Oméga-3 & végétalisation de l'alimentation

OBSERVATOIRE NATIONAL DES ALIMENTATIONS VÉGÉTALES

Fabien BADARIOTTI
1 mars 2024

PROGRAMME

Oméga-3 ? Oméga-6 ? Métabolisme / Rôles physiologiques

Occurrences alimentaires / Apports alimentaires observés

Recommandations / Données chez les omnivores

Santé des populations végétariennes

Recommandations d'apports ONAV (VG, population générale)

Données chez les personnes végétariennes enceintes / allaitantes

Recommandations d'apports ONAV (VG, population spécifique)

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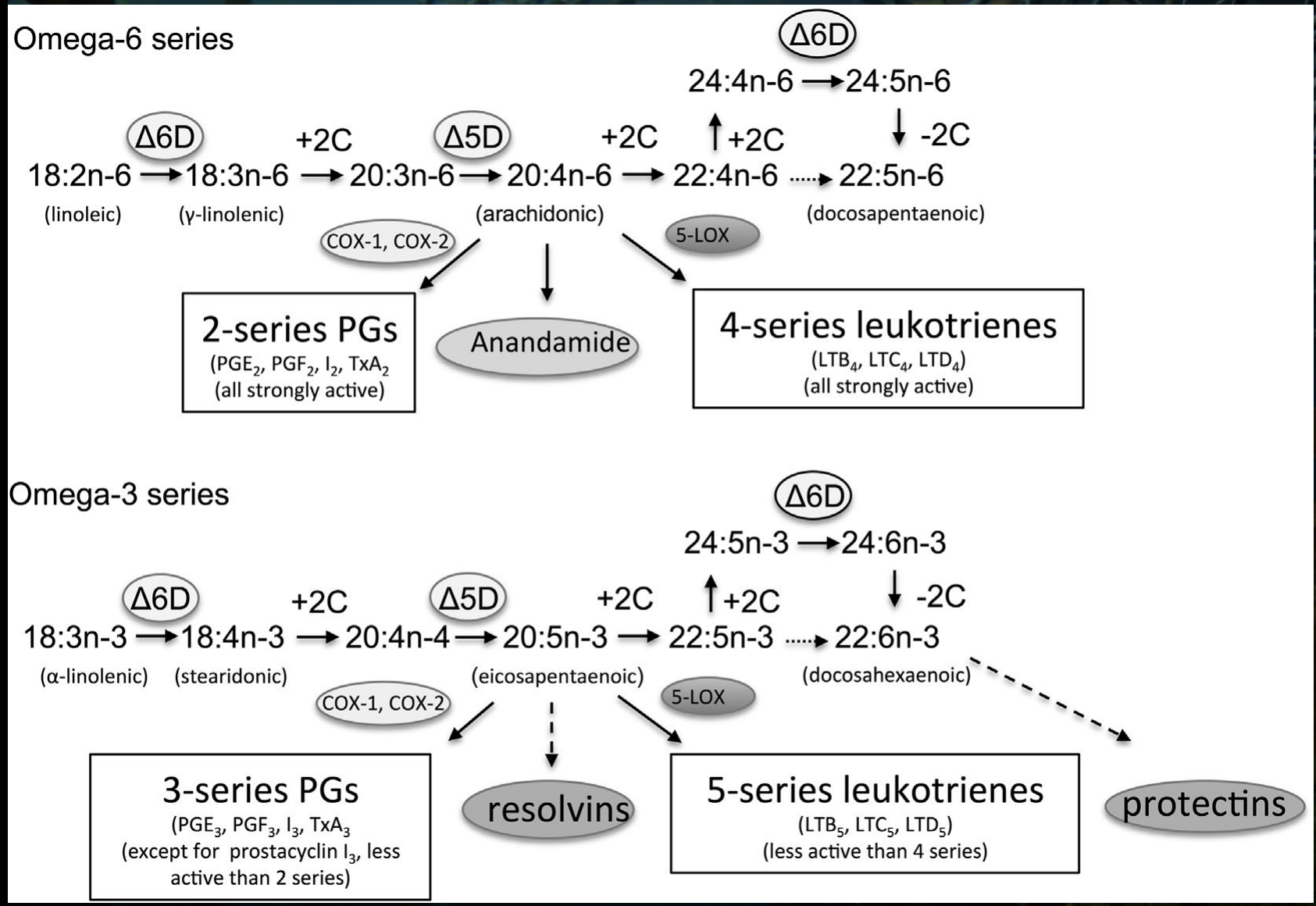
Oméga-3 ?



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Sanders, 2017 (DOI: 10.1016/B978-0-12-803968-7.00037-X)

Macronutriments & Micronutriments

Lipides + Glucides + Protéines

Oméga-3 ?

Acides gras saturés (AGS) + Acides gras insaturés (AGI)

AG monoinsaturés (AGMI)
+
AG polyinsaturés (AGPI)

Famille des Oméga-3 (ω-3)
+
Famille des Oméga-6 (ω-6)

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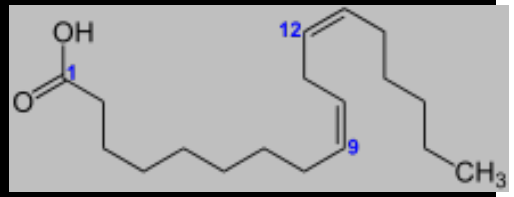
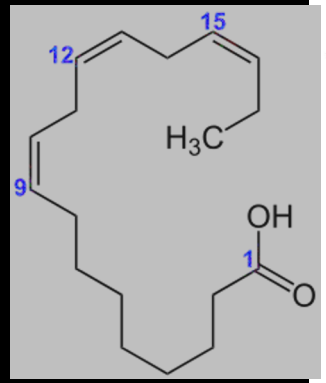
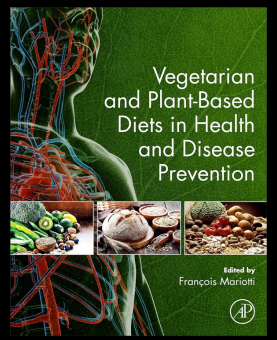


Oméga-6 & Oméga-3





Sanders, 2017 (DOI: 10.1016/B978-0-12-803968-7.00037-X)

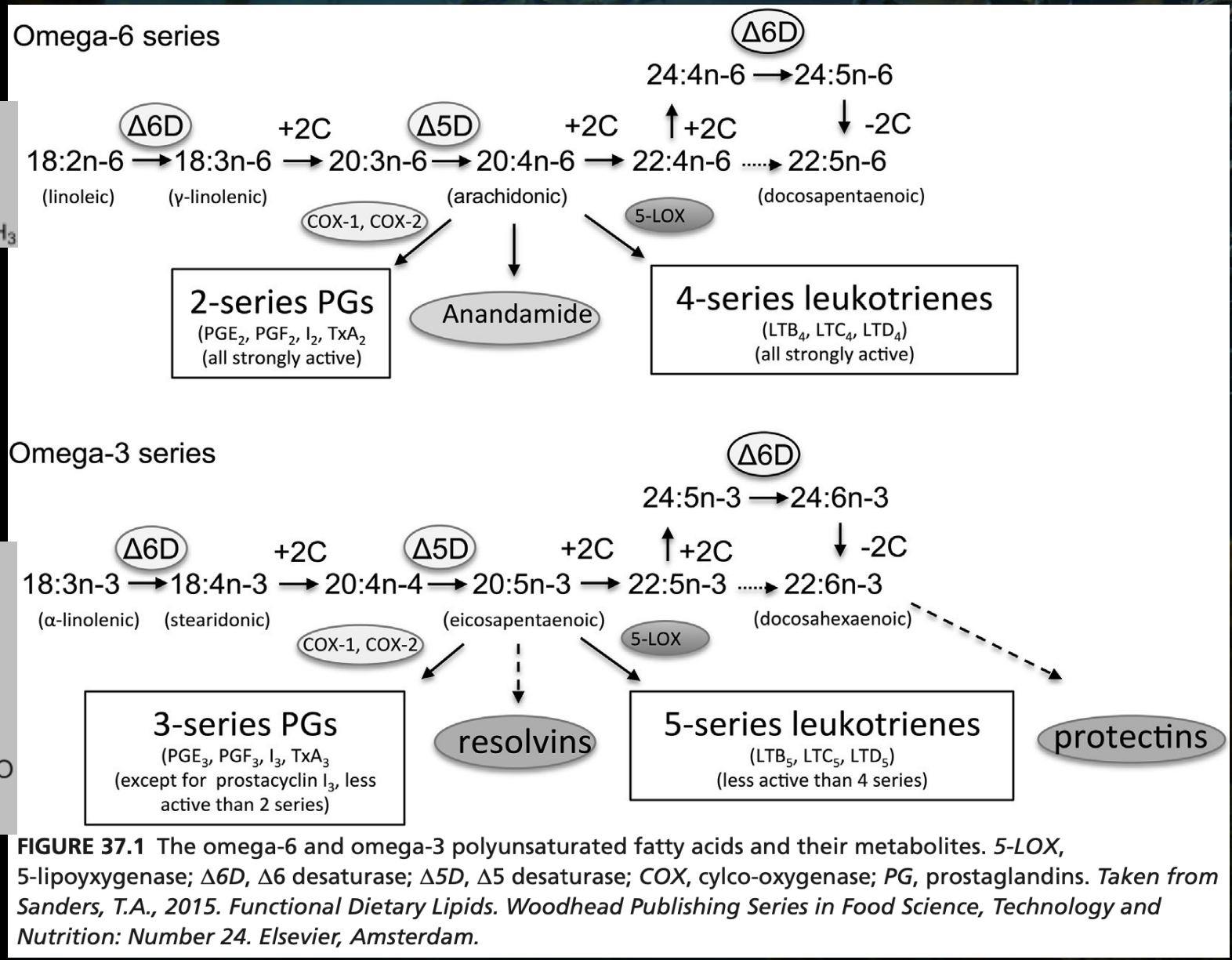


FIGURE 37.1 The omega-6 and omega-3 polyunsaturated fatty acids and their metabolites. *5-LOX*, 5-lipoyxygenase; *Δ6D*, Δ6 desaturase; *Δ5D*, Δ5 desaturase; *COX*, cylco-oxygenase; *PG*, prostaglandins. *Taken from Sanders, T.A., 2015. Functional Dietary Lipids. Woodhead Publishing Series in Food Science, Technology and Nutrition: Number 24. Elsevier, Amsterdam.*

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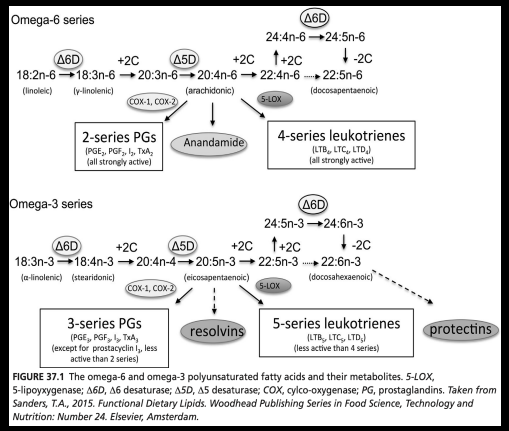

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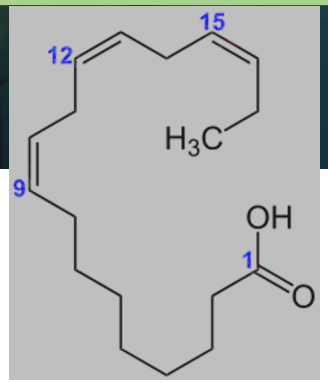
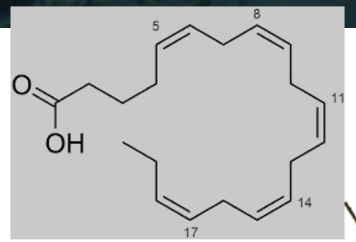
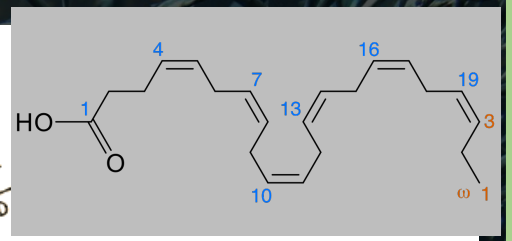
Oméga-3 & Oméga-6





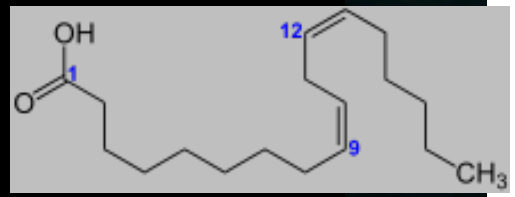
rétroconversion possible



Acide Alpha-Linolénique (ALA) (C18) → → → Acide EicosaPentaénoïque (EPA) (C20) → → Acide DocosaHexaénoïque (DHA) (C22) = **famille ω-3**

