

**Les oméga 3 sont-ils utiles dès la naissance?**

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## Déclaration d'intérêts en rapport avec la présentation

➤ **Activités de conseil, fonctions de gouvernance, rédaction de rapports**

*Non*

➤ **Essais cliniques, autres travaux, communications de promotion**

*Non*

➤ **Intérêts financiers (actions, obligations)**

*Non*

➤ **Liens avec des personnes ayant des intérêts financiers ou impliquées dans la gouvernance**

*Non*

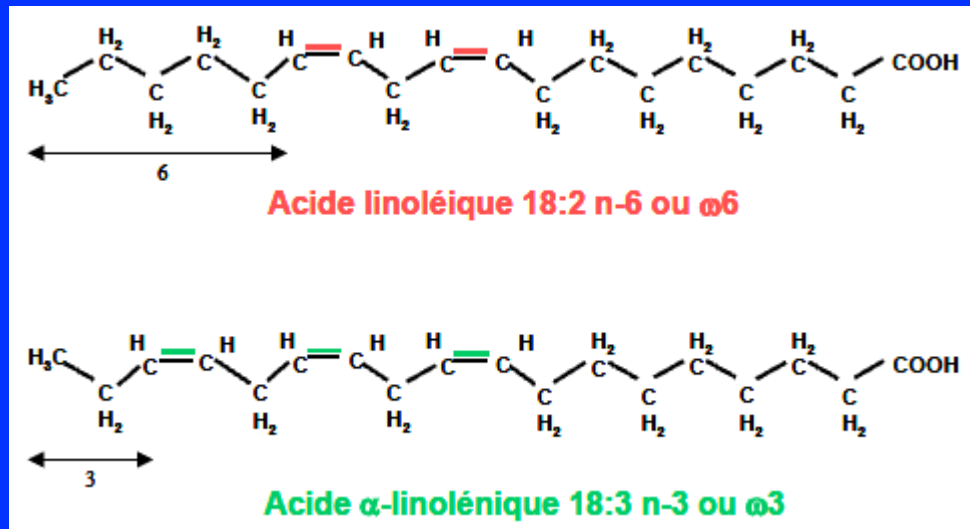
➤ **Réception de dons sur une association dont je suis responsable**

*Non*

➤ **Détention d'un brevet, rédaction d'un ouvrage utilisé par l'industrie**

*Non*

# Les acides gras poly-insaturés essentiels (AGE)



2 familles

Acide linoléique (AL) (n-6) (oméga 6)

Acide alpha-linolénique (AAL) (n-3) (oméga 3)

AGPI-LC : longue chaîne (n ≥ 20) EPA (20C) DPA (22C) DHA (22C)

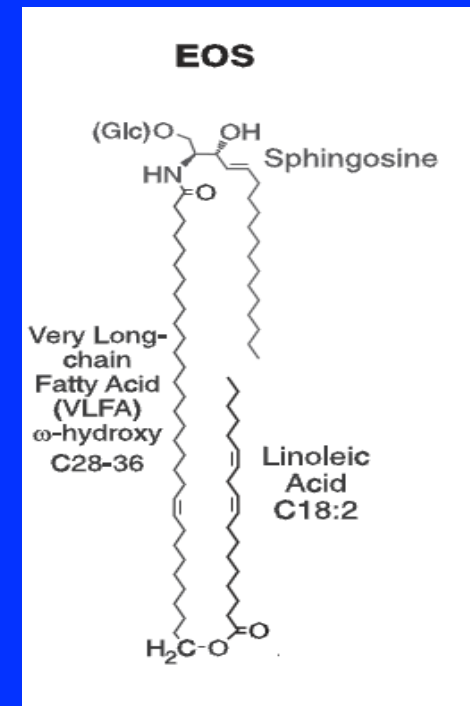
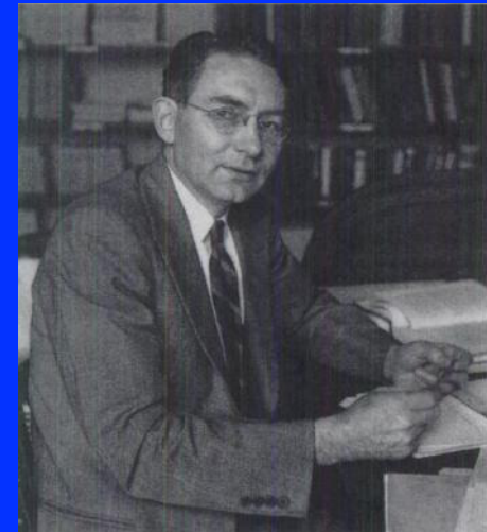
# AL (n-6) reconnu comme essentiel chez l'animal dans les années 30

Burr et Burr (1929-30)

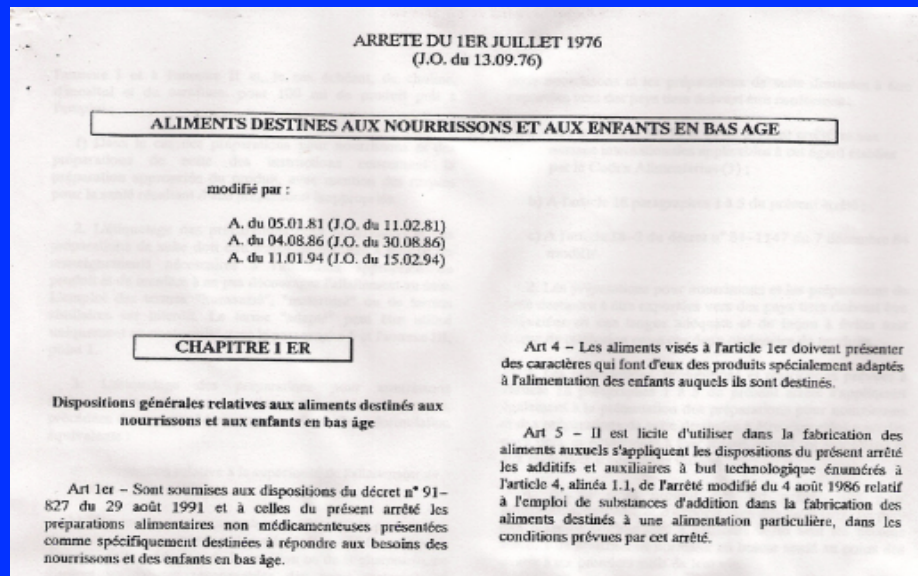
Acide linoléique (n-6) nécessaire à la synthèse d'une céramide (EOS) rendant la peau imperméable

AL reconnu comme essentiel chez l'enfant dans les années 60

AAL connu, mais non reconnu comme essentiel



## 1976 : présence d'AL dans les préparations pour nourrissons rendue obligatoire en France



**AL > 300 mg/100 kcal**

**1981 : présence d'AL obligatoire pour les préparations pour nourrissons dans le Codex Alimentarius**

**Pas de mention de l'AAL**

# **Années 50-80 : Travaux sur l'AAL chez l'animal**

**AL et AAL non interchangeables - Compétitions entre les deux familles pour les réactions d'élongation**

**Présence d'AGPI-LC (n-3) dans les tissus nerveux**

**Mais difficulté de montrer des lésions anatomiques ou une pathologie dues à une carence en AAL chez l'animal et chez l'adulte volontaire**

