THIAMINE ET TROUBLES COGNITIFS CHEZ LES PATIENTS PRESENTANT UN TROUBLE DE L'USAGE D'ALCOOL (TUAL)

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Conflits d’intérêt

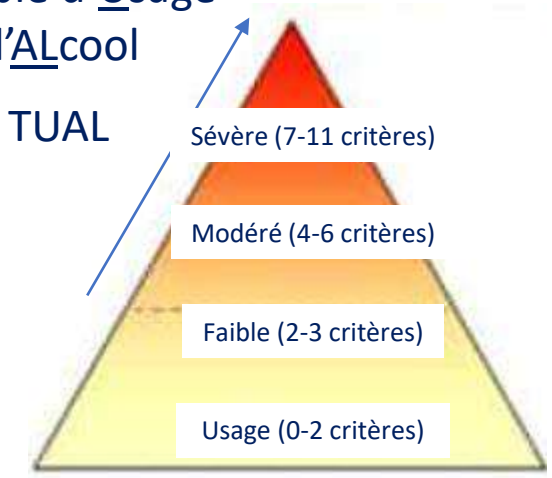
AUCUN

Troubles cognitifs et consommation d'alcool

Consommation chronique d'alcool → Encéphalopathie de Gayet-Wernicke → Syndrome de Korsakoff

Trouble d'Usage d'ALcool

TUAL



Critères DSM-5

Critères de Caine, 1997

- Confusion
- Signes oculomoteurs
- Syndrome cérébelleux
- Déficits nutritionnels documentés (IMC, dénutrition …)

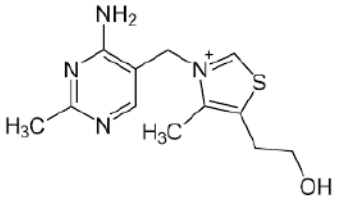
Consommation excessive

- Atteintes hépatiques et cardiovasculaires
- Troubles nutritionnels
- Vieillissement accéléré
- Troubles cognitifs : 50 à 80 % des patients TUAL

Syndrome de Korsakoff

- Troubles neurocognitifs sévères
- Conséquences sociales majeures
- 600 à 900 nouveaux cas par an en France
- Prévalence (sous-)estimée en France : 0,02% de la population
- Stigmatisation des patients
- Autres causes que alcool
- Etude APHP (2025) : Mortalité à 3 ans ~30%
- Carence de repérage, de diagnostic et de prise en charge

Facteurs influençant le développement des troubles cognitifs chez le patient avec un TUAL



Sévérité du sevrage

Fonction hépatique

Qualité du sommeil

Histoire de l'alcoolisation

Nutrition, Déficit en Thiamine et encéphalopathie de Gayet-Wernicke

Atteintes cérébrales et troubles cognitifs

La thiamine : aspects biochimiques

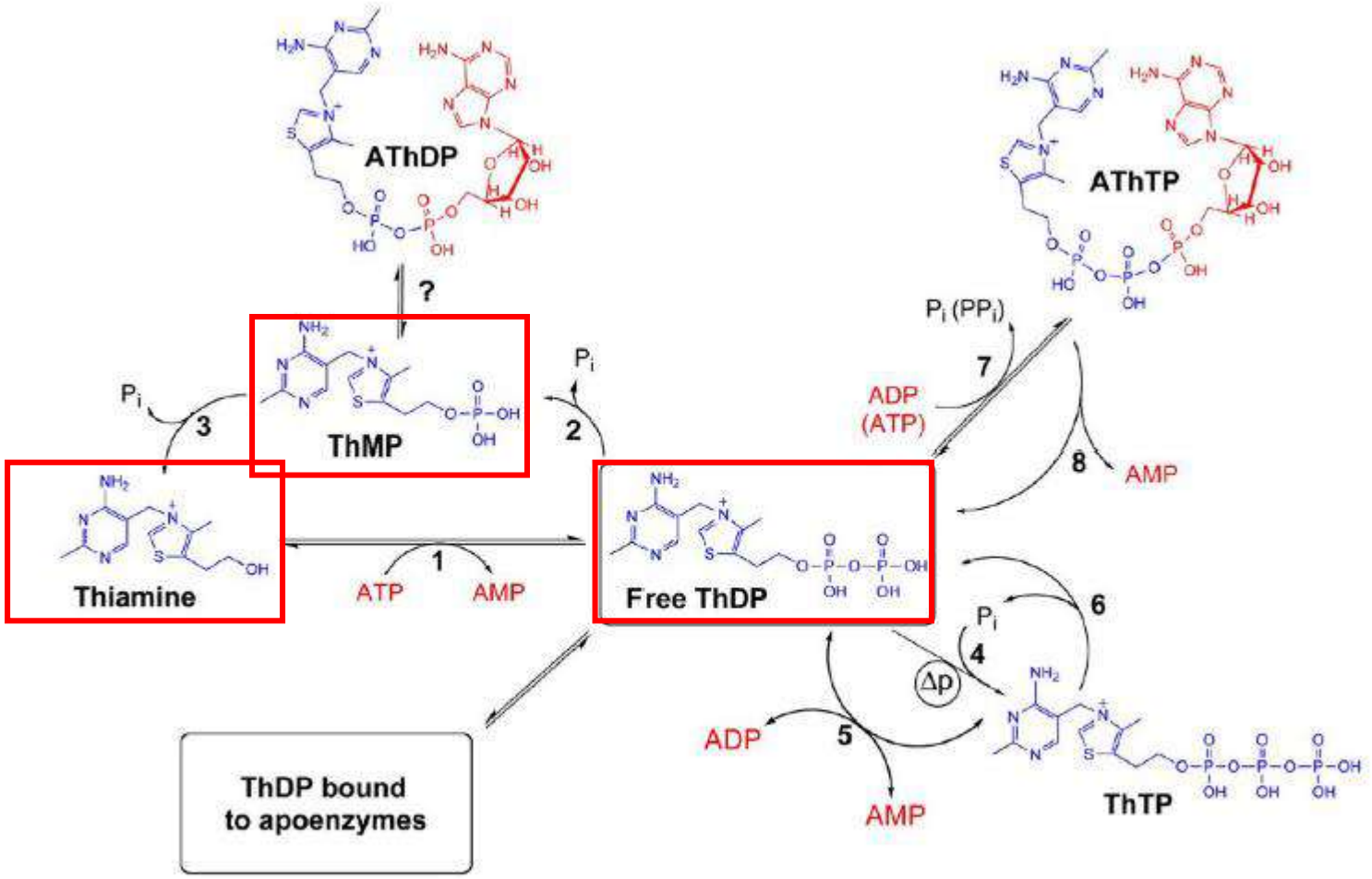
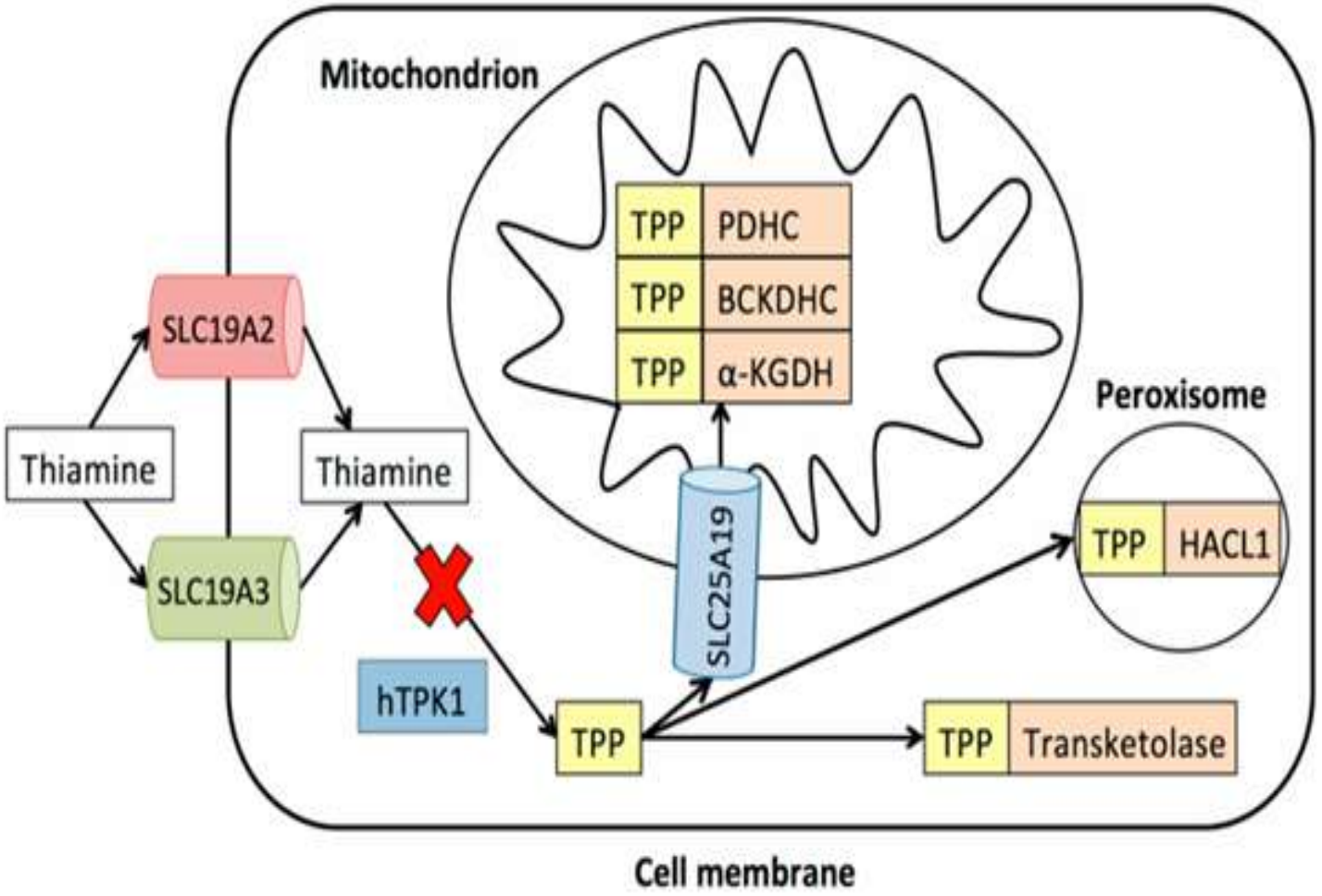


Figure 1. Interconversion of thiamine derivatives in a model human cell. 1, cytosolic thiamine diphosphokinase (TPK); 2, cytosolic thiamine diphosphatase (also hydrolyzes nucleoside diphosphates); 3, thiamine monophosphatase (hypothetical); 4, mitochondrial membrane-associated ThTP synthase; 5, cytosolic adenylate kinase; 6, cytosolic 25-kDa ThTPase; 7, cytosolic ThDP adenylyl transferase; 8, AThTP hydrolase (hypothetical). The mechanisms of AThDP synthesis and degradation are unknown. Δp, transmembrane H^+ gradient. Updated from [21].
doi:10.1371/journal.pone.0013616.g001

