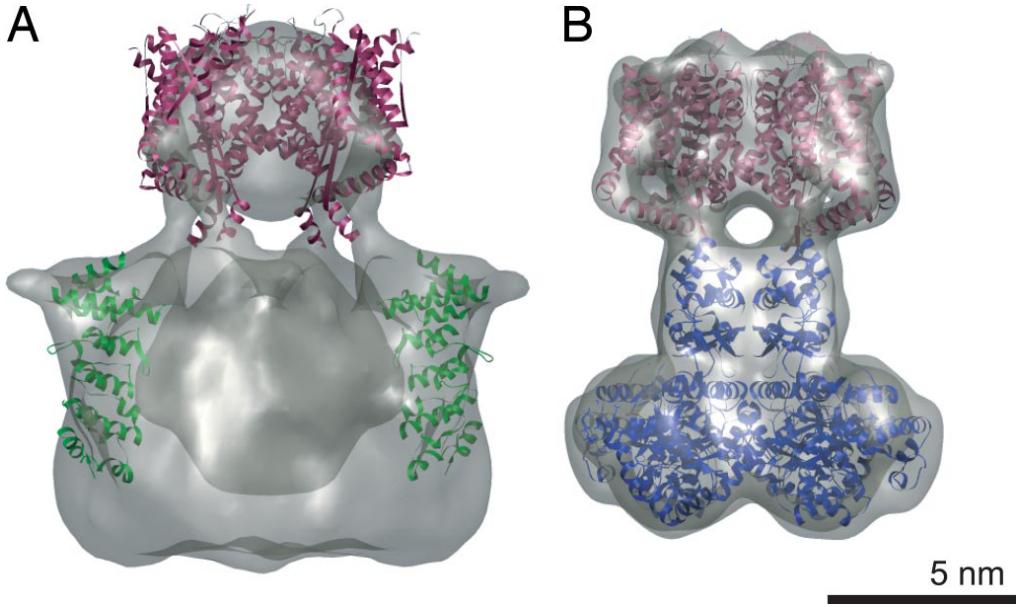


Figure 2: Polymodal nociceptors use a greater diversity of signal-transduction mechanisms to detect physiological stimuli than do primary sensory neurons in other systems. a, In mammals, light or odorants are detected by a convergent signalling pathway in which G-protein-coupled receptors modulate the production of cyclic nucleotide second messengers, which then alter sensory neuron excitability by regulating the activity of a single type of cation channel. b, In contrast, nociceptors use different signal-transduction mechanisms to detect physical and chemical stimuli. Recent studies suggest that TRP-channel family members (VR1 and VRL-1) detect noxious heat, and that ENaC/DEG-channel family detect mechanical stimuli. Molecular transducers for noxious cold remain enigmatic. Noxious chemicals, such as capsaicin or acid (that is, extracellular protons) may be detected through a common transducer (VR1), illustrating aspects of redundancy in nociception. At the same time, a single type of stimulus can interact with multiple detectors, as shown by the ability of extracellular protons to activate not only VR1, but also ASICs, which are also members of the ENaC/DEG-channel family