**Doutes sur l'essentialité de l'AAL**

# Présence d'AGPI-LC dans les tissus nerveux et dans la rétine

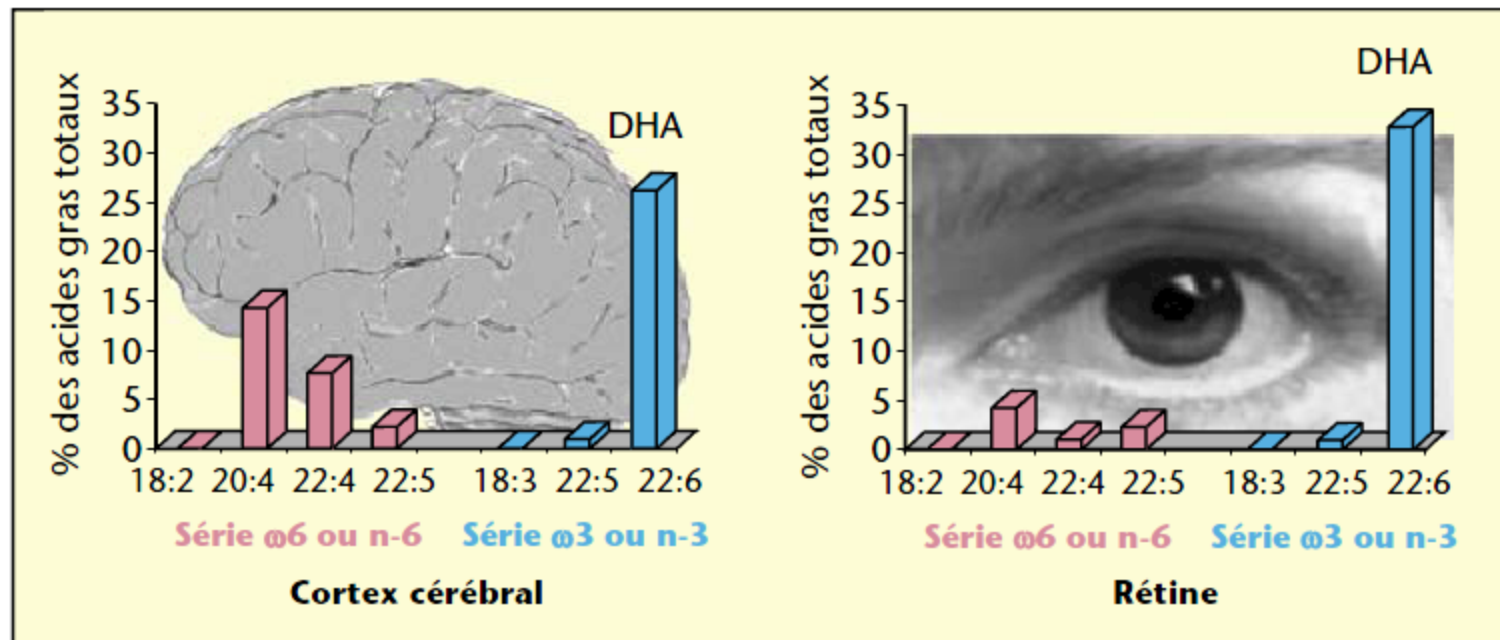


Figure 2. Teneurs des principaux acides gras polyinsaturés (AGPI) retrouvés des phospholipides des membranes du cerveau et de la rétine de l'homme : exemple de la phosphatidyléthanolamine (d'après [8]).

DHA : acide docosahexaénoïque (22:6ω3)

Guesnet et al, 2005, d'après Tinoco 1982

# AAL essentiel chez la truite arc-en-ciel

Effect of Dietary Lipids on Feed Efficiency and Mortality of Rainbow Trout

Diet no.	Lipid composition, %			Feed efficiency, <sup>a</sup> gain/feed	Accumulated mortality
	12:0	18:3 $\omega$ 3	22:6 $\omega$ 3		
1	2.00	0	0	0.32	17
2	1.75	0	0.25	0.80	0
3	1.50	0	0.50	0.88	1
4	1.00	0	1.00	0.91	1
5	1.50	0.50	0	0.84	3
6	1.00	1.00	0	0.90	1

<sup>a</sup>Feed efficiency is defined as units of weight gained per unit of dry feed consumed.

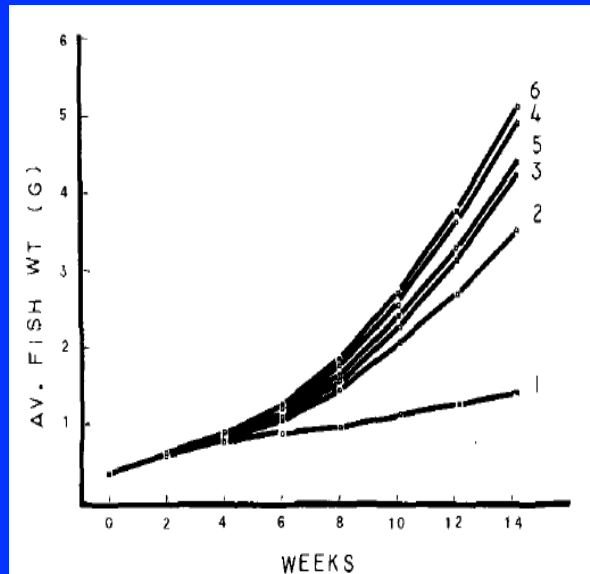
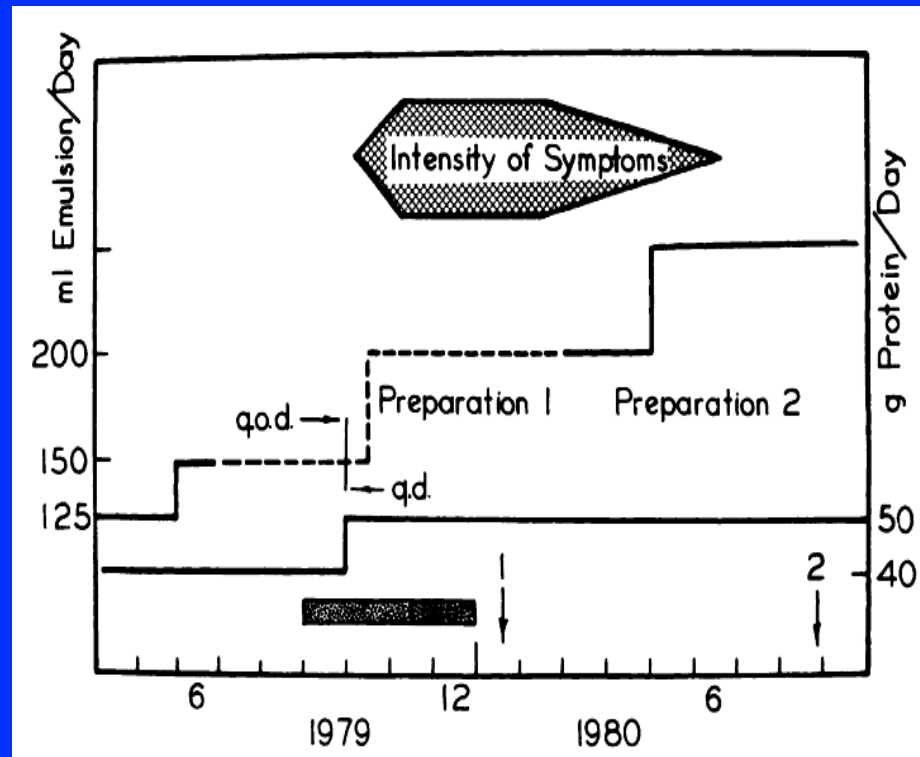


FIG. 1. Effect of dietary fatty acids on growth rate of rainbow trout. Curve 1, control trout diet containing no  $\omega$ 3 fatty acid. Curves 2, 3 and 4, diets containing 0.25, 0.5 and 1.0% 22:6 $\omega$ 3, respectively. Curves 5 and 6, diets containing 0.5 and 1.0% linolenic acid, respectively.