Gangolf *et al.*, PLOS One, 2010

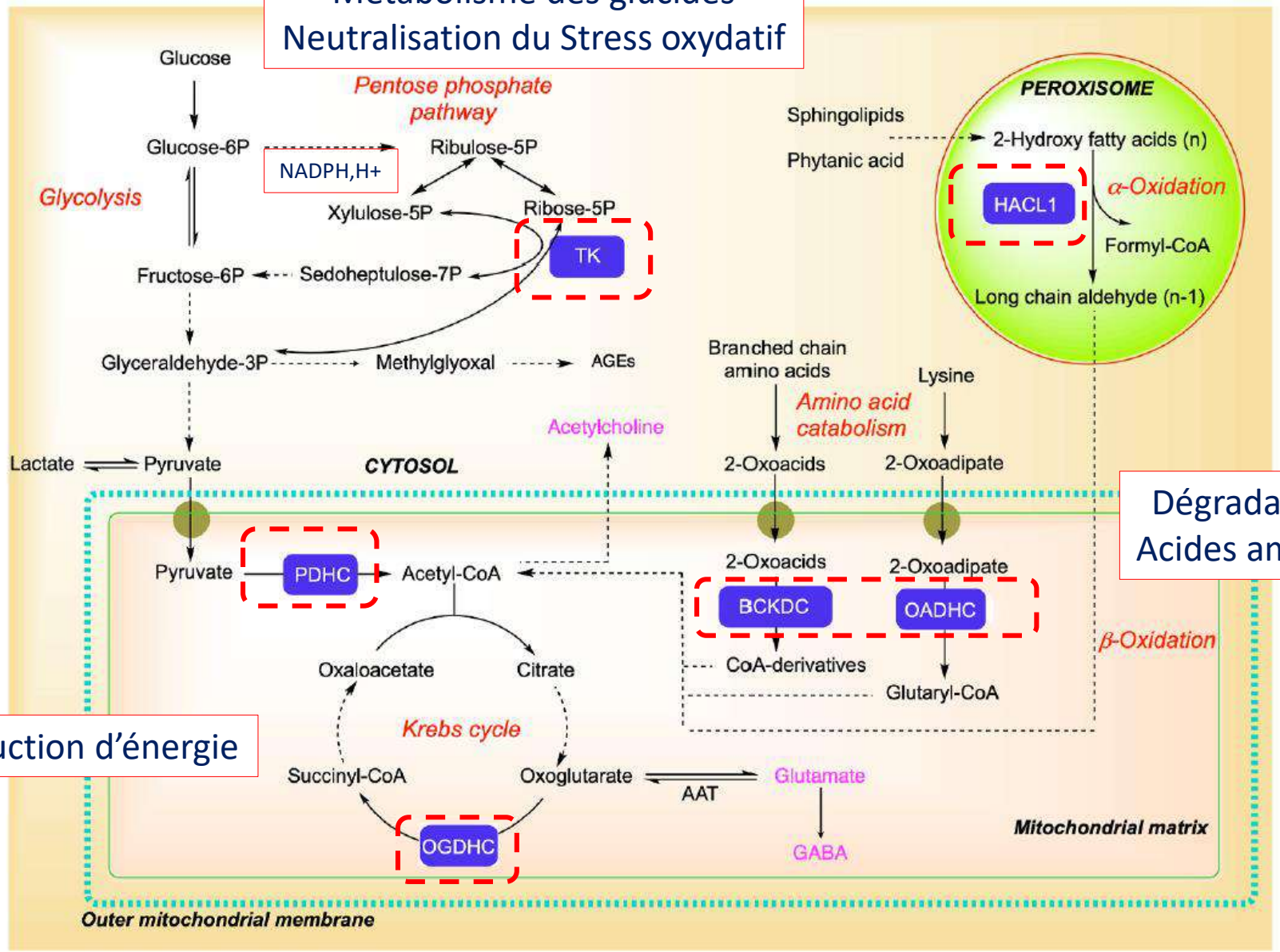
Thiamine et transport intracellulaire



Buggiardini *et al.*, J Clin Med, 2019

La thiamine : un acteur essentiel dans le métabolisme

Métabolisme des glucides
Neutralisation du Stress oxydatif



Dégradation
Acides gras

Dégradation
Acides aminés

Production d'énergie

Makarchikov *et al.*, Neurochem Int, 2025

Distribution tissulaire de la thiamine et des formes phosphorylées

Sang, plasma

Table 1. Distribution of thiamine derivatives in human whole blood, plasma and CSF.

(nmol/L)	Thiamine	ThMP	ThDP	ThTP	AThTP
Whole blood (7)	4±3	10±4	138±33	13±4	n. d.
Plasma (3)	11±3	5±2	n. d.	n. d.	n. d.
CSF (3)	19±5	30±5	n. d.	n. d.	n. d.

The blood was from 7 healthy volunteers aged from 25 to 49 years. Aliquots of three samples were centrifuged to obtain a plasma preparation. CSF was from three patients with no known neurological disorder. The results are expressed as means ± SD. The numbers in parentheses indicate the number of patients. n. d., not detected.

- Formes phosphorylées (TDP/TMP) majoritaires dans le sang total et les tissus
- TDP : Forme dosée dans le sang
- Thiamine ~65-70% des formes dans le plasma

Cerveau

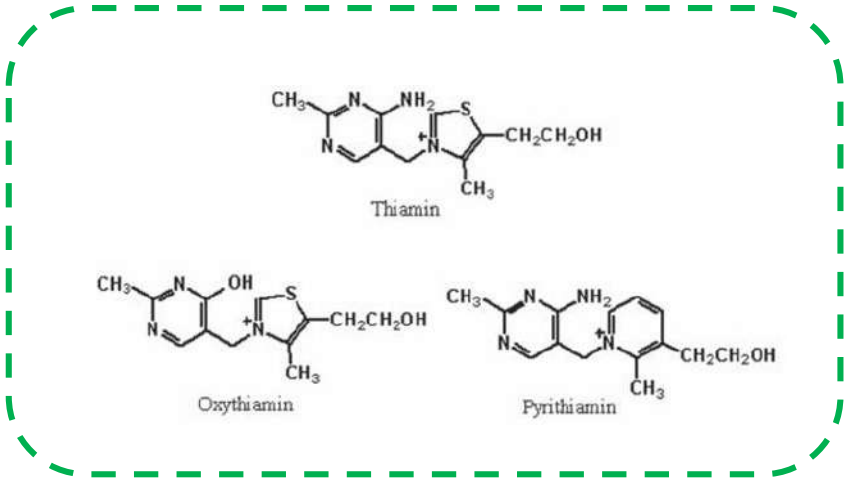
Table 7. Thiamine derivatives in brain samples.

	(pmol/mg of protein)	Thiamine	ThMP	ThDP	ThTP	AThTP
Pig	Cerebral cortex (3)	13±1	5.5±1.1	66±1	1.5±0.4	n. d.
Quail	Forebrain (3)	0.9±0.1	2.0±0.4	116±21	2.8±0.6	n. d.
Rat	Brainstem (5)	4.3±0.4	10±2	115±12	0.50±0.12*	0.2±0.3
	Right hemisphere (5)	4.4±0.6	9±1	153±65	0.35±0.09	0.1±0.2
	Left hemisphere (5)	5.2±1.6	9±2	148±8	0.36±0.08	0.2±0.3
	Cerebellum (5)	4.4±2.6	14±2	168±54	0.31±0.05	0.1±0.2
Human	Cerebral cortex[a] (5)	0.2±0.3	3.5±2.6	21±5	0.4±0.3	n. d.

[a]Peritumoral cerebral tissue, from patients with temporal lobe glioblastoma.
*$p<0.05$, compared with cerebellum.
The results are expressed as mean ± SD. n.d., not detected.

Gangolf *et al.*, PLOS One, 2010

Modèles expérimentaux de déficit en thiamine



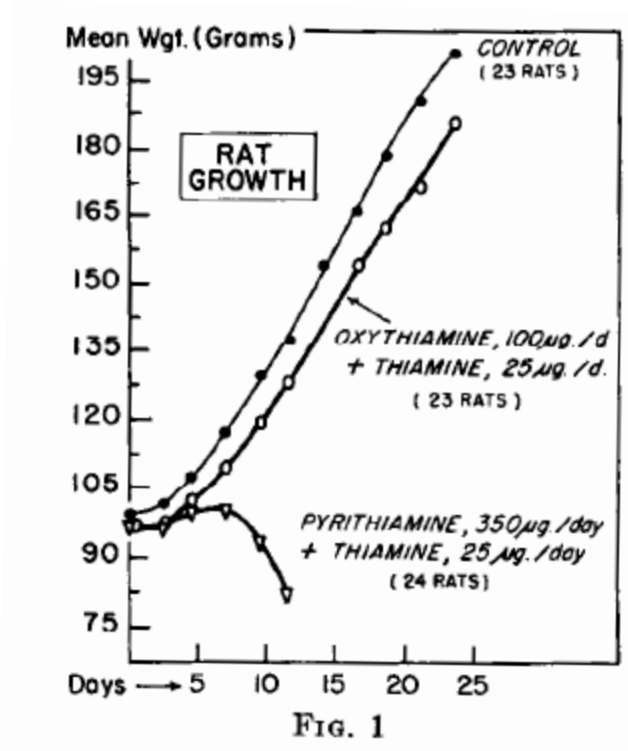


FIG. 1

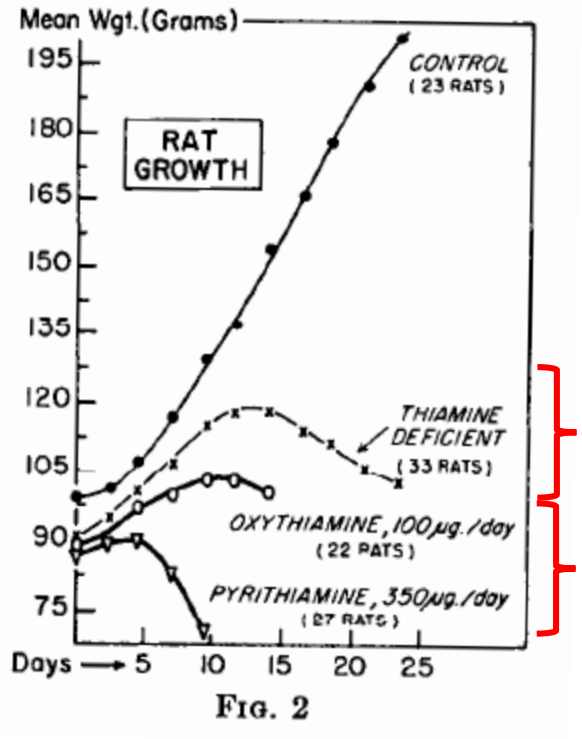


FIG. 2

➢ Effet sur la croissance

Wolfe SJ, J Biol Chem, 1957

Atteintes cérébrales dans un modèle expérimental de carence en thiamine

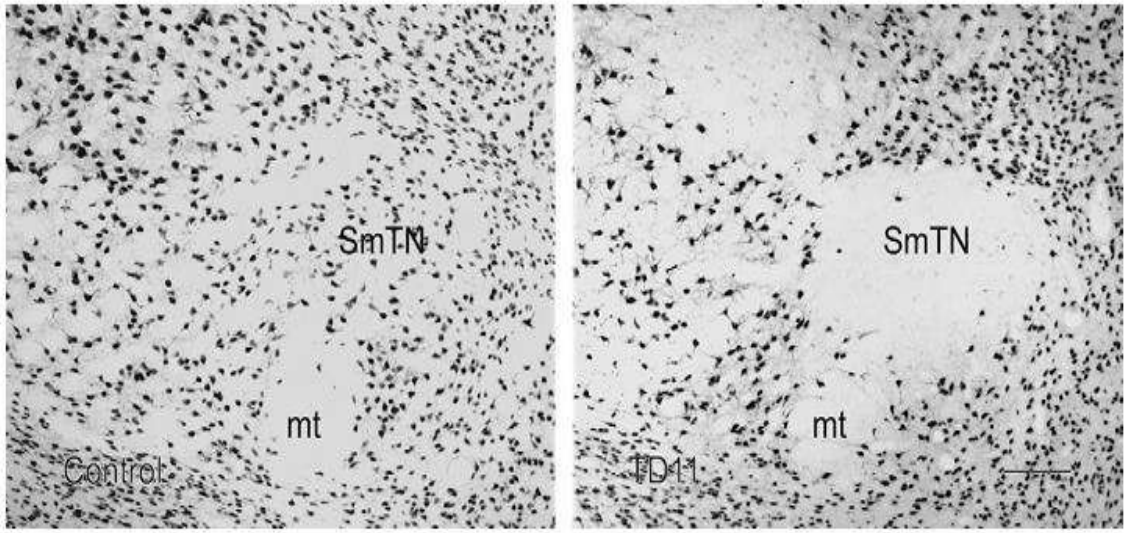


Fig. 1. NeuN immunoreactivity in the thalamus of C57BL/6 mice in control, and after 11 days of TD. The results show the pattern of neuronal loss in the SmTN during TD. SmTN lies medial to the mammillothalamic tract (mt). Scale bar = 150 μm.