3 étapes

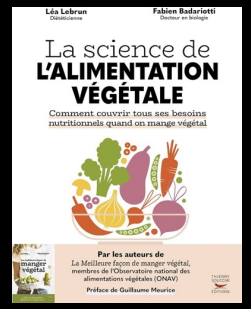
2 étapes



Acide Linoléique (AL) (C18) → → → Acide Arachidonique (AA) (C20) → → Acide DocosaPentaénoïque (DPA) (C22) = **famille ω-6**

3 étapes

2 étapes



Lebrun et Badariotti, 2022 et 2023 (ISBN: 978-2-36549-551-6 et 978-2-36549-722-0)

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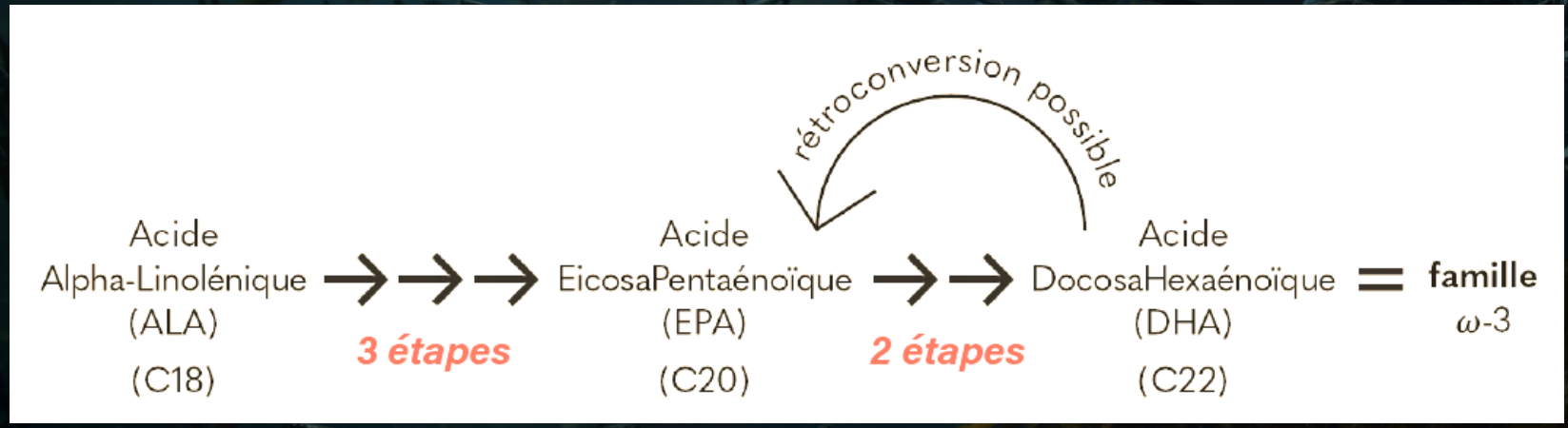
Conversion des AG Oméga-3

Taux faible de conversion ALA → DHA
0,05 - 10 %



♂ ≈ **8 - 10 %**
♀ ≈ **8 - 21 %**

♂ ≈ **0,6 % (40 %)**
♀ ≈ **50 %**

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Lebrun et Badariotti, 2022 et 2023 (ISBN: 978-2-36549-551-6 et 978-2-36549-722-0); Burdge, 2018 (DOI: 10.1017/S0007114518003707)

Rôles physiologiques

anses
agence nationale de sécurité sanitaire alimentation, environnement, travail

Tableau 1. Rôles physiologiques majeurs exercés par les AGPI n-6 et n-3.

	AGPI	Rôles physiologiques majeurs
	Série n-6	
AL	* Acide linoléique (18:2 n-6)	* AG indispensable, précurseur métabolique des AGPI n-6. Exerce des fonctions essentielles en tant que constituant majeur des lipides membranaires (rôle structural)
	* Acide gamma-linolénique (18:3 n-6)	* Pas de fonction démontrée. Possèderait des propriétés anti-inflammatoires en provoquant l'accumulation de l'acide dihomo-gamma-linolénique et en limitant la synthèse d'acide arachidonique (Chilton *et al.*, 2008)
	* Acide dihomo-gamma-linolénique (20:3 n-6)	* AG essentiel en tant que précurseur des prostanoïdes de la série 1 et des leucotriènes de la série 3
	* Acide arachidonique (AA, 20:4 n-6)	* AG essentiel. Il exerce trois fonctions majeures spécifiques comme constituant ubiquitaire des lipides de structure, précurseur de médiateurs lipidiques (prostanoïdes de la série 2, leucotriènes de la série 4, AG hydroxylés (HETE, EET, lipoxines) et endocannabinoïdes) et régulateur de l'expression génique
	* Acide adrénique (22:4 n-6)	* Pas de fonction démontrée
DPA	* Acide docosapentaénoïque (22:5 n-6)	* Synthétisé et incorporé dans les lipides membranaires en situation de carence alimentaire en AGPI n-3 (marqueur biologique spécifique)
	Série n-3	
ALA	* Acide α-linolénique (18:3 n-3)	* AG indispensable en tant que précurseur métabolique des AGPI n-3
	* Acide stéaridonique (18:4 n-3)	* Pas de fonction démontrée. Pourrait moduler la lipémie (réduction du taux de triglycérides circulants) en modulant l'expression des gènes de l'anabolisme lipidique au niveau du foie (Chilton *et al.*, 2008)
EPA	* Acide eicosapentaénoïque (EPA, 20:5 n-3)	* AG essentiel, précurseur de médiateurs lipidiques (prostanoïdes de la série 3, leucotriènes de la série 5, et AG hydroxylés (HETE, EET, lipoxines)) et régulateur de l'expression génique
	* Acide docosapentaénoïque (DPA n-3, 22:5 n-3)	* Pas de fonction démontrée
DHA	* Acide docosahexaénoïque (DHA, 22:6 n-3)	* AG indispensable. Il exerce trois fonctions majeures spécifiques comme constituant ubiquitaire des lipides de structure du système nerveux central (rôle structural), précurseur de médiateurs lipidiques (docosanoïdes, endocannabinoïdes), et régulateur de l'expression génique (effet direct ou indirect par ses dérivés)

AGPI, acides gras polyinsaturés ; HETE, acides hydroxyeicosatétraénoïques ; EET, acides époxyeicosatriénoïques.

Anses, 2011 (ISBN: 978-2-11-128598-9)

Occurrences alimentaires

ALA (g/100g)	**AL** (g/100g)
Huile de lin (53)	**Huile de pépin de raisin** (64)
Graine de lin (20)	**Huile de chanvre** (58)[a]
Huile de chanvre (19)[a]	**Huile de tournesol** (56)
Graine de chia (18)	**Huile de noix** (56)
Huile de noix de Grenoble (12)	**Huile de maïs** (54)
Huile de colza (8)	**Huile de soja** (52)
Cerneaux de noix de Grenoble (7,5)	**Huile de germe de blé** (47)
Huile de soja (7)	**Huile de sésame** (40)

Données issues du Ciqual de l'Anses (consulté en août 2021)
sauf [a]: Sanders, 2017 (DOI: 10.1016/B978-0-12-803968-7.00037-X)

EPA (mg/100g)	**DHA** (mg/100g)
Huile de foie de morue (8 000)	**Huile de foie de morue** (11 000)
Hareng fumé (3 000)	**Maquereau** (2 000)
Maquereau (800)	**Sardine à l'huile** (1 700)
Saumon (700)	**Saumon** (1 500)
Sardine à l'huile (700)	**Hareng fumé** (1 100)
Œuf « riche en ω-3 » (traces)[b]	**Œuf « riche en ω-3 »** (160)[b]
Œuf « ordinaire » (traces)	**Œuf « ordinaire »** (10-60)
Lait de vache (5)	**Lait de vache** (5)
Huile d'algue *Schizochytrium sp.* (6 000)[a]	**Huile algue *Schizochytrium sp.*** (41 000)[a]

Données issues du Ciqual de l'Anses (consulté en août 2021)
sauf [a] : Sanders, 2017 (DOI: 10.1016/B978-0-12-803968-7.00037-X)
et sauf [b] : Œufs « Bleu Blanc Cœur »: https://bleu-blanc-coeur.org/actualites/sante-humaine/volailles-ne-pas-mettre-tous-les-oeufs-dans-le-meme-panier/

Bach Virginie, 2022; Formation « *Alimentations végétariennes : accompagnement diététique optimisé* » (25h en e-learning);
Centre de Formation Diététique et Comportement (https://www.dietetiquecomportementale.com/copie-de-elearning-tca).

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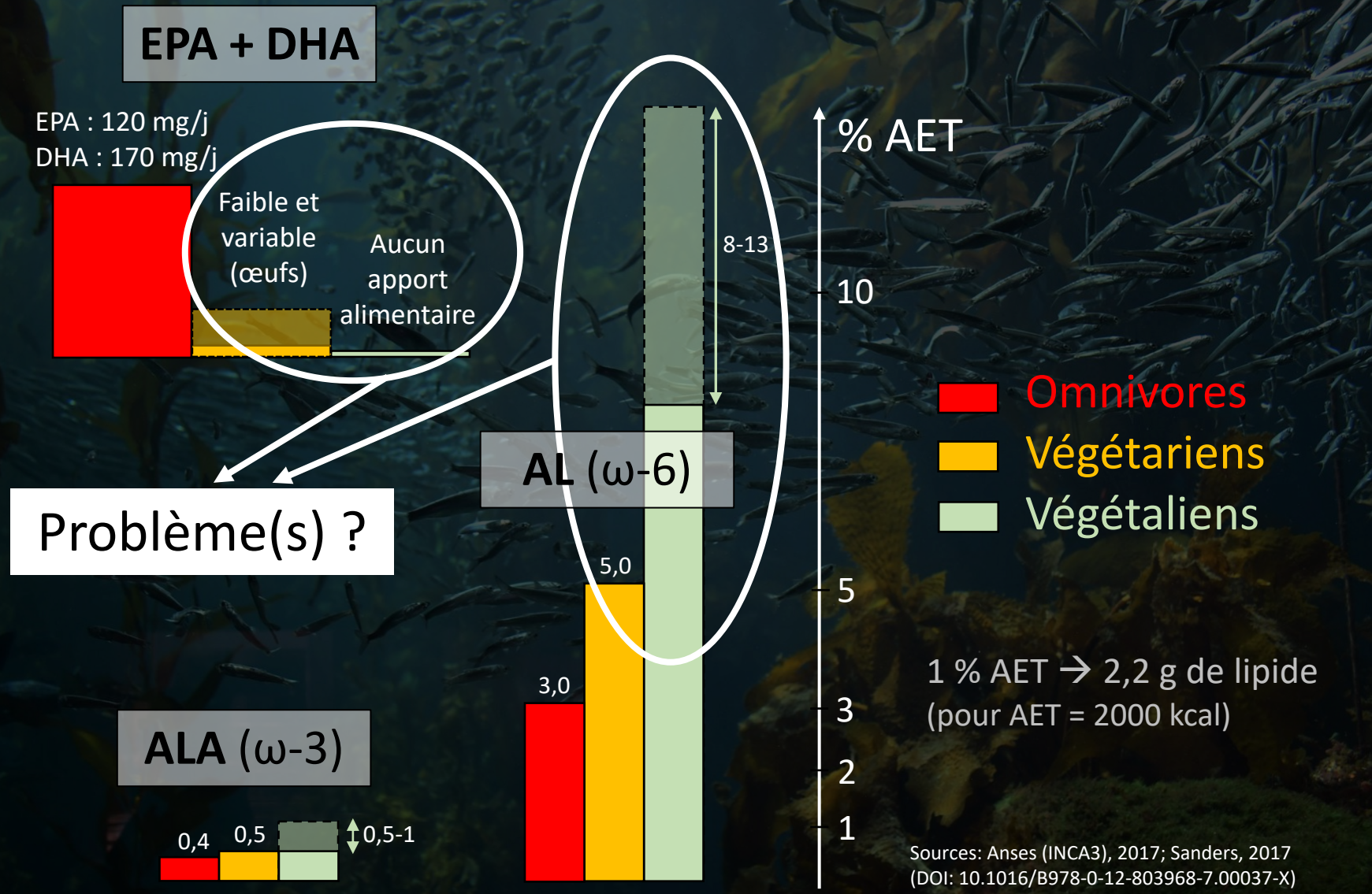
Consommations moyennes ?



Sources: Anses (INCA3), 2017; Sanders, 2017 (DOI: 10.1016/B978-0-12-803968-7.00037-X)

Bach Virginie, formation « *Alimentations végétariennes : accompagnement diététique optimisé* », 2022 (25h en e-learning); Centre de Formation Diététique et Comportement (https://www.dietetiquecomportementale.com/copie-de-elearning-tca).