Yu et Sinnhuber, Lipids. 1972;7:450-4

## 1982 : description du premier cas clinique de carence en AAL



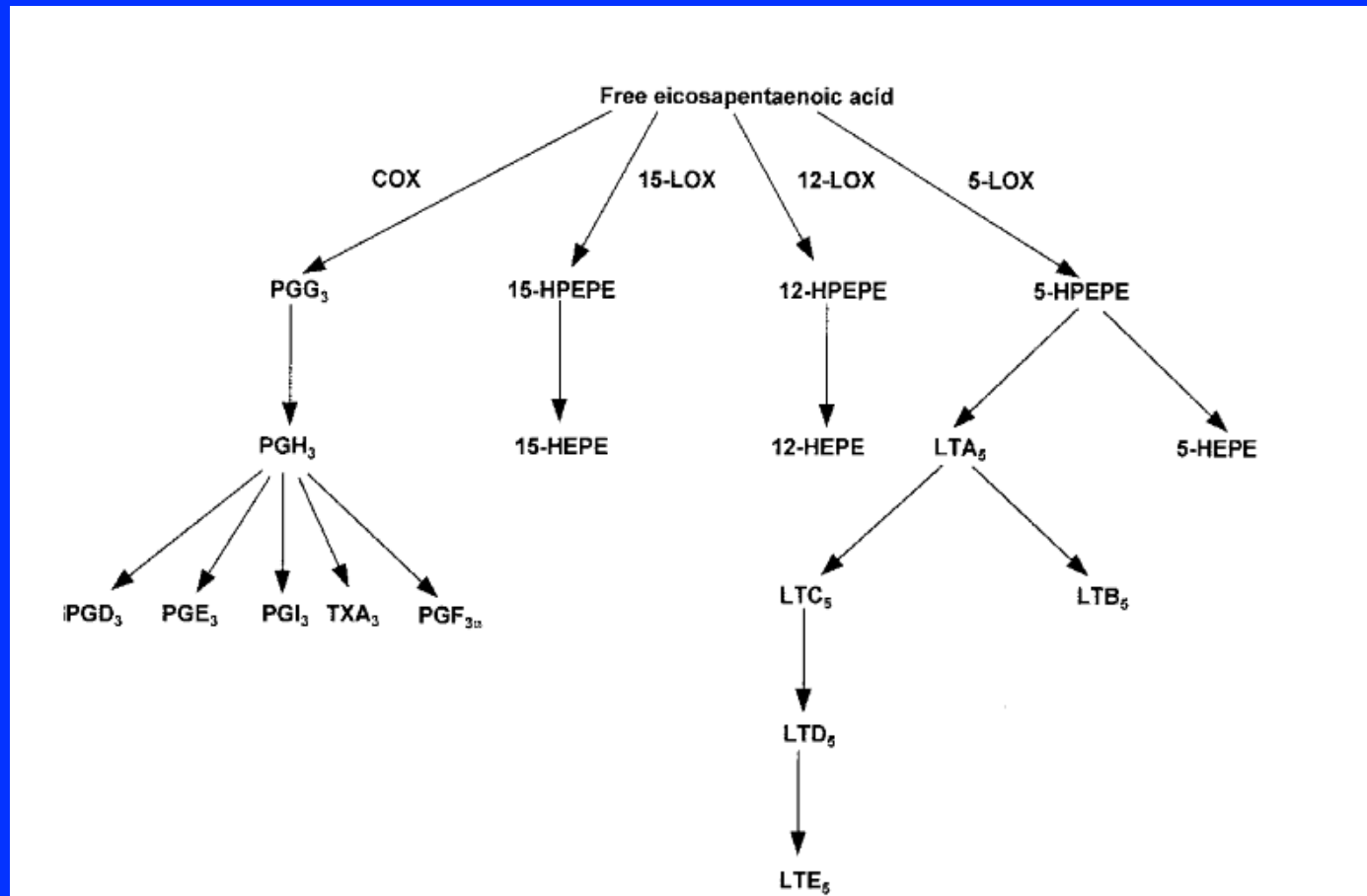
# **Années 75-90 : Effets des carences en AAL chez l'animal mieux connus**

**Diminution des concentration des AGPI-LC n-3 (EPA: acide eicosapentaénoïque; DHA: acide docosahexaénoïque) dans les tissus nerveux et dans la rétine**

**Troubles de l'apprentissage (revue en 2011 par T Brenna : 65 références)**

**Atteinte des voies de neurotransmission monoaminergique et cholinergique au niveau synaptique**

# L'EPA est à l'origine de toute une famille d'eicosanoïdes



Calder, Am J Clin Nutr. 2006;83(6 Suppl): 1505S-1519S.

# Incorporation d'AAL obligatoire dans les Préparations pour nourrissons (fin années 90)

## Acide linoléique

Unité	Minimum	Maximum	Limite indicative maximale
mg/100 kcal	300	-	1400
mg/100 kJ	70	-	330

## $\alpha$ -acide linoléique

Unité	Minimum	Maximum	Limite indicative maximale
mg/100 kcal	50	N.S.*	-
mg/100 kJ	12	N.S.	-

\*N.S. = non spécifié

## Codex alimentarius (FAO/OMS)

Normes fixées pour se rapprocher de la composition du lait de femme

Pas de mention des AGPI-LC (AA, EPA, DHA)

# Plus faible accumulation de DHA chez les enfants non allaités

Enfants non  
allaités  
nourris avec  
des  
préparations  
non  
enrichies en  
DHA

Accumulation of Docosahexaenoate (DHA) in Breast-Fed or Formula-Fed Term Infants from Birth to 6 mon<sup>a</sup>

	Term	6 mon	Change	Accumulation of DHA (mg/d) <sup>a</sup>
Brain (g)	400 <sup>c</sup>	650 <sup>c</sup>		
DHA (mg/g)	1.8 <sup>d</sup>	2.5 <sup>d</sup> (1.8 <sup>e</sup> )		
DHA (mg/brain)	720	1,625 (1,170)	+905 (+450)	5.0 (2.5)
Liver (g)	120 <sup>f</sup>	200 <sup>f</sup>		
DHA (mg/g)	2.8 <sup>g</sup>	1.8 <sup>g</sup> (1.0 <sup>g</sup> )		
DHA (mg/liver)	336	360 (200)	+24 (-136)	0.1 (-0.7)
Body fat (g)	560 <sup>h,i</sup>	2000 <sup>h,i</sup>		
Actual fat (wt%)	47 <sup>j</sup>	60 <sup>k</sup>		
DHA (%)	0.4 <sup>l</sup>	0.1 <sup>l</sup> (ND <sup>l</sup> )		
DHA (mg/total fat)	1,053	1,200 (0)	+147 (-1053)	0.8 (-5.8)
Remaining lean (g)	2,420 <sup>h</sup>	5,000 <sup>h</sup>		
DHA (mg/g)	0.7 <sup>m</sup>	0.5 <sup>m</sup> (0.3 <sup>m</sup> )		
DHA (mg/total lean)	1,694	2,500 (1,500)	+806 (-194)	4.4 (-1.1)
Total body (g)	3,500 <sup>n</sup>	7,850 <sup>n</sup>		
DHA (mg/body)	3,803 <sup>o</sup>	5,685 <sup>o</sup> (2,870 <sup>o</sup> )	+1,882 (-933)	10.3 (-5.1)

<sup>a</sup>Data for formula-fed infants are shown in parentheses.

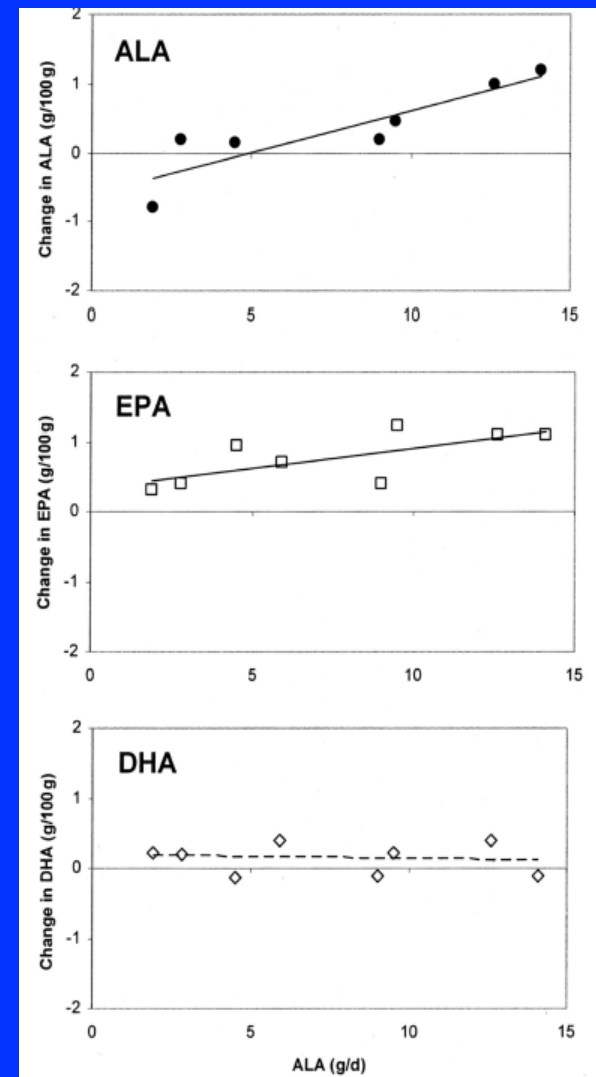
Cunnane et al, Lipids 2000;35:105-11.