- Mort des neurones dans des régions spécifiques
- Activation de la microglie

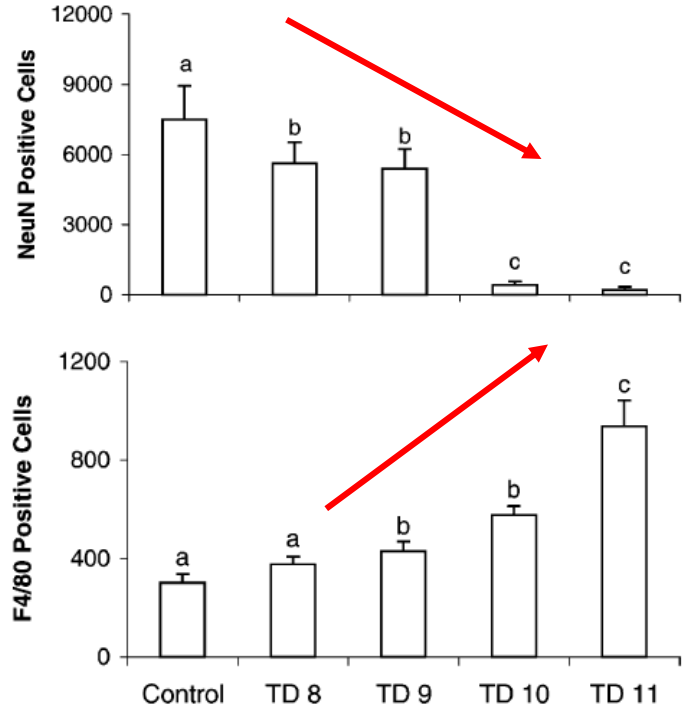


Fig. 2. Temporal response of neurons and microglia to TD. NeuN-positive neurons (top) and F4/80-positive microglia (bottom) in the SmTN at different stages of TD. Mice were made TD for the indicated number of days. Columns with different letters vary significantly from each other.

Ke ZJ and Gibson GE, Neurochem International, 2004

Atteintes cérébrales dans un modèle expérimental utilisant un inhibiteur métabolique

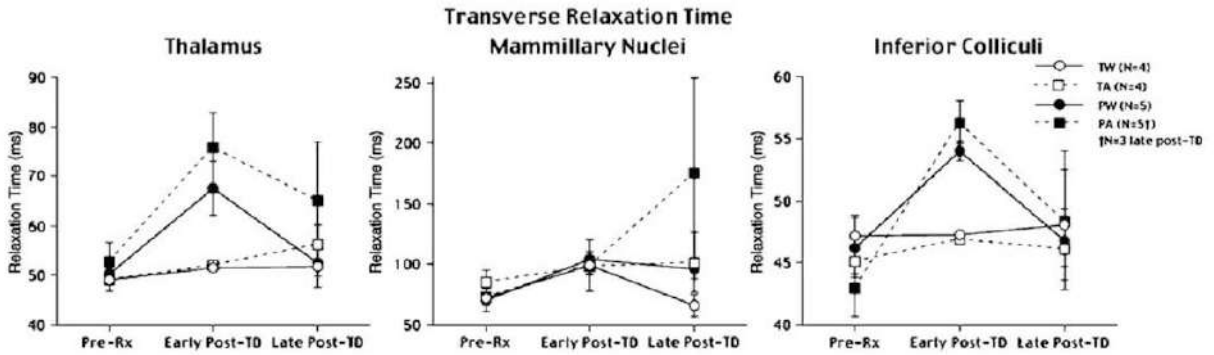


Figure 5 Mean ± SEM of transverse relaxation time in three brain structures at three experiment times for each of the four groups: TW = thiamine + water pre-exposure group; TA = thiamine + alcohol pre-exposure group; PW = pyrithiamine + water pre-exposure group; and PA = pyrithiamine + alcohol pre-exposure group. TD = thiamine deficiency.

- ○ Thiamine - water
- □ Thiamine – alcohol
- ● Pyrithiamine – Water
- ■ Pyrithiamine - alcohol

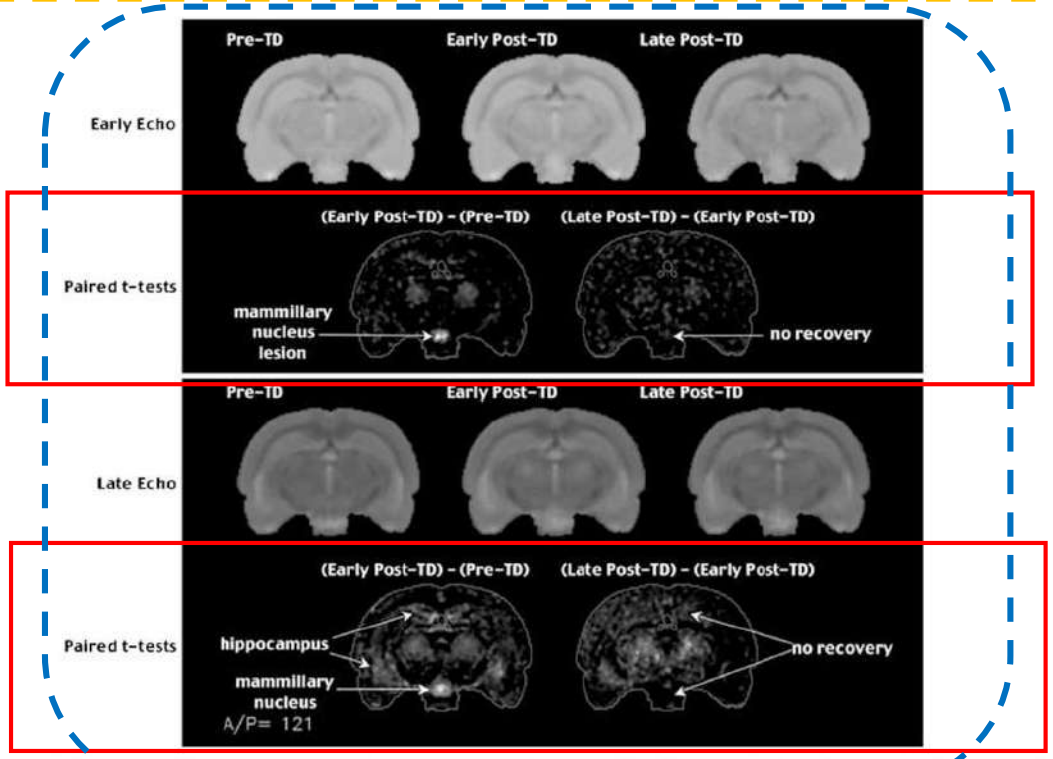


Figure 10 Top panel of five coronal images are based on early-echo data, and the bottom panel presents the late-echo data. The bottom two images in each panel show the outline of each image and the voxel-by-voxel *t*-test differences meeting a $p < 0.01$ statistical significance criterion between pre-TD and early post-TD images (left) and between early and late post-TD images.

- La carence en thiamine engendre des atteintes irréversibles des corps mamillaires et de l'hippocampe
- Effet aggravant de l'alcool

Pfefferbaum *et al.*, Neuropsychopharmacology, 2009

Concentrations tissulaires en thiamine dans un modèle expérimental de carence d'apport

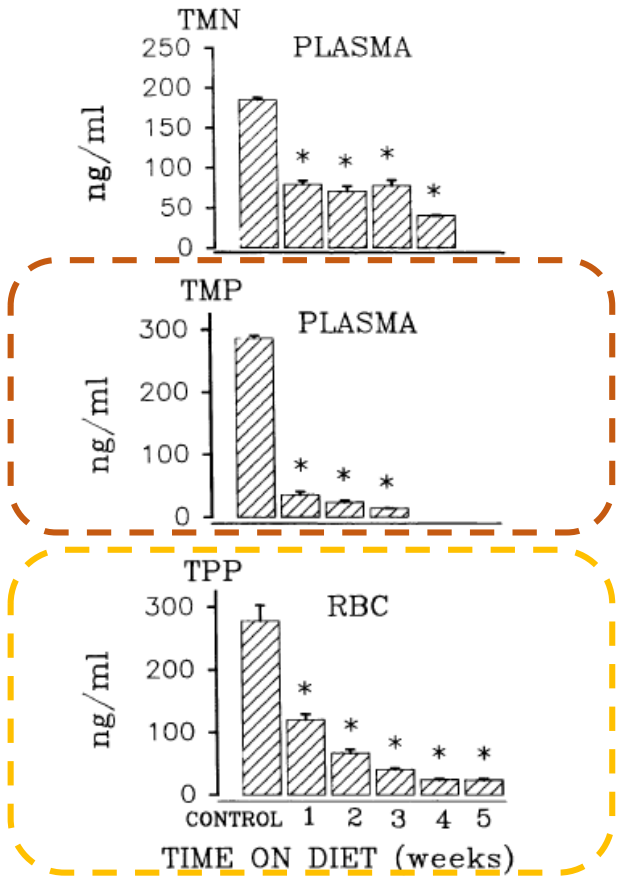


FIG. 2. Plasma TMN (top panel) and TMP (middle panel) and RBC TPP (lower panel) concentrations in control and deficient rats expressed as nanograms per milliliter over time on the diet. Values are means ± SEM. n = 6 to 7 per group; *$p < .05$ compared with control animals.

- Chute du TDP dans RBC
- Chute du TMP plasmatique

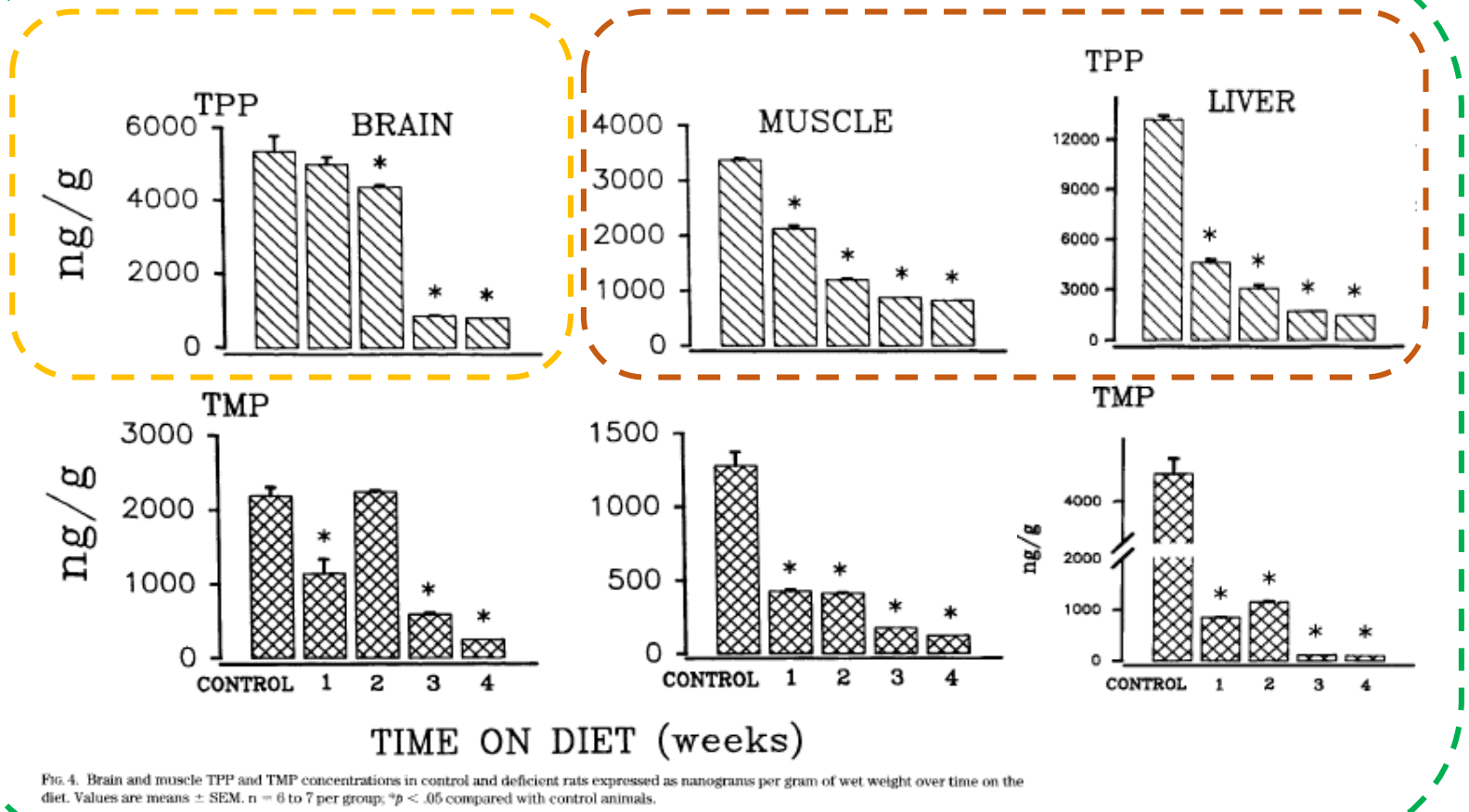


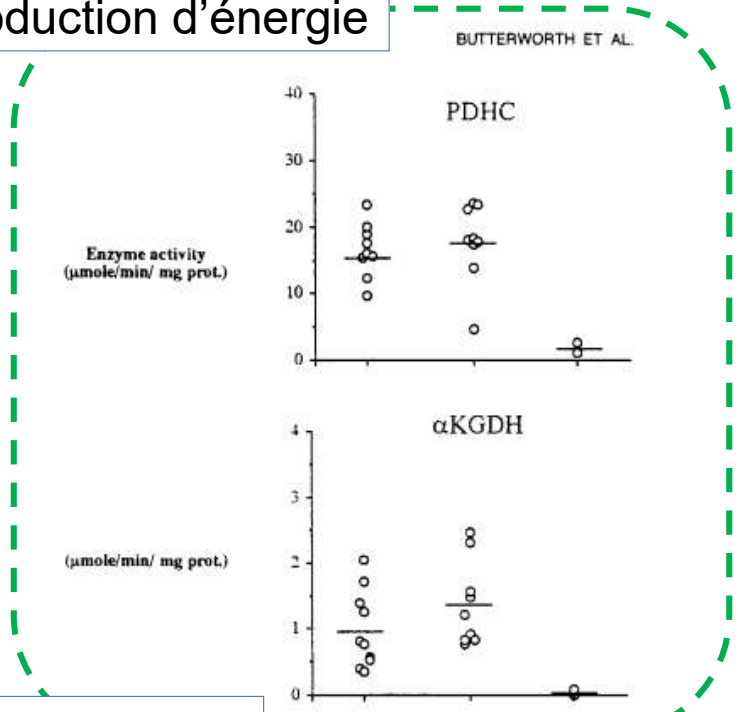
FIG. 4. Brain and muscle TPP and TMP concentrations in control and deficient rats expressed as nanograms per gram of wet weight over time on the diet. Values are means ± SEM. n = 6 to 7 per group; *$p < .05$ compared with control animals.

