23e CONGRÈS NATIONAL DE LA SFETD
SOCIÉTÉ FRANÇAISE D'ÉTUDE ET DE TRAITEMENT DE LA DOULEUR
PRÉSENTIEL ET VIRTUEL

Eskétamine, clé du récepteur NMDA

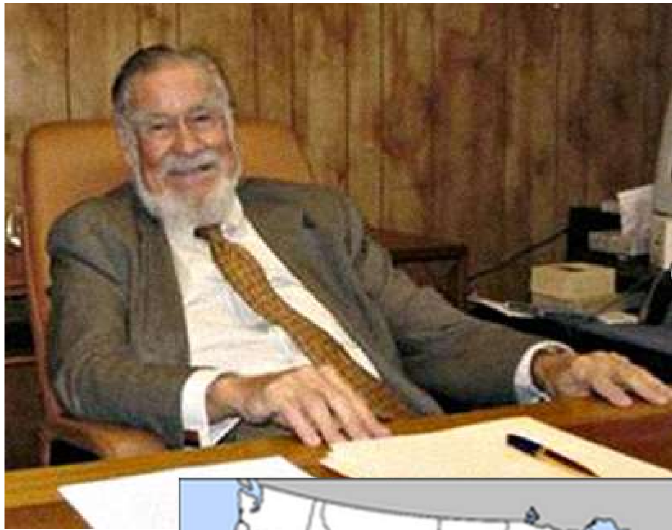
Georges Mion
Hôpital Cochin - Paris

22-24 NOVEMBRE 2023
St-Malo Grand Large
congres-sfetd.fr

Georges Mion est consultant pour International Drug Development (IDD)

EXPLOITATION ET STRATÉGIE RÉGLEMENTAIRE

Notre cœur de métier : le dossier d'AMM



Calvin Stevens (1923-2014)

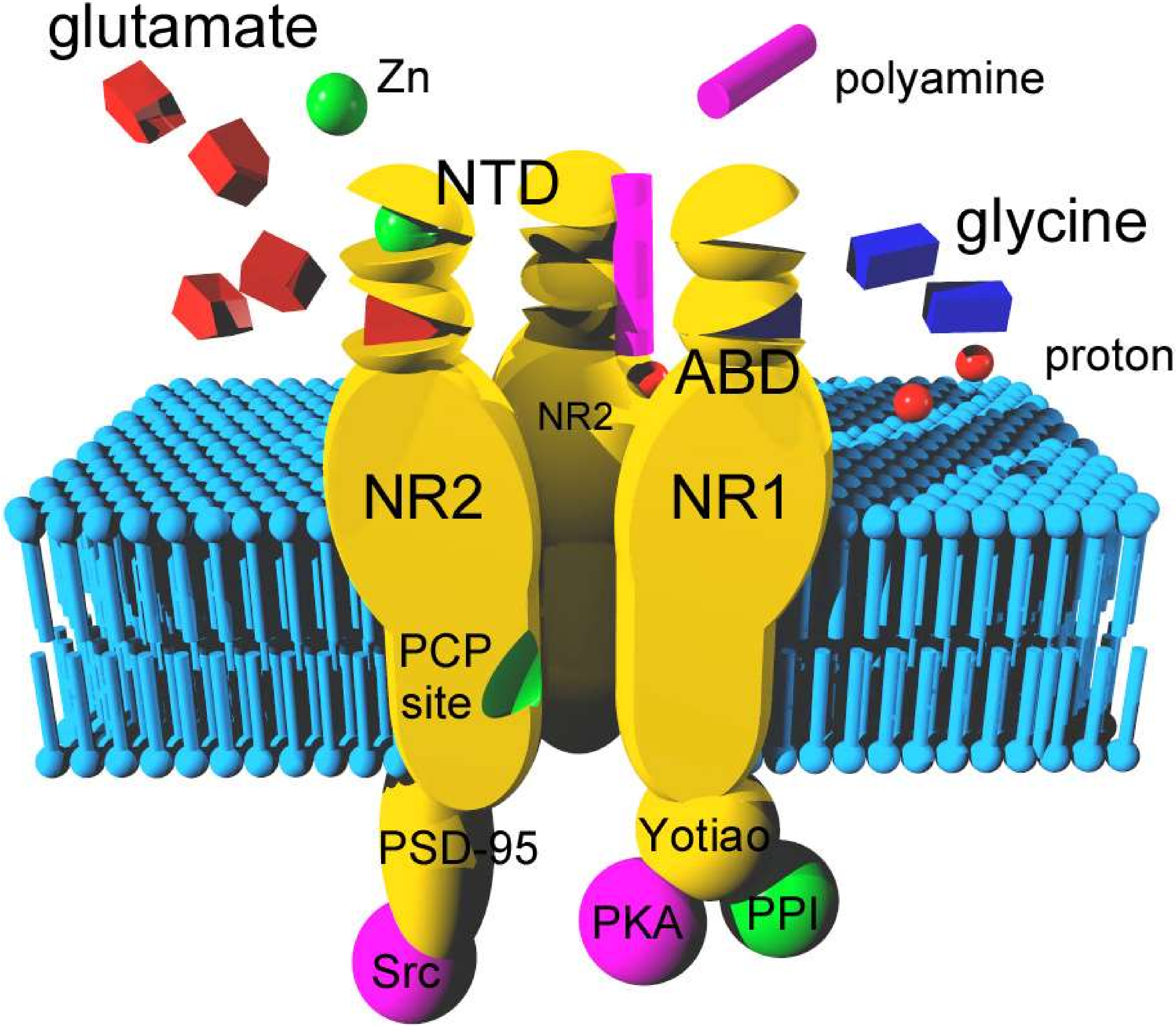
professeur de chimie à l'université de Wayne (Michigan)

synthétise le CI-581

aux laboratoires Parke Devis

Pharmacodynamie

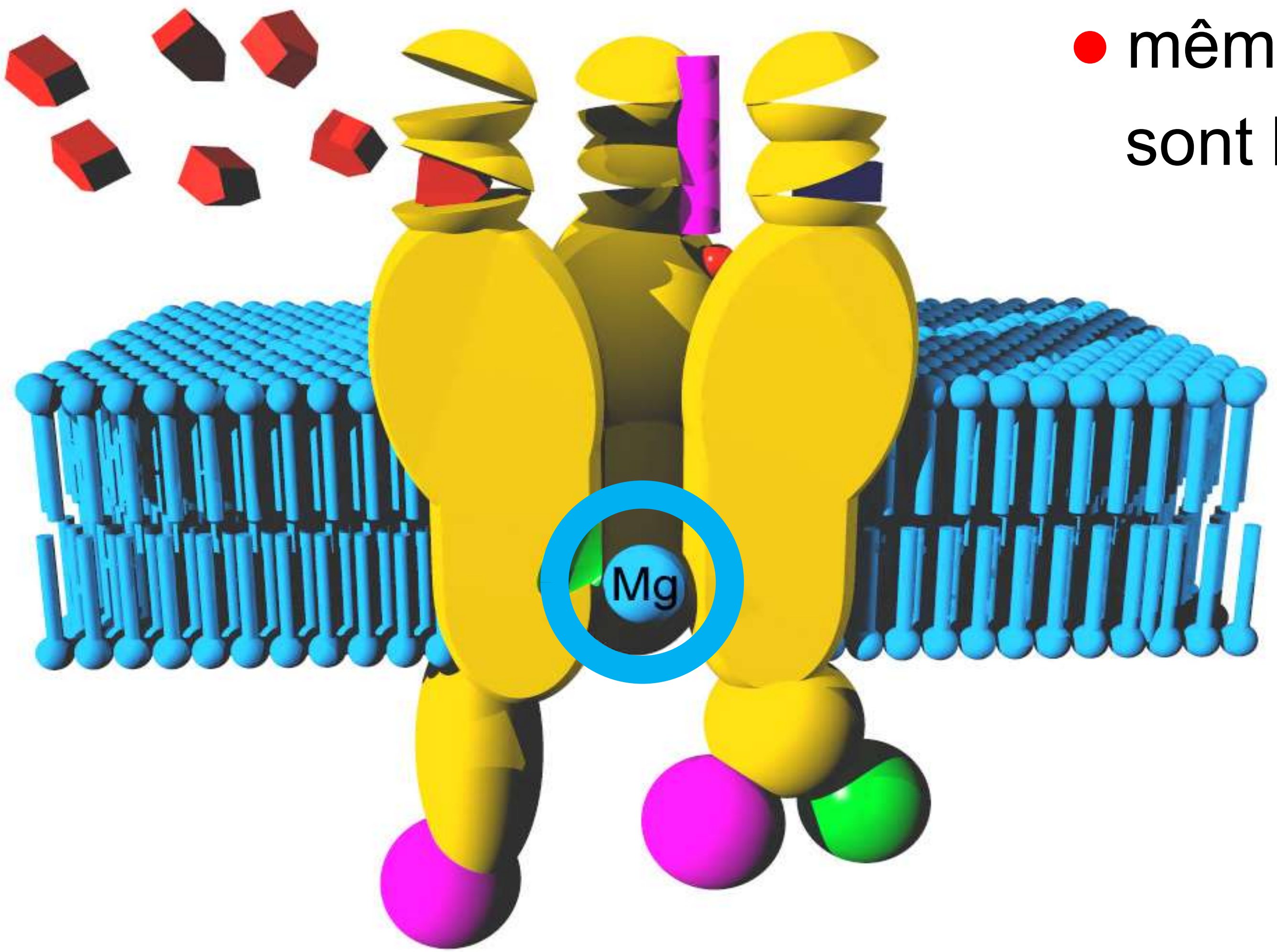
- **Inhibition** non compétitive **NMDA-R**
- **Inhibition** des canaux **HCN1**
- **Effet anticholinergique central** (SAC)
- **Anticalcique** (dilatation bronches et vaisseaux)
- **Activation syst. monoaminergiques desc.**
- **Effet anesthésique local** (canaux sodiques)
- **Effets anti-proinflammatoires**
- **Effets antidépresseurs** (facteurs neurotrophiques)
- **. . .**


glutamate
Zn
polyamine
NTD
glycine
proton
ABD
NR2
NR2
NR1
PCP
site
PSD-95
Yotiao
PKA
PPI
Src

Bloc magnésien *voltage-dépendant*

- quand la membrane est polarisée
  les ions Mg^{2+} obstruent le canal

- même si les co-agonistes
  sont liés au récepteur



Temporal dynamics of the pharmacological MRI response to subanaesthetic ketamine in healthy volunteers: A simultaneous EEG/fMRI study

Journal of Psychopharmacology
1–11 2019

Rebecca McMillan[1], Anna Forsyth[1], Doug Campbell[2], Gemma Malpas[2], Elizabeth Maxwell[2], Juergen Dukart[3,4,5], Joerg F Hipp[3] and Suresh Muthukumaraswamy[1]