# L'augmentation de l'AAL n'a pas d'effet sur le niveau de DHA

Méta-analyse chez l'adulte : pas d'effet de l'AAL sur les taux plasmatiques de DHA

Bloquage au niveau EPA

Arterburn et al, Am J Clin Nutr.  
2006;83(6 Suppl):1467S-1476S.



# Très faible conversion AAL -> DHA chez l'Homme

Rapport  
ANSES 2011

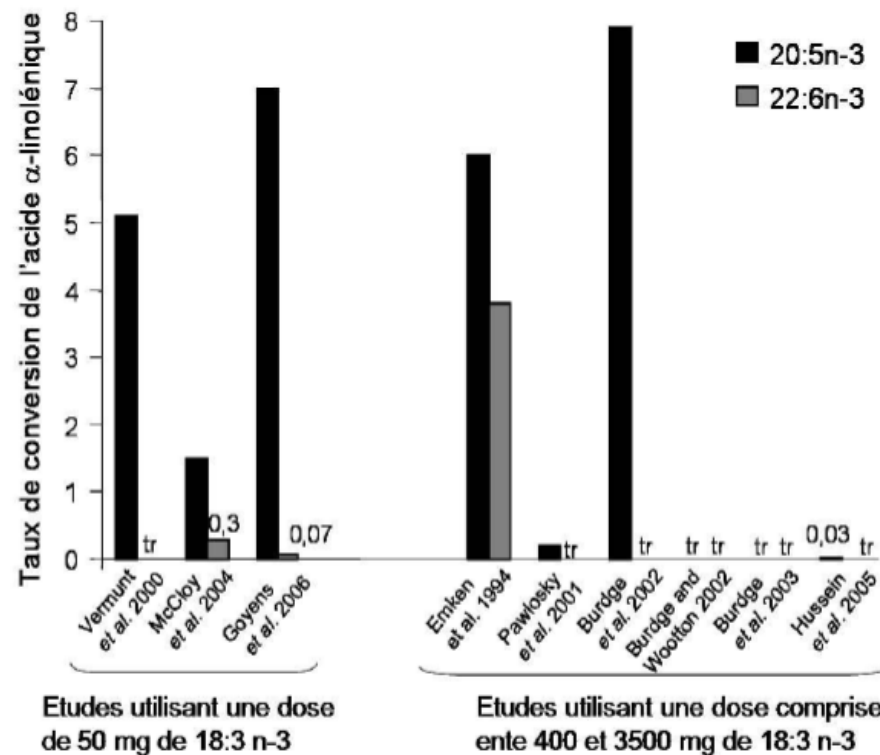
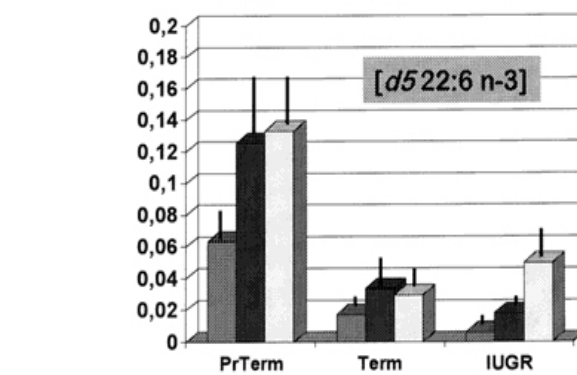
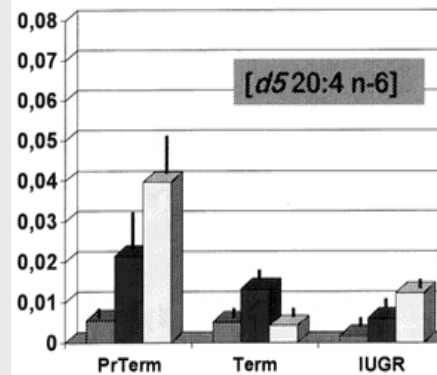
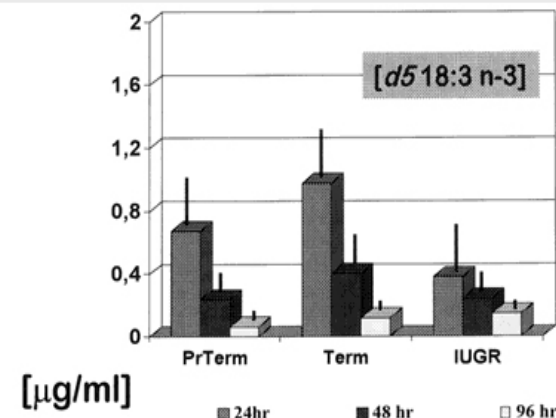
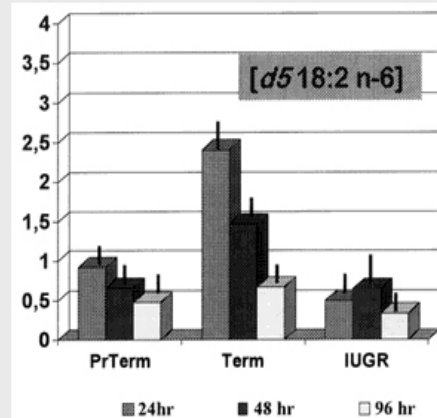


Figure 3. Taux de conversion (%) de l'acide  $\alpha$ -linoléique en EPA et DHA chez l'homme adulte omnivore en bonne santé estimés au moyen d'isotopes stables (adapté de Plourde et Cunnane, 2007).

# Très faible conversion AAL -> DHA chez l'enfant

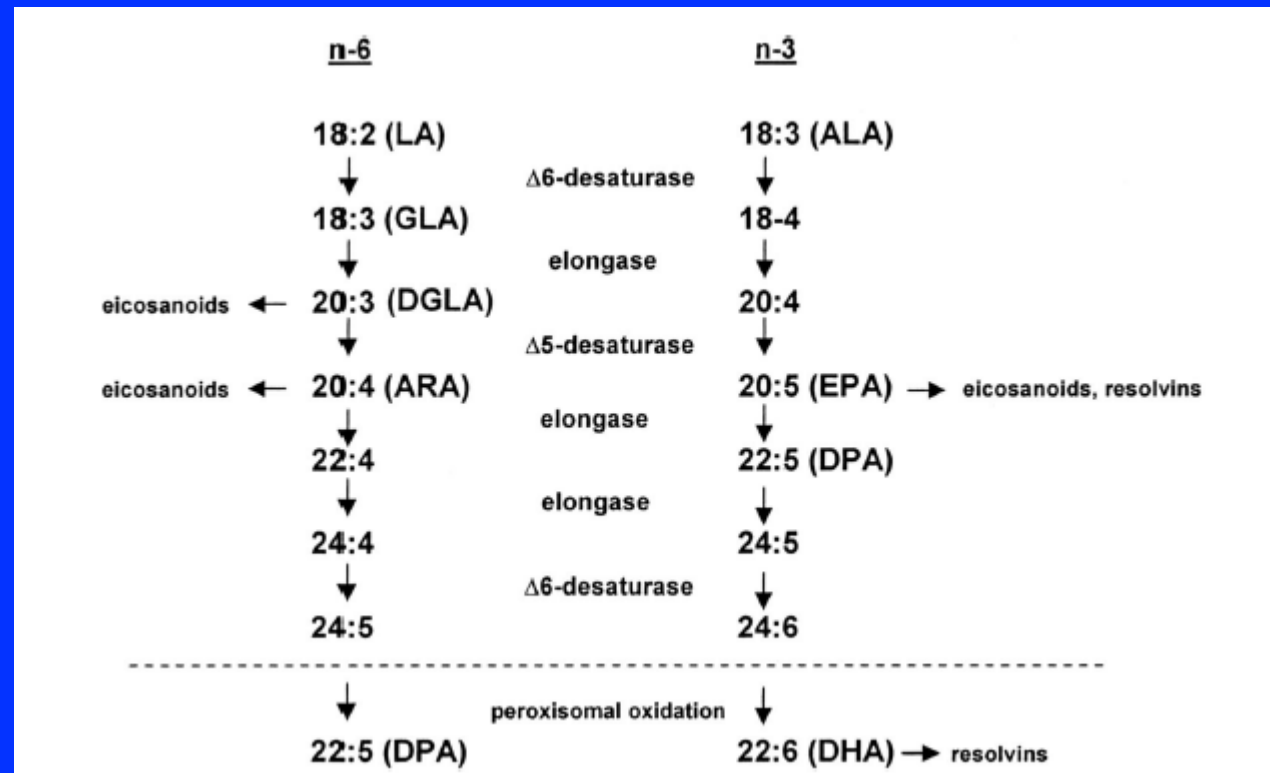
Seulement 3 à 5% de l'AAL converti en DHA après 96 heures