TrpV1 transient receptor potential cation channel subfamily V member 1

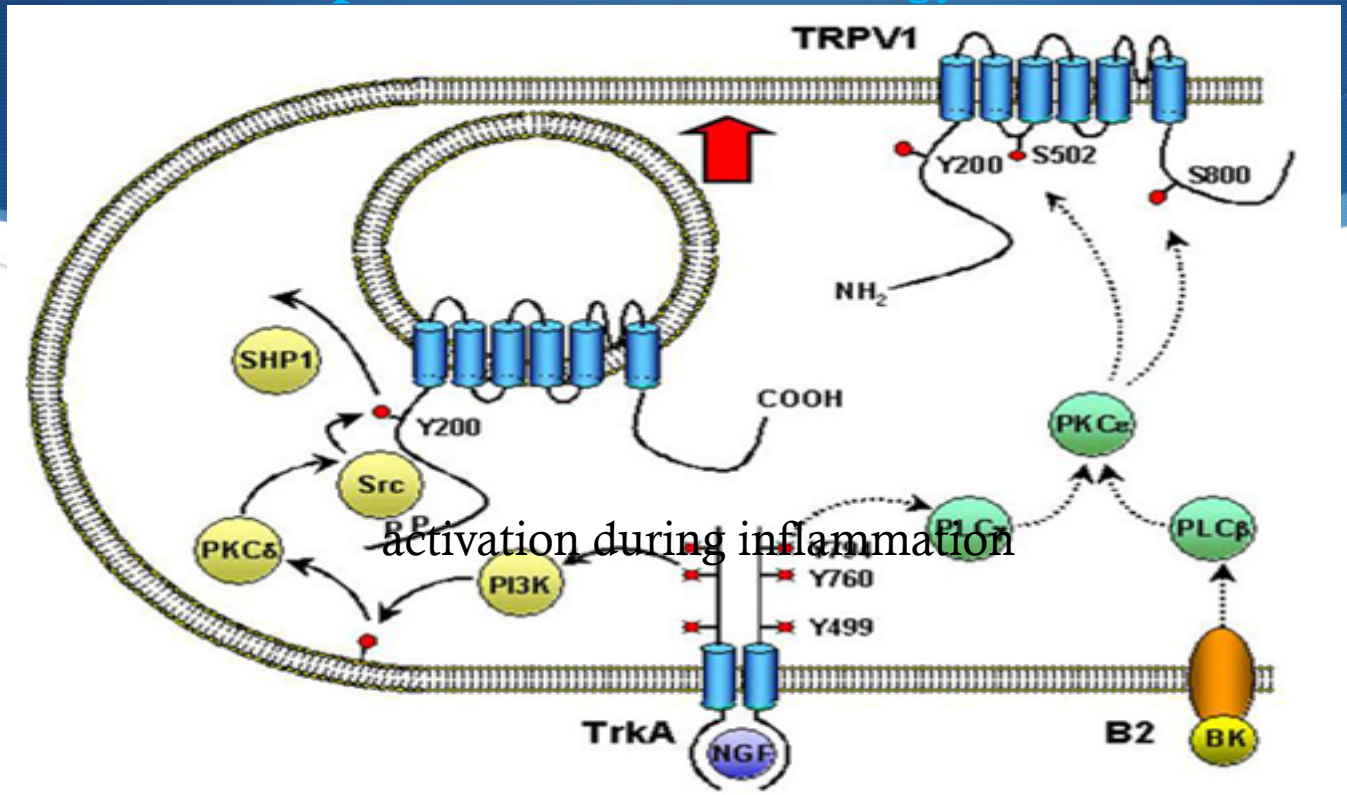




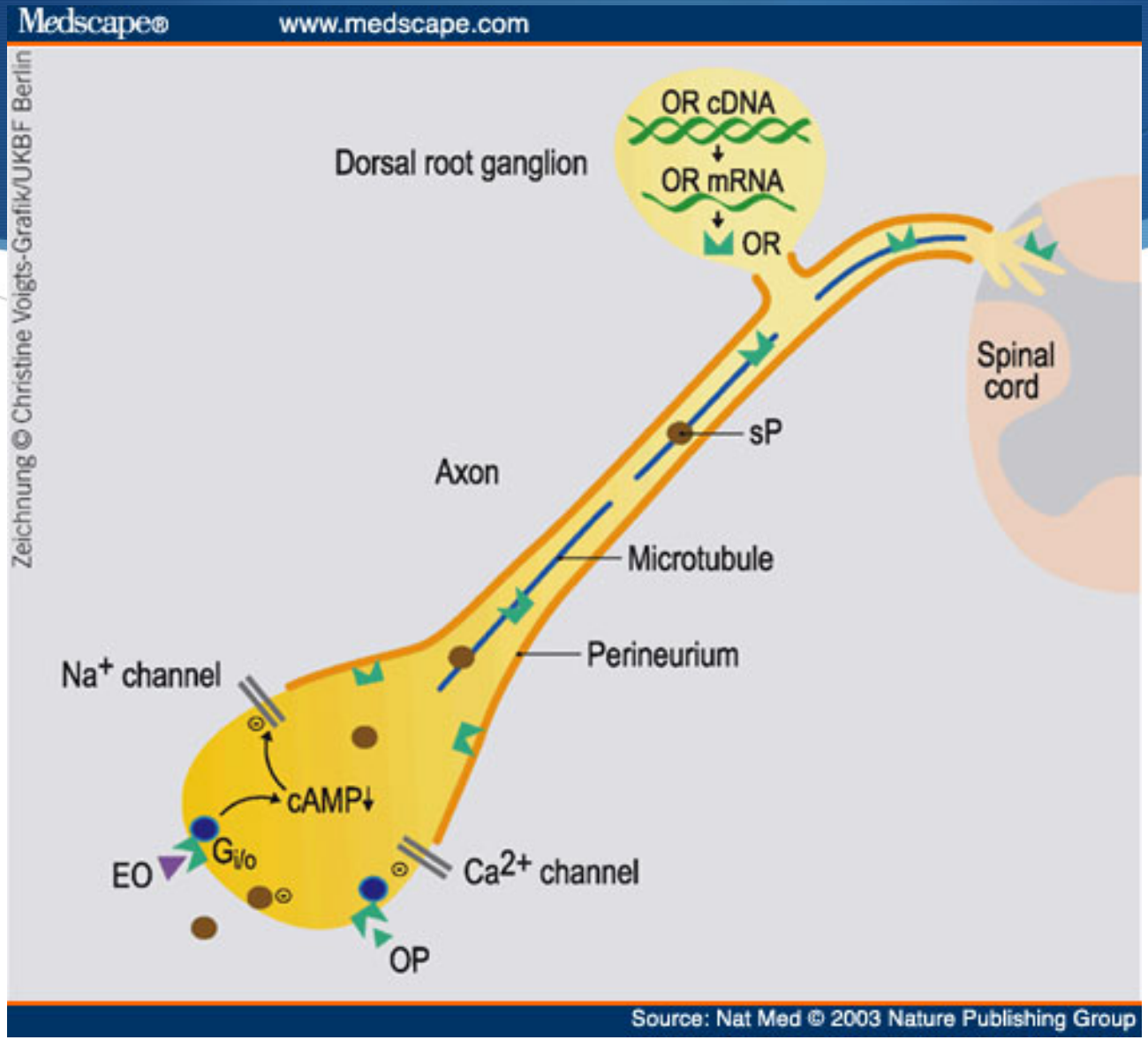
[Some like it hot! Structure of receptor for hot chili pepper and pain revealed]

a bio blog about genetics, genomics, and biotechnology

[Josh Hill]
May 20th, 2008



Schematic diagram of the signaling pathways important in sensitization of TRPV1 by TrkA. Functionally most significant pathway is shown at left (yellow, solid arrows). A smaller component of sensitization following exposure to NGF is mediated by phosphorylation of TRPV1 at residues S502 and S801, probably by the PLC-gamma/PKC-epsilon pathway (green, dashed arrows). PKC-epsilon is a crucial intermediate in sensitization of TRPV1 by bradykinin (pathway shown at lower right of diagram). See Zhang et al, 2005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OR cDNA
OR mRNA
OR
Dorsal root ganglion
Spinal cord
sP
Axon
Microtubule
Perineurium
Na+ channel
cAMP↓
Gi/o
EO
Ca2+ channel
OP
Source: Nat Med © 2003 Nature Publishing Group

Cellular/Molecular

Differential Control of Opioid Antinociception to Thermal Stimuli in a Knock-In Mouse Expressing Regulator of G-Protein Signaling-Insensitive Gαo Protein

Jennifer T. Lamberts1, Chelsea E. Smith1, Ming-Hua Li3, Susan L. Ingram3, Richard R. Neubig1,2, and John R. Traynor1

+ Author Affiliations

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2Center for the Discovery of New Medicines, University of Michigan Medical School, Ann Arbor, Michigan 48109, and
3Department of Neurological Surgery, Oregon Health and Science University, Portland, Oregon 97239

Author contributions: J.T.L. and J.R.T. designed research; J.T.L., C.E.S., M.-H.L., and S.L.I. performed research; R.R.N. contributed unpublished reagents/analytic tools; J.T.L., C.E.S., M.-H.L., S.L.I., and J.R.T. analyzed data; J.T.L. and J.R.T. wrote the paper.

Patogenesi del dolore neuropatico

Table 1 Mechanisms of neuropathic pain

Peripheral mechanisms	Central mechanisms
Changes in sodium channel distribution and expression	Central sensitization
Altered neuropeptide expression	Aβ fibre sprouting into lamina II of the dorsal horn
Sympathetic sprouting	Reduced inhibition of descending pathways
Peripheral sensitization	
Altered peripheral blood flow	
Axonal atrophy, degeneration or regeneration	
Damage to small fibres	
Glycaemic flux	

Adapted with permission from [16].