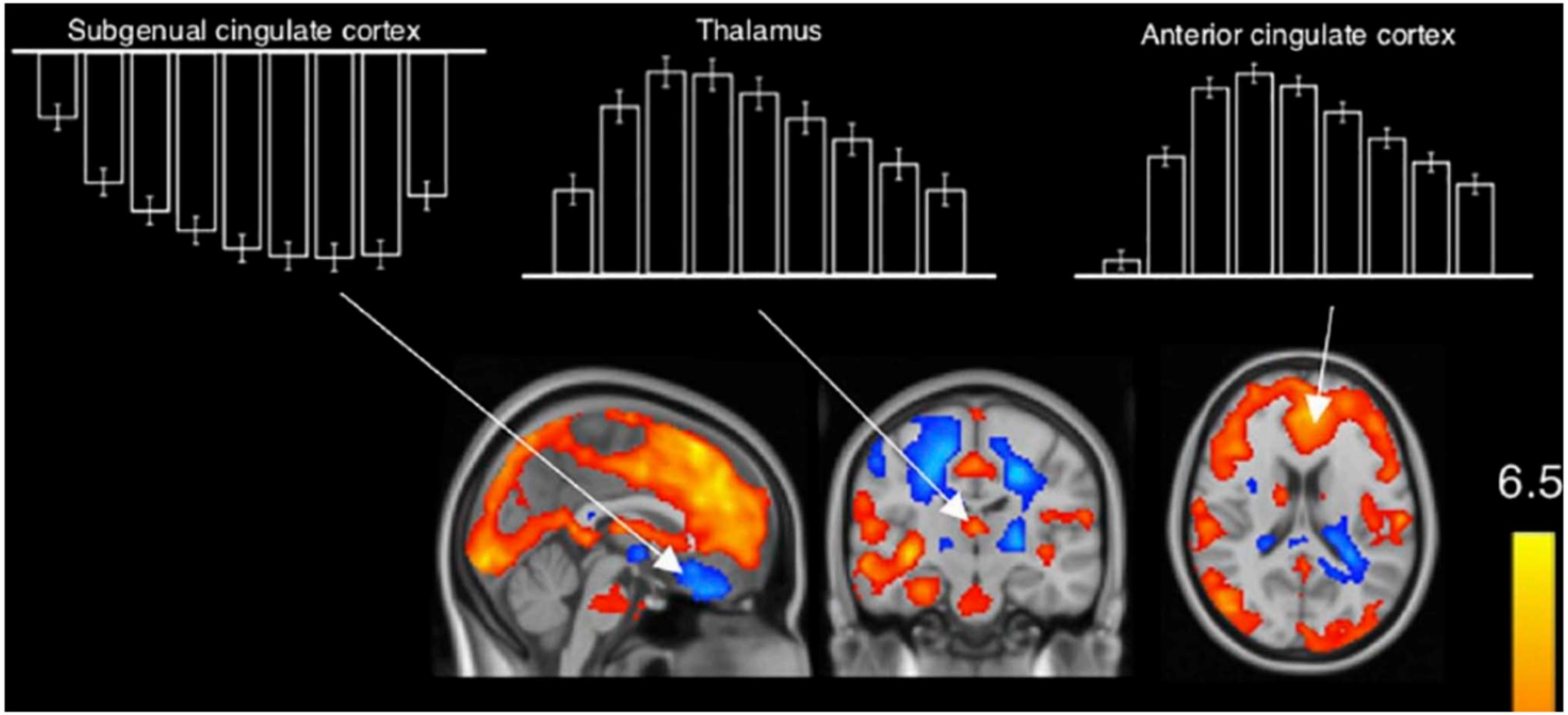


Figure 2. Group maps showing the locations of significant (*F*-ratio maps, $p < 0.05$, familywise error (FWE) cluster corrected) effects of ketamine on the blood-oxygenation-level-dependent (BOLD) signal during the first 9 minutes of ketamine infusion. Although these are *F*-ratio maps, for visual display purposes, areas that showed decreased BOLD responses post-hoc are coloured blue. A grey matter mask was applied to all maps. The histograms show the time series of the BOLD signal change in 1-minute increments from three selected regions showing a significant effect of ketamine. Error bars represent 90% confidence intervals.

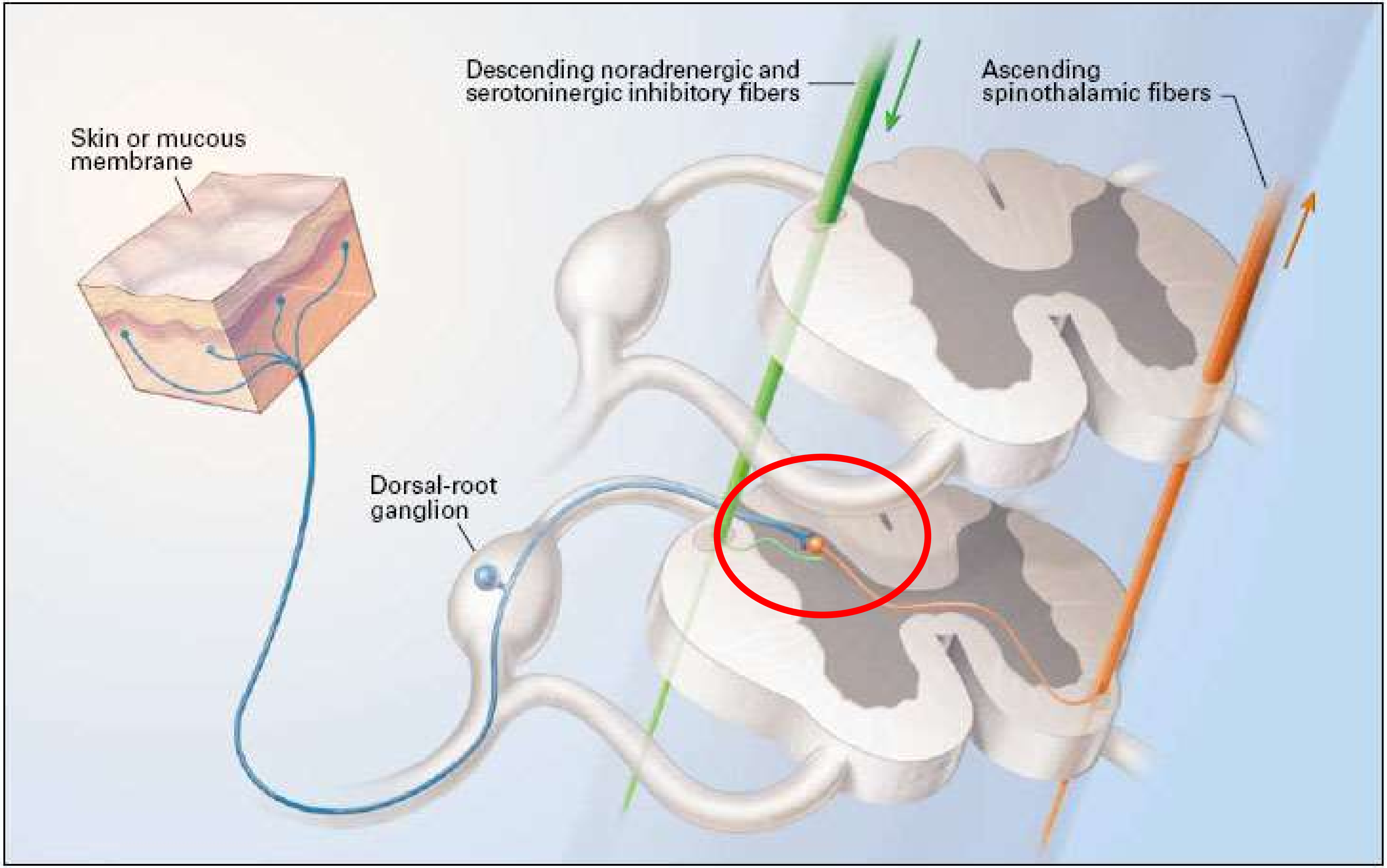
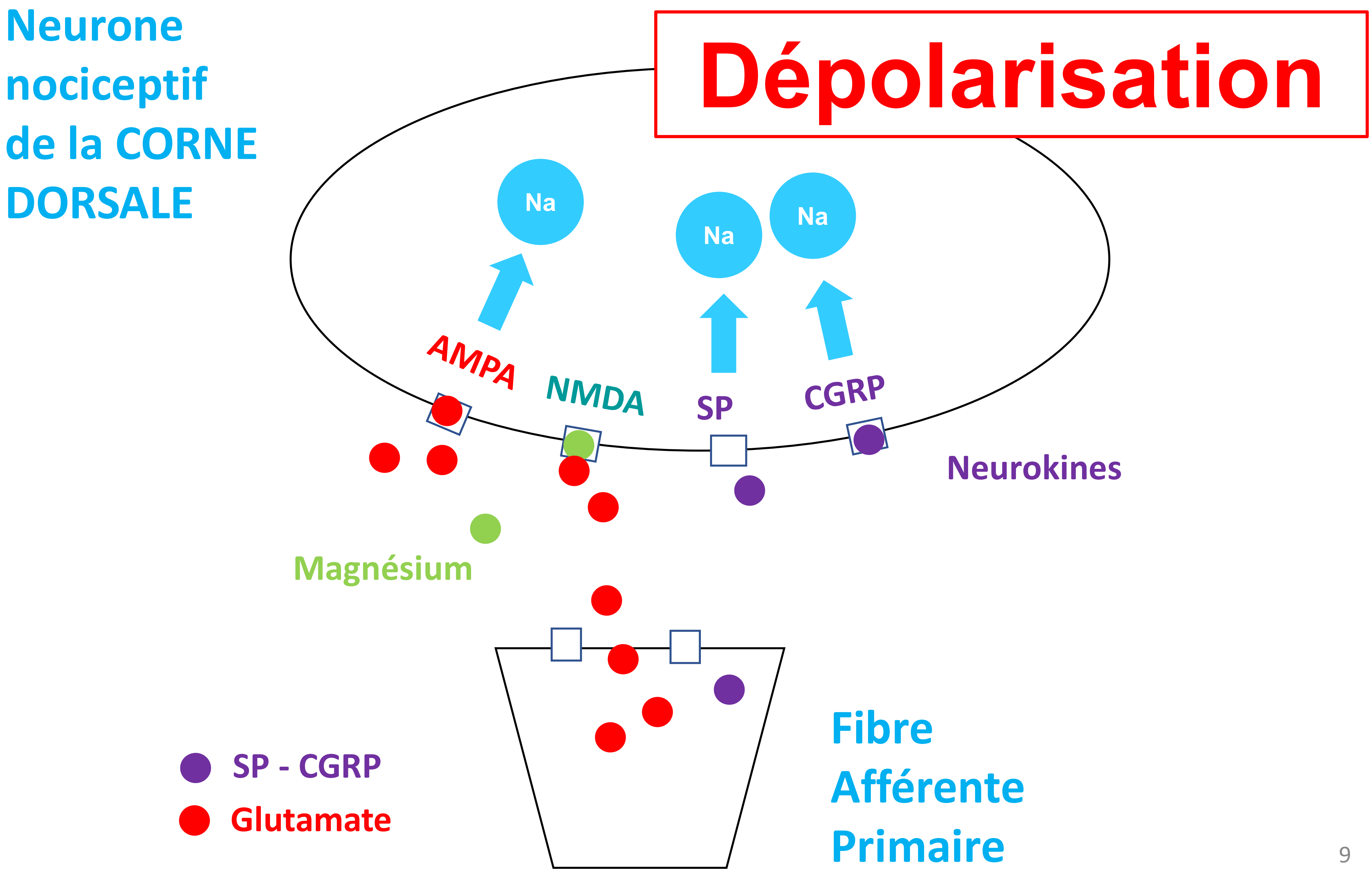


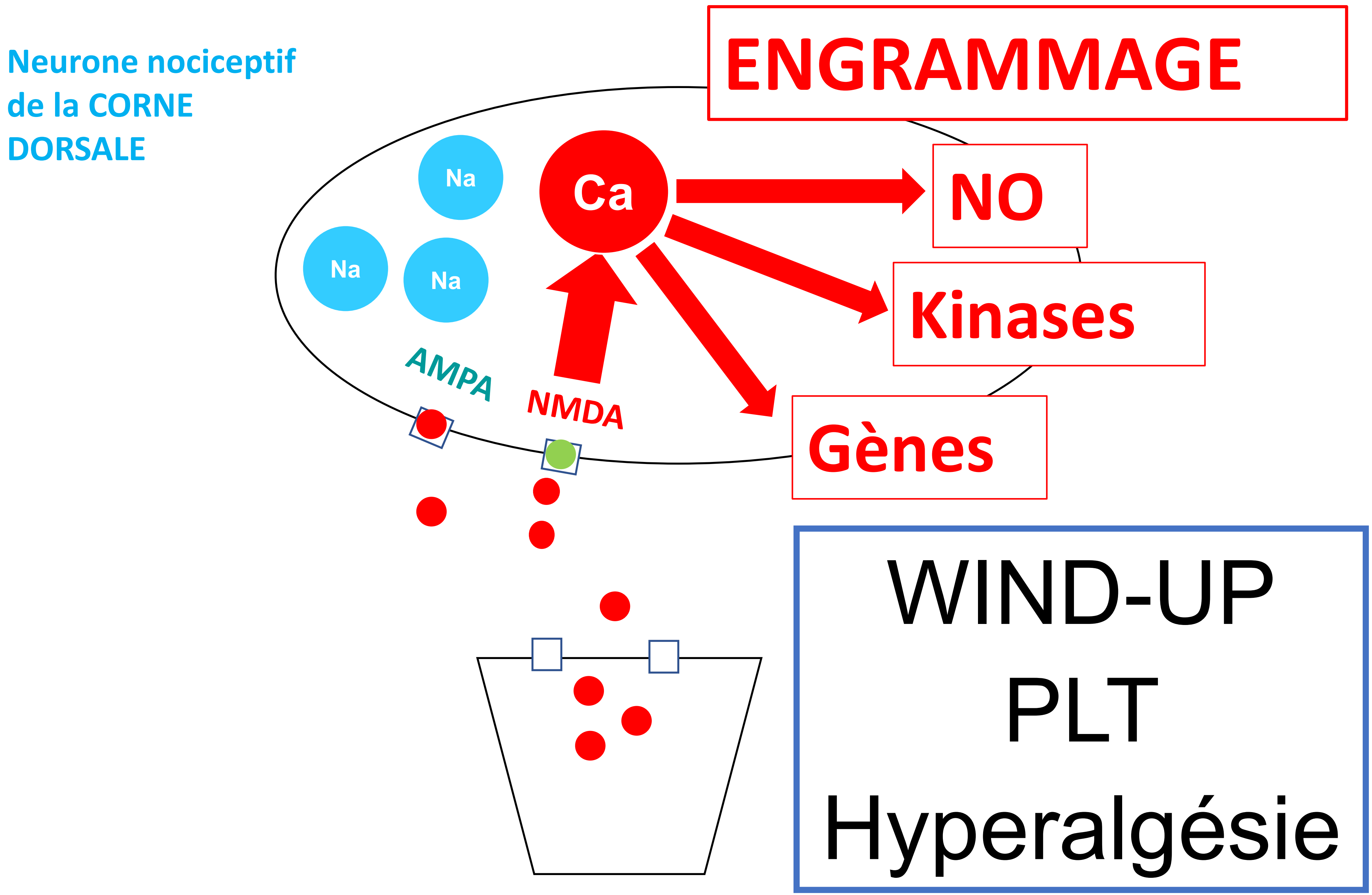
Figure 3. Pathway of Normal Pain Perception.