Uauy et al,  
Pediatric  
Research  
2000;47: 127–  
135



# Détail de la conversion AAL -> DHA (1)

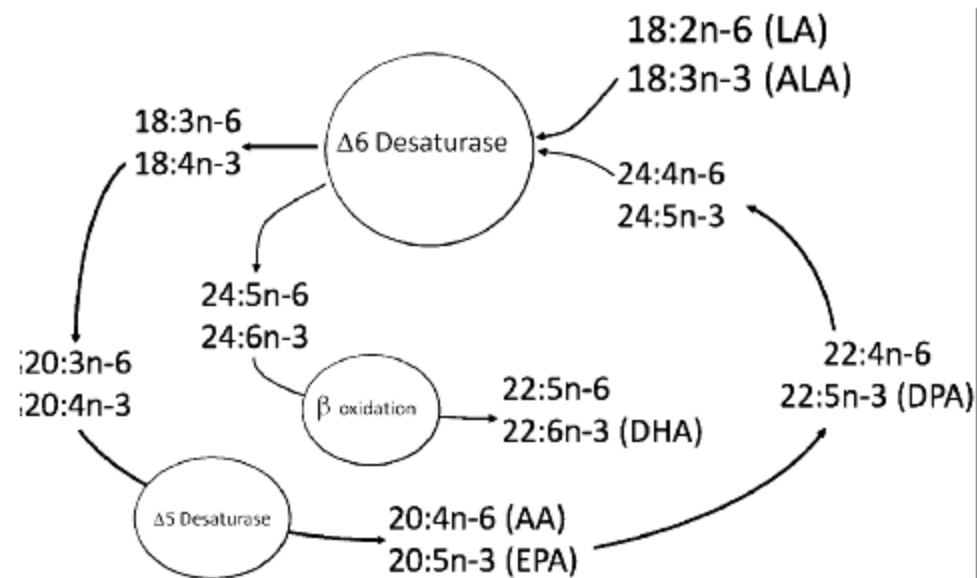
Désaturases et  
élongases  
communes à  
différentes  
réactions



## Détail de la conversion AAL -> DHA (2)

**$\Delta 6$ - désaturase  
commune à  
différentes  
réactions**

**Compétition  
entre AAL et  
DPA pour  $\Delta 6$   
désaturase**



**Fig. 1.** Diagrammatic representation of our current understanding of the synthetic pathway for n-3 and n-6 fatty acids. AA, arachidonic acid; ALA, alpha-linolenic acid; DHA, docosahexaenoic acid; EPA, eicosapentaenoic acid; LA, linoleic acid.

## Détail de la conversion AAL -> DHA (3)

Désaturase et  
élongases  
communes à  
différentes  
réactions

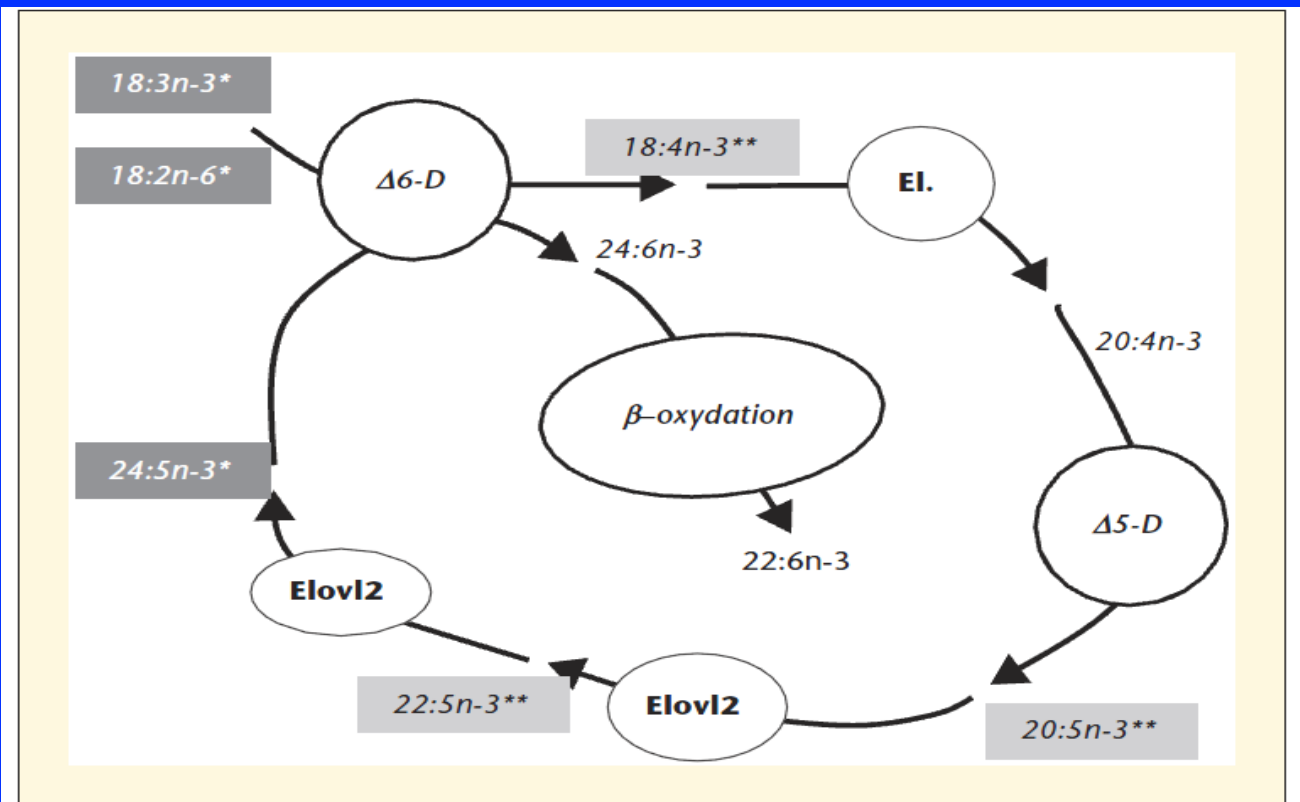


Figure 3. Voie de bioconversion de l'acide  $\alpha$ -linoléique ( $18:3n-3$ ) en acide docosahexaénoïque (DHA,  $22:6n-3$ ). Étapes pour lesquelles une régulation de type inhibition compétitive a été démontrée (adapté de Gibson et al., 2011, et de Grégory et al., 2011). \* Inhibition compétitive au niveau de la  $\Delta 6$  désaturase ; \*\* Inhibition compétitive au niveau de l'élongase Elov12. El. : élongase ; D6-D : D6 désaturase ; D5-D : D5 désaturase.