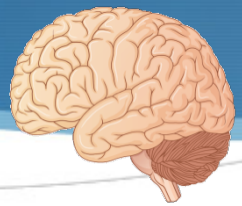
Current Opinion in Supportive and Palliative Care 2009, 3:136–143

Goals of Neuropathic Pain Treatment

- Primary goal: reduction in pain[1,2]
- Secondary goals[1,2]
  - Improvement in physical function
  - Reduction in affective distress
  - Improvement in quality of life
  - Maintenance of positive outcomes
  - Education of patient and providers
- Achieving these goals depends upon[1]
  - Accurate diagnosis of any underlying etiology
  - Preventive treatment of underlying etiology (eg, diabetes and joint inflammation) if possible

DPNP 1. Argoff et al. *Mayo Clin Proc* 2006;81(4 Suppl):S12-25.
2. Turk. *Clinical Journal of Pain* 2000;16:279-80.

Mechanisms of Pain Modulation and Pharmacological Treatments



TCAs, SNRIs, SSRIs
α-adrenergic blocking agents
Opioids
Tramadol
Cannabinoids

Peripheral sensitization

Na^+ channel modulators

- Carbamazepine
- Oxcarbazepine
- TCAs
- Topiramate
- Lamotrigine
- Lidocaine

Central sensitization

Ca^{2+} channel modulators

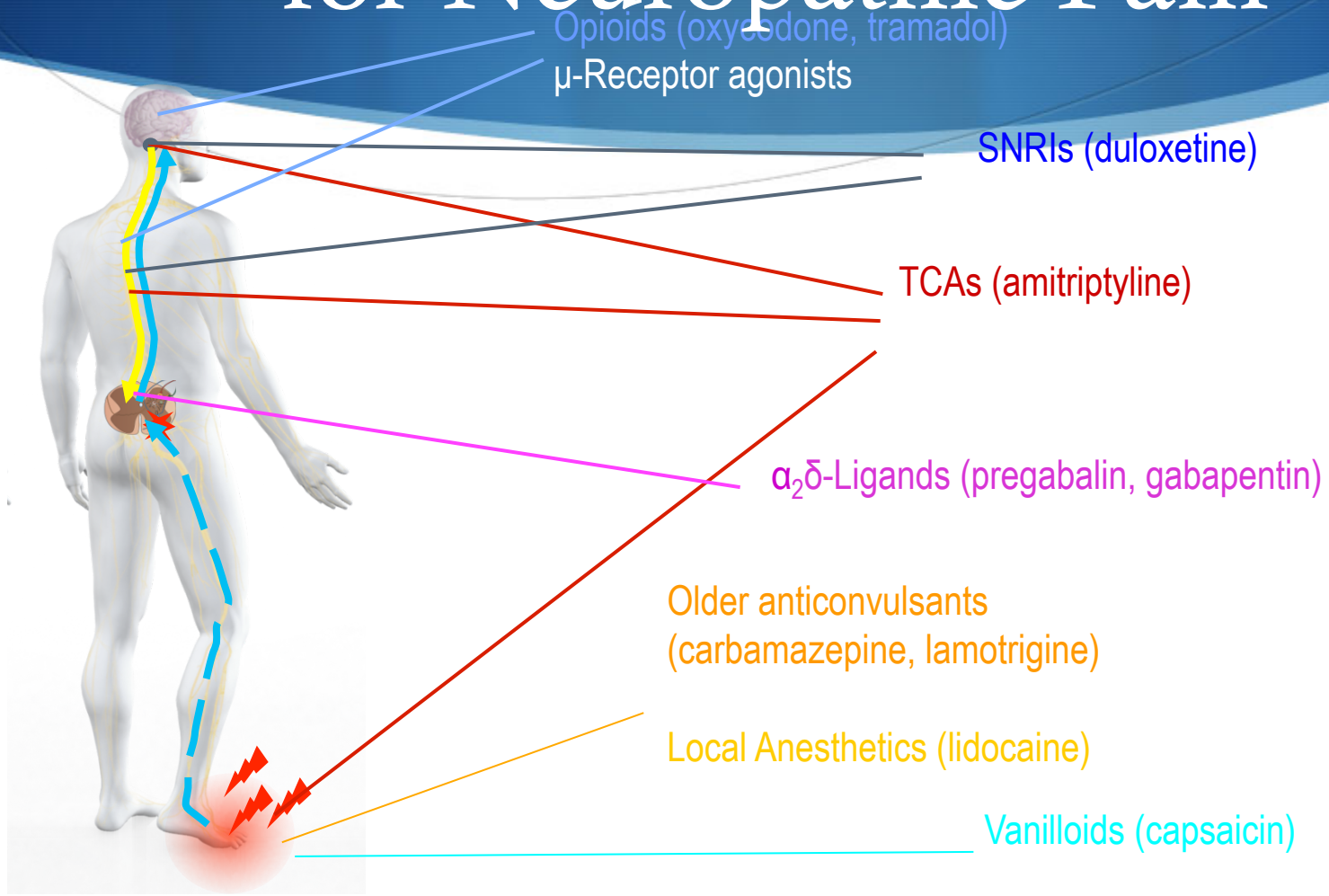
- Gabapentin
- Levetiracetam
- Oxcarbazepine
- Lamotrigine
- Pregabalin

NMDA antagonists

- Ketamine
- Dextromethorphan
- Methadone
- Memantine

DPNP
1. Beydoun et al. *J Pain Symptom Manage* 2003;25(5 Suppl):S18-30.
2. Raskin et al. *Pain Med* 2006;7(5):373-85.
3. Argoff et al. *Mayo Clin Proc* 2006;81(4 Suppl):S12-25.
4. Cole. *Pain Medicine* 2007;8(Suppl 2):S27-S32.