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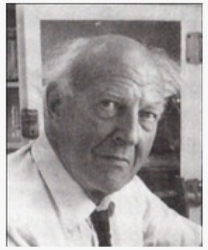

Hugh MacDonald Sinclair, ChB
(1910—1990)

Oméga-3

1953

Contexte historique

Recommandations d'apports

≈ 3200 kcal
Protéine: 377g (47% AET)
Glucide: 59g (7% AET)
Lipide: 162g (46% AET)

The Diet of Canadian Indians and Eskimos

By H. M. Sinclair, *Laboratory of Human Nutrition, University of Oxford*

Table 1. *Nutritional value of the adult Eskimo daily diet in* 1855

Foodstuff	Wt. of edible portion (g)	Calories (Cal.)	Total protein (g)	Animal protein (g)	Carbohydrate (g)	Fat (g)	Iron (mg)	Calcium (mg)	Phosphorus (mg)	Vitamin A (i.u.)	Carotenoids (μg)	Vitamin D (i.u.)	Thiamine (mg)	Nicotinic acid (mg)	Riboflavin (mg)	Ascorbic acid (mg)
Seal flesh	860	1686	163	163	26	103	23·2	95	1686	7740	0	0	0·95	42·1	1·2	69
Other flesh	225	441	43	43	7	27	6·1	25	441	2025	0	0	0·25	11·0	0·32	18
Capelin (salmon)	620	645	105	105	0	19	6·2	155	1500	508	0	6144	1·30	46·1	0·87	56
Other fish	370	444	61	61	0	11	3·3	67	699	56	0	0	0·15	8·5	0·18	7
Eggs	5	8	1	1	0	1	0·1	3	10	35	30	3	0·01	0	0·02	0
Berries	50	14	0	0	3	0	0·6	30	22	0	27	0	0·02	0·2	0·02	45
Bread	27	64	2	0	13	0	0·3	6	20	0	0	0	0·01	0·2	0·01	0
Barley and peas	6	14	1	0	2	0	0·4	11	19	0	0	0	0·03	0·1	0·02	0
Sugar	6	24	0	0	6	0	0	0	0	0	0	0	0	0	0	0
Coffee	6·5	19	1	0	2	1	0·3	9	10	0	0	0	0·06	0·6	0	0
Total		3359	377	373	59	162	40·5	401	4407	10364	57	6147	2·78	108·8	2·64	195
Oxford Nutrition Survey standard		3000	72	36	432	102	10	750	1000	833	3000	200	1·2	12	1·8	30
Proportion of standard met (%)		112	524	1036	14	159	405	54	441	1244	2	3074	232	907	147	650

Hypothèse :
Omega-3 et Omega-6 bénéfiques
/ santé cardio-vasculaire ?

The problem of cholesterol and atherosclerosis has been mentioned above. Wilber & Levine (1950) concluded that 'In the Alaskan Eskimos . . . there is a consistently high serum cholesterol on the one hand ; repeated clinical surveys, on the other, indicate an almost total absence of cardiovascular-renal diseases in the population' [their means are, however, 203 mg/100 ml. serum for males and 234 for

Sinclair, 1953 (DOI:10.1079/PNS19530016)

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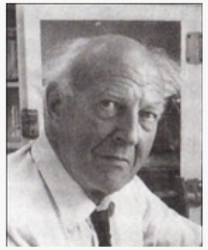

Hugh MacDonald Sinclair, ChB
(1910—1990)

1980

Contexte historique

Recommandations d'apports

Postgraduate Medical Journal (August 1980) **56**, 579–584

Prevention of coronary heart disease: the role of essential fatty acids

H. M. SINCLAIR
D.M., D.Sc., F.R.C.P.

International Institute of Human Nutrition, Sutton Courtenay, Oxon

Summary
There are 2 classes of essential fatty acids (EFA), the linoleic (n-6) and linolenic (n-3). They are required for the glycerophosphatides (phospholipids) of cellular

greatly decreased, very interesting changes in body lipids occurred, and there was probably toxicity of cetoleic acid (C22 : 1n-11).

It is suggested that the British diet should contain

Quality of fat and coronary thrombosis

The author concluded in 1956 (Sinclair, 1956) that atheroma and coronary thrombosis were related to a relative deficiency of EFA and not to the total fat in the diet (which at that time was regarded as the relevant factor by Keys (1953) and by Brock and Bronte-Stewart (1955)). On the one hand, Eskimoes on their traditional diet have the highest dietary fat in the world (Sinclair, 1953) but very rich in EFAs of the linolenic class, whereas the Japanese have very low dietary fat but this is also relatively rich in EFAs from fish (linolenic class) and soy-bean oil (51% linoleic acid and 7% linolenic).

Eskimoes have a diet very high in fat but relatively very rich in EFA; they do not get ischaemic heart disease. A study on the effect of the Eskimo diet on the author showed that platelet aggregation was

(C18 : 2n-6) and the linolenic (C18 : 3n-3); each can be desaturated and elongated. Certain fatty acids relevant to this discussion can be summarized thus:

Oméga-3

Obviously the next stage of this work is to see how small an amount of EFA of the linolenic class, for instance from occasional mackerel or other fatty fish, is sufficient to make platelets 'unsticky'. But the experiment has also indicated that cetoleic acid is toxic and that the linoleic class of EFA are definitely required; the Eskimoes have no doubt adapted to the toxicity of the former, and as in the author's experiment appear to conserve their limited amounts of arachidonic acid. Since C20 : 5n-3 inhibits the desaturation and elongation of linoleic to arachidonic acid and since our usual diets contain little of the latter, too much of the linolenic class may be undesirable. A balance is needed between the EFA of the linoleic and linolenic classes.

Hypothèse :
Equilibre entre
Omega-3 et Omega-6

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1 mars 2024

Sinclair, 1980 (DOI:10.1136 pgmj.56.658.579)

Recommandations AGPI - FAO & France -

Recommandations d'apports

Organisation des Nations Unies pour l'alimentation et l'agriculture

RÉPUBLIQUE FRANÇAISE
Liberté
Égalité
Fraternité

anses
agence nationale de sécurité sanitaire alimentation, environnement, travail

ADULTES

	ω-3			ω-6	AL /ALA
	ALA (% AET)	**EPA**	**DHA**	**AL** (% AET)	
FAO (2010)	> 0,5	250 mg/j		2-3	/
	0,5-2 (% AET)				
ANSES (2011, 2016)	1	250 mg/j	250 mg /j	4	< 5

FEMMES ENCEINTES ou ALLAITANTES

	ω-3			ω-6	AL /ALA
	ALA (% AET)	**EPA**	**DHA**	**AL** (% AET)	
FAO (2010)	> 0,5	***300 mg/j (DHA > 200 mg/j)***		2-3	/
	0,5-2 (% AET)				
ANSES (2011, 2016)	1	250 mg/j	250 mg /j	4	< 5

Anses, 2011 (ISBN: 978-2-11-128598-9); Anses, 2016 (ISBN: 979-10-286-0140-9); FAO, 2010 (ISBN:978-92-5-106733-8)

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1 mars 2024

Recommandations adultes EPA/DHA - autres comités d'experts-

Recommandations d'apports

LEGENDES

[1] = International Society for the Study of Fatty Acids and Lipids
[2] = World Association of Perinatal Medicine
[3] = World Gastroenterology Organisation

« CV » = recommandation explicitement établie pour la prévention des maladies cardio-vasculaires
« ω3-CL » = oméga-3 à chaîne longue
« p/sem » = portion / semaine
« AET » = de l'apport énergétique total
« + » = la quantité indiquée après ce signe doit être ajoutée à la quantité recommandée pour la population générale
« ♀ E/A » = femmes enceintes et allaitantes (en gras italique)
« **V » = recommandations s'adressant explicitement aux véganes** (en gras souligné)

Au sein d'un même pays (ou du même regroupement international), lorsque plusieurs quantités sont indiquées, cela signifie que différents comités ont établi des recommandations distinctes.

ISSFAL, 2014; Global Recommendations for EPA and DHA Intake (Rev 19 November 2014)

Apports recommandés en EPA et DHA chez l'adulte

(document réalisé à partir des données de l'ISSFAL, 2014)

Comités d'experts		Poisson	ω3 totaux	EPA + DHA	DHA
Mondiaux	OMS (2003)	/	1-2% AET	/	/
	FAO (2010)	/	0,5-2% AET	0,250g ***0,300g (♀ E/A)***	***> 0,200g (♀ E/A)***
	ISSFAL[1] (2004, 2007)	/	/	> 0,500g (CV)	***0,200g (♀ E/A)***
	WAPM[2] (2008)	/	/	/	***0,200g (♀ E/A)***
	WGO[3] (2008)	3-5 p/sem	/	/	/
Nationaux / Regroupements régionaux de nations	Australie et Nouvelle Zélande (2006, 2008)	/	/	0,500g (CV) 0,160g (♂) / 0,090g (♀) ***0,110-0,145g (♀ E/A)***	/
	Europe (2010, 2007, 2012)	/	/	0,250g	***+ 0,1-0,2g (♀ E/A)***
				/	***0,200g (♀ E/A)***
		≥ 2 p/sem, dont au moins 1 poisson gras (CV)			/
	Autriche (2008) Allemagne (2008) Suisse (2008)	/	0,5% AET	0,250g	***> 0,200g (♀ E/A)***
	Pologne (2014)	/	/	/	***> 0,6-1,0g (♀ E/A)***
	Belgique (2004)	2 p/sem dt poisson gras (CV)	/	/	***0,250g (♀ E/A)***
	Pays-Bas (2001, 2006)	/	/	0,450g ***20mg/kg/j (♀ E/A)***	/
	Scandinavie (2013)	/	> 1% AET	/	***0,200g (♀ E/A)***
	Royaume-Uni (2004, 2005, 2007, 2008, 2014)	≥ 2 p/sem, dont au moins 1 poisson gras	/	0,450g	/
		/		0,200g	
	Italie (2007)	/	/	/	***♀ E/A : supplémentation nécessaire (MAIS aucune valeur chiffrée) (V)***
	Russie (2010)	/	/	1,3g	700mg
	Brésil (2014)	/	/	/	***0,200g (♀ E/A)***
	USA (2005, 2014, 2009, 2008, 2002, 2003, 2012)	/	1,6g (♂) 1,1g (♀)	0,16g (♂) 0,11g (♀)	/
		≥ 2 p/sem, dont au moins 1 poisson gras	/	/	/
		/	/	0,500g	/
		/	/	/	***0,200g (♀ E/A)***
		/	/	/	***0,2-0,3g (♀ E/A) (V)***
	Canada (2007)	/	/	0,500g	/
	Chine (2014)	/	/	0,25-2g	***0,200g (♀ E/A)***
	Japon (2014)	/	2-2,4g (♂) 1,6-1,9g (♀) ***1,8g (♀ E/A)***	/	/
	Israël (2011)	/	/	0,5-1g (CV)	/
	Singapour (2014)	2 p/sem	/	/	/

Recommandations d'apports

RÉPUBLIQUE FRANÇAISE
Liberté
Égalité
Fraternité

anses

agence nationale de sécurité sanitaire
alimentation, environnement, travail

* **AGPI n-3 à longue chaîne (DHA et EPA).** Les données nouvelles, et en particulier celles relatives à la très faible conversion de l'acide α-linolénique en DHA chez l'Homme, conduisent donc le groupe de travail à définir un besoin physiologique minimal pour le DHA. La valeur retenue est de 250 $mg.j^{-1}$ pour un adulte (0,1 % de l'énergie). Elle rejoint la valeur recommandée par d'autres comités de nutrition (Simopoulos et al., 2000, ISSFAL, 2004) et correspond aux données de consommation pour la population française (Astorg et al., 2004). La valeur de ce besoin physiologique est 2 fois plus élevée que la valeur des ANC de 2001, mais on ne disposait pas alors de données précises de consommation et le taux de conversion de l'acide α-linolénique en DHA était surestimé. En ce qui concerne l'EPA, et bien que les fonctions qu'exerce cet AGPI n-3 soient essentielles (précurseur d'une famille d'eicosanoïdes), le groupe de travail ne dispose pas actuellement d'arguments suffisants pour le considérer comme rigoureusement indispensable et définir un besoin physiologique minimal. En effet, la conversion de l'acide α-linolénique en EPA est significative dès lors que les apports en acide α-linolénique (et en acide linoléique pour des raisons de compétition) sont adéquats.

Conclusion

La relation entre la consommation de poisson ou d'EPA-DHA et le risque cardiovasculaire dépend des paramètres évalués. Ainsi, la prévention des morts subites augmente rapidement avec les apports puis se stabilise. A l'inverse, la baisse de la triglycéridémie est linéaire et proportionnelle aux quantités consommées. Les études épidémiologiques et les essais d'intervention montrent que la consommation de poisson ou d'EPA et DHA diminue la mortalité cardio-vasculaire. Ces effets sont observés pour des apports compris entre 0,4 $g.j^{-1}$ et 1,8 $g.j^{-1}$ d'AGPI-LC n-3 (EPA-DHA) chez des patients avec des antécédents vasculaires, mais ils sont moins bien documentés en prévention primaire.
Par conséquent, un apport journalier de 500 mg d'EPA et DHA (soit 0,25 % de l'AE) semble justifié pour la population générale dans une perspective de prévention cardio-vasculaire. Cet apport, sur la base des études d'intervention peut atteindre 750 mg pour les sujets à haut risque cardiovasculaire (Mozaffarian et Rimm, 2006).