# Conversion AAL → DHA non prioritaire, même chez le rat avec carence extrême en AAL

**Table 3.** Lipid content and distribution of  $^{13}\text{C}$  in the brain of the control and n-3 PUFA-deficient (Deficient) rats 24 h after dosing with  $[\text{U-}^{13}\text{C}]\text{-}\alpha\text{-linolenate}$

	Lipid content (mg/g)		$^{13}\text{C}$ Enrichment (ng $^{13}\text{C}$ /brain)	
	Control	Deficient	Control	Deficient
n-3 PUFA				
Docosapentaenoate	$0.1 \pm 0.04^*$	$0.02 \pm 0.004^\dagger$	$33 \pm 7$	$115 \pm 41^\dagger$
Docosahexaenoate	$4.0 \pm 0.3$	$0.7 \pm 0.3^\dagger$	$129 \pm 44$	$157 \pm 103$
Sterols	$7.4 \pm 0.2$	$7.6 \pm 1.8$	$276 \pm 196$	$80 \pm 45^\dagger$
Other fatty acids <sup>‡</sup>				
Myristate	$0.4 \pm 0.02$	$0.5 \pm 0.1$	$9 \pm 3$	$4 \pm 3^\dagger$
Palmitate	$8.1 \pm 0.4$	$7.1 \pm 0.9^\dagger$	$388 \pm 94$	$239 \pm 91^\dagger$
Stearate	$3.6 \pm 0.2$	$3.1 \pm 0.7$	$112 \pm 31$	$49 \pm 39^\dagger$
Oleate	$4.0 \pm 0.3$	$3.4 \pm 0.4^\dagger$	$118 \pm 43$	$66 \pm 36^\dagger$
Sum other fatty acids <sup>‡</sup>	$16.1 \pm 0.7$	$14.1 \pm 1.8^\dagger$	$627 \pm 167$	$358 \pm 154^\dagger$
Recycling <sup>§</sup> /docosahexaenoate	NA	NA	$7.5 \pm 2.1$	$2.8 \pm 2.0^\dagger$

\* Mean  $\pm$  SEM of  $n = 10/\text{group}$ .

$^\dagger p < 0.05$  versus controls.

$^\ddagger$  Excludes all n-6 and n-3 polyunsaturates.

$^\S$  Recycling is the sum of  $^{13}\text{C}$  in sterols and other fatty acids excluding n-3 and n-6 polyunsaturates

NA, not applicable.

**Cunnane et al, Pediatr Res. 2006;59:107-10.**

# L'espèce humaine a évolué avec un régime bas en AL et très riche en AAL et AGPI-LC

## Estimated macronutrient and fatty acid intakes from an East African Paleolithic diet

Remko S. Kuipers<sup>1\*</sup>, Martine F. Luxwolda<sup>1</sup>, D. A. Janneke Dijck-Brouwer<sup>1</sup>, S. Boyd Eaton<sup>2</sup>, Michael A. Crawford<sup>3</sup>, Loren Cordain<sup>4</sup> and Frits A. J. Muskiet<sup>1</sup>

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<sup>2</sup>*Departments of Anthropology and Radiology, Emory University, Atlanta, GA, USA*

<sup>3</sup>*Department of Bio-molecular Medicine, Imperial College, London, UK*

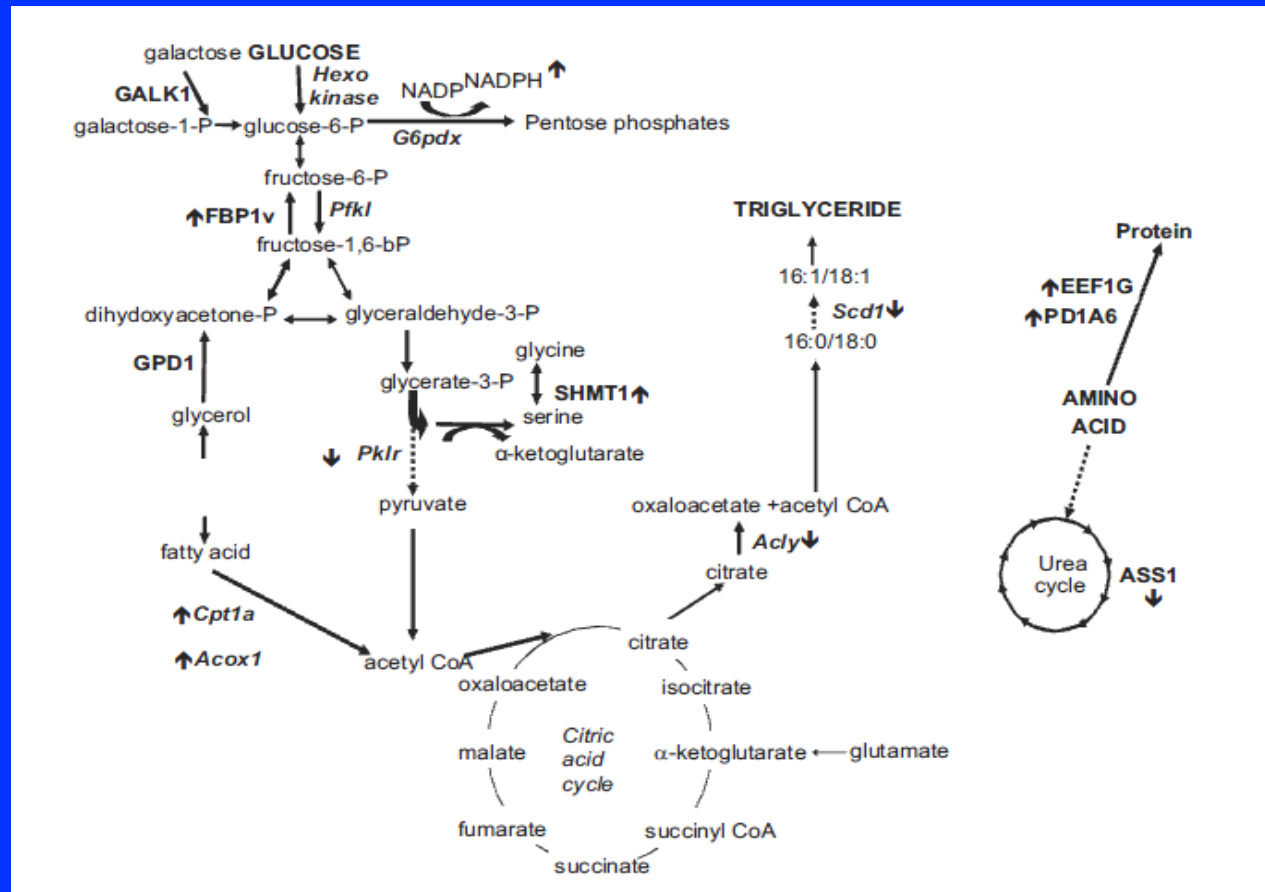
<sup>4</sup>*Department of Health and Exercise Science, Colorado State University, Fort Collins, CO, USA*

(Received 3 February 2010 – Revised 17 May 2010 – Accepted 18 May 2010 – First published online 23 September 2010)

Our genome adapts slowly to changing conditions of existence. Many diseases of civilisation result from mismatches between our Paleolithic genome and the rapidly changing environment, including our diet. The objective of the present study was to reconstruct multiple Paleolithic diets to estimate the ranges of nutrient intakes upon which humanity evolved. A database of, predominantly East African, plant and animal foods (meat/fish) was used to model multiple Paleolithic diets, using two pathophysiological constraints (i.e. protein < 35 energy % (en%) and linoleic acid (LA) > 1.0 en%), at known hunter–gatherer plant/animal food intake ratios (range 70/30–30/70 en%/en%). We investigated selective and non-selective savannah, savannah/aquatic and aquatic hunter–gatherer/scavenger foraging strategies. We found (range of medians in en%) intakes of moderate-to-high protein (25–29), moderate-to-high fat (30–39) and moderate carbohydrates (39–40). The fatty acid composition was SFA (11.4–12.0), MUFA (5.6–18.5) and PUFA (8.6–15.2). The latter was high in  $\alpha$ -linolenic acid (ALA) (3.7–4.7 en%), low in LA (2.3–3.6 en%), and high in long-chain PUFA (LCP; 4.75–25.8 g/d), LCP *n*-3 (2.26–17.0 g/d), LCP *n*-6 (2.54–8.84 g/d), ALA/LA ratio (1.12–1.64 g/g) and LCP *n*-3/LCP *n*-6 ratio (0.84–1.92 g/g). Consistent with the wide range of employed variables, nutrient intakes showed wide ranges. We conclude that compared with Western diets, Paleolithic diets contained consistently higher protein and LCP, and lower LA. These are likely to contribute to the known beneficial effects of Paleolithic-like diets, e.g. through increased satiety/satiation. Disparities between Paleolithic, contemporary and recommended intakes might be important factors underlying the aetiology of common Western diseases. Data on Paleolithic diets and lifestyle, rather than the investigation of single nutrients, might be useful for the rational design of clinical trials.