- Préservation du cerveau vis-à-vis de la déplétion
- Moindre utilisation de la thiamine dans le cerveau par rapport au foie ?
- Quid en présence d'alcool ?

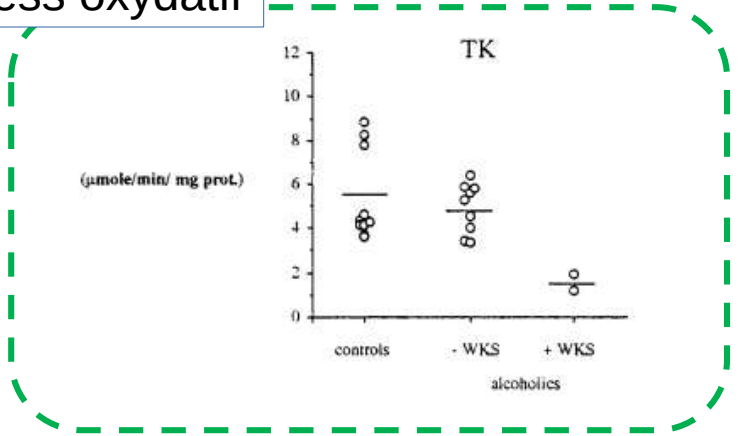
Molina *et al.*, JPEN, 1994

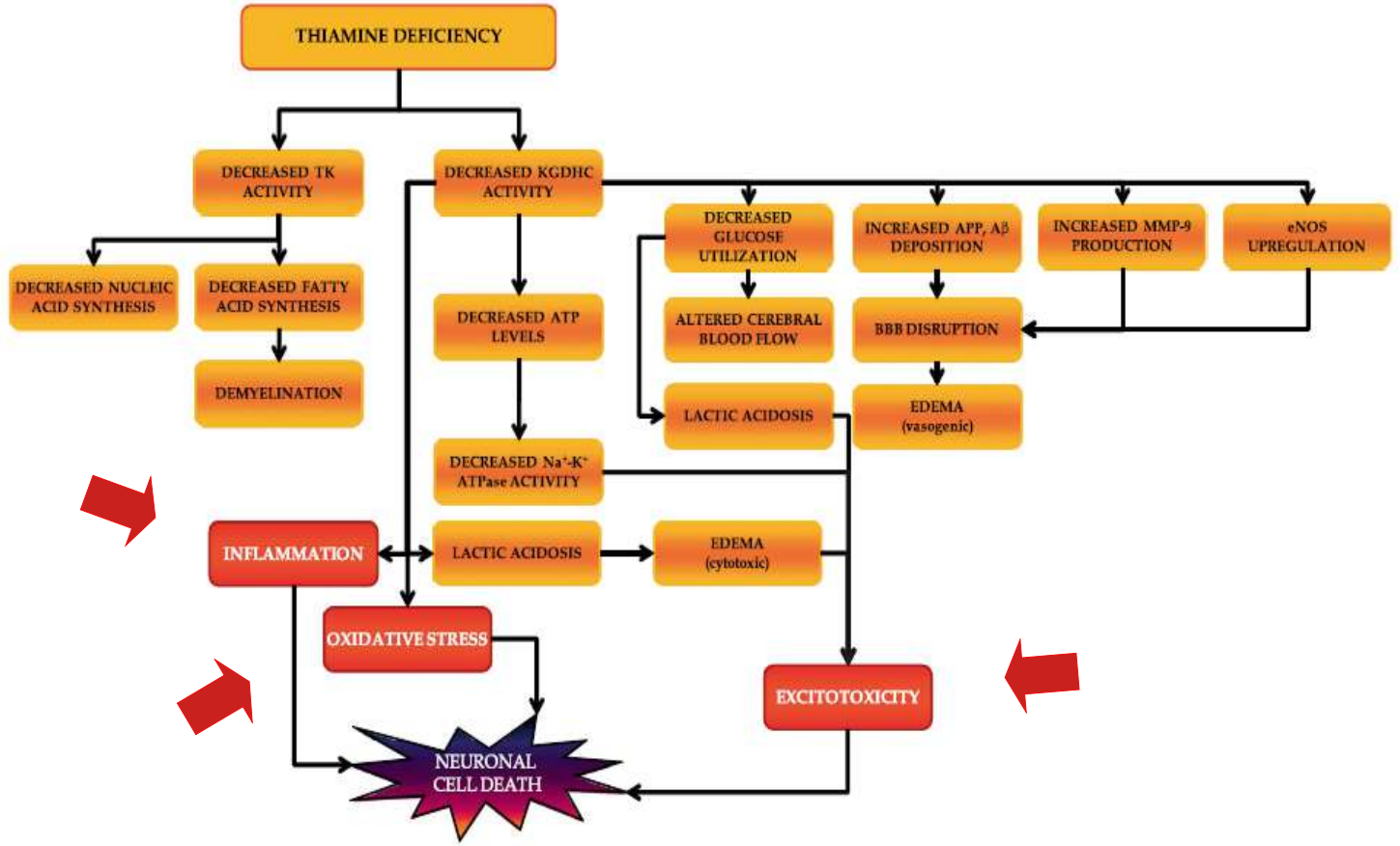
Mécanismes physiopathologiques au cours de la carence en thiamine

Production d'énergie



Stress oxydatif





Butterworth *et al*., ACER, 1993 ; Hazell *et al*., Neurochem Int, 2013

Consommation d'alcool et statut en thiamine

- Diminution du TDP sanguin avec la consommation chronique d'alcool
- Pas retrouvée chez tous les patients

Table 1
Comparison of transketolase activity (ETK activity), TDP effect, thiamine and its phosphate esters between erythrocytes from healthy and alcoholic subjects

Parameter	Healthy subjects	Alcoholic subjects		
		Groups I and II	Group I	Group II
n	52	85	53	32
TTP (nmol/l)	7.0 ± 6.0	4.9 ± 3.9[a]	4.8 ± 3.4[a]	5.1 ± 4.7[a]
TDP (nmol/l)	176 ± 28.0	148 ± 54.7[b]	182 ± 27.8[a]	90.8 ± 25.7[b]
TMP (nmol/l)	<2	<2	<2	<2
T (nmol/l)	4.0 ± 2.0	2.9 ± 2.7[a]	3.2 ± 2.7[a]	2.6 ± 2.6[a]
Total T (nmol/l)	186 ± 30.0	156 ± 55.6[b]	191 ± 27.8[a]	99 ± 20.9[b]
Percentage of phosphorylation[c]	98.1 ± 1.3	98.0 ± 2.6[a]	98.4 ± 1.9[a]	97.6 ± 2.5[a]
TDP/total T (%)	94.3 ± 4.1	93.4 ± 5.7[a]	95.5 ± 3.5[a]	91.8 ± 5.8[a]
ETK activity (units/l)	165 ± 20.6	153 ± 51.1[a]	172 ± 22.8[a]	122 ± 37.2[b]
TDP effect (%)	11.6 ± 10.0	22.4 ± 21.7[b]	10.6 ± 4.6[a]	41.8 ± 24.7[b]

Values are means ± S.D.
[a]Statistically non-significant difference ($P > 0.05$).
[b]Statistically significant difference ($P < 0.001$).
[c]Percentage of phosphorylation = [(TTP + TDP + TMP)/(total T)] × 100.

Hervé *et al.*, Alcohol Alcoholism, 1994

Statut en thiamine chez des patients avec une encéphalopathie de Gayet-Wernicke

Thiamine diphosphate TDP

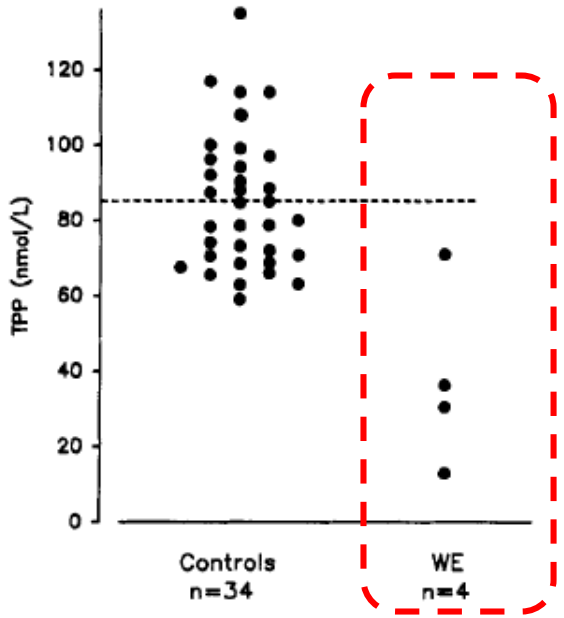


Fig. 3. Whole blood concentrations of TPP in controls (n = 34) and patients with Wernicke encephalopathy (WE; n = 4). The mean concentration of the controls is indicated as a dotted line.

Thiamine monophosphate TMP

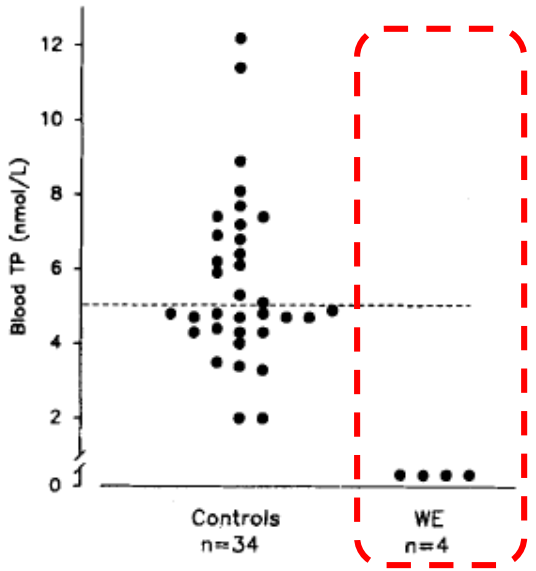


Fig. 2. Whole blood concentrations of TP in controls (n = 34) and patients with Wernicke encephalopathy (WE; n = 4). The mean concentration of the controls is indicated as a dotted line. The patients' values are all below detection level (1 nmol/liter).

Thiamine

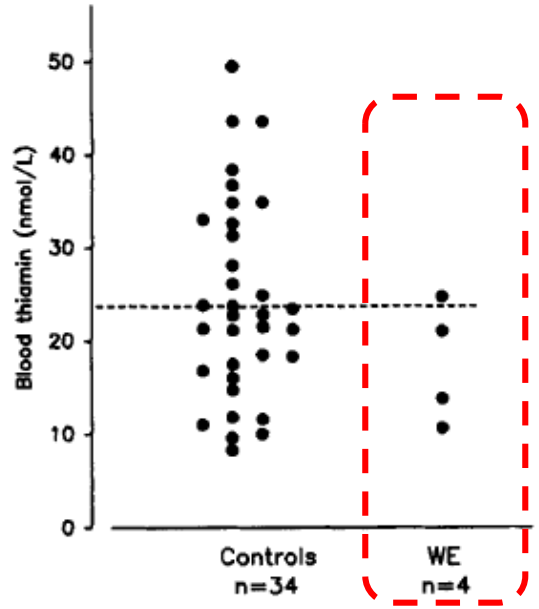


Fig. 1. Whole blood concentrations of T in controls (n = 34) and patients with Wernicke encephalopathy (WE; n = 4). The mean concentration of the controls is indicated as a dotted line.