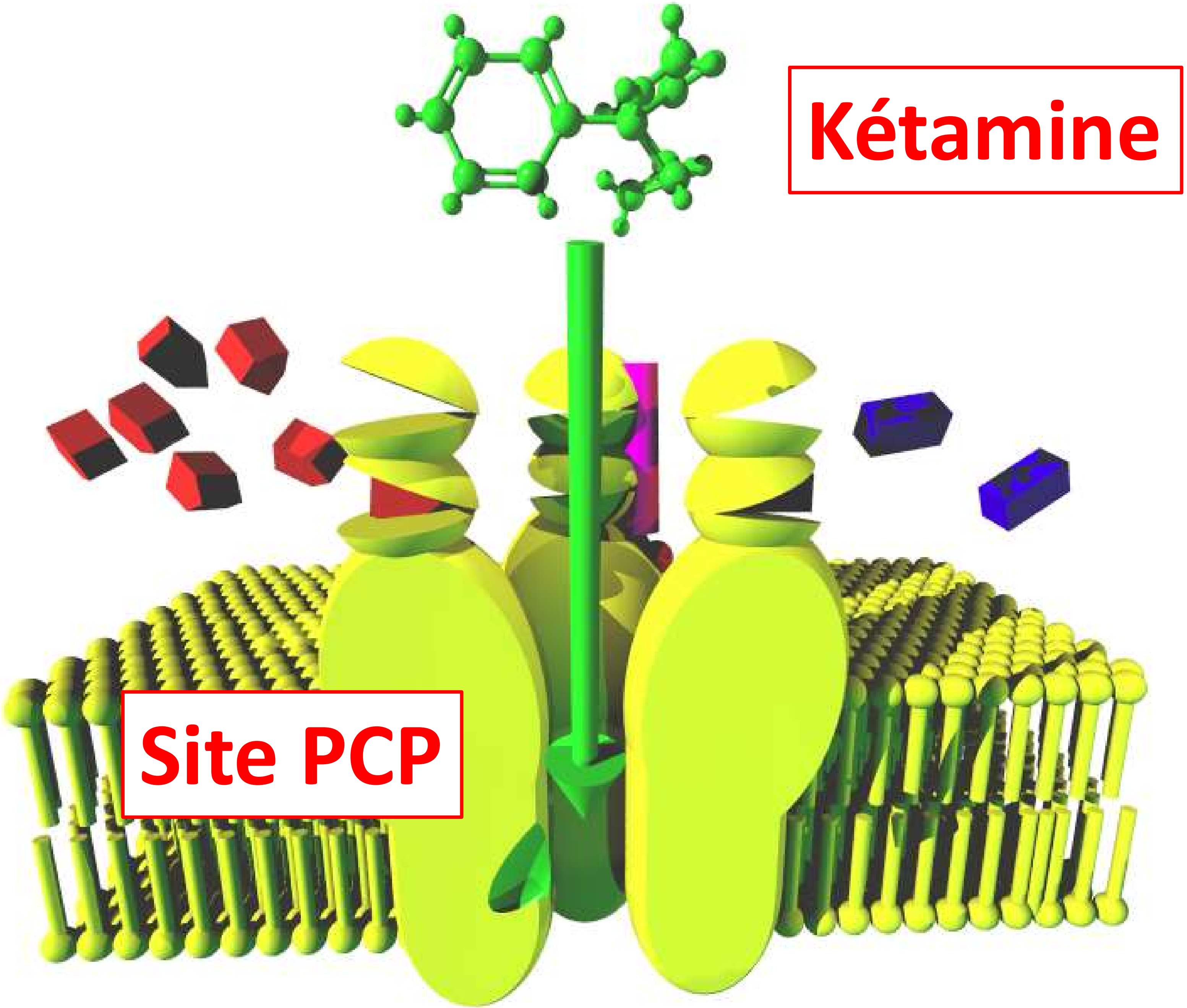
Noxious stimuli activate free nerve endings in the skin to generate signals that are conveyed through unmyelinated C fibers (blue) and small Aδ fibers to the neuronal bodies in the segmental dorsal-root ganglia, then proximally to the dorsal horn of the spinal cord, where they form synapses with second-order neurons.[68] Spinal cord neurons are subject to powerful descending inhibitory signals from the brain (green), mediated by the biogenic amines serotonin and norepinephrine. Drugs that potentiate the central effects of biogenic amines, such as tricyclic antidepressant drugs, may act by enhancing these descending pathways.[68] Endogenous opiates also contribute to descending inhibitory input. The net result of peripheral afferent input and descending inhibitory input is projected cephalad, joining other ascending fibers in the contralateral spinothalamic tract (orange). Information from the spinothalamic tract is integrated with input from brain-stem and cortical areas for the perception of specific aspects of pain, as well as more general affective components of pain perception.

Glutamate : AMPA et NMDA



Si la membrane est dépolarisée





Use dependance

Review

Ketamine and the Disinhibition Hypothesis: Neurotrophic Factor-Mediated Treatment of Depression

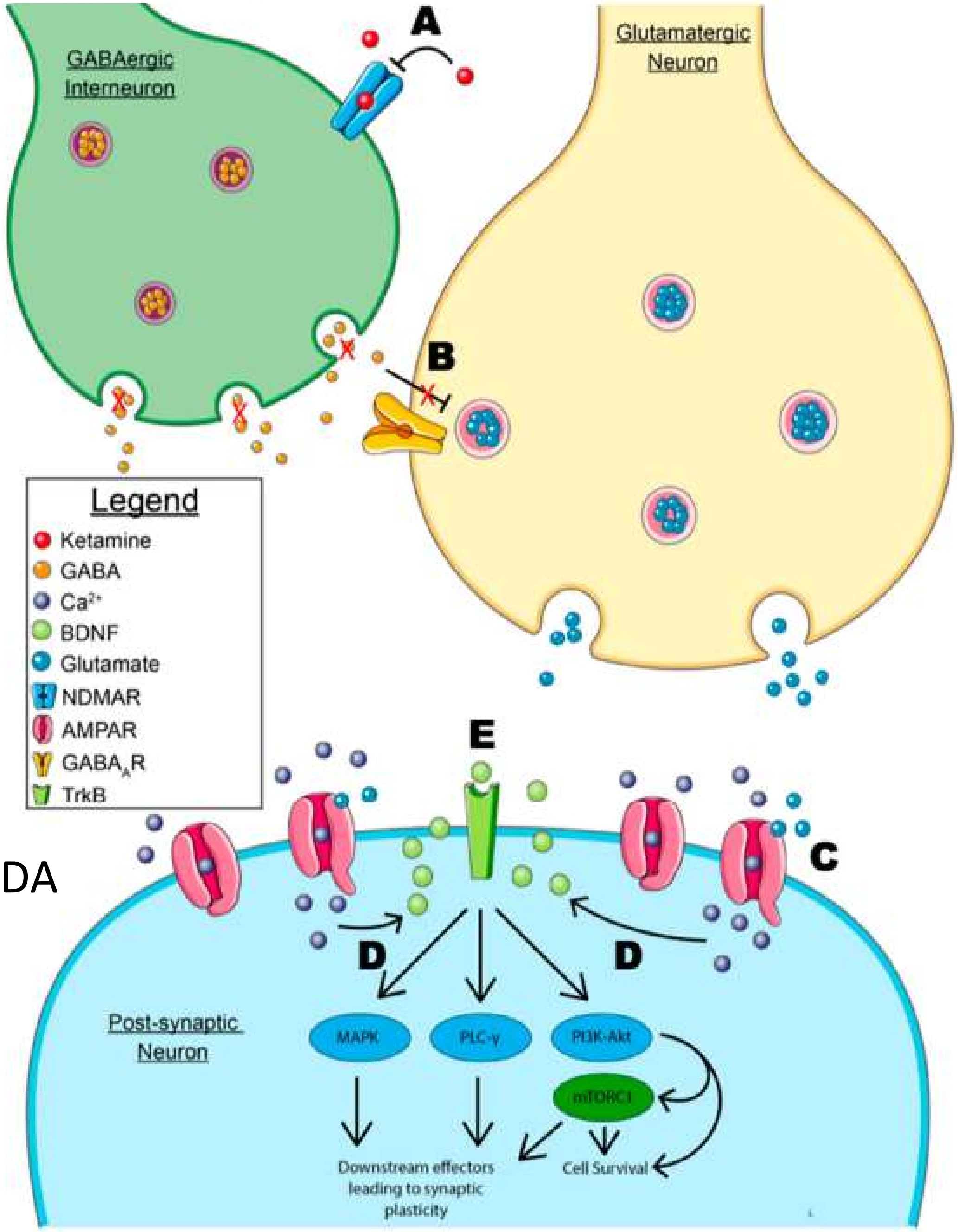
Philip Borsellino, Reese I. Krider, Deanna Chea, Ryan Grinnell and Thomas A. Vida *

Pharmaceuticals **2023**, *16*, 742

La kétamine bloque des récepteur NMDA situés sur des interneurones GABAergiques (inhibiteurs)

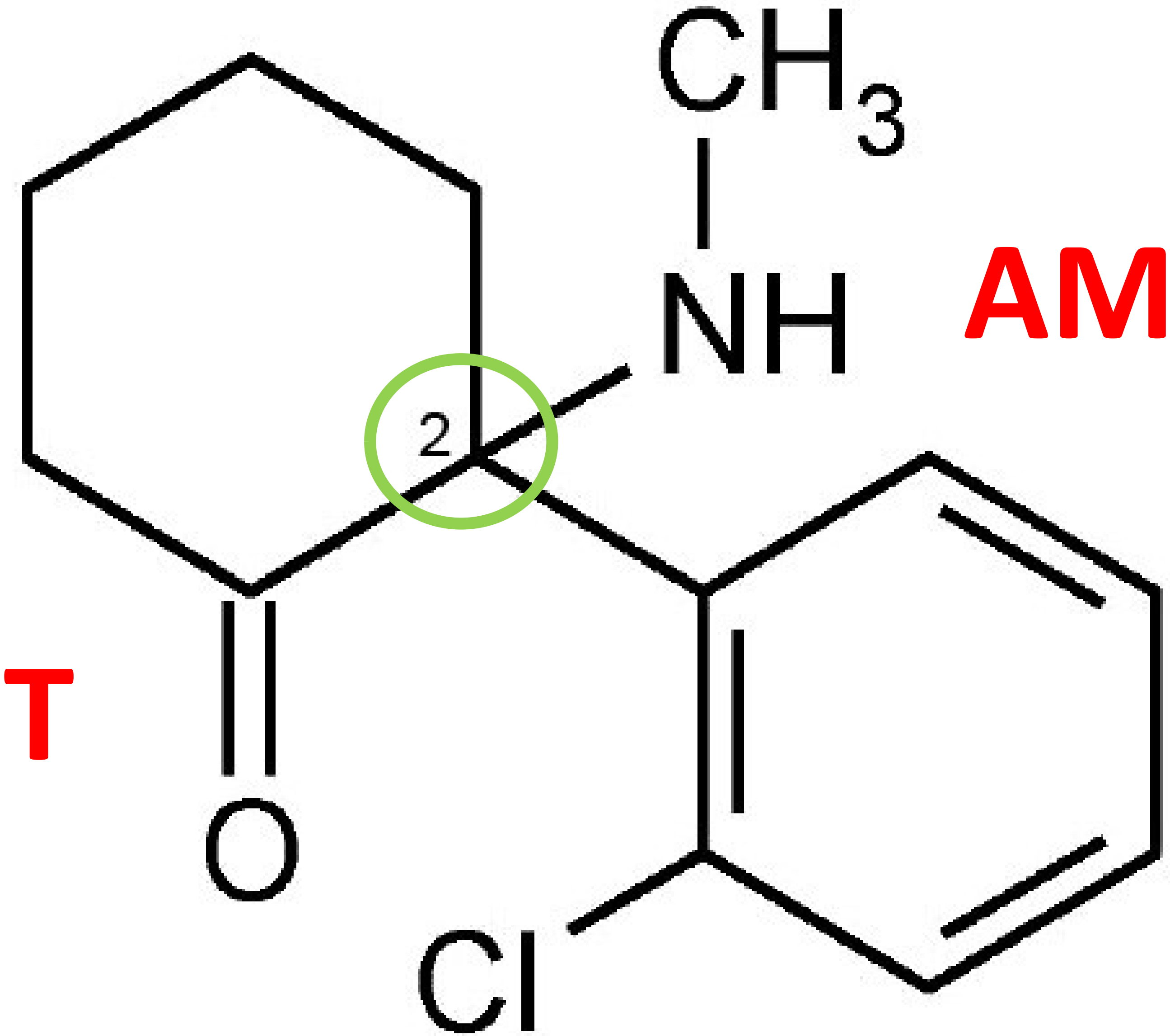
Cette inhibition d'interneurones inhibiteurs désinhibe des neurones excitateurs glutamatergiques (moins par moins égale plus)