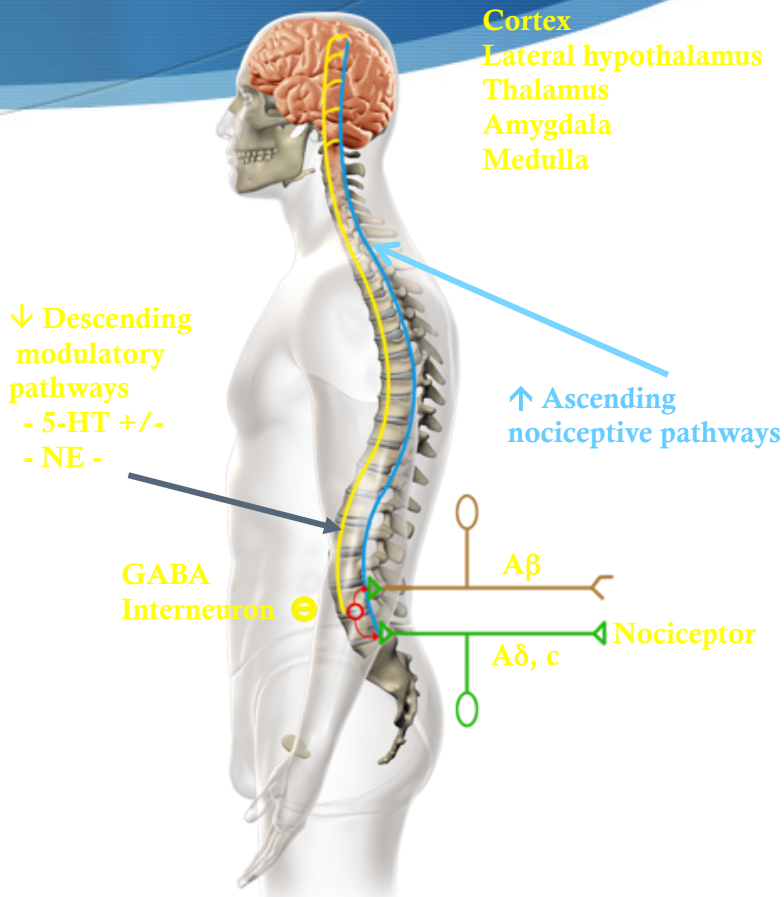
Mechanism-Orientated Therapy for Neuropathic Pain



1. Jensen et al. *Diabetes Vasc Dis Res* 2006;3(2):108-19. 2. Kroenke et al. *Gen Hosp Psychiatry* 2009;31(3):206-19.

Ruolo della serotonina (5-HT) e Noradrenalina (NE) nel Dolore Cronico

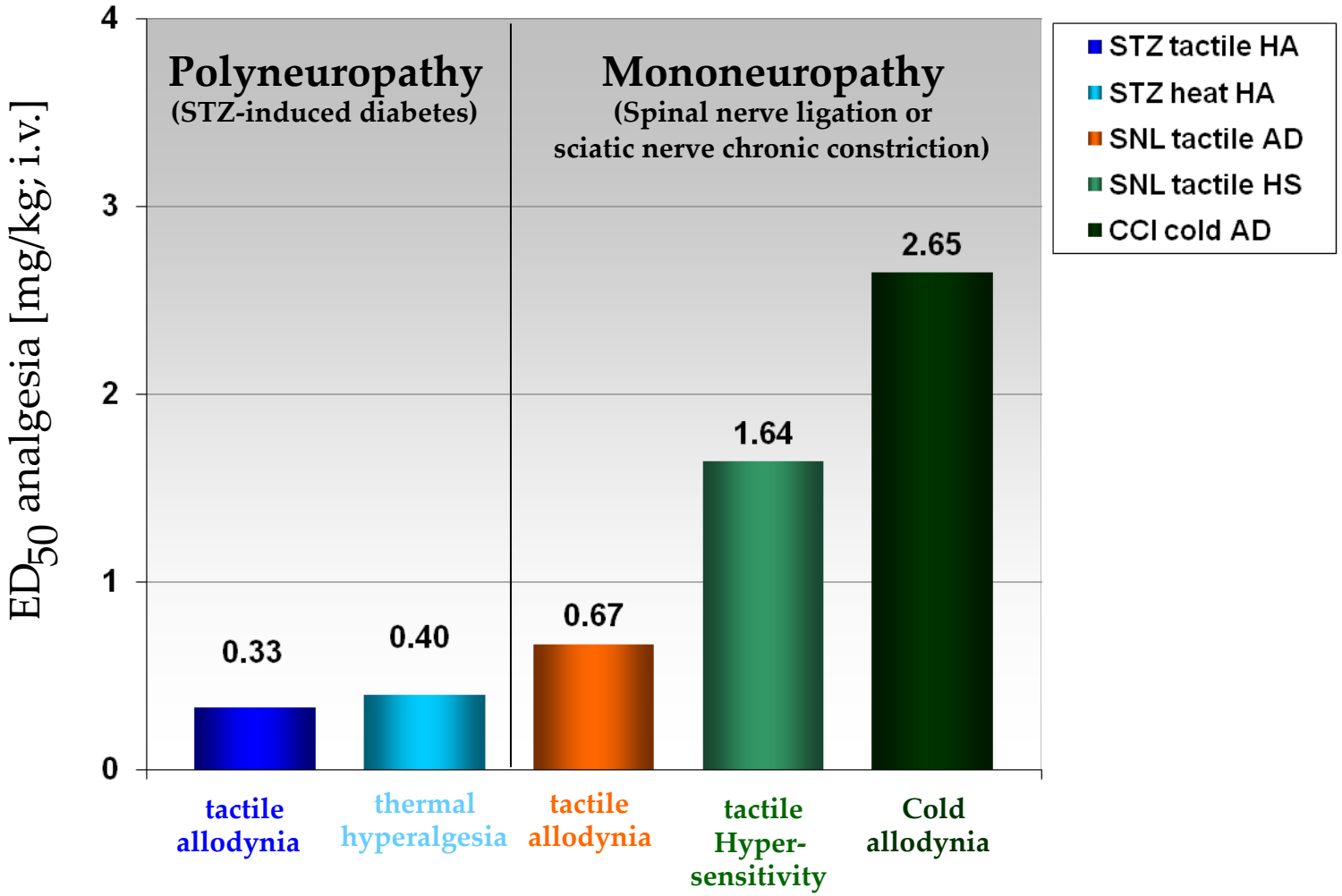
- Via della percezione del dolore[1,2]
  - Via nocicettiva ascendente
  - Vie discendenti modulatorie
- 5-HT e NE: neurotrasmettitori chiave nella modulazione delle vie discendenti inibitorie del dolore[1]
  - Parte del sistema endogeno analgesico del corpo
- Il potenziamento dell'attivita' 5HT e NE si ritiene provochi l'inibizione del dolore[3]



- CNS: central nervous system

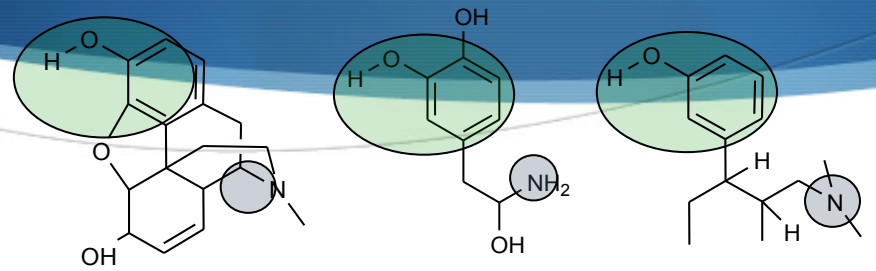
1. Iyengar et al. *J Pharmacol Exp Ther* 2004; 311(2): 576-84.
2. Woolf. *Ann Intern Med* 2004;140(6):441-51.
3. Arnold et al. *Arthritis Res Ther* 2006;8(4):212.