Tallaksen *et al.*, Alcohol Alcoholism, 1994

Alcool et statut nutritionnel : données récentes

Table 1. Biomarkers describing nutritional status and their association with MoCA score <26 points.

	Mean	SD	Median	*N*	NR	Above NR *N* (%)	Below NR *N* (%)	Association with MoCA score < 26[a] Khi^2 (*N*)	Sig.
Albumin (g/l)	39.5	5.56	38.7	80	38.5–47.5	3 (3.8)	37 (46.3)	0.667 (*N* = 52)	0.557
Prealbumin (g/l)	0.27	0.09	0.26	85	0.27–0.34	23 (27.1)	43 (50.6)	0.750 (*N* = 53)	0.562
Calcium (mmol/l)	2.39	0.14	2.37	43	2.2–2.55	6 (14)	2 (4.7)	0.633 (*N* = 29)	1
Phosphorus (mmol/l)	1.02	0.24	1.07	29	0.74–1.52	0 (0)	4 (13.8)	2.015 (*N* = 17)	0.471
Magnesium (mmol/l)	0.78	0.10	0.79	81	0.65–1.05	0 (0)	9 (11.1)	0.021 (*N* = 50)	1
Vitamin A (µmol/l)	2.62	1.24	2.06	14	1.5–2.7	4 (28.6)	1 (7.1)	-	-
Vitamin B1 nmol/l)	153	62.8	141	52	60–200	15.3	0 (0)	-	-
Vitamin B3 µmol/l)	130	46.2	126	12	55–98	9 (75)	0 (0)	-	-
Vitamin B6 nmol/l)	189	165	119	34	35–110	20 (58.8)	0 (0)	-	-
Vitamin B9 (µg/l)	6.35	2.51	5.7	87	4.6–18.7	0 (0)	25 (28.7)	0.162 (*N* = 54)	0.766
Vitamin B12 (ng/l)	451	252	398	82	197–771	6 (7.3)	9 (11)	2.931 (*N* = 51)	0.168
Vitamin C (mg/l)	5.33	4.41	3.2	15	5.0–15	0 (0)	8 (53.3)	2.744 (*N* = 10)	0.183
Vitamin D (ng/ml)	21.3	10.5	19.6	75	20–80	0 (0)	39 (52)	1.042 (*N* = 47)	0.371
CRP (mg/l)	9.64	27	0	83	<6	25 (30.1)	-	0.944 (*N* = 52)	0.529

66% des patients avec un score MOCA indiquant des troubles cognitifs

- Qualité de la prise en charge ? supplémentation intermittente ?

Gautron *et al.*, Alcohol Alcoholism, 2018

Syndrome de korsakoff : facteurs environnementaux et génétiques

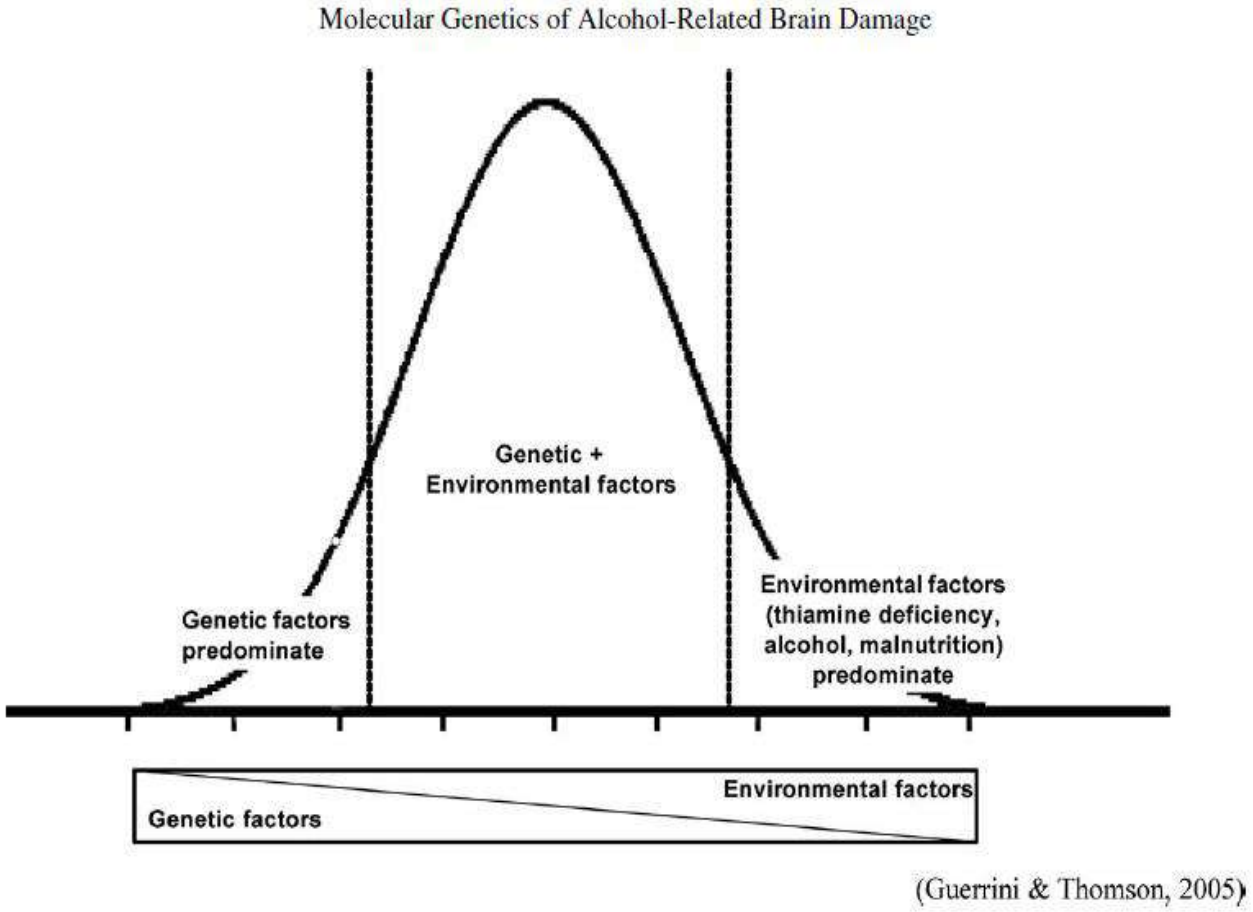


Fig. 2. Wernicke–Korsakoff syndrome—a pathogenetic model.

Guerrini *et al.*, Alcohol Alcoholism, 2005

Syndrome de korsakoff : aspects génétiques

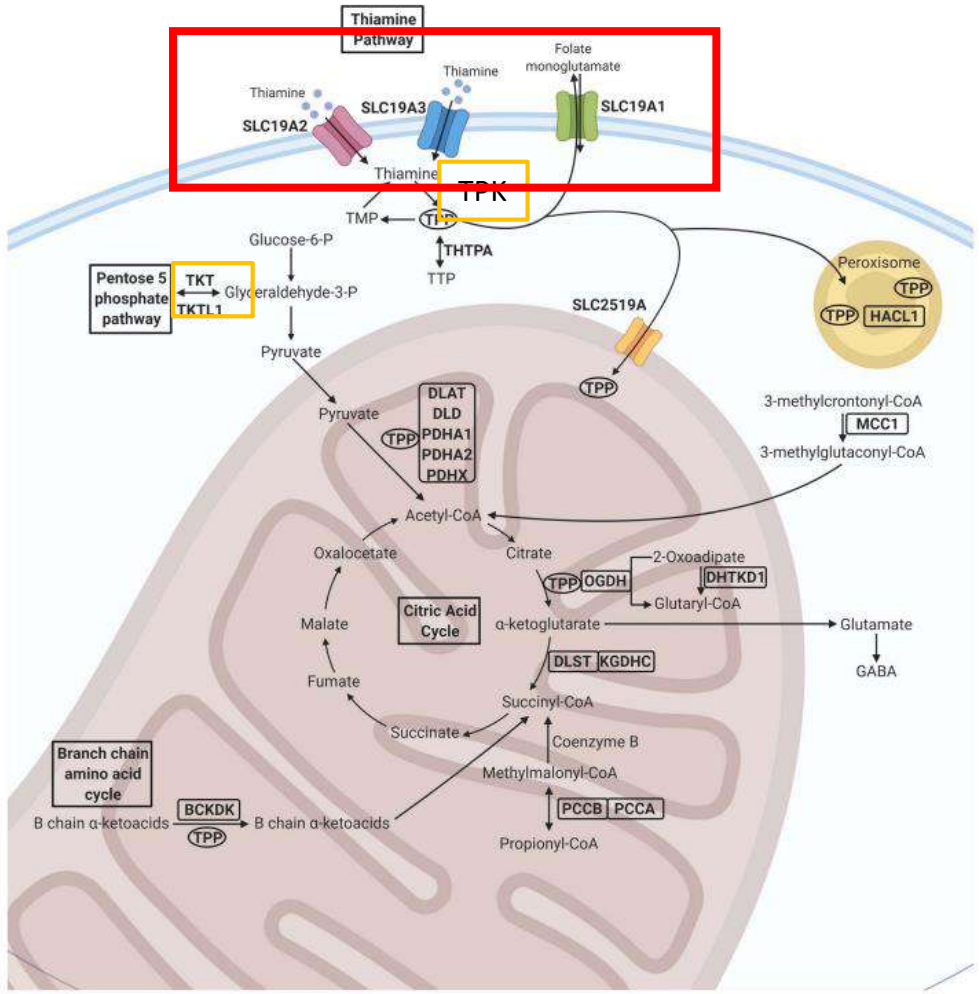


Fig. 1. Thiamine transport and metabolism pathways. Thiamine enters the cell via two transporters, SLC19A2 and SLC19A3. Within the cell, it is converted into its active form, TPP, which is required for several essential metabolic pathways. The SLC19A1 transporter is involved in the efflux of TPP and the influx of folate monoglutamate. The key genes involved in these metabolic processes are shown in bold.

JOURNAL ARTICLE

Molecular Genetics of Alcohol-Related Brain Damage

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Irene Guerrini ✉, Allan D. Thomson, Hugh M. Gurling

Alcohol and Alcoholism, Volume 44, Issue 2, March-April 2009, Pages 166–170,

› Am J Med Genet B Neuropsychiatr Genet. 2005 Aug 5;137B(1):17-9. doi: 10.1002/ajmg.b.30194.