Cette sécrétion de glutamate stimule des récepteurs AMPA (les récepteurs NMDA sont bloqués par la kétamine) et favorise la synthèse de facteurs neurotrophiques (BDNF, etc.)

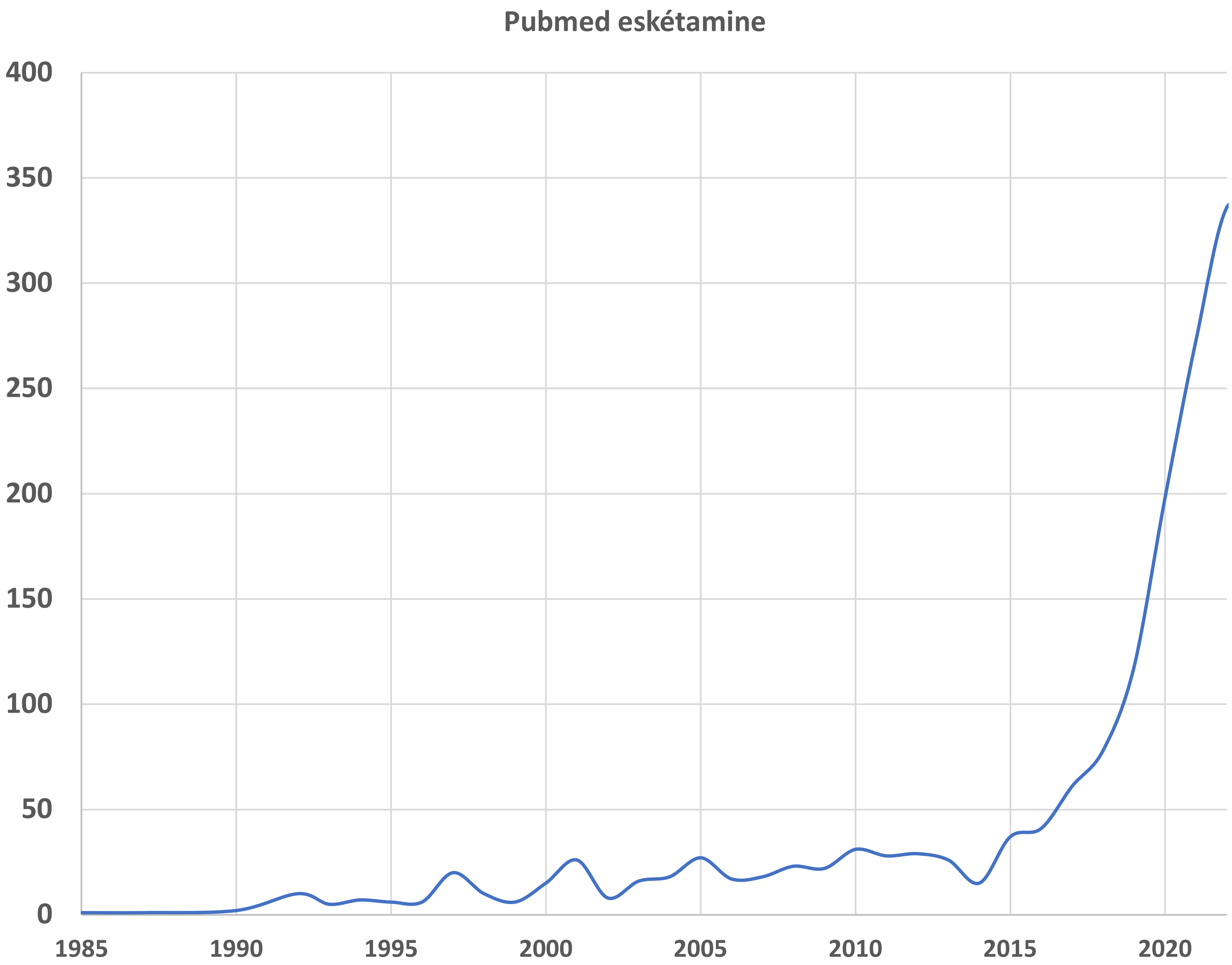


2 - ortho-chlorophényl - 2 - méthylamino - cyclohexanone

KET



AMINE

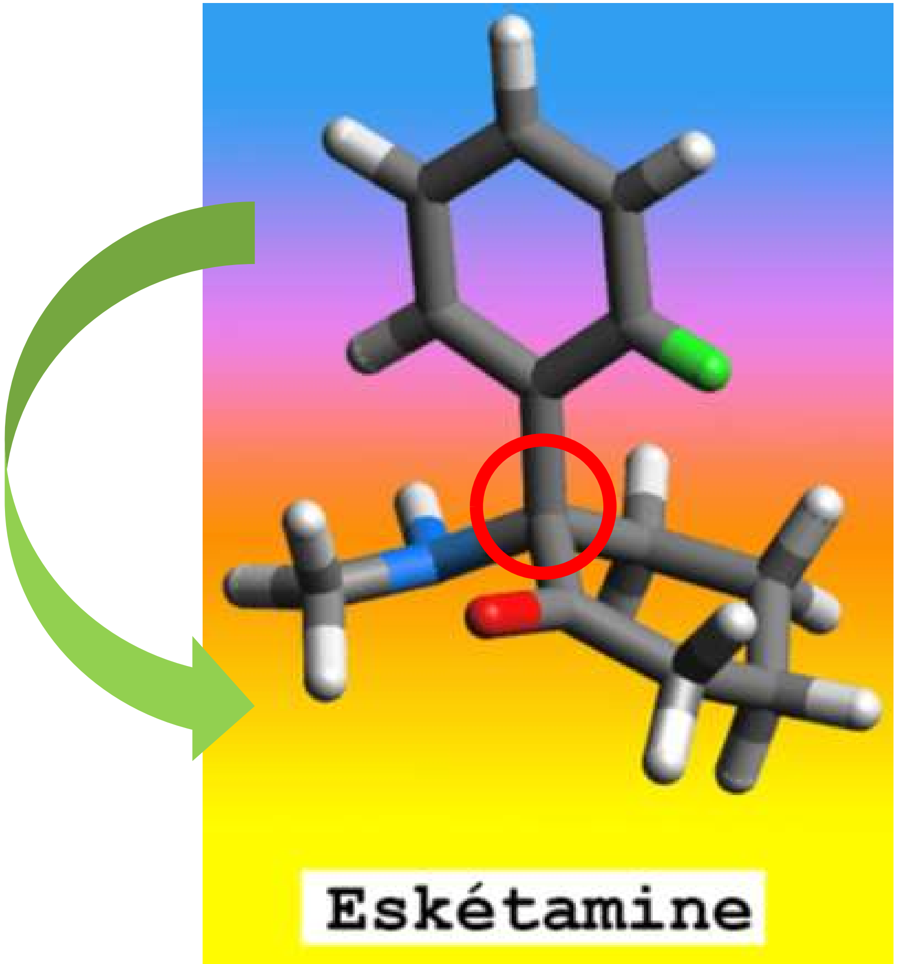

Pubmed eskétamine
400
350
300
250
200
150
100
50
0
1985
1990
1995
2000
2005
2010
2015
2020

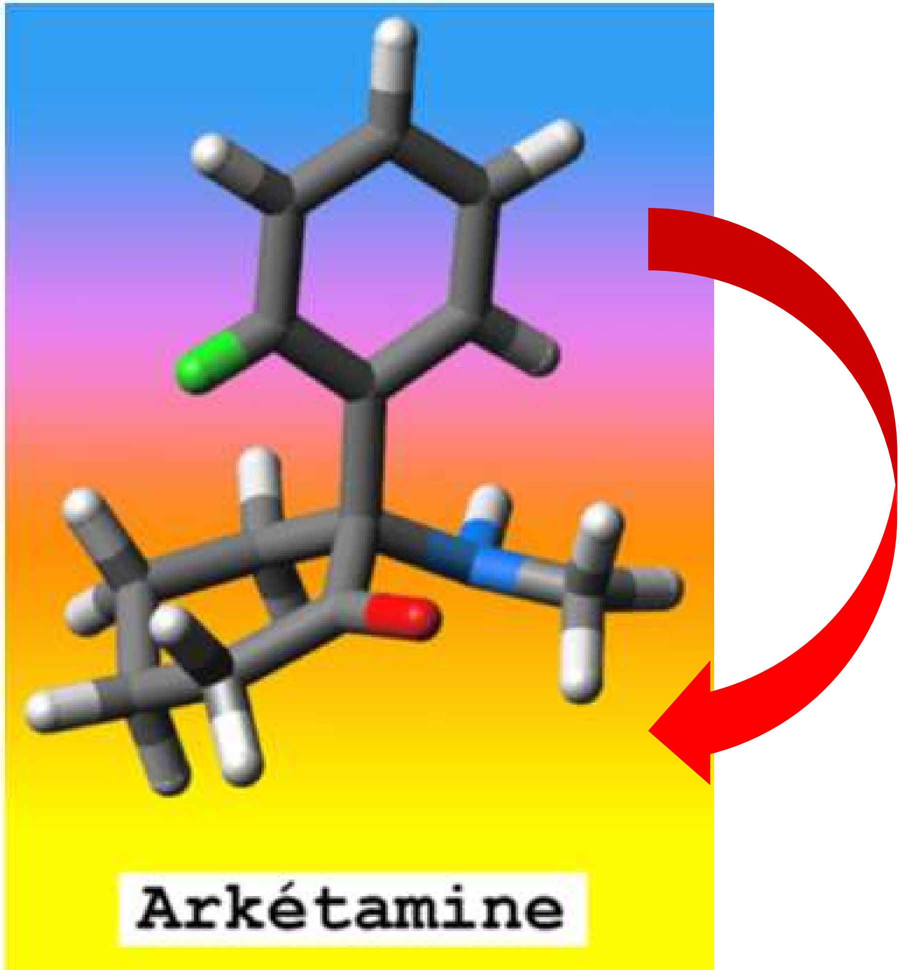
Que veut dire

?