?

Anses, 2011 (ISBN: 978-2-11-128598-9)

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Recommandations d'apports

Plant Sources. Alpha-linolenic acid (ALA) (18:3n-3) is an n-3 fatty acid present in flaxseed, canola, soybeans, and walnuts.[121] In humans, ALA is converted to EPA in small quantities (in women more than men); further conversion to DHA is very limited.[201] Consumption of ALA (eg, 2-3 g/d) may reduce cardiovascular risk[202] or affect neurodevelopment, but benefits are less established compared with those for EPA and DHA.

Optimal Intakes

Optimal intake of n-3 PUFAs may vary depending on population and outcome of interest. In the general population, 250 mg/d of EPA and DHA is a reasonable target intake to reduce CHD mortality. Because dietary n-3 PUFAs

CLINICAL REVIEW — CLINICIAN'S CORNER

Fish Intake, Contaminants, and Human Health
Evaluating the Risks and the Benefits

Dariush Mozaffarian, MD, DrPH
Eric B. Rimm, ScD

Context Fish (finfish or shellfish) may have health benefits and also contain contaminants, resulting in confusion over the role of fish consumption in a healthy diet.

Evidence Acquisition We searched MEDLINE, governmental reports, and meta-analyses, supplemented by hand reviews of references and direct investigator contacts, to identify reports published through April 2006 evaluating (1) intake of fish or fish oil and cardiovascular risk, (2) effects of methylmercury and fish oil on early neurodevelopment, (3) risks of methylmercury for cardiovascular and neurologic outcomes in adults, and (4) health risks of dioxins and polychlorinated biphenyls in fish. We concentrated on studies evaluating risk in humans, focusing on evidence, when available, from randomized trials and large prospective studies. When possible, meta-analyses were performed to characterize benefits and risks most precisely.

Evidence Synthesis Modest consumption of fish (eg, 1-2 servings/wk), especially species higher in the n-3 fatty acids eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), reduces risk of coronary death by 36% (95% confidence interval, 20%-50%; $P<.001$) and total mortality by 17% (95% confidence interval, 0%-32%; $P=.046$) and may favorably affect other clinical outcomes. Intake of 250 mg/d of EPA and DHA appears sufficient for primary prevention. DHA appears beneficial for, and low-level methylmercury may adversely affect, early neurodevelopment. Women of childbearing age and nursing mothers should consume 2 seafood servings/wk, limiting intake of selected species. Health effects of low-level methylmercury in adults are not clearly established; methylmercury may modestly decrease the cardiovascular benefits of fish intake. A variety of seafood should be consumed; individuals with very high consumption (≥5 servings/wk) should limit intake of species highest in mercury levels. Levels of dioxins and polychlorinated biphenyls in fish are low, and potential carcinogenic and other effects are outweighed by potential benefits of fish intake and should have little impact on choices or consumption of seafood (women of childbearing age should

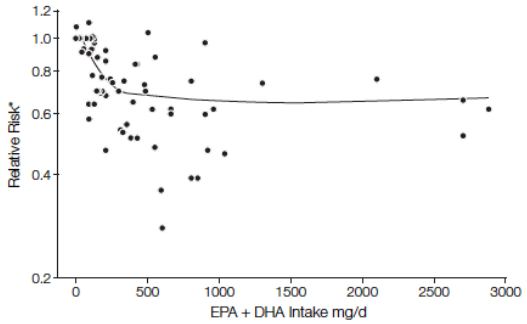
SINCE THE PUBLICATION OF PIONEERING studies demonstrating low rates of death from coronary heart disease (CHD) among Greenland Eskimos,[1] fish (used herein to refer to finfish or shellfish) has been considered a healthy food. During ensuing years, evidence from several research paradigms—including animal-experimental, observational, and clinical studies—further supported this hypothesis and identified 2 long-chain n-3 polyunsaturated fatty acids (n-3 PUFAs), eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), as the likely active constituents.[2-20] DHA also appears important for neurodevelopment during gestation and infancy.[21-26] Conversely, concern has arisen over potential harm from mercury, dioxins, and polychlorinated biphenyls (PCBs) present in some fish species.[27-34] The public is faced with seemingly conflicting reports on the

Conclusions of the evidenc… ceed the pote… take, exceptin… *JAMA.* 2006;296:…

topics for w… cern are g… MEDLINE, g… systematic re… to identify re… April 2006 ev… or fish oil ar… events and m… thylmercury … rodevelopme… mercury fo… neurologic ou… health risks o…

See also Patient Page.
CME available online at www.jama.com

Figure 2. Relationship Between Intake of Fish or Fish Oil and Relative Risks of CHD Death in Prospective Cohort Studies and Randomized Clinical Trials



The relationship between intake of fish or fish oil and relative risk of coronary heart disease (CHD) death in a pooled analysis of the prospective studies and randomized trials shown in Figure 1, evaluated nonparametrically using restricted cubic splines[38,39] and adjusted for each within-study relationship. Given the much higher reference group intakes in some studies, the reference relative risk was scaled by 0.7 for studies with reference group intakes between 150-500 mg/d of eicosapentaenoic acid (EPA) + docosahexaenoic acid (DHA) (n=5) and by 0.6 for studies with reference group intakes >500 mg/d (n=1) based on spline relationships prior to including these studies; exclusion of these studies, or of the few groups with intakes >1000 mg/d, had little effect on the pooled spline relationship. A significant threshold effect ($P<.001$) was evident at intake of 250 mg/d: between 0 and 250 mg/d, mortality risk was lower by 14.6% (95% confidence interval [CI], 8% to 21%) per each 100-mg/d greater intake (total risk reduction, 36%; 95% CI, 20% to 50%; $P<.001$), while at higher intakes, risk was not further lowered (0.0% change per each 100 mg/d; 95% CI, –0.9% to 0.8%; $P=.94$). *Relative risks in the control and intervention groups (for randomized trials) or relative risks in the reference group and multivariable-adjusted relative risks in the comparison groups (for cohort studies).

1. Aucun effet clinique propre au DHA

2. Apports conjoints « EPA+DHA »

0-250 mg/j ==> ↘ -36% mortalité coronarienne
(-14,6% par tranche de +100 mg/j)
> 250 mg/j ==> bénéfices sup. très modestes = **EFFET SEUIL**

3. Fortes consommations ALA (> 1% AET) pourraient également être bénéfique mais manque de données dispo…

Fabien BADARIOTTI
1 mars 2024

Anses, 2011 (ISBN: 978-2-11-128598-9); Mozzafarian et Rimm, 2006 (DOI: 10.1001/jama.296.15.1885)

Étude observationnelle ?

Maladie coronarienne

Recommandations d'apports

60 g/j = conso. optimale
(risque minimum / santé du cœur)

Effet de l'apport en EPA+DHA ?

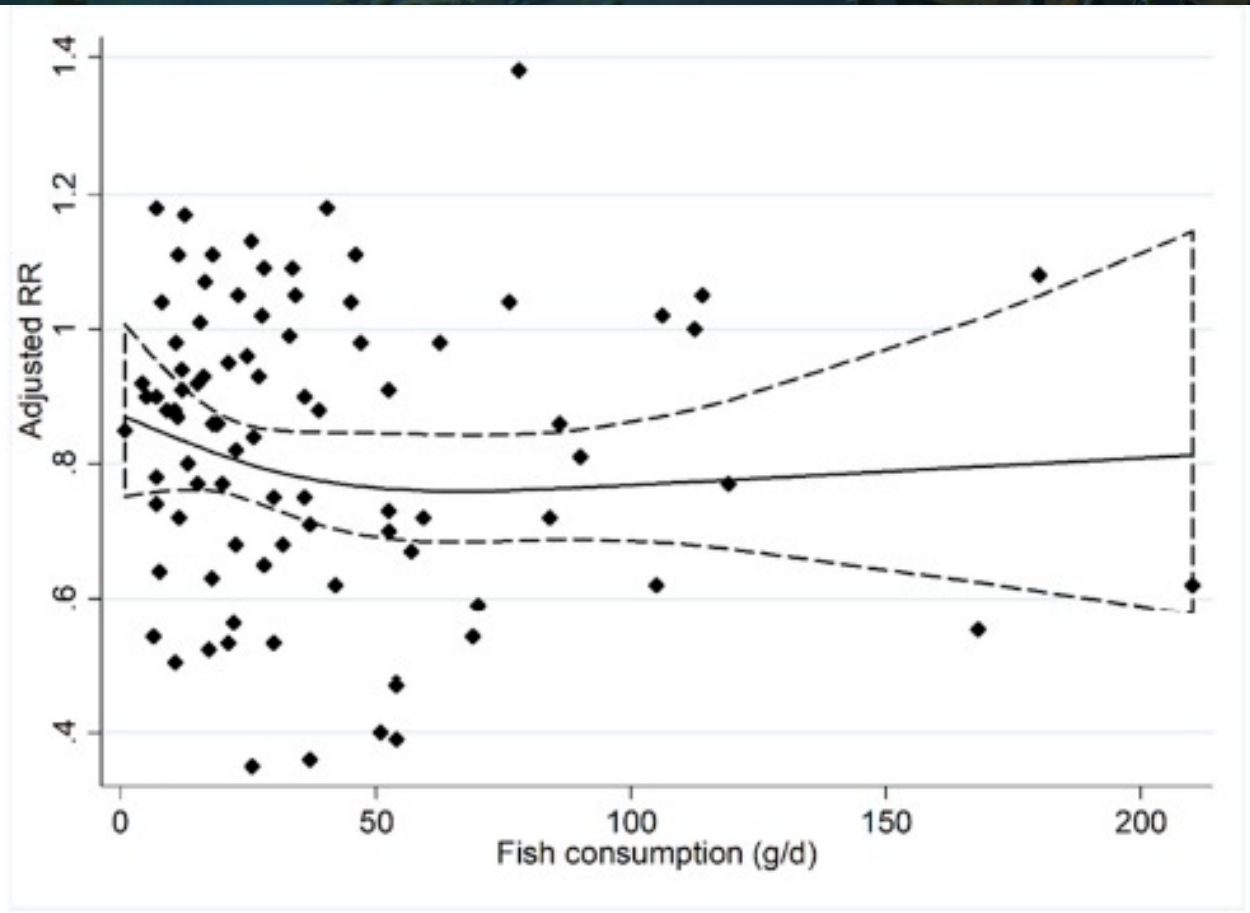


Figure 5. The dose–response analysis between the fish consumption and the CHD mortality. The diamonds represented the adjusted RRs for each exposure quantile of fish consumption in the included individual studies. The solid line and the long dashed line represent the estimated relative risk (RR) and its 95% confidence interval (CI). Abbreviations: g/d, grams per day.

Zhang *et al.*, 2020 (DOI:10.3390 nu12082278)

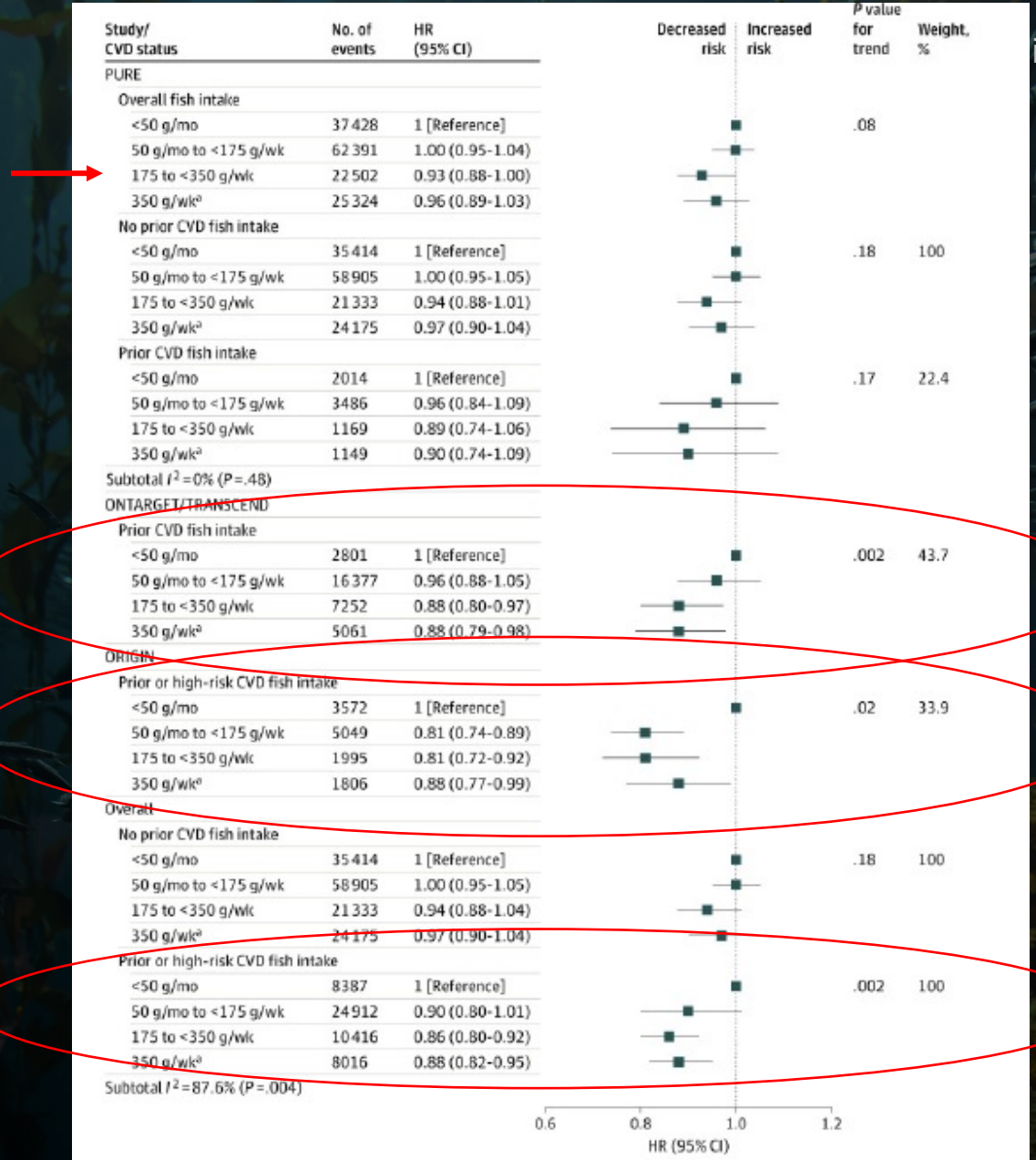
Étude observationnelle

Maladie cardiovasculaire

Recommandations d'apports

Bénéfices significatifs uniquement en prévention II
(risque cardiovasculaire)

Effet de l'apport en EPA+DHA ?