Direct genomic PCR sequencing of the high affinity thiamine transporter (SLC19A2) gene identifies three genetic variants in Wernicke Korsakoff syndrome (WKS)

Irene Guerrini [1], Allan D Thomson, Cristopher C H Cook, Andrew McQuillin, Vishal Sharma, Michael Kopelman, Gerald Reynolds, Pramod Jauhar, Clive Harper, Hugh M D Gurling

Alcohol and Alcoholism, 2022, **57**(5), 581–588
https://doi.org/10.1093/alcalc/agac032
Advance access publication date 12 August 2022
Article

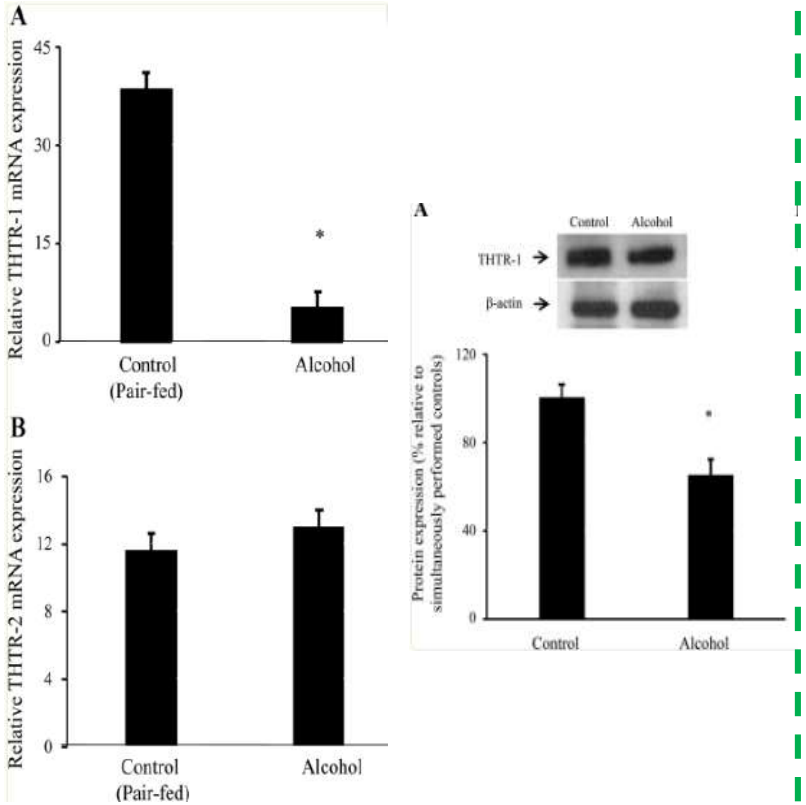
OXFORD

SLC19A1 Genetic Variation Leads to Altered Thiamine Diphosphate Transport: Implications for the Risk of Developing Wernicke–Korsakoff's Syndrome

Niamh L. O'Brien[1], Giorgia Quadri[1], Iain Lightley[2], Sally I. Sharp[1], Irene Guerrini[3,4], Iain Smith[5], Mathis Heydtmann[6], Marsha Y. Morgan[7], Allan D. Thomson[1,4], Nicholas J. Bass[1], Patrick C. McHugh[2,†], and Andrew McQuillin[1,*,†]

Effet de l'alcool sur l'expression des transporteurs de la thiamine (vit B1)

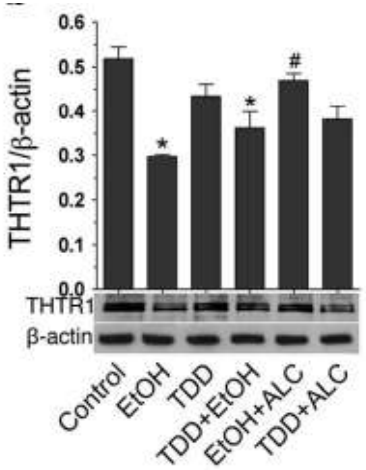
Entérocytes

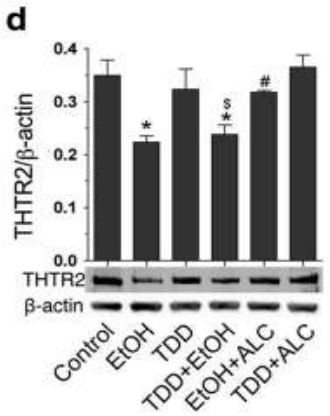


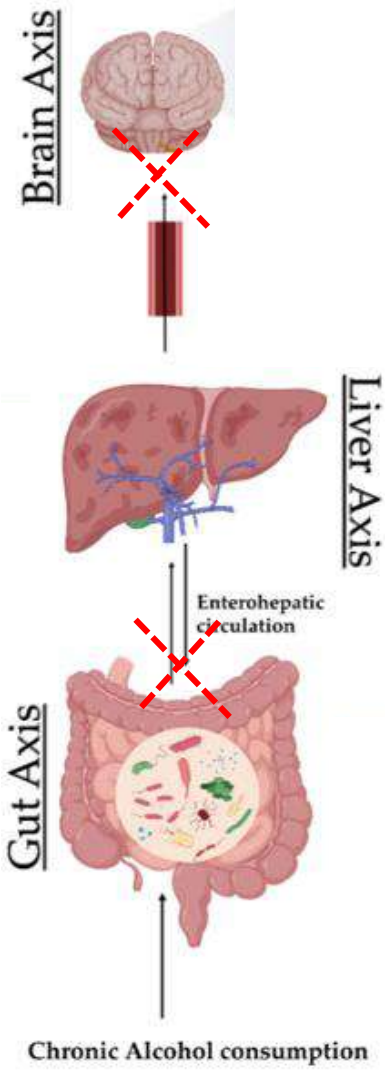
THTR-1 : SLC19A2
THTR-2 : SLC19A3

Subramanya *et al.*, Am. J Physiol Liver Physiol, 2010

Cellules de la barrière hémato-encéphalique

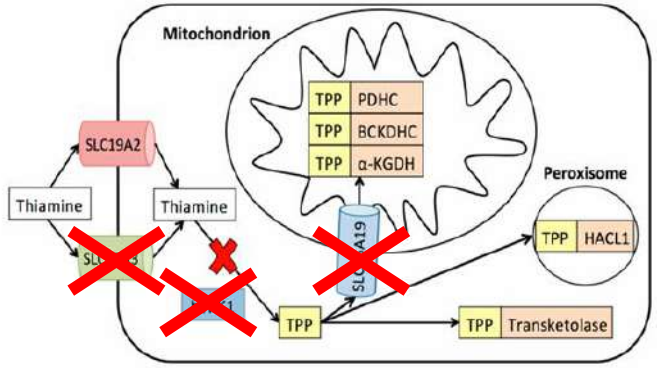


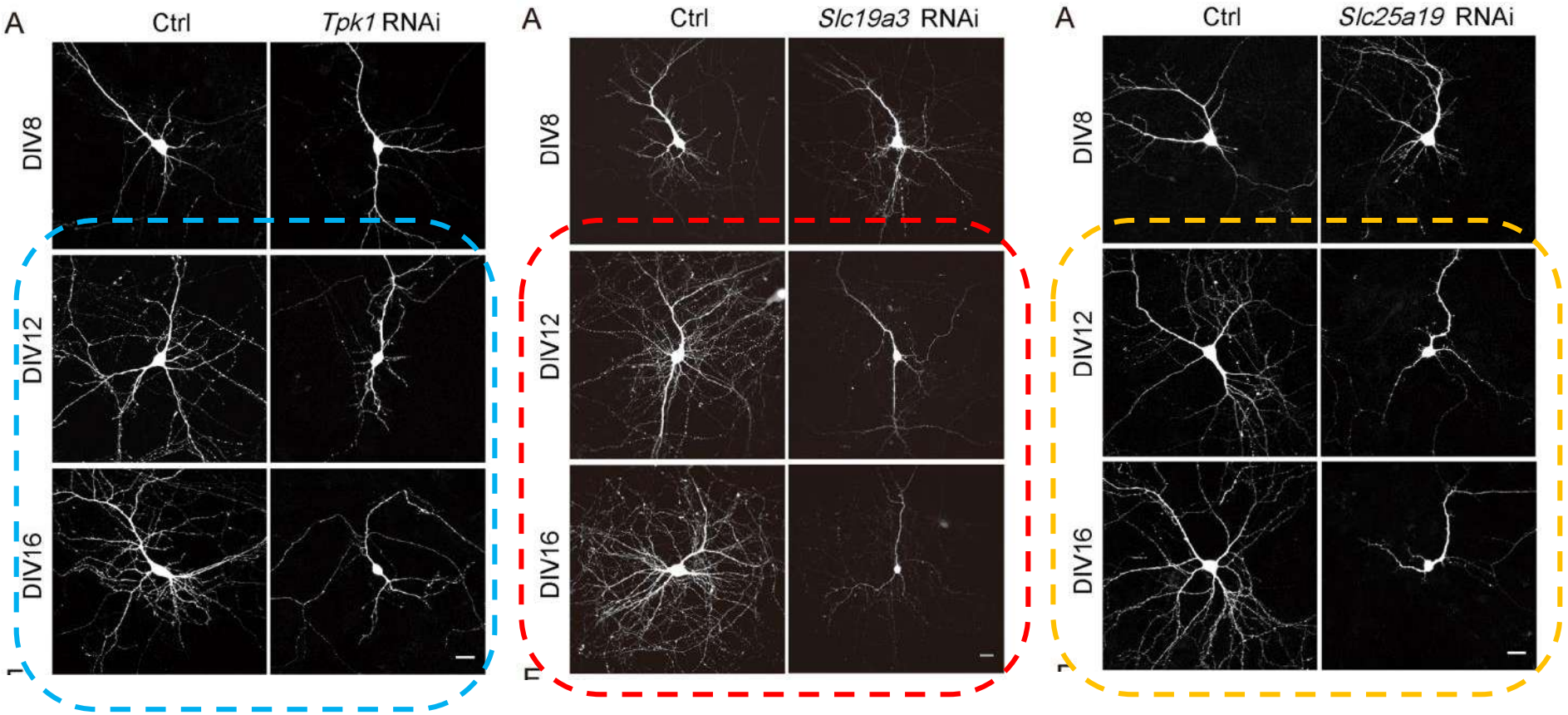




Muneer *et al.*, Mol Neurobiol., 2022

Perturbations du métabolisme ou du transport de la thiamine et atteintes neuronales





Liu *et al.*, Scientific Reports, 2017

Alcool et Métabolisme/Distribution de la thiamine

Table 2. The Concentrations of Free Thiamin (T), Thiamin-Monophosphate (TP), -Diphosphate (TPP) and -Triphosphate (TPPP) in Serum and Whole Blood in Patients (ALC) and Controls (Contr) before and 24 hr after Thiamin Injection. Means ± SD

		Serum (nmol/liter)				Whole Blood (nmol/liter)			
		T	TP	TPP	TPPP	T	TP	TPP	TPPP
Men									
Contr before	Mean	15.2	5.9	4.3	<1	36.2	12.1	179	<2
n = 25	SD	3.4	3.1	1.8	–	13.3	5.1	40	–
Contr after	Mean	29.7*	19.5*	5.8	<1	73.1*	27.7*	289*	11.5
n = 10	SD	5.8	8.1	3.2	–	12.6	10.7	18	2.2
ALC before	Mean	17.5	2.9†	3.5	<1	53.8†	4.1†	149	<2
n = 24	SD	11.2	2.3	2.8	–	33.3	3.7	64	–
ALC after	Mean	546[a]	8.1*†	5.1	<1	178[b]	8.6*†	238*	10.2
n = 24	SD	1741	5.1	4.1	–	324	5.9	88	10.9
Women									
Contr	Mean	10.9	8.3	3.2	<2	29.6	9.7	122	<2
n = 15	SD	2.9	1.5	1.7	–	10.0	2.3	30	–
ALC before	Mean	14.9	2.5†	3.6	<2	40.6	5.3†	130	<2
n = 6	SD	8.1	1.5	4.6	–	18.9	5.6	30	–
ALC after	Mean	1208[c]	12.2*	4.1	<2	243[d]	12.4*	272*	9.5
n = 6	SD	2837	4.0	4.1	–	323	3.8	61	8.0

* Statistically different from before treatment ($p < 0.05$).

† Statistically different from controls ($p < 0.05$).

Median (range): [a] 40.0 (7.9–7400), [b] 74.5 (23.0–1370), [c] 53.1 (18.9–7000), [d] 89.2 (79.1–900).

- Amplitude d'augmentation de la concentration en thiamine très différente entre les sujets contrôles et les patients TUAL après supplémentation
- Observé dans le plasma et dans le sang total

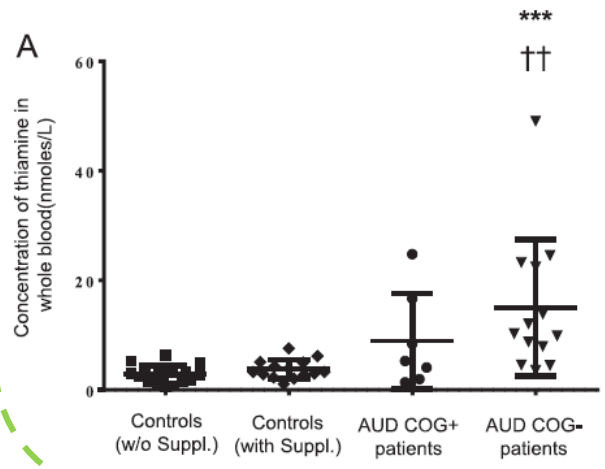
- Ralentissement du métabolisme (Th > TDP) ?
- Ralentissement de la distribution dans les tissus ?