S

R



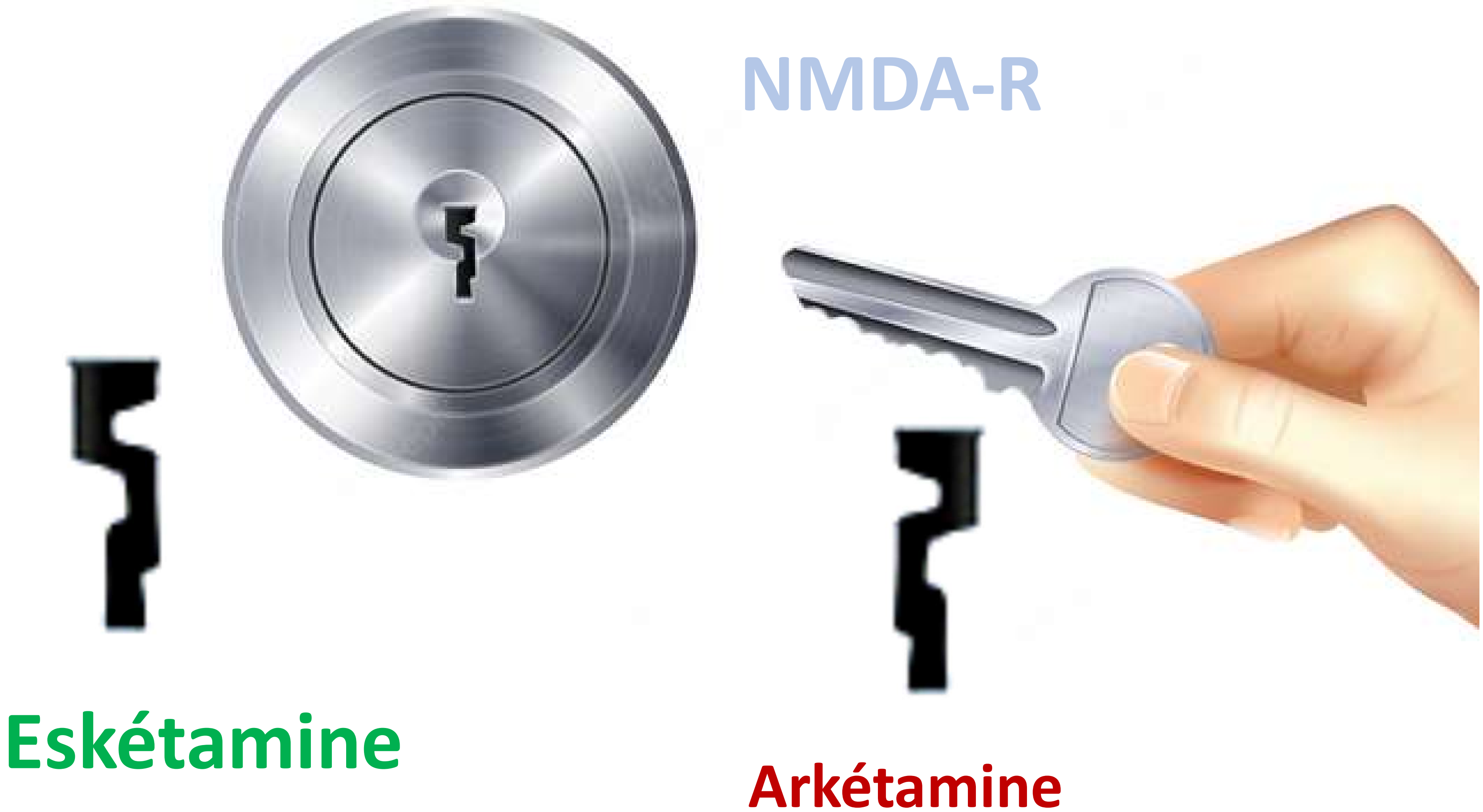


Dextrogyre (+)

Lévogyre (-)

16

Eskétamine : la clé du NMDA-R

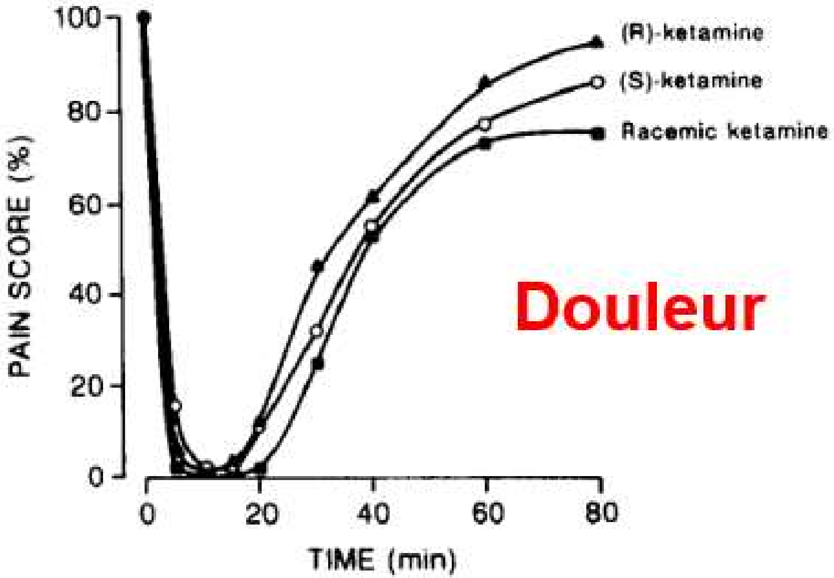


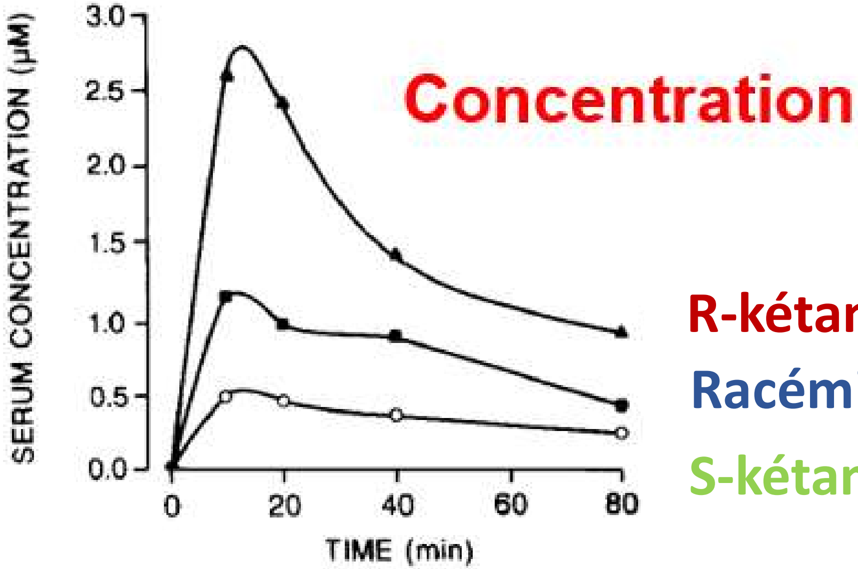
Effect of ketamine, an NMDA receptor inhibitor, in acute and chronic orofacial pain

Lene Cecilie Mathisen [a,1], Per Skjelbred [b], Lasse A. Skoglund [c] and Ivar Øye [a,*]

Pain, 61 (1995) 215–220

The various forms of ketamine were given on an individual basis, either i.m. or intravenously (i.v.) as a single dose sometimes followed by continuous infusion. The patients recorded their pain on the VAS described above and the treatment goal was to obtain analgesia for a time period of at least 15 min. This study was regarded as a pilot investigation. It was not blinded, placebo was not included, and the





R-kétamine
Racémique
S-kétamine

Cognitive Impairment after Small-dose Ketamine Isomers in Comparison to Equianalgesic Racemic Ketamine in Human Volunteers

Ernst G. Pfenninger, M.D., Marcel E. Durieux, M.D., Ph.D.,† Sabine Himmelseher, M.D.‡*

Anesthesiology 2002; 96:357–66

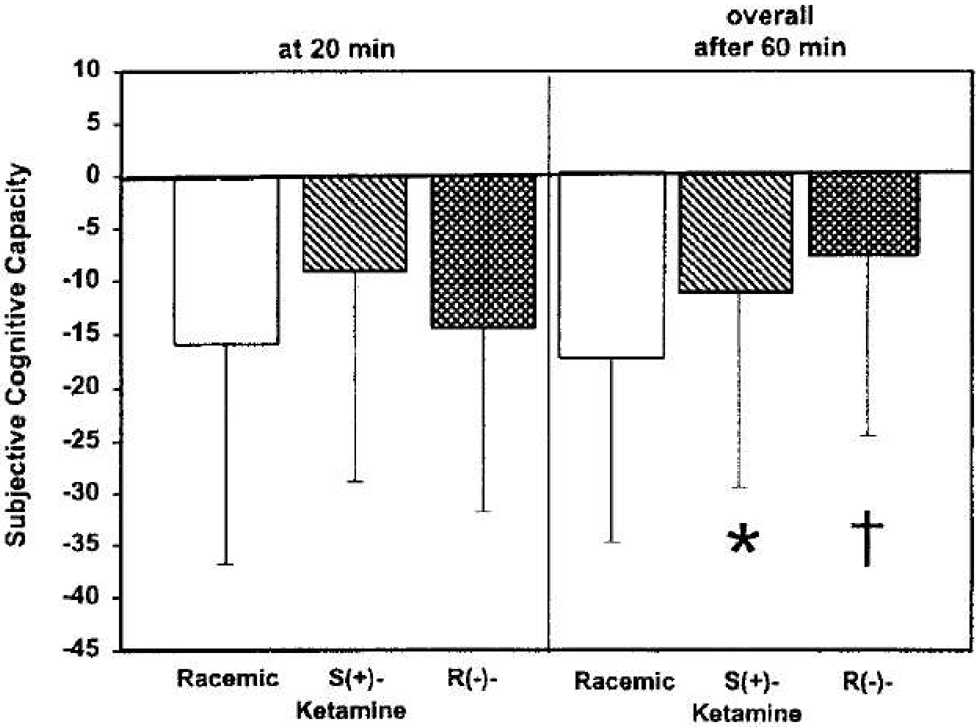


Fig. 3. Analysis of change of clustered rating scores for subjective cognitive capacity revealed less decline after $R(-)$- (P = 0.040) or $S(+)$-ketamine (P = 0.042) than after racemic ketamine for the overall period at 60 min. Bars show the mean ± SD. *Significant difference between racemic ketamine and $S(+)$-ketamine. †Significant diffference between racemic ketamine and $R(-)$-ketamine.

Methods: Twenty-four subjects received intravenous 0.5 mg/kg racemic, 0.25 mg/kg $S(+)$-, and 1.0 mg/kg $R(-)$-ketamine in a prospective, randomized, double-blind, crossover study. Hemodynamic variables, mood, and cognitive capacities were assessed for 60 min.

Plus de problèmes cognitifs avec le racémique

BEHAVIORAL AND ELECTROENCEPHALOGRAPHIC EFFECTS OF ATROPINE AND RELATED COMPOUNDS

V. G. LONGO

Pharmacological Reviews June 1966, 18 (2) 965-996;

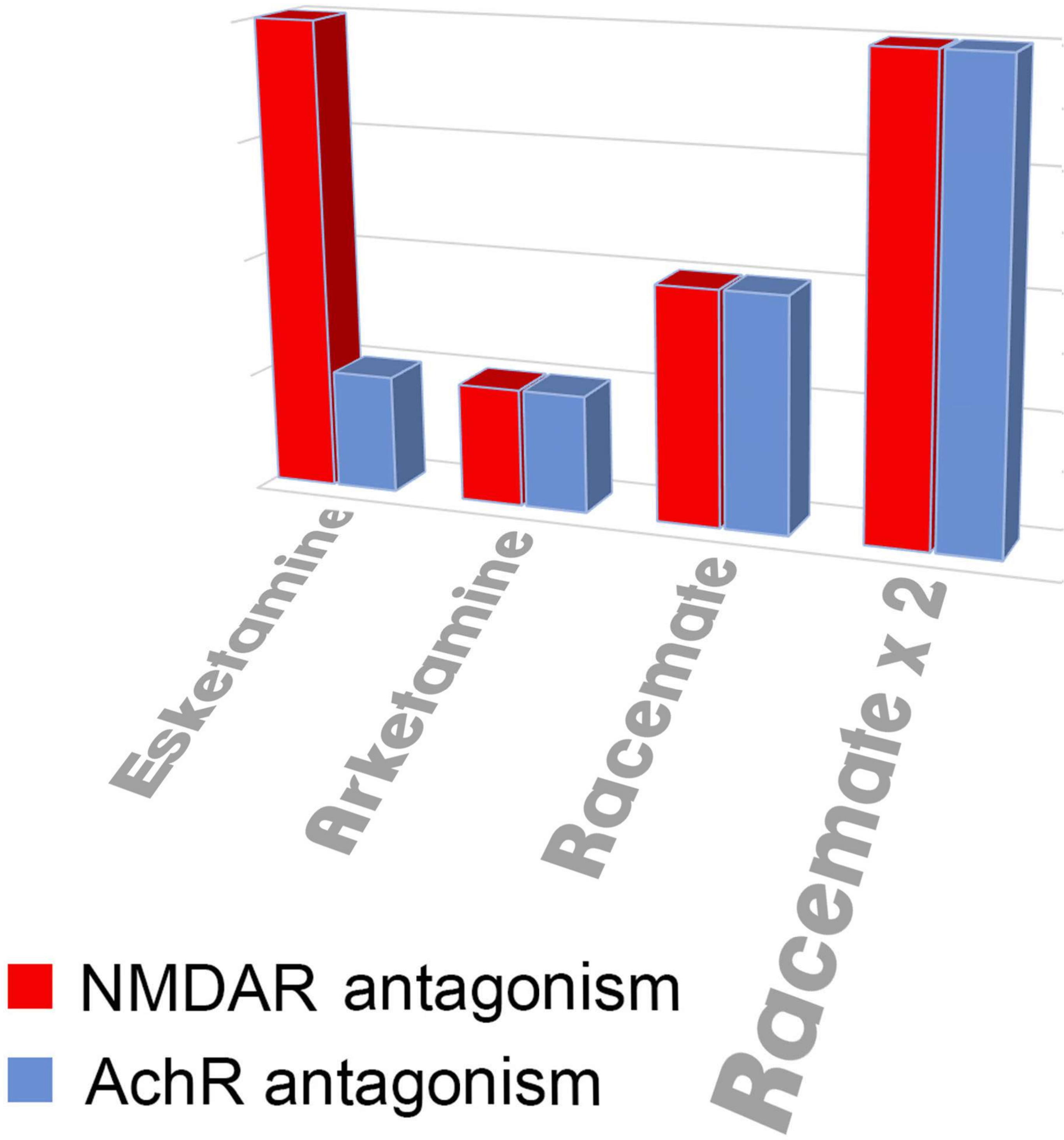
This review is concerned primarily with atropine and scopolamine; other anti-cholinergic compounds will be referred to when necessary. The effects observed in man will be reviewed in an attempt to set forth the essential and characteristic elements which make up the "central anticholinergic syndrome." In a separate