Fish Intake vs Risk of Composite of Death or Major Cardiovascular Disease (CVD) by Study and by Prior Cardiovascular Disease

[a]Adjusted for age, sex, study center (random effect), body mass index, educational level, smoking status, physical activity, alcohol intake, urban vs rural location, history of diabetes, cardiovascular disease, cancer, use of statin or antihypertension medication, and intake of fruit, vegetables, red meat, poultry, dairy, and total energy.

Mohan *et al.*, 2021 (DOI:10.1001/jamainternmed.2021.0036)

Fabien BADARIOTTI
1 mars 2024

Études observationnelles

Maladie coronarienne
et cardio-vasculaire

Recommandations d'apports

Santé cardiaque
→ seule la conso. de poisson GRAS est bénéfique

Santé cardio-vasculaire globale
→ aucun effet significatif

→ **Effet probable de EPA+DHA**
(aucune quantification)

Giosuè *et al.*, 2022 (DOI: 10.1093 advances nmac006)

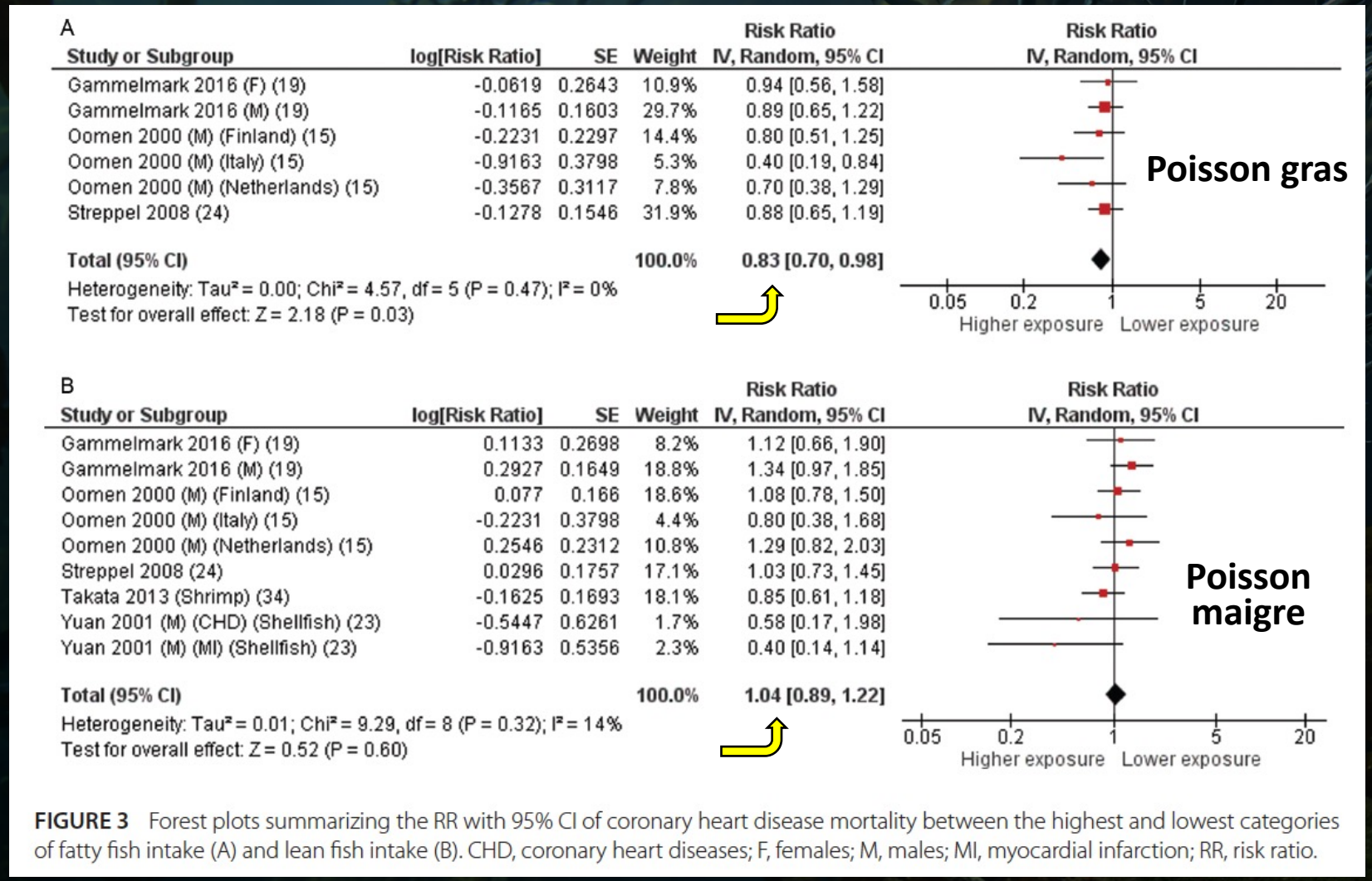


FIGURE 3 Forest plots summarizing the RR with 95% CI of coronary heart disease mortality between the highest and lowest categories of fatty fish intake (A) and lean fish intake (B). CHD, coronary heart diseases; F, females; M, males; MI, myocardial infarction; RR, risk ratio.

Fabien BADARIOTTI
1 mars 2024

Études interventionnelles

Maladie coronarienne
& cardio-vasculaire

Recommandations d'apports

Santé cardiaque &
cardio-vasculaire globale
→ Légère baisse du risque
(prévention I^aire^)

Résultat positif pour EPA+DHA
et pour EPA seul
(mais pas pour DHA seul)

Apports médicamenteux d'EPA+DHA (études interventionnelles)

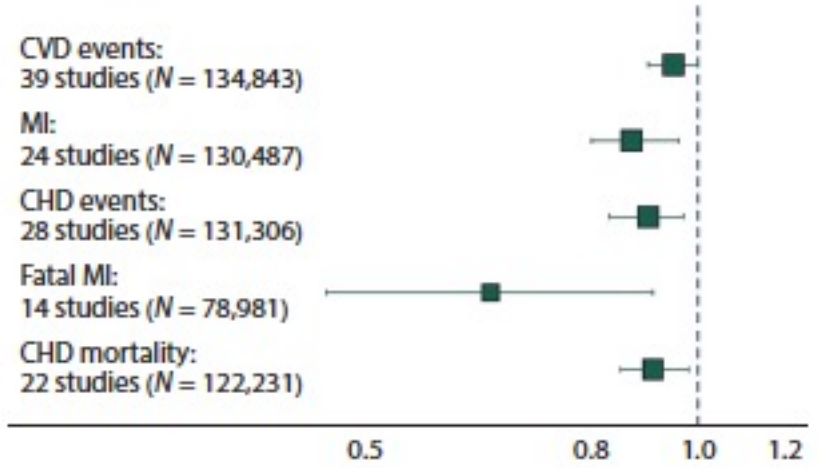


Figure 2

Pooled results from meta-analysis of randomized controlled trials of long-chain omega-3 polyunsaturated fatty acids and cardiovascular outcomes. The figure shows the pooled estimate of relative risk and 95% confidence interval, as well as the number of studies and combined number of participants. Abbreviations: CHD, coronary heart disease; CVD, cardiovascular disease; MI, myocardial infarction. Figure reprinted from Reference 76; copyright 2021 Mayo Foundation for Medical Education and Research, with permission from Elsevier.

Quantités très élevées
→ 2-10 g/j d'EPA+DHA

Utilisation médicamenteuse :
HS / cette présentation

Djuricic *et al.*, 2023 (DOI: 10.1146 annurev-pharmtox-051921-090208)

Fabien BADARIOTTI
1 mars 2024

Étude observationnelle

Risque d'AVC

Recommandations d'apports

Diminution du risque d'AVC ischémique (pas hémorragique)

-19% pour le
3ème quintile d'EPA
(% des lipides sanguins)

Table. Hazard Ratios (95% CI) for Stroke by FA Quintile (Versus Quintile 1) Excluding the UK Biobank

Fatty acid		Total stroke	Ischemic stroke	Hemorrhagic stroke
DHA	Quintile 1 (reference)	1.0	1.0	1.0
	Quintile 2	0.92 (0.85–1)*	0.9 (0.82–0.99)*	1.08 (0.82–1.42)
	Quintile 3	0.93 (0.86–1.02)	0.9 (0.82–0.99)*	1.03 (0.78–1.36)
	Quintile 4	0.90 (0.83–0.98)*	0.88 (0.8–0.96)†	1.02 (0.77–1.36)
	Quintile 5	0.88 (0.81–0.96)†	0.86 (0.78–0.95)†	1.09 (0.82–1.46)
	P value for trend	0.04	0.03	0.79
EPA	Quintile 1 (reference)	1.0	1.0	1.0
	Quintile 2	1.00 (0.92–1.08)	0.99 (0.9–1.08)	0.96 (0.73–1.26)
	Quintile 3	0.82 (0.75–0.89)‡	0.81 (0.74–0.9)‡	0.82 (0.62–1.09)
	Quintile 4	0.91 (0.83–0.99)*	0.91 (0.82–1.00)	0.86 (0.65–1.14)
	Quintile 5	0.83 (0.76–0.91)‡	0.82 (0.74–0.91)‡	0.9 (0.67–1.21)
	P value for trend	0.001	0.002	0.45
DPA	Quintile 1 (Reference)	1.0	1.0	1.0
	Quintile 2	1.00 (0.91–1.11)	1.04 (0.94–1.16)	0.83 (0.61–1.13)
	Quintile 3	1.03 (0.93–1.13)	1.04 (0.93–1.16)	0.64 (0.47–0.89)†
	Quintile 4	1.01 (0.91–1.12)	1.04 (0.94–1.16)	0.89 (0.66–1.22)
	Quintile 5	0.89 (0.8–0.99)*	0.93 (0.83–1.05)	0.79 (0.57–1.09)
	P value for trend	0.21	0.47	0.45
EPA+DHA	Quintile 1 (Reference)	1.0	1.0	1.0
	Quintile 2	0.94 (0.86–1.02)	0.93 (0.85–1.02)	1.14 (0.86–1.51)
	Quintile 3	0.92 (0.84–0.99)*	0.88 (0.8–0.97)*	1.00 (0.75–1.35)
	Quintile 4	0.92 (0.84–0.99)*	0.89 (0.81–0.98)*	1.17 (0.87–1.57)
	Quintile 5	0.83 (0.76–0.91)‡	0.82 (0.74–0.91)‡	1.04 (0.76–1.42)
	P value for trend	0.007	0.006	0.82

Adjusted for age (continuous), sex (men/women), race (binary: White/non-White), field center (categories), body mass index (continuous), education (less than high school graduate, high school graduate, at least some college or vocational school), occupation (if available), smoking (current, former, never), physical activity (kcal/wk, METS/wk, or h/d), alcohol intake (drinks or servings/d, g/d, or mL/d), prevalent DM (treated or physician-diagnosed), prevalent hypertension (treated or physician-diagnosed), prevalent dyslipidemia (treated or physician-diagnosed), prevalent atherosclerotic CVD, history of AF, and circulating omega-6 fatty acid levels (ie, the sum of linoleic and arachidonic acids). DHA quintiles including the UKBB data is shown in Figure 1. AF indicates atrial fibrillation; CVD, cardiovascular disease; DHA, docosahexaenoic acid; DM, diabetes; EPA, eicosapentaenoic acid; and METS, metabolic equivalents.