Tallaksen *et al.*, Alcohol Alcoholism, 1994

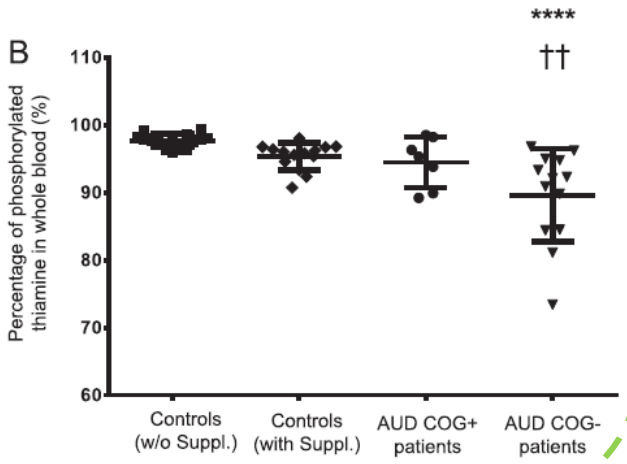
Alcool, Métabolisme/Distribution de la thiamine, et troubles cognitifs

Sang total

Thiamine

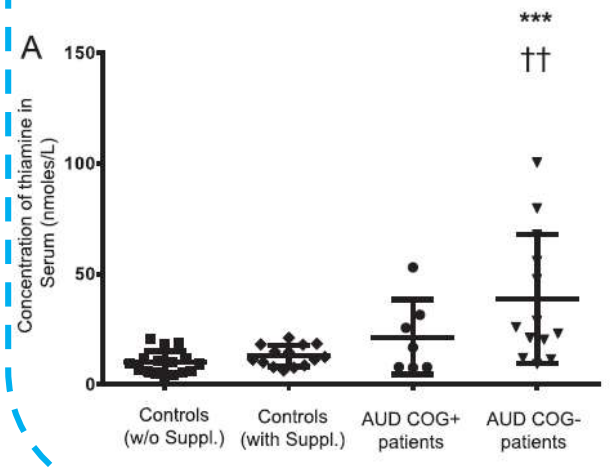


Ratio $\frac{\text{TDP+TMP}}{\text{TDP+TMP+Thiamine}}$

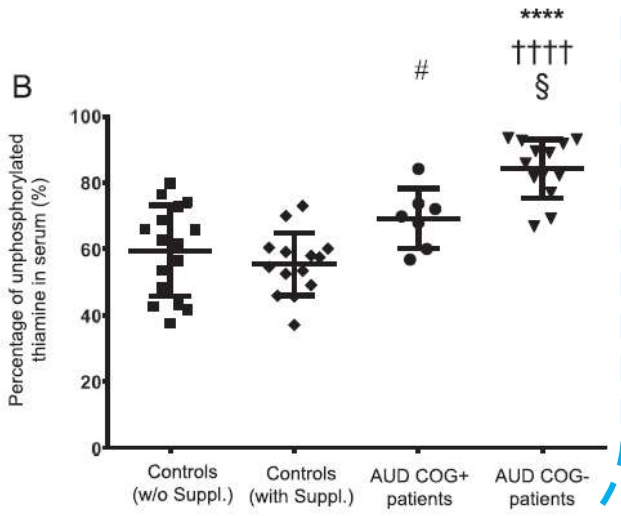


Sérum

Thiamine



Ratio $\frac{\text{Thiamine}}{\text{TDP+TMP+Thiamine}}$



- Après supplémentation, les patients TUAL avec des troubles cognitifs présentent des concentrations en thiamine plus élevées et des ratios métaboliques significativement différents
- Anomalie de formation de la forme active (TDP) ? de distribution de la thiamine dans les tissus ?

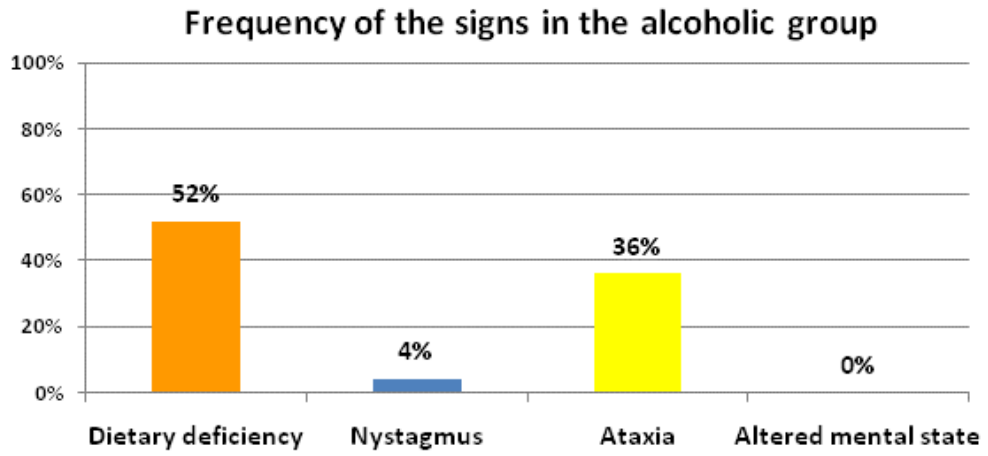
Coulbault *et al.*, Nutr Neurosc, *2019*

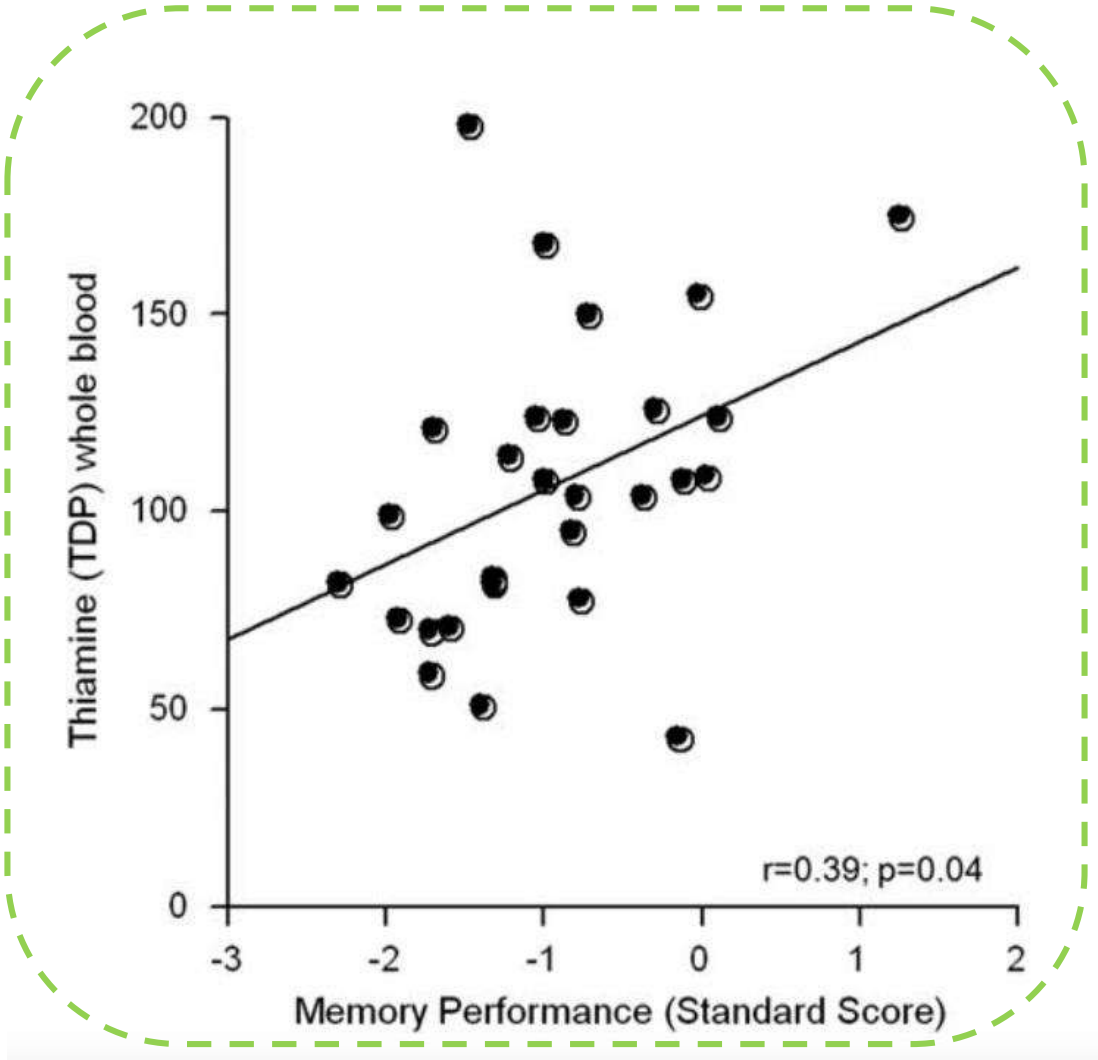
Thiamine et troubles cognitifs

Signs of Preclinical Wernicke's Encephalopathy and Thiamine Levels as Predictors of Neuropsychological Deficits in Alcoholism without Korsakoff's Syndrome

Anne-Lise Pitel[1], Natalie M Zahr[1,2], Karen Jackson[2], Stephanie A Sassoon[2], Margaret J Rosenbloom[1,2], Adolf Pfefferbaum[1,2] and Edith V Sullivan*[,1]

[1]Department of Psychiatry and Behavioral Sciences, Stanford University School of Medicine, Stanford, CA, USA; [2]Neuroscience Program, SRI International, Menlo Park, CA, USA





Pitel *et al.*, Neuropsychopharmacology, 2011

Thiamine et troubles cognitifs (2)

Further evidence of relationship between thiamine blood level and cognition in chronic alcohol-dependent adults: Prospective Pilot Study of an inpatient detoxification with oral supplementation protocol

Udo Bonnet [a, b, *], Laura Pohlmann [a], Heath McAnally [c, d], Benedikt Bernd Claus [a, e]