**Paleolithic diet: Land–water ecosystem: Hunter–gatherers: Evolutionary medicine: Macronutrients: Arachidonic acid: Linoleic acid:  $\alpha$ -Linolenic acid: Docosahexaenoic acid: Cholesterol: Long-chain PUFA**

# Le DHA influence le métabolisme hépatique à des niveaux multiples



Innis et al, Prostaglandins Leukot Essent Fatty Acids. 2013 Jan;88(1):91-5

# Le DHA à l'origine de résolvines et protectines régulant l'inflammation

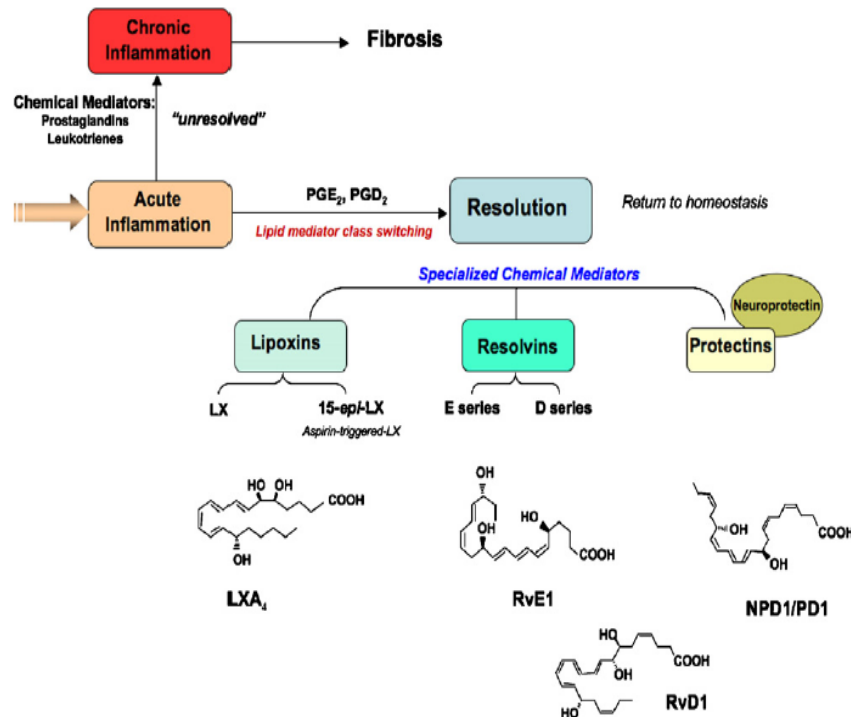


Fig. 2. Biosynthesis of specialized mediators in resolution of acute inflammation. Local mediators include the lipoxins (LX) derived from arachidonic acid (AA); resolvins derived from omega-3 PUFAs eicosapentaenoic acid (EPA; resolvins E series) and docosahexaenoic acid (DHA; resolvins D series); and protectins enzymatically derived from DHA. The structures of each of these key mediators have been confirmed by total organic synthesis (see text for details).

Serhan, Prostaglandins Leukot Essent Fatty Acids. 2008;79:157-63.

# Le DHA influence l'expression d'un millier de gènes au niveau du cortex cérébral

## Differential Cerebral Cortex Transcriptomes of Baboon Neonates Consuming Moderate and High Docosahexaenoic Acid Formulas

Kumar S. D. Kothapalli<sup>1</sup>, Joshua C. Anthony<sup>2</sup>, Bruce S. Pan<sup>1</sup>, Andrea T. Hsieh<sup>1</sup>, Peter W. Nathanielsz<sup>3</sup>, J. Thomas Brenna<sup>1\*</sup>

<sup>1</sup> Division of Nutritional Sciences, Cornell University, Savage Hall, Ithaca, New York, United States of America, <sup>2</sup> Mead Johnson and Company, Evansville, Indiana, United States of America, <sup>3</sup> Center for Pregnancy and Newborn Research, University of Texas Health Science Center, San Antonio, Texas, United States of America

**Background.** Docosahexaenoic acid (DHA, 22:6n-3) and arachidonic acid (ARA, 20:4n-6) are the major long chain polyunsaturated fatty acids (LCPUFA) of the central nervous system (CNS). These nutrients are present in most infant formulas at modest levels, intended to support visual and neural development. There are no investigations in primates of the biological consequences of dietary DHA at levels above those present in formulas but within normal breastmilk levels. **Methods and Findings.** Twelve baboons were divided into three formula groups: Control, with no DHA-ARA; "L", LCPUFA, with 0.33%DHA-0.67%ARA; "L3", LCPUFA, with 1.00%DHA-0.67%ARA. All the samples are from the precentral gyrus of cerebral cortex brain regions. At 12 weeks of age, changes in gene expression were detected in 1,108 of 54,000 probe sets (2.05%), with most showing <2-fold change. Gene ontology analysis assigns them to diverse biological functions, notably lipid metabolism and transport, G-protein and signal transduction, development, visual perception, cytoskeleton, peptidases, stress response, transcription regulation, and 400 transcripts having no defined function. *PLA2G6*, a phospholipase recently associated with infantile neuroaxonal dystrophy, was downregulated in both LCPUFA groups. *ELOVL5*, a PUFA elongase, was the only LCPUFA biosynthetic enzyme that was differentially expressed. Mitochondrial fatty acid carrier, *CPT2*, was among several genes associated with mitochondrial fatty acid oxidation to be downregulated by high DHA, while the mitochondrial proton carrier, *UCP2*, was upregulated. *TIMM8A*, also known as deafness/dystonia peptide 1, was among several differentially expressed neural development genes. *LUM* and *TIMP3*, associated with corneal structure and age-related macular degeneration, respectively, were among visual perception genes influenced by LCPUFA. *TIA1*, a silencer of *COX2* gene translation, is upregulated by high DHA. Ingenuity pathway analysis identified a highly significant nervous system network, with epidermal growth factor receptor (*EGFR*) as the outstanding interaction partner. **Conclusions.** These data indicate that LCPUFA concentrations within the normal range of human breastmilk induce global changes in gene expression across a wide array of processes, in addition to changes in visual and neural function normally associated with formula LCPUFA.

Kothapalli et al. PLoS One 2007;2:e370

# Recommendations FAO-OMS 2010

n-3 PUFA				
ALA	0-6 mo	AI	0.2-0.3%E <sup>b</sup>	Convincing
	6-24 mo	AI	0.4-0.6%E	Probable
	6-24 mo	U-AMDR	<3%E	Probable
DHA	0-6 mo	AI	0.1-0.18%E <sup>b</sup>	Convincing
	0-6 mo	U-AMDR	no upper value within the HM range up to 0.75%E	Convincing
	0-6 mo	Comment	conditionally essential due to limited synthesis from ALA	Probable
	6-24 mo	AI	10-12 mg/kg	Probable
	0-24 mo	Comment	critical role in retinal and brain development	Convincing
EPA+DHA	2-4 yr	AI	100-150 mg (age adjusted for chronic disease prevention) <sup>c</sup>	Probable
	4-6 yr	AI	150-200 mg (bridged from an infant value of 10 mg/kg)	Probable
	6-10 yr	AI	200-250 mg (to the adult value assigned at age 10 years)	Probable

# Recommandations ANSES 2011

**Tableau 22. ANC en AGPI précurseurs et à longue chaîne pour le nouveau-né/nourrisson (6 premiers mois).**

	Acide linoléique	Acide $\alpha$ -linolénique	Acide arachidonique	Acide docosa-hexaénoïque	AGPI-LC n-3 (EPA+DHA)
Nouveau-né/nourrisson	2,7 % AE	0,45 % AE	0,5 % AGT	0,32 % AGT	EPA < DHA