Elevata potenza del tapentadolo nel dolore neuropatico

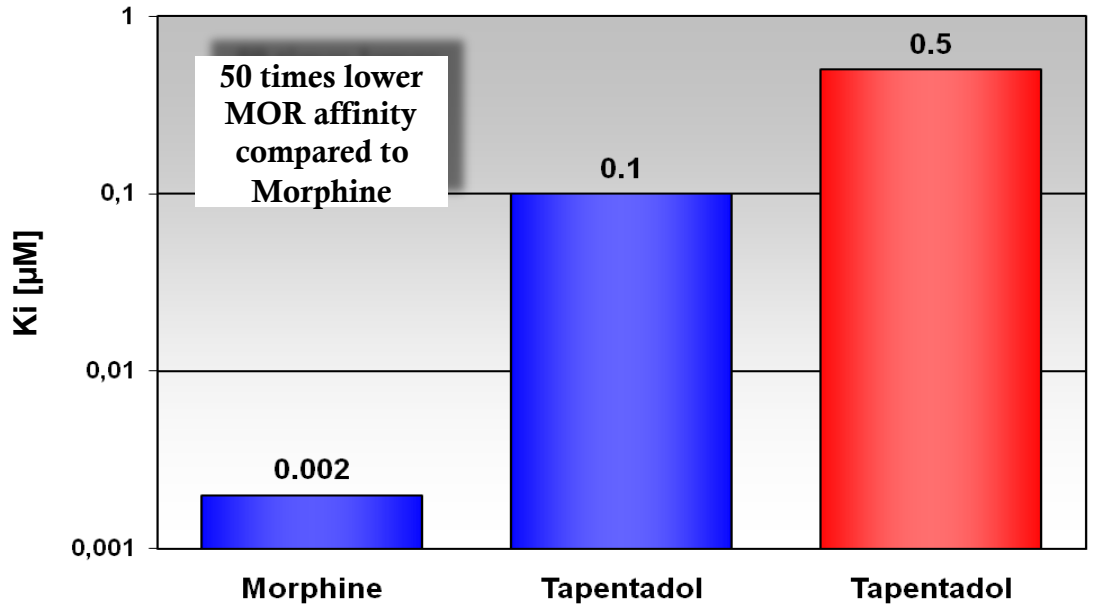


Christoph et al (2010) Neuroscience Lett 470:91-94;

Tapentadolo: *"more than MOR"*

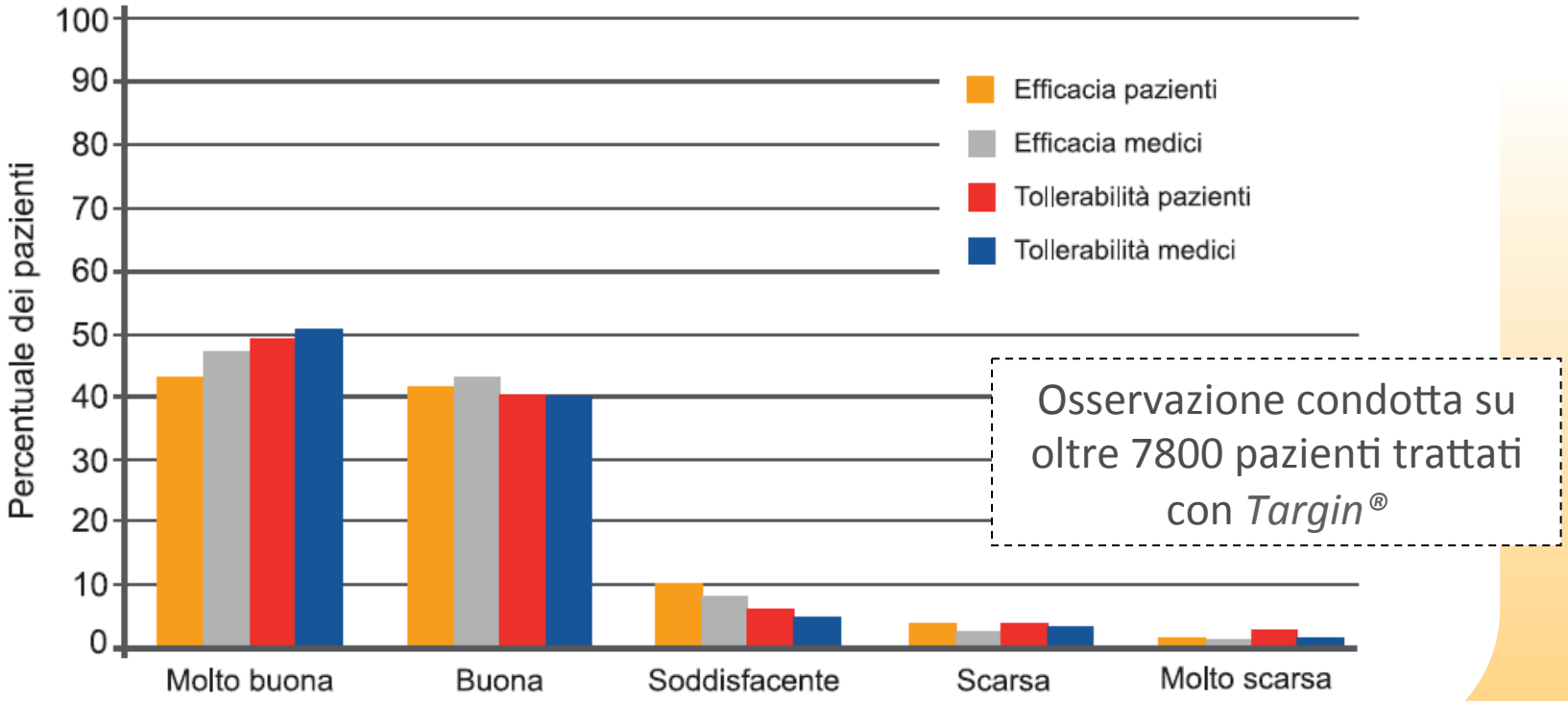


Morfina Noradrenalina Tapentadolo



Tapentadolo: agonista MOR e inibitore della ricaptazione di noradrenalina (NRI)

Medici e pazienti confermano l'elevata efficacia e la tollerabilità di Targin®



Osservazione condotta su oltre 7800 pazienti trattati con *Targin®*

Schutter U, Grunert S, Meyer C, Schmidt T, Nolte T. Innovative pain therapy with a fixed combination of prolonged-release oxycodone/naloxone: a large observational study under conditions of daily practice. Curr Med Res Opin. 2010 Jun;26(6):1377-87.