*$P<0.05$.
†$P<0.01$.
‡$P<0.001$.

Supplemental Table 4. 10th and 90th percentiles of n-3 fatty acids (% of total fatty acids in the lipid fraction) by cohort and by lipid fraction†

		EPA			DPA			DHA			EPA+DHA		
Cohort	Lipid fraction	10th	50th	90th	10th	50th	90th	10th	50th	90th	10th	50th	90th
Diet Cancer and Health	Adipose	0.05	0.10	0.16	0.18	0.27	0.39	0.15	0.26	0.45	0.21	0.36	0.61
3C	Plasma	0.30	0.90	1.80	0.30	0.50	0.60	1.60	2.40	3.50	2.20	3.30	5.10
CCCC	Plasma	0.20	0.33	0.60	N/A	N/A	N/A	1.05	1.70	2.61	1.27	2.07	3.15
CIRCS	Plasma	0.32	0.80	1.84	0.04	0.12	0.26	0.54	1.07	1.98	0.92	1.88	3.72
Hisayama	Plasma	0.99	2.07	3.95	0.43	0.62	0.89	3.18	4.58	6.49	4.30	6.68	10.23
Inchianti	Plasma	0.41	0.59	0.84	N/A	N/A	N/A	1.38	2.24	3.27	1.85	2.86	4.07
HPFS	Plasma	0.22	0.37	0.74	0.25	0.35	0.45	0.62	1.01	1.72	0.94	1.43	2.47
NHS	Plasma	0.22	0.41	0.75	0.23	0.35	0.51	0.74	1.25	2.09	1.05	1.70	2.86
KIHD	Plasma	0.85	1.47	2.66	0.43	0.54	0.69	1.63	2.35	3.36	2.60	3.79	5.93
UKBiobank	Plasma	N/A	N/A	N/A	N/A	N/A	N/A	1.56	1.93	2.36	N/A	N/A	N/A
PDPS	Plasma	0.69	1.27	2.72	0.40	0.57	0.80	1.64	2.74	4.36	2.42	3.91	6.98
Rotterdam study	Plasma	N/A	N/A	N/A	N/A	N/A	N/A	0.85	1.24	1.85	N/A	N/A	N/A
60YO	Plasma CE	1.13	1.87	3.25	N/A	N/A	N/A	0.62	0.89	1.24	1.81	2.76	4.43
AOC	Plasma CE	0.61	1.06	2.22	0.00	0.00	0.08	0.43	0.67	1.00	1.12	1.73	3.16
MORGEN	Plasma CE	0.29	0.65	1.16	N/A	N/A	N/A	0.27	0.45	0.76	0.68	1.09	1.81
ULSAM50	Plasma CE	0.70	1.27	2.16	N/A	N/A	N/A	0.47	0.69	0.97	1.23	1.95	3.08
AgeCoDe	Plasma PL	0.50	0.92	1.77	0.63	0.84	1.11	2.40	3.64	5.14	3.08	4.61	6.65
AGESR	Plasma PL	1.31	2.38	5.19	0.92	1.14	1.43	4.51	6.14	8.39	5.94	8.57	13.40
CHS	Plasma PL	0.30	0.51	0.93	0.63	0.82	1.05	1.96	2.89	4.36	2.40	3.40	5.19
CSHA	Plasma PL	0.31	0.63	1.09	1.19	2.10	2.73	1.84	3.57	5.12	2.20	4.22	6.00
EPIC-Norfolk	Plasma PL	0.59	1.06	2.10	3.32	4.90	7.26	0.96	1.36	1.89	4.09	6.00	9.19
HCS	Plasma PL	1.30	2.10	4.15	0.49	0.77	1.04	3.04	4.59	7.02	4.66	6.64	10.74
MESA	Plasma PL	0.36	0.67	1.65	0.66	0.91	1.20	2.11	3.59	5.83	2.61	4.26	7.38
VITAL	Plasma PL	0.30	0.50	1.00	N/A	N/A	N/A	1.30	1.90	2.90	1.70	2.50	3.80
PIVUS	Plasma PL	1.21	1.92	3.60	0.88	1.12	1.41	3.74	5.15	6.81	6.14	8.24	11.40
EPIC - Spain	RBC PL	0.53	0.88	1.34	N/A	N/A	N/A	5.85	7.55	9.15	6.55	8.44	10.44
Framingham	RBC PL	0.37	0.60	1.16	2.21	2.67	3.27	3.14	4.60	6.60	3.59	5.20	7.72
METSIM	RBC PL	0.91	1.42	2.30	2.12	2.59	3.10	4.73	6.20	7.58	5.74	7.64	9.77
WHIMS	RBC PL	0.33	0.60	1.17	1.96	2.50	3.05	2.88	4.35	6.40	3.37	4.97	7.38
	Mean	10th	50th	90th	10th	50th	90th	10th	50th	90th	10th	50th	90th
	Plasma CE	0.68	1.21	2.20	N/A	N/A	N/A	0.45	0.68	0.99	1.21	1.88	3.12
	Plasma	0.54	1.06	2.06	0.32	0.47	0.65	1.49	2.25	3.31	2.22	3.50	5.60
	Plasma PL	0.69	1.19	2.39	1.09	1.57	2.15	2.43	3.65	5.27	3.65	5.38	8.19
	RBC PL	0.53	0.88	1.49	2.10	2.59	3.14	4.15	5.67	7.43	4.81	6.56	8.83
	Adipose	0.05	0.10	0.16	0.18	0.27	0.39	0.15	0.26	0.45	0.21	0.36	0.61

Mean	10th	50th	90th
Plasma CE	0.68	1.21	2.20
Plasma	0.54	1.06	2.06
Plasma PL	0.69	1.19	2.39
RBC PL	0.53	0.88	1.49
Adipose	0.05	0.10	0.16

?

O'Keefe *et al.*, 2024 (DOI: 10.1161/STROKEAHA.123.044281)

Fabien BADARIOTTI
1 mars 2024

Étude interventionnelle

Evaluation du niveau de conversion ALA → EPA (omni ♂ uniquement)

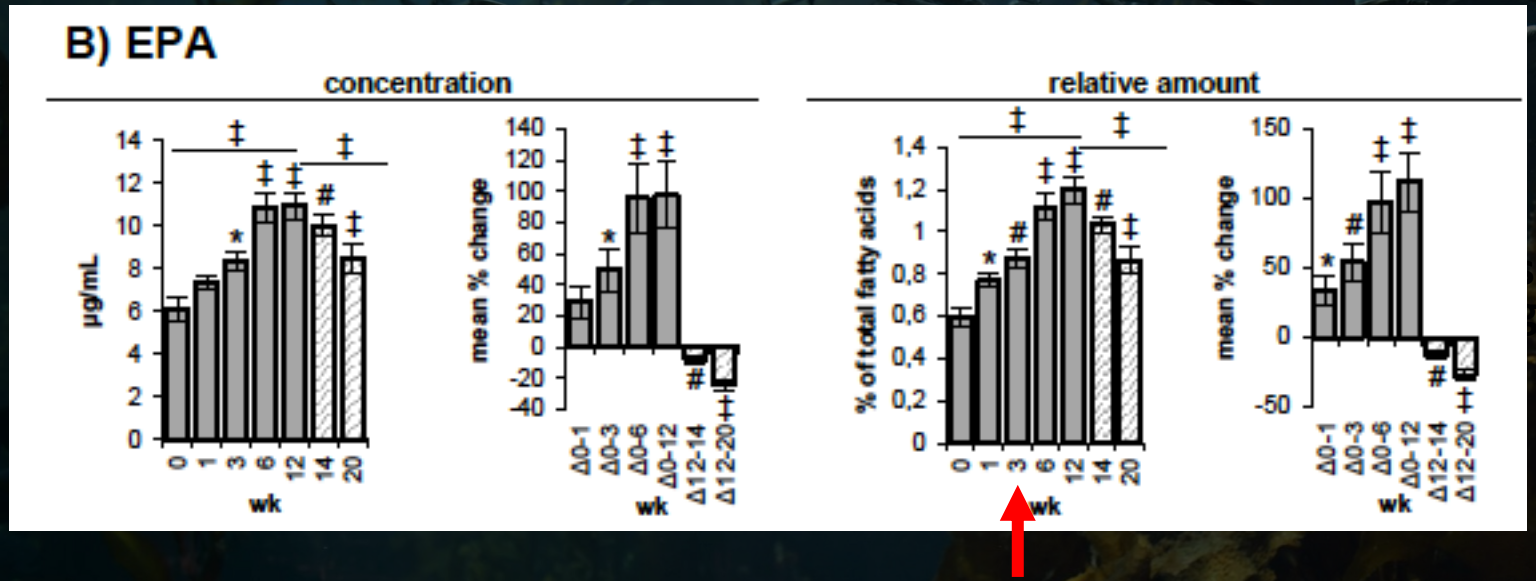
Recommandations d'apports

+ 14g ALA/j (22g/j huile de lin) pendant 20 semaines

ALA seul permet d'augmenter très significativement le % d'EPA dans les lipides des globules rouges

Table 3: Concentration and relative amount of fatty acids in red blood cells.

	wk 0			wk 1			t-test[a]	wk 3			t-test[a]	wk 6			t-test[a]	wk 12			t-test[a]	An reM[b]
	mean	±	SE	mean	±	SE	p (wk 1 - wk 0)	mean	±	SE	p (wk 3 - wk 0)	mean	±	SE	p (wk 6 - wk 0)	mean	±	SE	p (wk 12 - wk 0)	p
C20:5n3 µg/mL	6.13	±	0.51	7.33	±	0.33	n.s.	8.38	±	0.42	**0.021**	10.9	±	0.67	**<0.001**	11.0	±	0.64	**<0.001**	**<0.001**
% of total FA	0.60	±	0.04	0.77	±	0.03	**0.006**	0.87	±	0.04	**0.002**	1.12	±	0.06	**<0.001**	1.20	±	0.06	**<0.001**	**<0.001**



Action d'une forte consommation d'ALA via la synthèse endogène d'EPA ?

Greupner *et al.*, 2018 (DOI: 10.1039/C7FO01809F)

Fabien BADARIOTTI
1 mars 2024

Revue

Maladies cardio-vasculaires (MCV)

Recommandations d'apports

Activités biologiques distinctes de l'EPA et du DHA

Rôle prépondérant de l'EPA dans la prévention des MCV ?

Role of Omega-3 Fatty Acids in Cardiovascular Disease: the Debate Continues

Samuel C. R. Sherratt[1,2] · Peter Libby[3] · Matthew J. Budoff[4] · Deepak L. Bhatt[5] · R. Preston Mason[2,3]

Accepted: 15 October 2022 / Published online: 29 December 2022

Abstract

Purpose of Review The omega-3 fatty acids (n3-FAs), eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), have recently undergone testing for their ability to reduce residual cardiovascular (CV) risk among statin-treated subjects. The outcome trials have yielded highly inconsistent results, perhaps attributable to variations in dosage, formulation, and composition. In particular, CV trials using icosapent ethyl (IPE), a highly purified ethyl ester of EPA, reproducibly reduced CV events and progression of atherosclerosis compared with mixed EPA/DHA treatments. This review summarizes the mechanistic evidence for differences among n3-FAs on the development and manifestations of atherothrombotic disease.

Recent Findings Large randomized clinical trials with n3-FAs have produced discordant outcomes despite similar patient profiles, doses, and triglyceride (TG)-lowering effects. A large, randomized trial with IPE, a prescription EPA only formulation, showed robust reduction in CV events in statin treated patients in a manner proportional to achieved blood EPA concentrations. Multiple trials using mixed EPA/DHA formulations have not shown such benefits, despite similar TG lowering. These inconsistencies have inspired investigations into mechanistic differences among n3-FAs, as EPA and DHA have distinct membrane interactions, metabolic products, effects on cholesterol efflux, antioxidant properties, and tissue distribution. EPA maintains normal membrane cholesterol distribution, enhances endothelial function, and in combination with statins improves features implicated in plaque stability and reduces lipid content of plaques.

Summary Insights into reductions in residual CV risk have emerged from clinical trials using different formulations of n3-FAs. Among high-risk patients on contemporary care, mixed n3-FA formulations showed no reduction in CV events. The distinct benefits of IPE in multiple trials may arise from pleiotropic actions that correlate with on-treatment EPA levels beyond TG-lowering. These effects include altered platelet function, inflammation, cholesterol distribution, and endothelial dysfunction. Elucidating such mechanisms of vascular protection for EPA may lead to new interventions for atherosclerosis, a disease that continues to expand worldwide.

Sherratt *et al*. 2023 (DOI: 10.1007 s11883-022-01075-x/ptr.6393)

Étude interventionnelle

ALA / Risque MCV chez patients DT2

Recommandations d'apports

Huile de lin (ALA) &
Huile de poisson (EPA+DHA)
=
Effets protecteurs comparables / MCV (chez patients DT2)

Effets propres à l'ALA et/ou EPA (conversion) ?