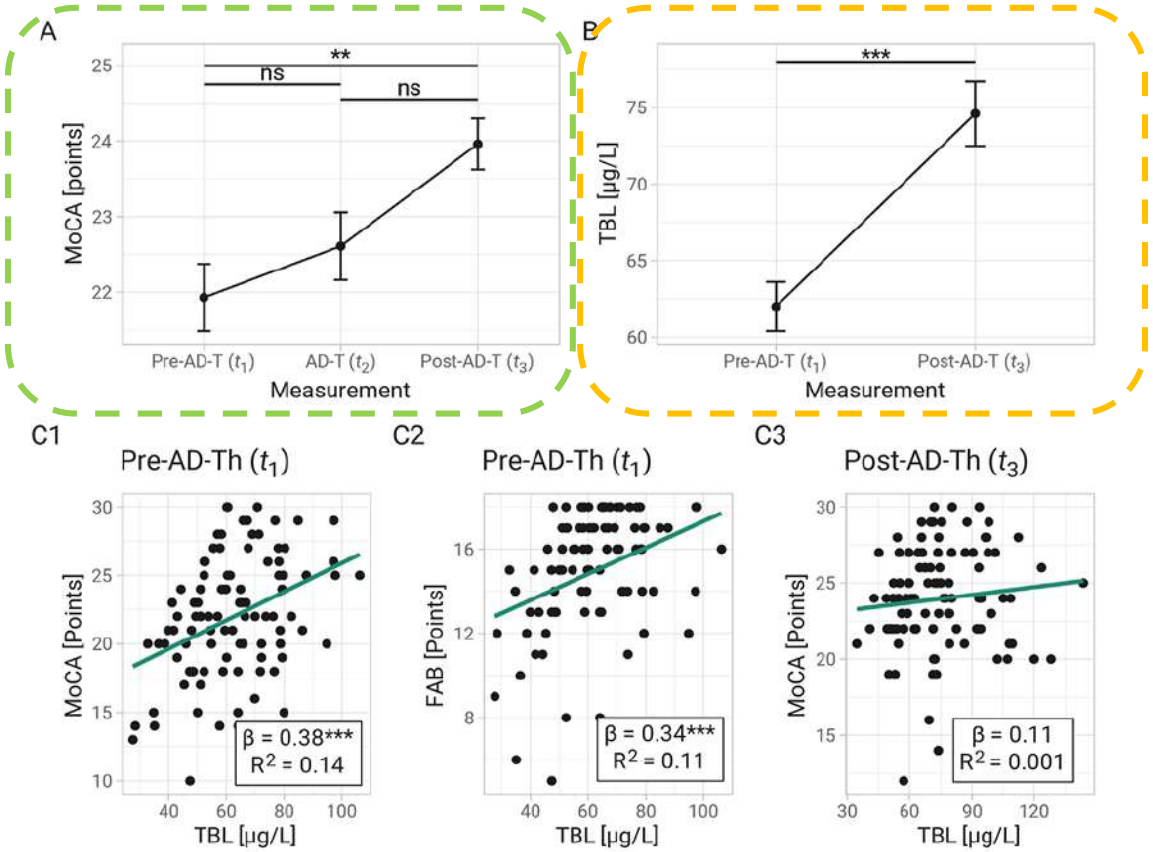
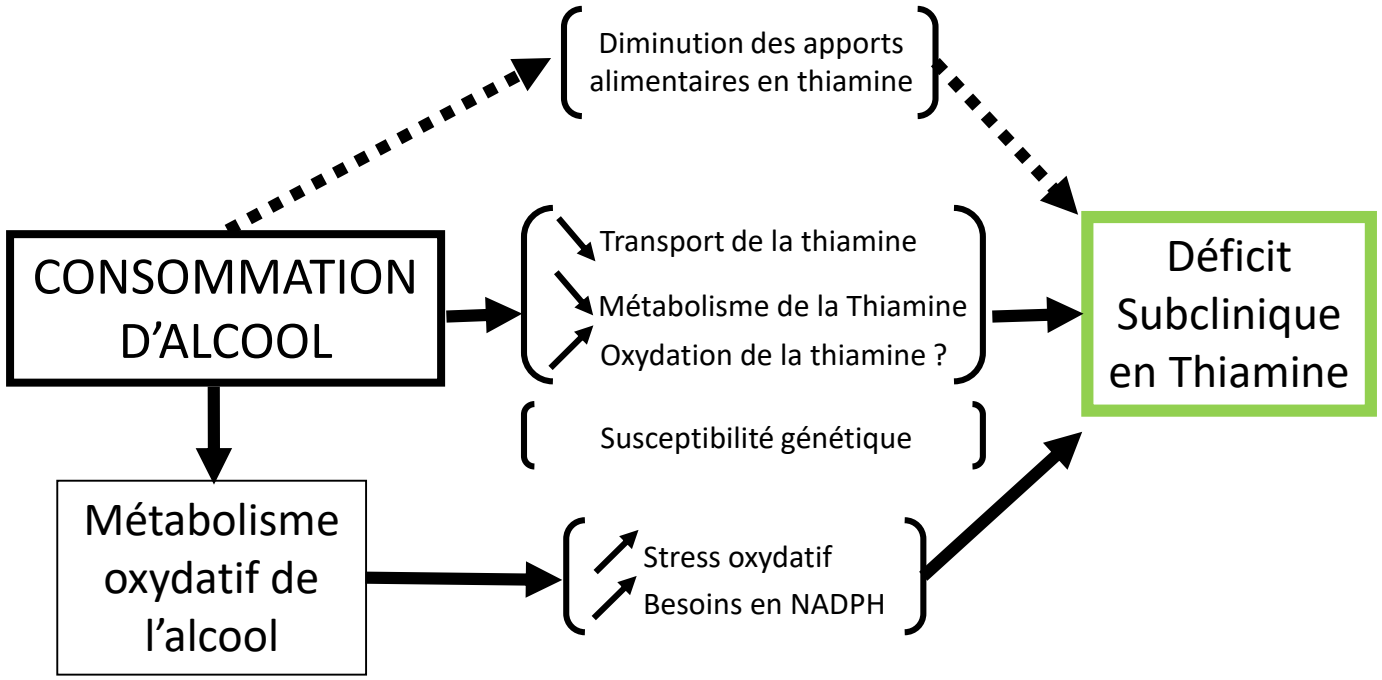


Fig. 2. Significant improvement of MoCA (**A**) and thiamine blood level (TBL) (**B**) from admission baseline (t_1: pre-AD + Th) to study endpoint/discharge (t_3: post-AD + Th), on average 16 days post-admission. Error bars indicate the standard error (SE). MoCA improved between t_1 and t_2, as well as between t_2 and t_3, but not significantly (**A**). Significant association between TBL and MoCA (**C1**) and FAB (**C2**) at t_1 with medium effect sizes (β = 0.38, 95% CI [0.19, 0.56] and β = 0.34, 95% CI [0.14, 0.54], respectively). No significant association between TBL and MoCA (**C3**) at t_3. *q < 0.05, **q < 0.01, ***q < 0.001.

TBL : TDP sang total

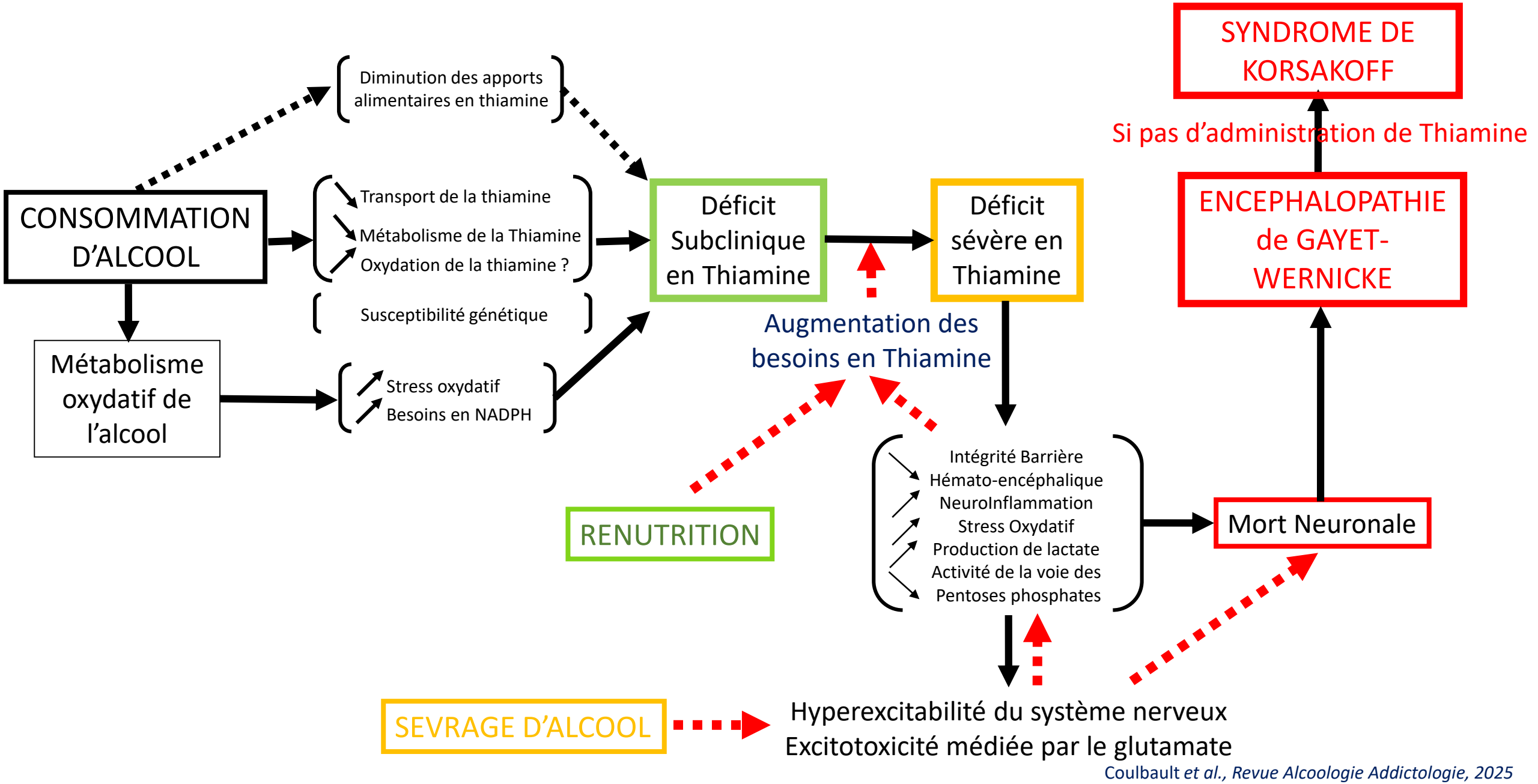
Bonnet *et al.*, Alcohol, 2023

Thiamine, sevrage et troubles cognitifs



Coulbault *et al., Revue Alcoologie Addictologie, 2025*

Thiamine, sevrage et troubles cognitifs



Coulbault *et al., Revue Alcoologie Addictologie, 2025*

Effet de la supplémentation en thiamine : expérience australienne

- Prévalence importante du syndrome de KSK en Australie
- Enrichissement des farines avec de la thiamine à partir de 1991

Study	No. of brain autopsies	No. with WKS	Prevalence of WKS	χ^2	*P* (two-tailed)	Relative risk (95% CI)	
This study	2212	25	1.1%				1996-1997
Harper (forensic cases)	1783	83	4.7%	45.304	<0.0001	1.8 (1.6–2.0)	1973-1981

Harper *et al*., Med J Aust, 1998

Supplémentation en thiamine et démence

Risk of all-cause dementia associated with thiamine use in patients with alcohol use disorder (N = 10,108).

	Case no	Per 1000 person year	Crude hazard ratio (95% CI)	p value	Adjusted hazard ratio (95% CI)	p value
VIT-B1 user (−)	163	7.45	Ref.		Ref.	
VIT-B1 user (+)	130	5.65	0.76 (0.60–0.96)	0.019	0.54 (0.43–0.69)	<0.001
Cumulative DDDs						
<23	45	5.90	0.79 (0.57–1.10)	0.166	0.80 (0.58–1.12)	0.199
23–121	41	5.35	0.72 (0.51–1.01)	0.058	0.53 (0.37–0.75)	<0.001
>121	44	5.69	0.77 (0.55–1.07)	0.116	0.40 (0.28–0.57)	<0.001

Adjusted for age, insurance range, and comorbidities.

- Supplémentation associée à la diminution du risque de démence

Chou *et al.*, Clinical Nutrition, 2019

Quelles recommandations ?

Recommandations	Situation clinique	Doses/durée
Australian Government Department of Health and Ageing. Guideline for the treatment of alcohol problems, 2009 [20]	Suspicion d'encéphalopathie de Gayet-Wernicke	200 mg trois fois par jour, en IV plutôt qu'en IM
	Malnutrition	3–5 jours, puis 100 mg par jour pendant 1 à 2 semaines. 300 mg par jour par voie orale pendant 3–5 jours, puis 100mg pendant 1 à 2 semaines
	Absence de malnutrition	Au moins 500 mg par jour IM ou IV pendant 5 jours puis 300 mg par jour par voie orale pendant plusieurs semaines
European Federation of Neurological Societies (EFNS, 2010) [12]	Encéphalopathie de Gayet-Wernicke diagnostiquée ou suspectée	100 mg trois fois par jour par voie orale pendant 3–5 jours, puis 100 mg par jour pendant 4–9 jours
British Association for Psychopharmacology (BAP, 2012) [22]	Encéphalopathie de Gayet-Wernicke diagnostiquée ou suspectée	500–1500 mg par jour IM ou IV pendant 3–5 jours, puis 250 mg par jour pendant 3–5 jours
	Risque élevé, patient dénutri	250 mg par jour IM ou IV pendant 3–5 jours
	Risque faible	> 300 mg par jour par voie orale
Société française d'alcoologie (SFA, 2015) [1]	Suspicion d'encéphalopathie de Gayet-Wernicke	1000 mg par jour pendant cinq jours puis relais par voie orale
	Signes carentiels et de malnutrition consommation importante, troubles cognitifs, maladie alcoolique du foie	250 mg deux fois par jour par voie parentérale, pendant cinq jours puis relais per os jusqu'à reprise d'une alimentation équilibrée
	Absence de malnutrition	500 mg par jour par voie orale pendant cinq jours, puis 250 mg par jour pendant deux semaines

- Hétérogénéité des pratiques
  - ✓ Dose
  - ✓ Voie d'administration
  - ✓ Fréquence d'administration
  - ✓ Durée de traitement
- Améliorer la nutrition (magnésium)

Dervaux *et al*., 2017; Collet *et al*. 2020

Conclusions

- Trouble d'usage d'alcool, syndrome de korsakoff et santé publique
- Prise en charge à améliorer avec notamment la prévention au moyen d'une supplémentation systématique chez les patients avec un TUAL et pas uniquement dans un contexte de prise en charge en urgence
- Absence de prise en charge du traitement par les tutelles actuellement
- Quelle place pour le dosage ? Valeurs de référence ?

SETViB
Société francophone
Eléments Traces, Vitamines et Biofacteurs

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Merci de votre attention !!

Team Alcool INSERM U1237 NEUROPRESAGE

SCAN ME