Molta domande: quali risposte?

- Qual è il miglior farmaco prima linea?
- Quale combinazione di farmaci di prima linea è migliore?
- Quando una risposta è clinicamente significativa per una monoterapia e quanto tempo aspettare per vedere se il farmaco è efficace?
- Switch o combinare?
- Combinazione precoce di farmaci che agiscono con meccanismi diversi vs dosi massime di monoterapia?

Sintesi delle linee guida internazionali

Organizzazione	Raccomandazioni di prima linea
The International Association for the Study of Pain (IASP) 2010	**Nel dolore neuropatico:** Duloxetine, Pregabalin, Gabapentin, Venlafaxine, Nortriptyline, Desipramine, Topical lidocaine
NICE (UK) 2010	**Nel dolore neuropatico:** Pregabalin, **Nel DPNP** Duloxetine
The American Society of Pain Educators (ASPE) 2006	**Nel DPNP:** Duloxetine, Pregabalin, Gabapentin, Venlafaxine, Amitryptiline, Desipramine, Opioids, Topical capsaicin, Topical lidocaine
The European Federation of Neurological Societies (EFNS) 2010	**Nel DPNP:** Duloxetine, Pregabalin, Gabapentin, Venlafaxine, TCAs

Linee guida dell'AAN 2011

Evidence-Based Treatment for DPN[1]

Livello	Raccomandazioni
A	Pregabalin
B	Gabapentin, Duloxetine, Venlafaxine, Sodium valproate, Amitriptyline, Tramadol, Oxycodone, Capsaicin, Dextromethorphan, Morphine sulphate
Non raccomandato	Oxcarbazepine, Lamotrigine, Lacosamide, Clonidine, Pentoxifylline, Mexiletine

Livello A: stabilito come efficace, inefficace o nocive (o come utile / predittivo o non utile / non predittiva) per la condizione indicata nella popolazione specificata sulla base di almeno 2 studi di Classe 1(RCTs)[2]

Livello B: probabilmente efficace, inefficace o nocive (o probabilmente utile / predittivo o non utili / predittiva) per la condizione indicata nella popolazione specificata basata su almeno 1studio di classe 1(RCT)[2]

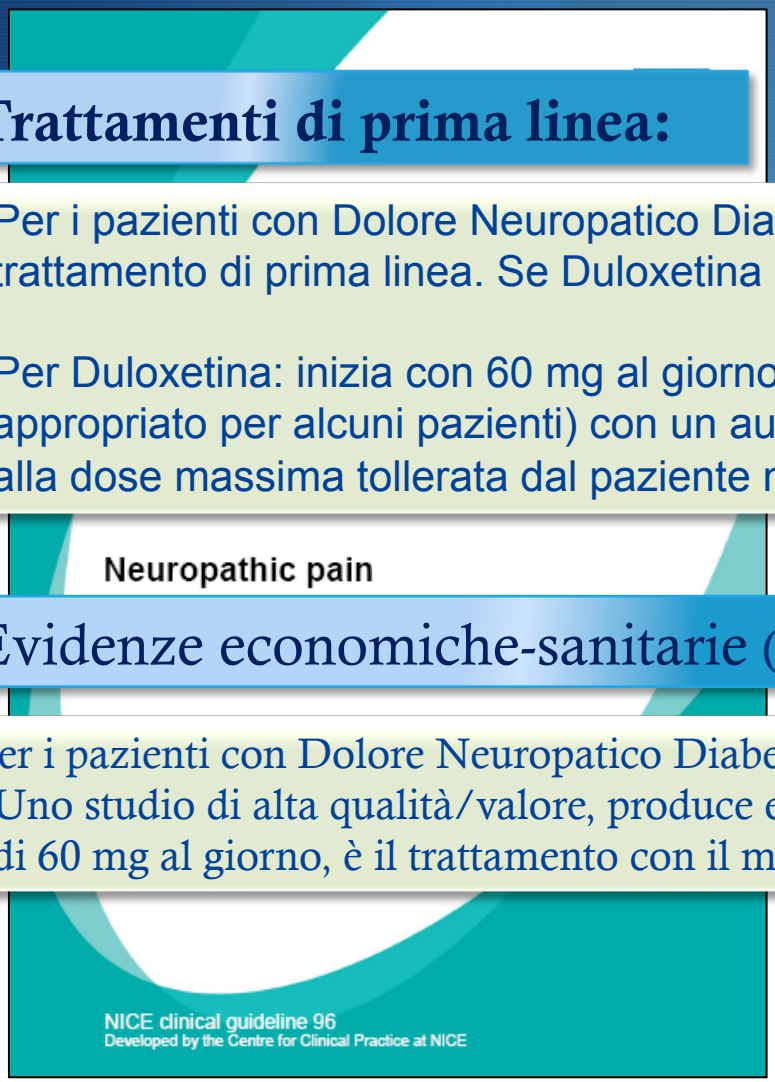
AAN = American Academy of Neurology; DPN = diabetic peripheral neuropathy; RCT = randomized controlled trial

1. Bril et al. *Neurology* 2011;76:1-8
2. AAN Classification of Recommendations. Available at http://www.neurology.org/site/misc/NeurologyFiller.pdf.

LINEE GUIDA NICE
(NATIONAL INSTITUTE FOR HEALTH AND CLINICAL EXCELLENCE)

Trattamenti di prima linea:

- Per i pazienti con Dolore Neuropatico Diabetico, proponi Duloxetina orale come trattamento di prima linea. Se Duloxetina è controindicata, offri amitriptilina*
- Per Duloxetina: inizia con 60 mg al giorno (un più basso dosaggio potrebbe essere appropriato per alcuni pazienti) con un aumento del dosaggio fino alla dose efficace o alla dose massima tollerata dal paziente non superiore ai 120 mg al giorno



Evidenze economiche-sanitarie (rapporto costo-beneficio)

Per i pazienti con Dolore Neuropatico Diabetico
- Uno studio di alta qualità/valore, produce evidenze che Duloxetina, specialmente alla dose di 60 mg al giorno, è il trattamento con il miglior rapporto costo-efficacia

17.NICE Clinical Guideline.
Neuropathic pain.
March 2010.
www.nice.org.uk/guidance/CG96.