Physostigmine Antagonizes Ketamine

TORO-MATOS, A., RENDON-PLATAS, A. M., AVILA-VALDEZ, E., AND VILLARREAL-GUZMAN, R. A.

Anesth Analg 59:764–767, 1980.

In a random cross-over double-blind trial, the effects of intravenous physostigmine salicylate (2.0 mg) and placebo were observed in seven healthy volunteers 10 minutes after the intravenous administration of 1.5 mg/kg of ketamine. Recovery time was significantly shorter after physostigmine than after placebo. Nystagmus and blurred vision, which followed ketamine anesthesia, disappeared more rapidly when physostigmine was given. This study confirms previous observations that physostigmine counteracts some of the manifestation of ketamine aftereffects which resemble the so-called central anticholinergic syndrome. Nausea and vomiting were significantly more frequent after physostigmine administration.



Esketamine
Arketamine
Racemate
Racemate x 2
NMDAR antagonism
AchR antagonism

Pharmacokinetics of ketamine and its major metabolites norketamine, hydroxynorketamine, and dehydronorketamine: a model-based analysis

Jasper Kamp*, Kelly Jonkman, Monique van Velzen, Leon Aarts, Marieke Niesters, Albert Dahan and Erik Olofsen

Recevied: 7 March 2020 **Accepted:** 19 June 2020

Results: The pharmacokinetics of ketamine and metabolites were adequately described by a seven-compartment model with two ketamine, norketamine, and hydroxynorketamine compartments and one dehydronorketamine compartment with metabolic compartments in-between ketamine and norketamine, and norketamine and dehydronorketamine main compartments. Significant differences were found between S- and R-ketamine enantiomer pharmacokinetics, with up to 50% lower clearances for the R-enantiomers, irrespective of formulation. Whilst SNP had a significant effect on ketamine clearances, simulations showed only minor effects of SNP on total ketamine pharmacokinetics.
Conclusions: The model is of adequate quality for use in future pharmacokinetic and pharmacodynamic studies into the efficacy and side-effects of ketamine and metabolites.
Clinical trial registration: Dutch Cochrane Center 5359.

Effects of Ketamine and Its Isomers on Ischemic Preconditioning in the Isolated Rat Heart

Andrei Molojavyi, M.D.,* Benedikt Preckel, M.D., D.E.A.A.,† Thomas Comfère,‡ Jost Müllenheim, M.D.,†
Volker Thämer, M.D., Ph.D.,§ Wolfgang Schlack, M.D., Ph.D., D.E.A.A.||

Anesthesiology 2001; 94:623

CPK

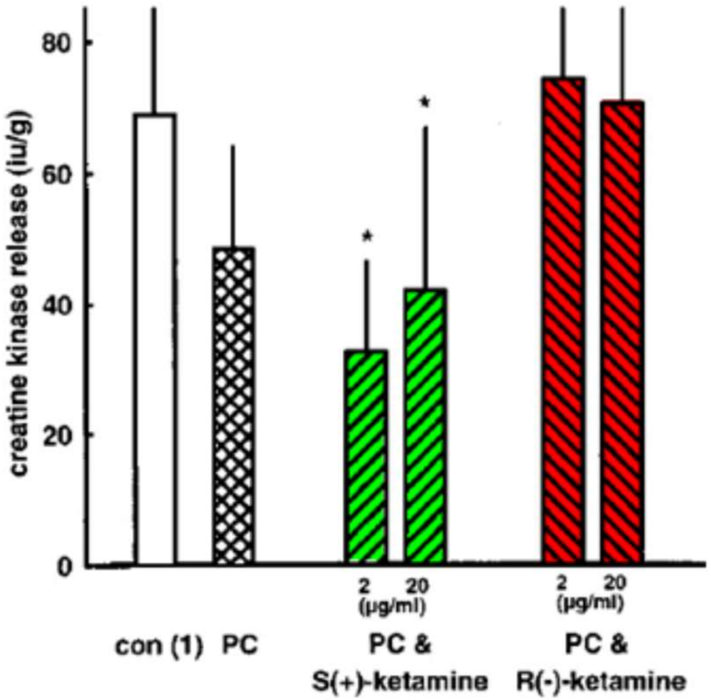


Fig. 3. Cumulative creatine kinase release during reperfusion as a variable of cellular damage. Data are mean values and standard deviation, n = 8; *$P < 0.05$ *versus* control group;

PC = ischemic preconditioning.

Methods: Isolated rat hearts (n = 80) underwent 30 min of no-flow ischemia and 60 min of reperfusion. Two groups with eight hearts each underwent the protocol without intervention (control-1 and control-2), and, in eight hearts, preconditioning was elicited by two 5-min periods of ischemia before the 30 min ischemia. In the six treatment groups (each n = 8), ketamine, *R*(−)- or *S*(+)-ketamine were administered at concentrations of 2 or 20 µg/ml before preconditioning. Eight hearts received 20

After administration of 20 µg/ml ketamine and 2 or 20 µg/ml *R*(−)-ketamine, the protective effects of preconditioning were abolished (LV developed pres-

The Effects of Ketamine-Isomers on Neuronal Injury and Regeneration in Rat Hippocampal Neurons

Sabine Himmelseher, MD, Ernst Pfenninger, MD, and Michael Georgieff, MD

(Anesth Analg 1996;83:505–12)

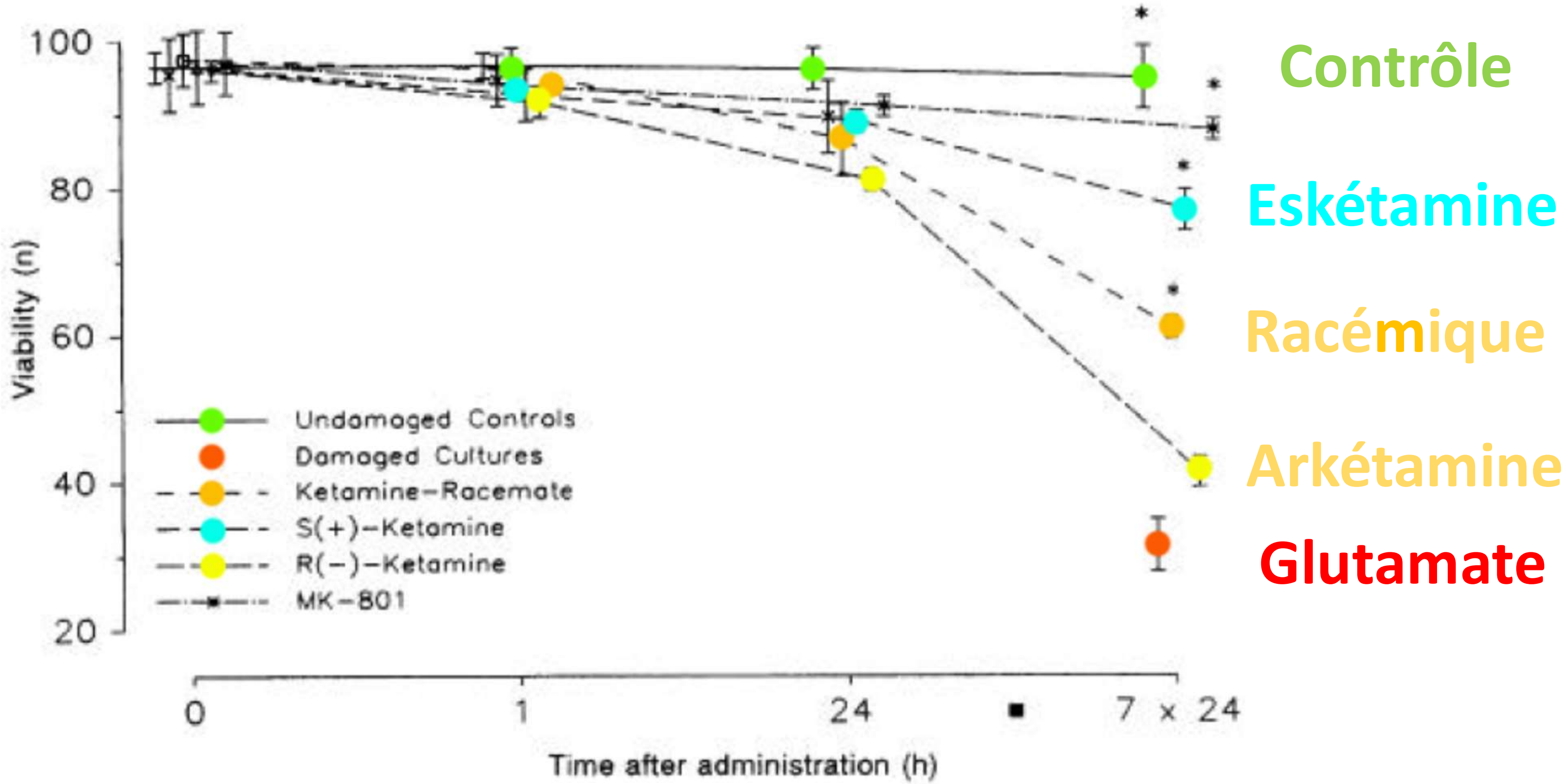


Figure 2. Survival of undamaged controls, glutamate-induced reduction in live cells of glutamate-exposed, untreated cultures, and glutamate-exposed cells treated with 10^{-4} M ketamine-racemate, 10^{-4} M S(+)-ketamine, 10^{-4} M R(−)-ketamine, and 10^{-6} M MK-801. Drugs were added after glutamate-exposure, and on postinjury

Conclusion

- **l’eskétamine est deux fois plus active** que le racémique en termes d’analgésie et de narcose
- **L’eskétamine est mieux tolérée au niveau psychique**
- L’eskétamine a un **meilleur profil sur les systèmes circulatoire et nerveux**
- l’eskétamine a une **clairance plus élevée** que le racémique. L’émergence est plus rapide et plus calme

Eskétamine, Analgésie périopératoire

Georges Mion
Hôpital Cochin - Paris

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Société Française d'Étude et de Traitement de la Douleur
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Eskétamine,
analgésie postopératoire