Raygan *et al.*, 2019 (DOI: 10.1002/ptr.6393)

A comparison between the effects of flaxseed oil and fish oil supplementation on cardiovascular health in type 2 diabetic patients with coronary heart disease: A randomized, double-blinded, placebo-controlled trial

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Funding information
Research Deputy of Kashan University of Medical Sciences (KAUMS)

This study compared the effects of flaxseed and fish oil supplementation on cardiovascular risk parameters in diabetic patients with coronary heart disease. Participants were randomly allocated into three intervention groups to receive either 1,000 mg of omega-3 fatty acids from fish oil or 1,000 mg of omega-3 fatty acids from flaxseed oil or placebo (n = 30 each group) twice a day for 12 weeks. A significant reduction in insulin levels (.04) was observed following flaxseed oil and fish oil supplementation compared with the placebo. In addition, a significant reduction in high-sensitivity C-reactive protein (.02) was seen after flaxseed oil supplementation compared with the placebo and a significant increase in total nitrite (.001) was seen after flaxseed oil and fish oil intake compared with placebo. Additionally, a significant increase in total antioxidant capacity (p < .001) after consuming flaxseed oil and fish oil compared with placebo and glutathione levels (.001) after consuming fish oil compared with flaxseed oil and placebo was observed. Overall, our study revealed the beneficial effects of flaxseed oil and fish oil supplementation on few metabolic profiles. This study suggests that the effect of flaxseed oil in reducing insulin and increasing total nitrite and total antioxidant capacity is similar to fish oil.

KEYWORDS
coronary heart disease, fish oil, flaxseed oil, metabolic status, type 2 diabetes mellitus

Poisson gras

Poisson maigre

Revue narrative

Conso. ALA
/ Risques MCV et Cognition

Recommandations d'apports

Rôles protecteurs de l'ALA
/ MCV
/ déclin cognitif

Effets propres à l'ALA
et/ou EPA (conversion) ?

Impact of α-Linolenic Acid, the Vegetable ω-3 Fatty Acid, on Cardiovascular Disease and Cognition

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ABSTRACT

Given the evidence of the health benefits of plant-based diets and long-chain n–3 (ω-3) fatty acids, there is keen interest in better understanding the role of α-linolenic acid (ALA), a plant-derived n–3 fatty acid, on cardiometabolic diseases and cognition. There is increasing evidence for ALA largely based on its major food sources (i.e., walnuts and flaxseed); however, this lags behind our understanding of long-chain n–3 fatty acids. Meta-analyses of observational studies have shown that increasing dietary ALA is associated with a 10% lower risk of total cardiovascular disease and a 20% reduced risk of fatal coronary heart disease. Three randomized controlled trials (RCTs) [AlphaOmega trial, Prevención con Dieta Mediterránea (PREDIMED) trial, and Lyon Diet Heart Study] all showed benefits of diets high in ALA on cardiovascular-related outcomes, but the AlphaOmega trial, designed to specifically evaluate ALA effects, only showed a trend for benefit. RCTs have shown that dietary ALA reduced total cholesterol, LDL cholesterol, triglycerides, and blood pressure, and epidemiologic studies and some trials also have shown an anti-inflammatory effect of ALA, which collectively account for, in part, the cardiovascular benefits of ALA. A meta-analysis reported a trend toward diabetes risk reduction with both dietary and biomarker ALA. For metabolic syndrome and obesity, the evidence for ALA benefits is inconclusive. The role of ALA in cognition is in the early stages but shows promising evidence of counteracting cognitive impairment. Much has been learned about the health benefits of ALA and with additional research we will be better positioned to make strong evidence-based dietary recommendations for the reduction of many chronic diseases. *Adv Nutr* 2022;13:1584–1602.

Statement of Significance: This narrative review updates the evidence from epidemiologic studies, randomized controlled trials, and meta-analyses about dietary α-linolenic acid and cardiovascular disease and its intermediate risk factors, as well as metabolic syndrome, type 2 diabetes, and cognition.

Sala-Vila *et al.*, 2022 (DOI: 10.1093 advances nmac016)

Méta-analyse études interventionnelles

Alimentation OMNIVORE

Complémentation OMEGA-3 / Risques MCV

Recommandations d'apports

Augmenter sa consommation de poisson, ALA, EPA, DHA
→ pas ou peu d'effet

⇩

HYPOTHESES personnelles :

- « Mangeur de poisson » bénéficient d'un environnement favorable / MCV ?
  → facteurs confondants ? (données socio-économiques, habitudes « santé »…)
- Conso. actuelles d'OMEGA 3 sont proches des quantités optimales ?
  → reco. ANSES pour EPA/DHA trop élevées ?… interroge la nécessité des apports en EPA et/ou DHA ?

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[Intervention Review]

Omega-3 fatty acids for the primary and secondary prevention of cardiovascular disease

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Editorial group: Cochrane Heart Group.
Publication status and date: New search for studies and content updated (conclusions changed), published in Issue 3, 2020.

Citation: Abdelhamid AS, Brown TJ, Brainard JS, Biswas P, Thorpe GC, Moore HJ, Deane KHO, Summerbell CD, Worthington HV, Song F, Hooper L. Omega-3 fatty acids for the primary and secondary prevention of cardiovascular disease. *Cochrane Database of Systematic Reviews* 2020, Issue 3. Art. No.: CD003177. DOI: 10.1002/14651858.CD003177.pub5.

Increasing EPA and DHA has little or no effect on deaths and cardiovascular events (high-certainty evidence) and probably makes little or no difference to cardiovascular death, stroke, or heart irregularities (moderate-certainty evidence). However, increasing EPA and DHA may slightly reduce risk of coronary death and coronary events (low-certainty evidence, coronary events are illnesses of arteries supplying the heart). To prevent one person having a coronary event, 167 people would need to increase their EPA and DHA, and 334 people would need to increase their EPA and DHA to prevent one person dying from coronary disease. EPA and DHA reduce triglycerides by about 15% but do not affect fatness or other lipids (high-certainty evidence).

Eating more ALA (for example, by increasing walnuts or enriched margarine) probably makes little or no difference to all-cause, cardiovascular or coronary deaths or coronary events but probably slightly reduces cardiovascular events and heart irregularities (moderate- or low-certainty evidence). To prevent one person having a coronary event, 500 people would need to increase their ALA, 91 people to prevent one person having arrhythmia.

There is little evidence of effects of eating fish. EPA and DHA reduce triglycerides. EPA, DHA and ALA may be slightly protective of some heart and circulatory diseases.

Fabien BADARIOTTI
1 mars 2024

Abdelhamid *et al.*, 2020 (DOI: 10.1002 14651858.CD003177.pub5.); Yu *et al.*, 2022 (DOI: 10.1097 MD.0000000000029556)

Angleterre (région: Norfolk)

Études observationnelles

Statuts sanguins OMEGA-3
(personnes non complémentées)

Santé des populations végétariennes

Les statuts ne reflètent pas du tout les niveaux d'apports

Le taux de conversion de l'ALA en ω-3 chaine longue est plus élevé (2x) chez les végétaliens

ALA/(EPA+DPA+DHA) = 20-25% vs 9-15%

Welch *et al.*, 2010 (DOI:10.3945/ajcn.2010.29457)

Dietary intake and status of n−3 polyunsaturated fat
a population of fish-eating and non-fish-eating meat
vegetarians, and vegans and the precursor-product
acid to long-chain n−3 polyunsaturated fatty acids:
EPIC-Norfolk cohort[1–3]

Alimentation

Statuts

Alimentation

Statuts

Ailsa A Welch, Subodha Shakya-Shrestha, Marleen AH Lentjes, Ni

The American Journal of Clinical Nutrition

ABSTRACT

Background: Intakes of n−3 (omega-3) polyunsaturated fatty acids (PUFAs) are important for health. Because fish is the major source of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), non-fish-eaters may have suboptimal n−3 PUFA status, although the importance of the conversion of plant-derived α-linolenic acid (ALA) to EPA and DHA is debated.

Objective: The objective was to determine intakes, food sources, and status of n−3 PUFAs according to dietary habit (fish-eaters and non-fish-eating meat-eaters, vegetarians, or vegans) and estimated conversion between dietary ALA and circulating long-chain n−3 PUFAs.

Design: This study included 14,422 men and women aged 39–78 y from the EPIC (European Prospective Investigation into Cancer and Nutrition)-Norfolk cohort with 7-d diary data and a substudy in 4902 individuals with plasma phospholipid fatty acid measures. Intakes and status of n−3 PUFAs were measured, and the precursor-product ratio of ALA to circulating n−3 PUFAs was calculated.

Results: Most of the dietary intake of EPA and DHA was supplied by fish; however, meat was the major source in meat-eaters, and

PUFAs are either plant-derive
(Al
acid
ma
hemp seeds and their oils; i
smaller amounts in soya oil a

Although conversion from A
is limited (9–11). Because fis
centrated sources of EPA and
fish or fish oils (eg, vegans an
meat-eaters) could be at risk
stat
und
of adequate n−3 PUFA statu
ALA was possible this could
and help preserve the fish sup
pan
proportion of the population d
eat oily fish (16–18). Fish int

TABLE 5
Characteristics, dietary intakes, and circulating plasma phospholipid n−3 polyunsaturated fatty acids (PUFAs) and plasma linoleic acid in the substudy in 4902 men and women aged 39–78 y with plasma measures of n−3 PUFAs who were not fish-oil-supplement consumers[1]

			Non-fish-eaters			
	All	Fish-eaters	Meat-eaters	Vegetarians	Vegans	P^2
Men						
n	2646	2257	359	25	5	
Age (y)	64.4 ± 7.7[3,4]	64.7 ± 7.6	63.3 ± 8.3	61.4 ± 9.9	54.4 ± 11.8	<0.001
Weight (kg)	80.6 ± 11.8[3]	80.5 ± 11.7	81.3 ± 12.5	79.6 ± 9.6	80.9 ± 10.5	0.43
Height (cm)	173.0 ± 6.6[3]	173.0 ± 6.6	173.2 ± 6.5	172.1 ± 6.4	172.8 ± 4.7	0.83
BMI (kg/m²)	26.9 ± 3.4	26.9 ± 3.4	27.0 ± 3.6	26.8 ± 2.8	27.1 ± 3.5	0.44
Current smokers [% (*n*)]	11.9 ± 315	11.0 ± 248	17.6 ± 63	8.0 ± 2	40.0 ± 2	<0.001
Diet (g/d)						
Total n−3 PUFAs	1.46 ± 0.57[3]	1.52 ± 0.57	1.13 ± 0.45	1.16 ± 0.55	0.87 ± 0.61	<0.001
α-Linolenic acid	1.19 ± 0.41[3]	1.21 ± 0.4	1.09 ± 0.45	1.15 ± 0.55	0.84 ± 0.61	<0.001
Eicosapentaenoic acid	0.12 ± 0.15[3]	0.13 ± 0.16	0.02 ± 0.02	0.01 ± 0.01	0.009 ± 0.008	<0.001
Docosahexaenoic acid	0.16 ± 0.21[3]	0.18 ± 0.22	0.02 ± 0.02	0.002 ± 0.007	0	<0.001
Linoleic acid	11.92 ± 4.87[3]	11.99 ± 4.63	11.43 ± 6	13.46 ± 6.5	8.53 ± 9.3	0.135
Plasma (μmol/L)						
α-Linolenic acid	11.1 ± 6.0[3]	10.9 ± 5.7	11.8 ± 7.0	13.6 ± 10.1	15.8 ± 9.7	<0.001
Eicosapentaenoic acid	56.1 ± 41.8[3]	57.5 ± 43.2	47.4 ± 30.3	55.9 ± 45.3	65.1 ± 45.5	0.001
Docosapentaenoic acid	67.7 ± 30.1[3]	67.3 ± 29.4	70.0 ± 33.4	77.5 ± 38.8	67.2 ± 26.8	0.038
Docosahexaenoic acid	236.2 ± 105.5[3]	239.7 ± 106.2	215.6 ± 96.4	222.2 ± 138.4	195.0 ± 58.8	<0.001
Total long-chain n−3 PUFAs	360.0 ± 163.3[3]	364.5 ± 164.8	333.0 ± 147.7	355.5 ± 211.1	327.4 ± 123.6	0.002
Linoleic acid	1171.0 ± 331.4	1164.1 ± 329.5	1207.9 ± 333.3	1238.2 ± 421.6	1337.7 ± 414.1	<0.001
Women						
n	2256	1891	309	51	5	
Age (y)	62.3 ± 8.8	62.4 ± 8.7	61.8 ± 9.3	60.1 ± 9.2	48.4 ± 5.0	0.002
Weight (kg)	68.8 ± 11.9	68.8 ± 11.8	69.1 ± 12.7	66.1 ± 11.5	69.4 ± 9.5	0.53
Height (cm)	160.4 ± 6.2	160.4 ± 6.2	160.1 ± 6.1	160.7 ± 6.6	164.3 ± 6.3	0.91
BMI (kg/m²)	26.8 ± 4.4	26.7 ± 4.4	27.0 ± 4.7	256 ± 4.1	25.9 ± 4.7	0.70
Current smokers [% (*n*)]	11.7 ± 263	11.5 ± 217	12.3 ± 38	15.7 ± 8	0 ± 0	0.65
Diet (g/d)						
Total n−3 PUFAs	1.18 ± 0.46	1.24 ± 0.46	0.89 ± 0.34	0.87 ± 0.39	0.72 ± 0.33	<0.001
α-Linolenic acid	0.97 ± 0.33	0.99 ± 0.32	0.86 ± 0.33	0.86 ± 0.39	0.71 ± 0.33	<0.001
Eicosapentaenoic acid	0.09 ± 0.12	0.1 ± 0.13	0.02 ± 0.01	0.01 ± 0.01	0.002 ± 0.004	<0.001
Docosahexaenoic acid	0.13 ± 0.17	0.15 ± 0.18	0.01 ± 0.01	0.002 ± 0.007	0 ± 0	<0.001
Linoleic acid	9.18 ± 3.86	9.33 ± 3.73	8.25 ± 3.94	9.02 ± 5.81	10.89 ± 10.86	<0.001
Plasma (μmol/L)						
α-Linolenic acid	12.5 ± 6.3	12.4 ± 6.1	13.1 ± 7.3	12.3 ± 4.8	13.71 ± 8.10	0.22
Eicosapentaenoic acid	63.4 ± 43.0	64.7 ± 43.4	57.1 ± 38.4	55.1 ± 52.5	50.0 ± 29.4	0.001
Docosapentaenoic acid	72.3 ± 30.4	71.8 ± 29.6	74.7 ± 34.2	75.0 ± 32.2	90.6 ± 54.0	0.056
Docosahexaenoic acid	266.0 ± 113.8	271.2 ± 113.1	241.3 ± 109.6	223.5 ± 137.8	286.4 ± 211.7	<0.001
Total long-chain n−3 PUFAs	401.7 ± 170.2	407.7 ± 169.3	373.1 ± 166.2	353.5 ± 191.5	426.8 ± 284.0	<0.001
Linoleic acid	1244.0 ± 334.3	1236.9 ± 328.4	1271.2 ± 373.9	1325.9 ± 278.6	1406 ± 162.1	<0.001