Rationale for Combination Therapy

The NEW ENGLAND JOURNAL of MEDICINE

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EDITORIAL

Combination Therapy for Neuropathic Pain — Which Drugs, Which Combination, Which Patients?

Srinivasa N. Raja, M.D., and Jennifer A. Haythornthwaite, Ph.D.

N Engl J Med 2005; 352:1373-1375 | March 31, 2005

Rationale for Combination Therapy

The NEW ENGLAND JOURNAL of MEDICINE

N ENGL J MED 352;13 WWW.NEJM.ORG MARCH 31, 2005

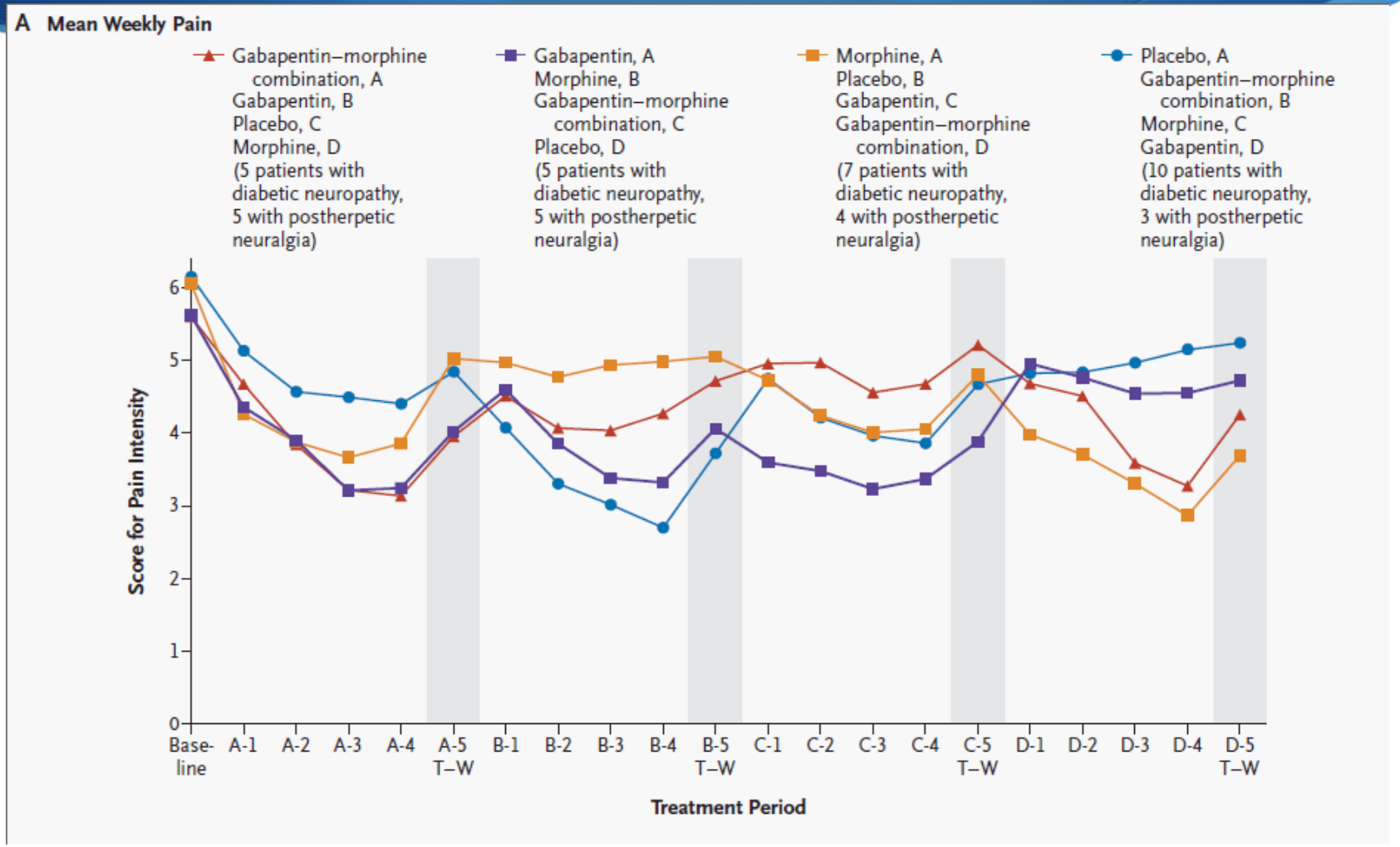
ORIGINAL ARTICLE

Morphine, Gabapentin, or Their Combination for Neuropathic Pain

Ian Gilron, M.D., Joan M. Bailey, R.N., M.Ed., Dongsheng Tu, Ph.D.,
Ronald R. Holden, Ph.D., Donald F. Weaver, M.D., Ph.D.,
and Robyn L. Houlden, M.D.

Rationale for Combination Therapy

Randomized, double-blind, active placebo-controlled, four-period crossover trial.
35 patients with diabetic neuropathy, 22 with postherpetic neuralgia, 41 completers.



Gilron, I. et al.; N ENGL J MED; 352;13 March 31, 2005

Rationale for Combination Therapy

Nortriptyline and gabapentin, alone and in combination for neuropathic pain: a double-blind, randomised controlled crossover trial

Ian Gilron, Joan M Bailey, Dongsheng Tu, Ronald R Holden, Alan C Jackson, Robyn L Houlden

Lancet. 2009 Oct 10;374(9697):1252-61.

International Association for the Study of Pain
IASP
Working together for pain relief
PAIN CLINICAL UPDATES
Vol. XVIII, Issue 9 November 2010

Pharmacological Management of Neuropathic Pain

The management of patients with chronic neuropathic pain is challenging,[4–8] despite several attempts to develop a more rational therapeutic approach.[8,9] Most studies have been performed in postherpetic neuralgia (PHN) and painful diabetic neuropathy (PDN). These trials mainly studied the effects of monotherapy and were placebo controlled. Outcome measures were generally restricted to a global assessment of pain by

The management of patients with chronic neuropathic pain is challenging, despite several attempts to develop a more rational therapeutic approach

the patient, and the quality of pain was seldom taken into account. However, newer studies have appeared that may allow us to revise this statement. Thus, studies have recently been performed in indications that were previously neglected, such as central pain and painful radiculopathies; combination studies and head-to-head comparative studies have appeared; and finally, a comprehensive assessment of patients, including the quality of their pain, is increasingly being performed in clinical trials. This issue of *Pain: Clinical Updates* will address new developments in the therapeutic management of neuropathic pain.