Author, Year	Surgery	Study Size	Esketamine Dose: Induction	Esketamine Dose: Intraop	Esketamine Dose: Postop / Duration	Postoperative: Opioid Consumption	Postoperative: Pain Relief
		N	mg/kg	mg.kg^{-1}.h^{-1}	mg.kg^{-1}.h^{-1} / h	% Reduction *vs* Control	
Jaksch et al; 2002[108]	Arthr. knee ligament repair	30	0.5	0.12	0.12 / 2 h	NS *vs* Control	NS *vs* Control
Argiriadou et al;	Major Abdominal surgery,	45	0.5			36 %	NS *vs* Control
2004[109]	*two esket groups*		0.5 and 0.2 mg/kg B every 20 min			63%	Esket > Control
Lahtinen et al; 2004[98]	Cardiac surgery	90	0.075	0.075	0.075 / 48 h	18%	NS *vs* Control
Snijdelaar et al; 2004[97]	Radical Prostatectomy	28	0.1	0.12	PCA: 0.5 mg B / 48 h	35%	Esket > Control
Becke et al; 2005[105]	Kids: Urological surgery	30	0.2	0.3		NS *vs* Control	Esket > Control
Spreng et al; 2010[107]	Hemorrhoidectomy	77	0.35	0.3		NS *vs* Control	NS *vs* Control
Argiriadou et al; 2011[93]	Thoracotomy	53	0.5	0.4		44%	Esket > Control
Mendola et al; 2012[100]	Thoracotomy	66		0.1	0.1 / 60 h	NS *vs* Control	Esket > Control
Suppa et al; 2012[99]	Cesarean section, SPA	56	0.5 IM B 10 min after birth then 0.12		0.12 / 12 h	31%	NS *vs* Control
Bornemann-Cimenti	Abdomial surgery,	56	0.25	0.125	0.125 / 48 h	41%	NS *vs* Control
et al; 2016[94]	*two esket groups*			0.015	0.015 / 48 h	45%	NS *vs* Control
Miziara et al; 2016[95]	Lap-scop. Cholecystectomy	48		0.3		31%	Esket > Control
Nielsen et al; 2017[102]	Spinal fusion surgery	147	0.5	0.25		35%	Esket > Control
Brinck et al; 2021[103]	Major lumbar fusion	100			PCA: 0.25 mg B / 24 h	NS *vs* Control	NS *vs* Control
	surgery,				PCA: 0.5 mg B / 24 h	NS *vs* Control	NS *vs* Control
	three esket groups				PCA: 0.75 mg B / 24 h	36%	Esket > Control
Brinck et al; 2021[104]	Major lumbar fusion	198	0.5	0.12		13%	NS *vs* Control
	surgery, *two esket groups*		0.5	0.6		10%	NS *vs* Control

Développement de l'OFA
Eskétamine + dexmed + AL

Différentes études randomisées ont montré :

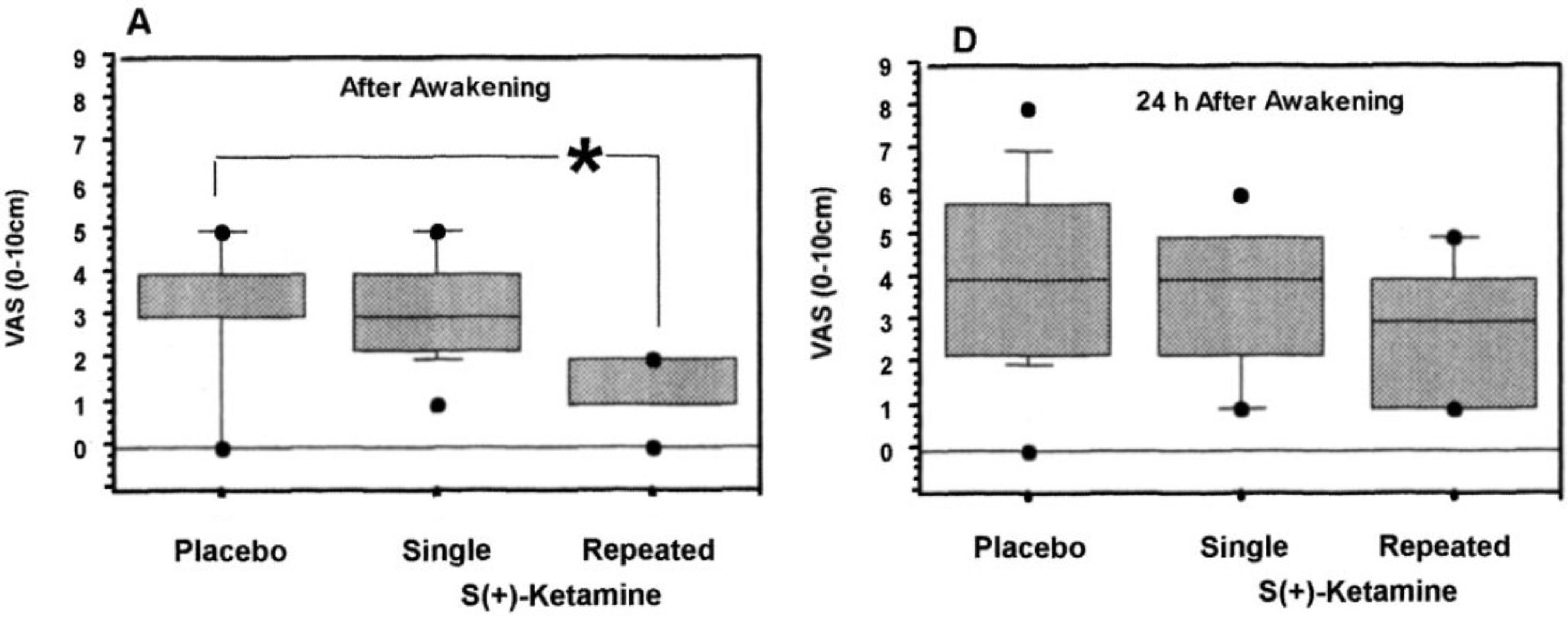
- Meilleure analgésie, diminution conso. de morphine
- Diminution des douleurs persistantes
- Moins de NVPO
- Plus grande stabilité hémodynamique
- Meilleurs paramètres respiratoires
- Reprise plus précoce de l'alimentation
- Diminution des paramètres inflammatoires
- Diminution des complications (chir. pancréatique)

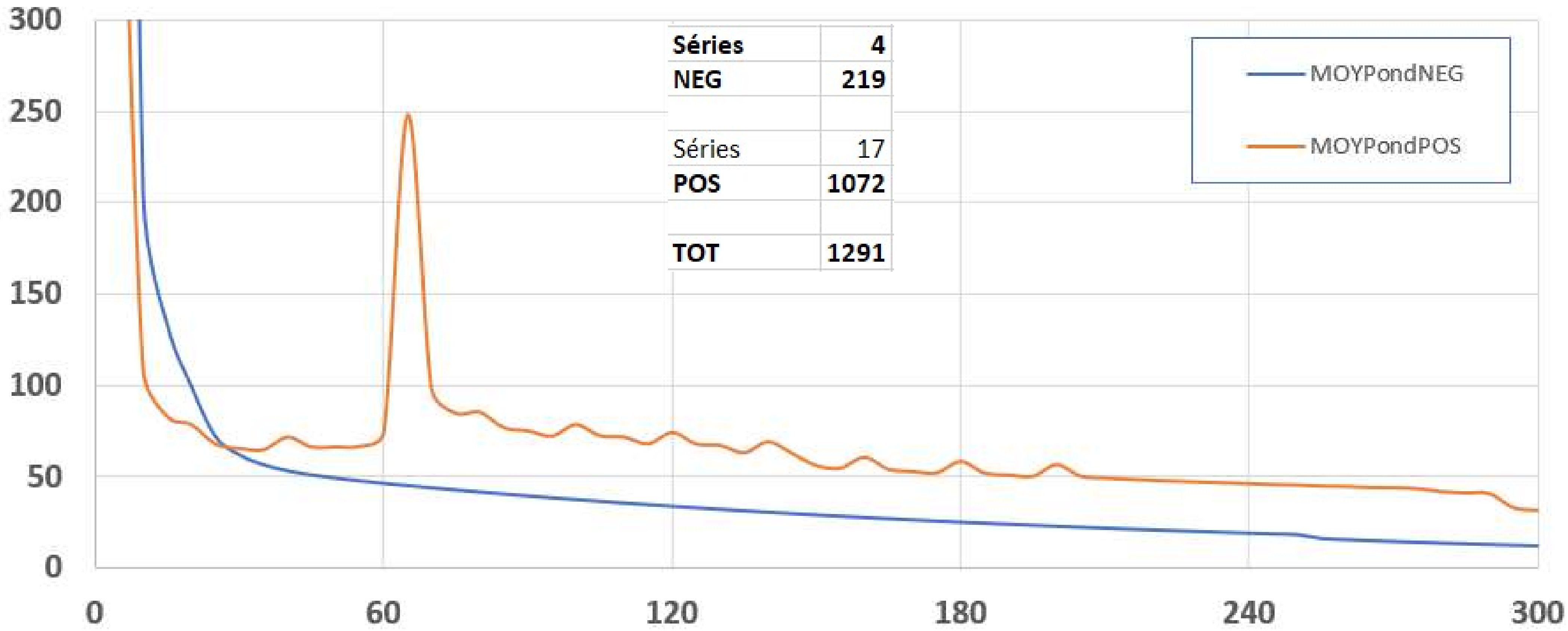
Improvement of Pain Treatment After Major Abdominal Surgery by Intravenous S(+)-Ketamine

Helena Argiriadou, MD*, Sabine Himmelseher, MD†, Pinelopi Papagiannopoulou, MD‡, Mary Georgiou, MD‡, Fotios Kanakoudis, MD‡, Maria Giala, MD*, and Eberhard Kochs, MD, PhD†

(Anesth Analg 2004;98:1413–8)

stereoisomer of ketamine. Patients were randomized to receive placebo, 0.5 mg/kg preincisional S(+)ketamine, or 0.5 mg/kg preincisional and 0.2 mg/kg intraoperative S(+)-ketamine repeated at 20-min intervals. In the postoperative period, epidural ropivacaine (2 mg/mL; 0.12 mL $\cdot$ kg^{-1} $\cdot$ h^{-1}) was infused for pain therapy. Pa-





Argiriadou	**2011**	thoracotomy
Argiriadou	**2004**	Abdominal
Argiriadou 2	***2004***	*3e groupe*
Bornemann-(	**2016**	Abdominal
Borneman 2	**2016**	*2e dose*
Lahtinen	**2004**	CABG)
Mendola	**2012**	thoracotomy
Miziara	**2016**	laparoscopic cholecystectomy
Nielsen 2017	**2017**	spinal fusion
Snijdelaar	**2004**	radical prostatectomy
Brinck	**2021**	lumbar spinal fusion surgery
Brinck 2	**2021**	
Becke	**2005**	Children, major urological surgery
Wang	**2020**	laparoscopic hysterectomy
		wang 2 racemic
		wang 3 esk faible dose
Qiu	**2022**	gynecological laparoscopic surgery
Yuan	**2022**	thoracoscopic surgery
		Yuan 2 double dose
Zhu	**2022**	Modified Radical Mastectomy
		Zhu 2 double dose
Zhang J	**2023**	Laparoscopic Bariatric Surgery
Chen	**2023**	thoracoscopic lung lobectomy
Zhou	**2023**	breast or thyroid surgery

Jaksch	**2002**	knee ligament repair
Spreng	**2010**	haemorrhoidectomy
Zhang	**2022**	laparoscopic cholecystectomy
Zhou R	**2023**	thoracoscopic lung lobectomy

Proposition de protocole en analgésie préventive

- **Bolus initial** :
  immédiatement (15 sec) avant le propofol (supprime la douleur à l'injection) ou avant l'incision
  IVL **0,2 à 0,35 mg/Kg**

- **Relais au PSE** : **0,125 à 0,5 mg/Kg/h**
  On interrompt la perfusion ~ 30 min avant la fin du geste chirurgical