## **Situation en 2013**

**La majorité des préparations pour nourrissons sur le marché en France n'est pas enrichie en AGPI-LC**

**Celles qui le sont, sont souvent à des niveaux inférieurs à ceux recommandés par FAO-OMS ou ANSES**

# **Pourquoi peu de préparations pour nourrissons sont enrichies en AGPI-LC ?**

**Enrichissement en AGPI-LC autorisé, mais pas obligatoire**

**Pas d'effet fonctionnel clair montré par les essais randomisés en dehors d'un effet transitoire sur la vision (RCT)**

**Mais:**

**Réglementation toujours en retard sur données scientifiques**

**RCT, hétérogènes, peu performants pour déterminer la dose optimale d'un nutriment (non utilisés pour AL et AAL)**

**Pas d'effet délétère**

**Rapport bénéfice risque en faveur d'une supplémentation**

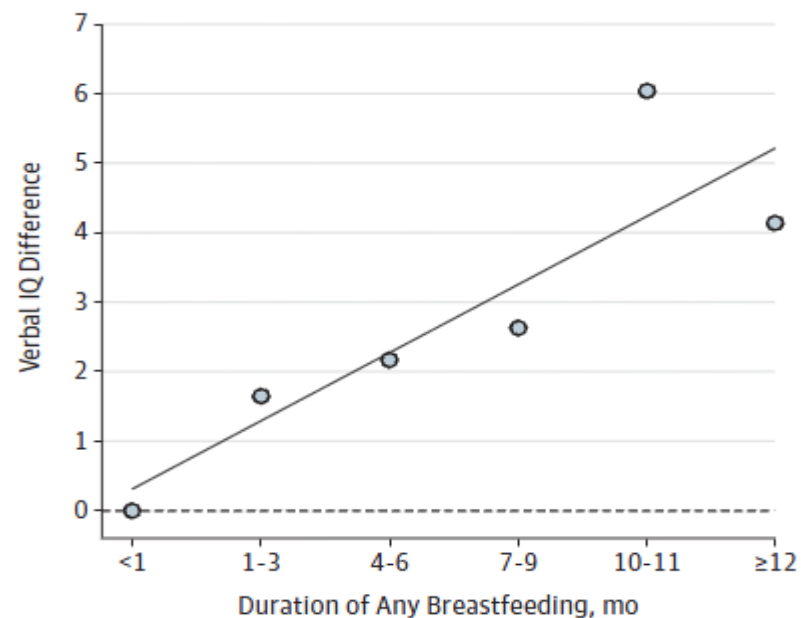
# Allaitement maternel

La référence +++

Belfort et al, 2013

JAMA Pediatr.  
2013;167:836-844.

Figure. Differences in Kaufman Brief Intelligence Test–Second Edition Verbal Scores at Age 7 Years According to Duration of Any Breastfeeding, With Linear Trend Line

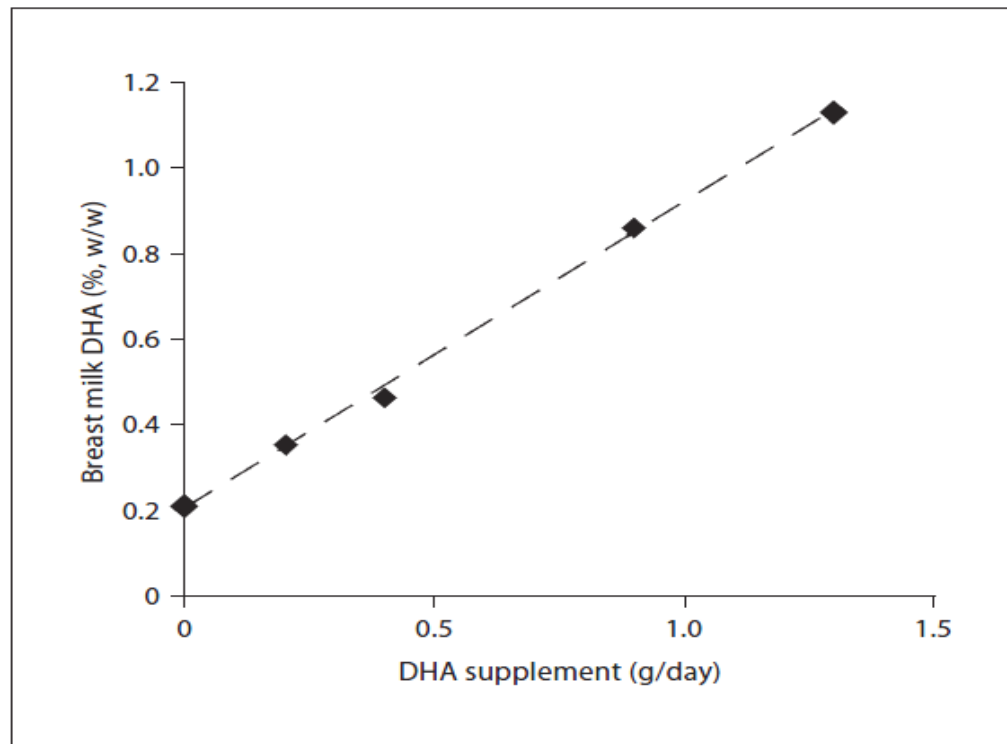


Estimates are adjusted for child age, sex, fetal growth, gestational age, race/ethnicity, and primary language and for maternal age, parity, smoking status, IQ, depression, employment, and child care at 6 months' post partum, as well as for parental education level, annual household income, and Home Observation Measurement of the Environment short form score.

# Le niveau du DHA dans le lait de femme dépend des apports maternels

Effet +++ sur le statut lipidique de l'enfant

Apports recommandés  
ANSES : 250 mg/j



**Fig. 2.** Regression analysis of breast milk DHA (B) concentration vs. DHA intake (I).  $B = (0.72 \times I) + 0.20$  ( $r^2 = 0.998$ ). Data from Gibson et al. [1997].

# Allaitement maternel

Table 6. Adjusted Associations of Duration of Breastfeeding With Cognitive Test Scores at Ages 3 and 7 Years by Postpartum Maternal Fish Intake<sup>a</sup>

Score	Points (95% CI) per Month Breastfed		P Value for Interaction
	<2 Servings of Fish per Week	≥2 Servings of Fish per Week	
At Age 3 y			
PPVT-III	0.10 (−0.15 to 0.35)	0.30 (0.03 to 0.58)	.22
WRAVMA total	−0.01 (−0.22 to 0.20)	0.24 (0.00 to 0.47)	.16
At Age 7 y			
KBIT-II verbal	0.31 (0.06 to 0.56)	0.35 (0.05 to 0.65)	.82
KBIT-II nonverbal	0.36 (0.02 to 0.69)	0.15 (−0.25 to 0.56)	.95
WRAVMA total	−0.04 (−0.40 to 0.32)	−0.06 (−0.47 to 0.35)	.92
WRAML	0.06 (−0.03 to 0.16)	0.04 (−0.06 to 0.14)	.75

Abbreviations: HOME-SF, Home Observation Measurement of the Environment short form; KBIT-II, Kaufman Brief Intelligence Test–Second Edition; PPVT-III, Peabody Picture Vocabulary Test–Third Edition; WRAML, Wide Range Assessment of Memory and Learning; WRAVMA, Wide Range Assessment of Visual Motor Abilities.

<sup>a</sup> Estimates are adjusted for child age, sex, fetal growth, gestational age, race/ethnicity, and primary language and for maternal age, parity, smoking status, IQ, depression, employment, and child care at 6 months' post partum, as well as for parental educational level, annual household income, and HOME-SF score.

L'impact de l'allaitement sur le développement cognitif pourrait dépendre des apports en DHA de la mère

Belfort et al, JAMA Pediatr. 2013;167:836-844.

# Conclusions

**Il semble prudent de considérer les omégas 3, AAL et AGPI-LC, comme étant nécessaires pour une alimentation optimale du nourrisson**

**Il est important de s'assurer également que les apports en AGPI-LC (DHA) soient suffisants chez les femmes enceintes et allaitantes**