Raccomandazioni per Gestione dei Farmaci Oppioidi nell' uso prolungato

Supporto a Medici non Specialisti della Terapia del dolore

Uso Cronico Oppioidi a basse / Alte Dosi

Gestione della sospensione acuta di oppioide

Gestione dello stato morboso da sovradosaggio

Gestione della Rotazione oppioide

Utente nata nel 1919

Sicurezza

Interazioni Biologiche

Ricoveri programmati per Prevenzione

Dipendenza- Tolleranza

Abbattere i pregiudizi sugli oppioidi

Sono associati a tolleranza dell' effetto analgesico?

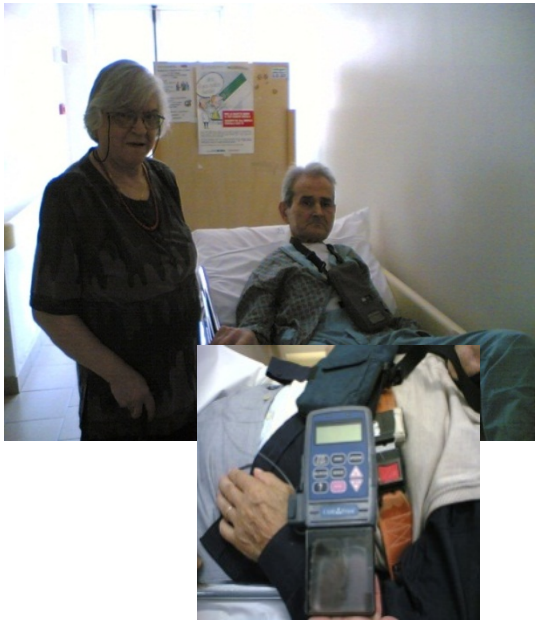
Tolleranza o assuefazione.

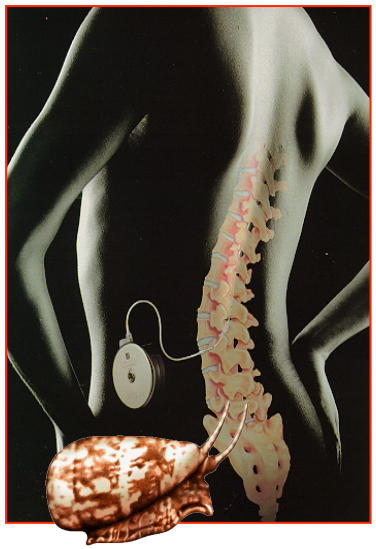
È il processo di adattamento dell'organismo a un farmaco, per cui per ottenere il medesimo effetto nel tempo è necessario aumentarne la dose.

- Tennant et al: survey di **12 anni su 52 pazienti**; adeguata analgesia 88%, parziale analgesia 12%, **nessun incremento di dose**
- Zenz et al: 100 pazienti monitorati per 6 mesi; 51 pazienti con buon controllo del dolore e 28 con parziale controllo del dolore; stabilizzazione della dose per la maggioranza dei pazienti e per alcuni anche riduzione
- Roth et al: pazienti con **severa osteoartrosi monitorati per 18 mesi;** maggioranza dei pazienti con **stabile controllo del dolore a dose stabile di una formulazione a rilascio controllato**

Cicala R, 2004

Divulgare le numerose potenzialità e sicurezza delle cure





Deer T, et al. Polyanalgesic Consensus Conference 2007: Recommendations for the Management of Pain by Intrathecal (Intraspinal) Drug Delivery: Report of an Interdisciplinary Expert Panel. *Neuromodulation*. 2007;10(4):300-328

Kress HG, Varassi G. Intrathecal therapy: what has changed with the introduction of ziconotide. *Pain practice*. 2009;5:338-347

Raffaeli W. Et al : Implantable intrathecal pumps for the treatment of No-Cancer Chronic Pain in elderly population: drug dose and clinical efficacy. Neuromodulation 32- 36; 2008

Raffaeli W, et al Italian Ziconotide Group. Italian registry on long-term intrathecal ziconotide treatment. *Pain Physician*. Jan;14(1):15-24. 2011

I Registri Oppioidi FEDERDOLORE/ISAL

- 2004 – **costruzione del registro** fase pilota: organizzazione e strutturazione dei file report

2007-08

CAMPAGNA per la FACILITAZIONE dell' USO degli OPPIOIDI nel DOLORE ONCOLOGICO

678 PTs trattati

Progetto FEDERDOLORE/ISAL
coordinato da W. Raffaeli

DOLORE CRONICO NON DA CANCRO

Registro Italiano Buon Uso degli Oppioidi

860 Pts trattati 680 utenti

per 6 mesi : beneficio > 70 %

W. Raffaeli, C. Bonezzi, et al **Analisi di sicurezza ed efficacia della Buprenorfina Transdermica nel Dolore Cronico non da Cancro**. Giornale Italiano di Terapia del Dolore e Cure Palliative n. 01 maggio 2008, 30-44.

W. Raffaeli, C. Bonezzi, et al . **Analisi di sicurezza ed efficacia della Buprenorfina Transdermica nel Dolore Oncologico**. Giornale Italiano di Terapia del Dolore e Cure Palliative 01 ottobre 2008: 22-32

Clinical Governance

La Gestione dei Rischi

Farmaci Oppioidi ad Alte Dosi

Età

Alterazioni Omeostasi

Neuro-endocrina

Opioids use in Chronic non–cancer-related pain (CNCP)

their use to ameliorate CNCP is still controversial because of the side effects of opioids,

the physical tolerance they build up (with the related withdrawal reactions and possibility of addiction) and anxiety over disapproval by regulatory bodies

Collett BJ. Chronic opioid therapy for non-cancer pain.

Br J Anaesth 2001;87:133-43.

Quale Monitoraggio Clinico-Laboratoristico - Sociale

CHANGES AFTER LAW 38/2010 IN ITALY

Articolo 7

Legge N.38 del 15/03/2010

(Obbligo di riportare la rilevazione del dolore all' interno della cartella clinica)

"Considerando una scala da 0 a 10 in cui a 0 corrisponde l' assenza di dolore e a 10 il massimo di dolore immaginabile, quanto valuta l' intensità del suo dolore?"

Divinum opus est sedare dolorem

Sin dai tempi remoti, come ricorda il riportato frammento – da taluni attribuito ad Ippocrate, da tal'altri a Galeno –, la scienza medica ha individuato uno dei suoi più alti fini nella sedazione del dolore.

…… grazie per l'attenzione!!!!

Thank you