- **Chez les patients les plus douloureux**
  les 2 premiers jours postopératoires
  - on peut continuer à administrer l'eskétamine au PSE
    0,06 à 0,15 mg/kg/h (4 à 10 mg/h)
  - ou, plus simplement, en PCA morphine **(1 mg/1 mg)**

esketiva

Go to HELP

Go to OFA

Go to Calculator

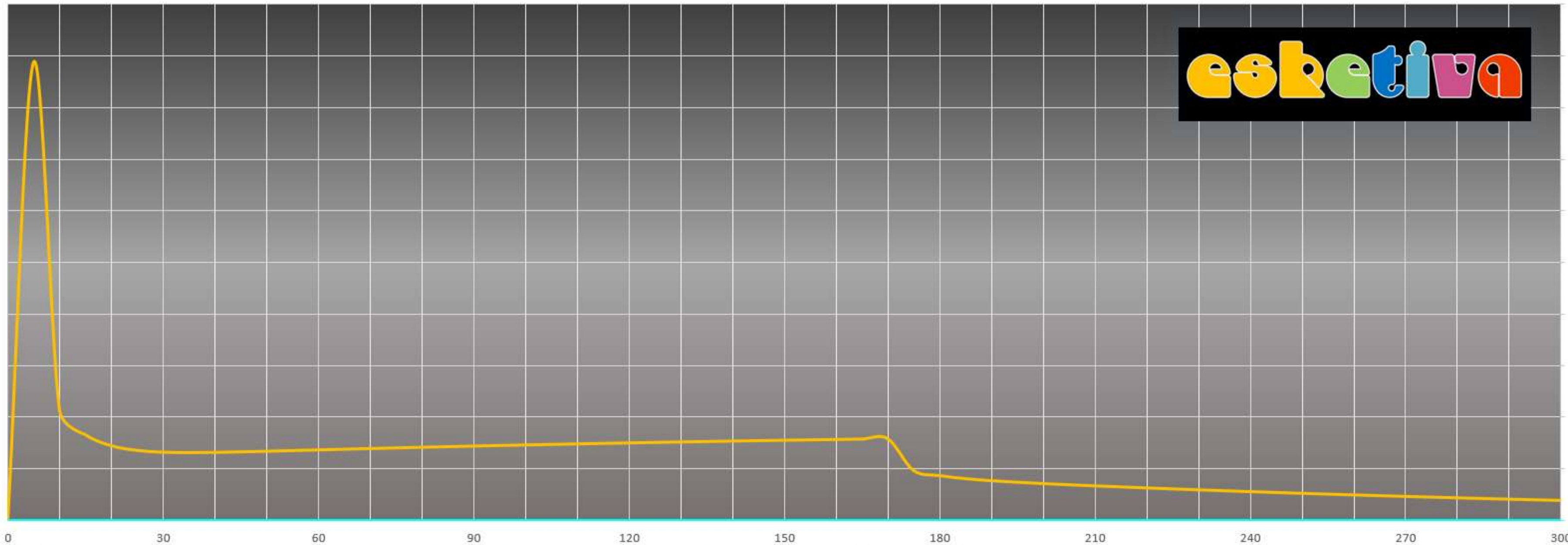
Go to Drugs

No label at all

New simulation

PATIENT

Age	40	years
Weight	70	Kg
Height	170	cm
BMI	24.2	
Gender	1	M=1 F=2



5 Esketamine clear
mg/kg/h clear
µg/kg/min clear
Scale mL
Labels No Yes ng/mL

TIME (Min)	To	5	10	15	20	25	30	35	40	45	50	55	60	65	70	75	80	85	90	95
Bolus mg		25																		
Infusion			0.30	0.30	0.30	0.30	0.30	0.30	0.30	0.30	0.30	0.30	0.30	0.30	0.30	0.30	0.30	0.30	0.30	0.30
Infusion																				
Volume		5.0	5.4	5.7	6.1	6.4	6.8	7.1	7.5	7.8	8.2	8.5	8.9	9.2	9.6	9.9	10.3	10.6	11.0	11.3
Cp		888	213	166	145	136	132	131	132	133	134	135	137	138	139	140	142	143	144	145

TIME (Min)	100	105	110	115	120	125	130	135	140	145	150	155	160	165	170	175	180	185	190	195
Bolus mg																				
Infusion	0.30	0.30	0.30	0.30	0.30	0.30	0.30	0.30	0.30	0.30	0.30	0.30	0.30	0.30	0.30					
Infusion																				
Volume	11.7	12.0	12.4	12.7	13.1	13.4	13.8	14.1	14.5	14.8	15.2	15.5	15.9	16.2	16.6	16.6	16.6	16.6	16.6	16.6
Cp	146	147	148	149	150	151	152	153	154	155	155	156	157	158	158	97	87	81	77	74

TIME (Min)	200	205	210	215	220	225	230	235	240	245	250	255	260	265	270	275	280	285	290	295	300
Bolus mg																					
Infusion																					
Infusion																					
Volume	16.6	16.6	16.6	16.6	16.6	16.6	16.6	16.6	16.6	16.6	16.6	16.6	16.6	16.6	16.6	16.6	16.6	16.6	16.6	16.6	16.6
Cp	71	69	67	65	63	61	59	58	56	54	53	51	50	48	47	45	44	43	41	40	39

The Effect of Ketamine Infusion in the Treatment of Complex Regional Pain Syndrome: a Systemic Review and Meta-analysis

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Study	Year	Intervention	Concentrations	Duration of interventions (day)	*N*	*N* (man)	Weight	Age	Baseline (pain score)	Treatment end (pain score)	Immediate response (% relief)	Follow-up effectiveness (% relief in 1-3 month)	None effect (% none relief)	History (pain duration)	Side effect
Correl, G et al	2004	S+ketamine	23.4±9.6mg/hr	4.7	33	25	-	40.5±13.8	-	-	76	54	6	28.1±47 months	Elevations of liver enzymes, Inebriation, Hallucinations
Webster, L et al	2006	S+ketamine	7–8mg/hr	16.4	13	8	-	38.8±12.8	7.7±1.8	4.8±2.3	62	54	38.5	Fulfill CRPS diagnosis criteria	Irritation; fatigue, dizziness, spinal pain, no hallucinations
Kiefer, R et al	2008	S+ketamine	3–7mg/kg/hr	5	20	2	68.4±18.7	30.4±10.4	8.97±0.3	0.5±0.8	93.5	89.4	0	49.4±25.6 months	Psychotropic side effects (anxiety, dysphoria, nightmares, difficulties with sleep), muscular weakness
Kiefer, R et al	2008	S+ketamine	50–500mg/d	10	4	0	-	25.3±10.3	85.1±1.9	89±0.1	0	0	100	58±20 months	Nausea, tiredness, dizziness, insomnia, headaches, weakness, euphoria, disorientation
Schwartzman, R et al	2009	S+ketamine	0.35mg/kg/hr	10	19(26)	1	-	41.9±9.8	7.7±1.5	6.01±0.6	26.3	Effective	5	6.6±5.8 months	Nausea, headache, tiredness or dysphoria ;none reported agitation, blurred vision, hallucinations, delusions
Goldberg, M et al	2010	S+ketamine	10–40mg/hr	5	16	1	67.9±19.3	33±10.2	8.5±1.6	5.4±0.8	62.5	0.6	37.5	Fulfill CRPS diagnosis criteria	No evidence of significant tachycardia, arterial oxygen desaturation, hallucinatory responses
Patil, S et al	2012	S+ketamine	0.9±0.4mg/kg	43.8	18	7	79.9±21.3	42±8.5	8.2±1.7	0.8±0.6	77	38	27	5.62 years (6 months unrelenting pain)	Hypertension and sedation
Goebel, A et al	2015	Racemic ketamine	44.6±21.9mg/hr	4.5	5	3	-	44.6±6.9	8.2±1.2	4.5±3.7	83.3	33.3	17.7	>1 year	Liver enzymes were mildly raised
Koffler, S et al	2007	S+ketamine	0.25–0.3mg/dl	4.5	9	1	-	29.1±9.43	54.44±9.77	29.0±20.26	-	-	-	55.6±24.75 months	Muscle weakness, dizziness, fatigue, episodes of hyperhidrosis
Schilder, J et al	2013	S+ketamine	22.2±2.0 mg/hr/70kg	4.5	15	2	-	42.3±13.7	6.9±1.3	2±0.8	-	-	-	9.6±9.9 months	No direct effect on motor function over 12 weeks follow up
Sheehy, K et al	2015	S+ketamine	0.1–0.3 mg/kg/hr	3	23	-	-	14.8±1.25	5.63±2.3	4.53±2.46	-	-	-	Fulfill CRPS diagnosis criteria	Psychiatric/psychological disorders
Sigtermans, M et al	2009	S+ketamine	22.2±2.0 mg/hr/70 kg	4.5	30	8	78.9±17.8	43.7±11.5	7.2±1.2	2.68±0.51	-	-	-	9.4±8.0 months	Psychomimetic side effects, nausea, vomiting
Sigtermans, M et al	2010	S+ketamine	0.08–0.26mg/hr/kg	1	10	0	68.9±10.8	43.2±13.0	6.2±0.2	0.4±0.3	-	-	-	8.4±6.1 years	Dose-dependent antinociception, psychomimetic side effects
Dahan, A et al	2011	S+ketamine	20±4mg/hr/70 kg	4.2	30	8	79±19	46±12	7.29±0.16	4.38±0.29	67	-	32	Fulfill CRPS diagnosis criteria	Psychomimetic side effects
Kiefer, R et al	2002	S+ketamine	0.15–7mg/kg/hr	5	6	-	-	-	-	-	-	66.7	0	Fulfill CRPS diagnosis criteria	-

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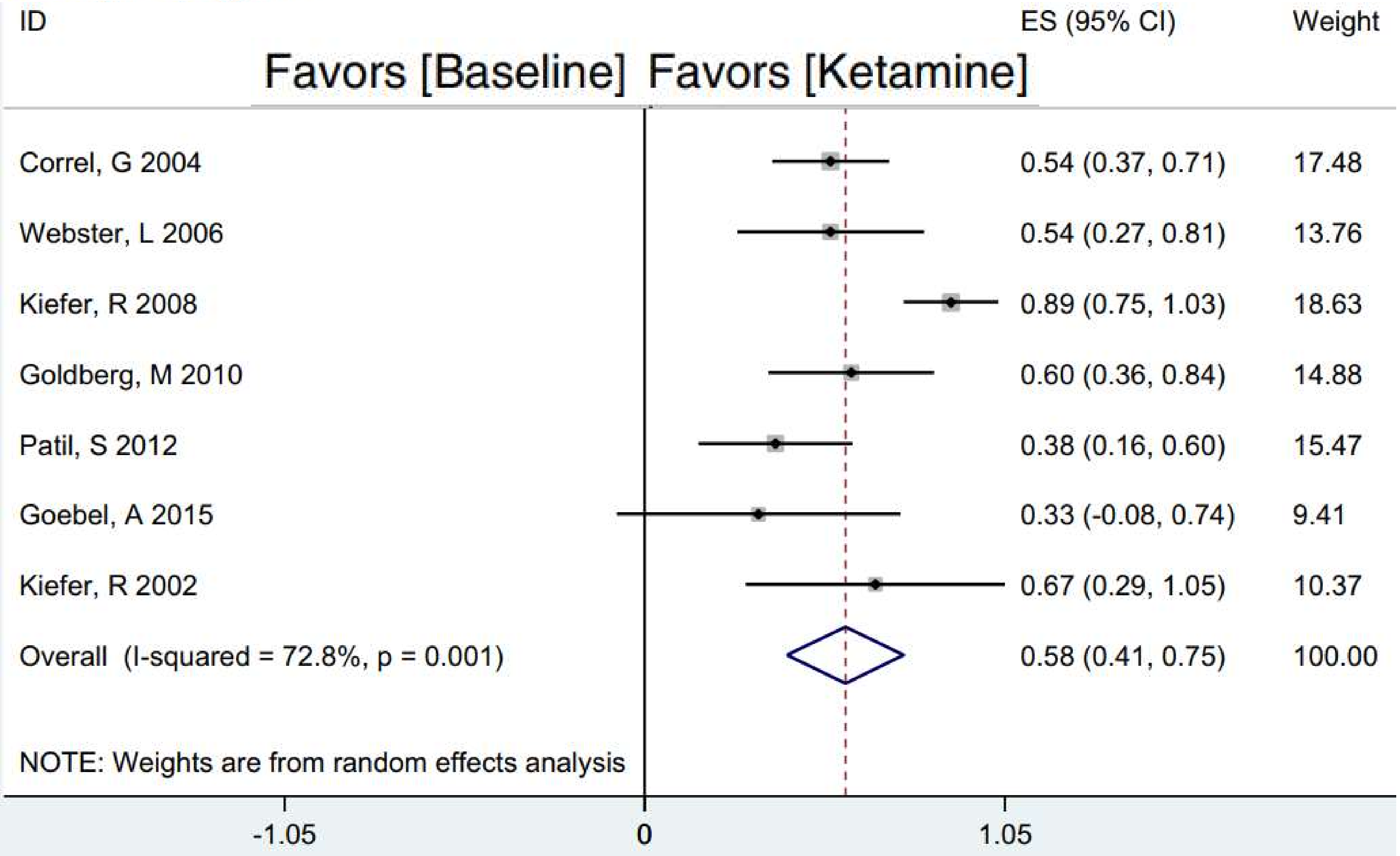
The forest plot for pain relief event rate at 1–3 months follow-ups. All studies assessed pain relief rates



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Proposition de protocoles d'utilisation (respectant l'AMM)

Proposition de protocoles pour la douleur chronique (hors AMM)

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A propos de la note eskétamine de la SFAR

Merci