TABLE 6
The precursor-product ratio of circulating eicosapentaenoic acid, docosapentaenoic acid, and docosahexaenoic acid to dietary α-linolenic acid (converted to μmol/d) in different dietary-habit groups of 4902 men and women aged 39–78 y[1]

		Fish-eaters			Non-fish-eaters								
					Meat-eaters			Vegetarians			Vegans		
	n	Mean ± SE	95% CI	*n*	Mean ± SE	95% CI	*n*	Mean ± SE	95% CI	*n*	Mean ± SE	95% CI	P^2
Men	2257	—	—	359	—	—	25	—	—	5	—	—	—
Unadjusted	—	0.093 ± 0.001	0.091, 0.096	—	0.101 ± 0.004	0.093, 0.108	—	0.108 ± 0.012	0.085, 0.132	—	0.199 ± 0.027	0.146, 0.252	<0.001
Adjusted[3]	—	0.093 ± 0.001	0.091, 0.096	—	0.101 ± 0.003	0.095, 0.107	—	0.111 ± 0.012	0.088, 0.135	—	0.206 ± 0.027	0.153, 0.258	<0.001
Adjusted[4]	—	0.092 ± 0.001	0.090, 0.095	—	0.108 ± 0.003	0.102, 0.114	—	0.105 ± 0.011	0.083, 0.128	—	0.193 ± 0.025	0.144, 0.248	<0.001
Women	1891	—	—	309	—	—	51	—	—	5	—	—	—
Unadjusted	—	0.129 ± 0.002	0.125, 0.133	—	0.142 ± 0.005	0.132, 0.153	—	0.141 ± 0.011	0.117, 0.164	—	0.230 ± 0.037	0.158, 0.303	0.002
Adjusted[3]	—	0.127 ± 0.002	0.123, 0.131	—	0.141 ± 0.005	0.132, 0.151	—	0.152 ± 0.011	0.130, 0.173	—	0.249 ± 0.037	0.177, 0.320	0.002
Adjusted[4]	—	0.128 ± 0.002	0.124, 0.131	—	0.152 ± 0.005	0.142, 0.161	—	0.136 ± 0.011	0.114, 0.159	—	0.235 ± 0.035	0.165, 0.304	<0.001

[1] The analysis was performed by using linear regression.
[2] *P* for difference between different dietary-habit groups by ANOVA.
[3] Model 1 adjusted for age, BMI, and smoking habit.
[4] Model 2 adjusted for age, BMI, smoking habit, circulating plasma linoleic acid, dietary eicosapentaenoic acid and docosahexaenoic acid, and meat.

Pologne

Études observationnelles

Statuts sanguins OMEGA-3
(femmes non complémentées)

Santé des populations végétariennes

Populations consommant

très peu

***ω*-3 EPA+DHA**

Omnivores: < 100 mg/j

Gogga *et al*. 2024 (DOI: 10.3390/nu16040516)

Table 3. Daily intake of selected FAs in study groups.

FA Intake [g]	Diet Type	Mean ± SD	n	*p*					
				Vgn vs. Vgt	Vgn vs. Psc	Vgn vs. Omv	Vgt vs. Psc	Vgt vs. Omv	Psc vs. Omv
SFAs	Vgn	15.09 ± 7.05	30	**0.049**	**0.010**	**0.030**	0.430	0.760	0.600
	Vgt	19.26 ± 5.01	24						
	Psc	21.02 ± 8.19	12						
	Omv	19.92 ± 6.67	25						
MUFAs	Vgn	15.09 ± 7.05	30	0.070	**0.030**	**0.001**	0.600	0.110	0.250
	Vgt	19.06 ± 5.01	24						
	Psc	22.85 ± 6.51	11						
	Omv	25.19 ± 6.49	27						
Total PUFAs	Vgn	15.58 ± 7.33	30	**0.040**	0.080	**0.050**	0.610	0.820	0.750
	Vgt	11.75 ± 2.81	23						
	Psc	12.66 ± 4.16	11						
	Omv	12.14 ± 3.99	27						
n-3 PUFAs	Vgn	1.53 ± 0.98	28	0.570	**0.030**	0.770	**0.010**	0.420	**0.040**
	Vgt	1.33 ± 0.77	23						
	Psc	2.35 ± 1.44	11						
	Omv	1.63 ± 1.26	26						
n-6 PUFAs	Vgn	8.91 ± 5.11	27	0.380	0.880	0.120	0.340	0.430	0.100
	Vgt	7.62 ± 4.09	24						
	Psc	9.13 ± 3.01	11						
	Omv	6.44 ± 4.89	26						
EPA + DHA *	Vgn	0.00 ± 0.00	30	0.500	0.120	**0.001**	1.000	0.400	1.000
	Vgt	0.02 ± 0.04	23						
	Psc	0.03 ± 0.43	12						
	Omv	0.10 ± 0.19	24						
ALA	Vgn	1.73 ± 1.12	29	0.910	**0.004**	0.650	**0.010**	0.710	**0.010**
	Vgt	1.78 ± 1.05	24						
	Psc	3.00 ± 2.45	12						
	Omv	1.93 ± 0.91	27						
LA	Vgn	11.13 ± 6.27	30	0.280	0.270	0.260	0.920	0.880	0.950
	Vgt	9.58 ± 2.20	22						
	Psc	9.45 ± 2.93	10						
	Omv	9.36 ± 3.20	27						
n-6/*n*-3	Vgn	6.53 ± 2.42	26	0.120	0.060	**0.030**	0.670	0.450	0.700
	Vgt	5.30 ± 2.19	20						
	Psc	4.97 ± 1.67	8						
	Omv	4.67 ± 2.02	26						

Data are shown as means ± standard deviation and were analyzed using one-way ANOVA and Duncan's test, except for EPA + DHA (*), for which presented values are medians ± interquartile ranges and were analyzed using Kruskal–Wallis's test due to incompliance of the data with a normal distribution. Statistically significant results are bolded. FA—fatty acid; SD—standard deviation, Vgn—vegan, Vgt—vegetarian, Psc—pescatarian, Omv—omnivore, SFAs—saturated fatty acids, MUFAs—monounsaturated fatty acids, *n*-3 PUFAs—*n*-3 polyunsaturated fatty acids, *n*-6 PUFAs—*n*-6 polyunsaturated fatty acids, EPA—eicosapentaenoic acid, DHA—docosahexaenoic acid, ALA—alpha-linolenic acid, and LA—linoleic acid.

Pologne

Études observationnelles

Statuts sanguins OMEGA-3
(femmes)

Santé des populations végétariennes

Statuts plus faibles en EPA et DHA chez végétariens

Pas d'état inflammatoire, bien au contraire
(cf. CRP)

Table 2. Serum levels of selected FAs in different diet groups.

FAs Levels [%]	Diet Type	Mean ± SD	n	*p* Vgn vs. Vgt	Vgn vs. Psc	Vgn vs. Omv	Vgt vs. Psc	Vgt vs. Omv	Psc vs. Omv
n-3 PUFAs	Vgn	0.87 ± 0.37	30	0.310	**<0.001**	**<0.001**	**0.010**	**0.010**	1.000
	Vgt	1.00 ± 0.48	28						
	Psc	1.27 ± 0.41	12						
	Omv	1.35 ± 0.41	29						
n-6 PUFAs	Vgn	34.61 ± 4.60	30	**0.040**	0.560	0.200	0.110	0.360	0.430
	Vgt	31.59 ± 4.70	28						
	Psc	33.85 ± 3.59	13						
	Omv	32.80 ± 4.11	31						
EPA	Vgn	0.21 ± 0.10	30	0.660	**0.020**	**<0.001**	**0.040**		
	Vgt	0.23 ± 0.11	27						
	Psc	0.30 ± 0.10	11						
	Omv	0.34 ± 0.12	30						
DHA	Vgn	0.22 ± 0.17	30	**0.050**	**<0.001**	**<0.001**			
	Vgt	0.48 ± 0.27	28						
	Psc	0.74 ± 0.36	13						
	Omv	0.63 ± 0.23	29						
LA	Vgn	31.12 ± 3.93	30	**0.020**	0.770	**0.030**			
	Vgt	27.90 ± 0.39	28						
	Psc	30.73 ± 1.41	9						
	Omv	28.05 ± 3.99	31						
ARA	Vgn	2.54 ± 0.99	30	0.740	0.130	**<0.001**	0.210	**<0.001**	0.056
	Vgt	2.62 ± 0.98	28						
	Psc	3.04 ± 1.09	9						
	Omv	3.66 ± 0.90	31						
ALA	Vgn	0.15 ± 0.06	29	0.110	0.860	0.280	0.120	**0.010**	0.240
	Vgt	0.11 ± 0.06	27						
	Psc	0.14 ± 0.05	13						
	Omv	0.17 ± 0.08	30						

Data are shown as means ± standard deviation and analyzed using one-way ANOVA and Duncan's test. Statistically significant results are bolded. FAs—fatty acids; SD—standard deviation; Vgn—vegan, Vgt—vegetarian, Psc—pescatarian, Omv—omnivore, ECSFAs—even-chained fatty acids, OCFAs—odd-chain fatty acids, BCFAs—branched-chain saturated fatty acids; Iso-BCFAs—iso-branched-chain saturated fatty acids; Anteiso-BCFAs—anteiso-branched-chain saturated fatty acids; SFAs—saturated fatty acids, MUFAs—monounsaturated fatty acids, *n*-3 PUFAs—*n*-3 polyunsaturated fatty acids, *n*-6 PUFAs—*n*-6 polyunsaturated fatty acids, EPA—eicosapentaenoic acid, DHA—docosahexaenoic acid, ALA—alpha-linolenic acid, LA—linoleic acid, and ARA—arachidonic acid.

Consommations

EPA + DHA *	Vgn	0.00 ± 0.00	30
	Vgt	0.02 ± 0.04	23
	Psc	0.03 ± 0.43	12
	Omv	0.10 ± 0.19	24
ALA	Vgn	1.73 ± 1.12	29
	Vgt	1.78 ± 1.05	24
	Psc	3.00 ± 2.45	12
	Omv	1.93 ± 0.91	27

The median serum CRP levels were 0.01 ± 0.00 mg/dL in vegans, vegetarians, and pescatarians, and 0.01 ± 0.29 mg/dL in omnivores. We found that vegans had significantly lower levels of this protein than omnivores ($H_{3,79} = 28.87$; $p = 0.04$), while there were no differences between omnivores and pescatarians ($p = 0.26$). The difference in serum CRP levels between omnivores and vegetarians was close to reaching significance ($p = 0.06$).

Gogga *et al*. 2024 (DOI: 10.3390/nu16040516)

Fabien BADARIOTTI
1 mars 2024

Monde

Revue

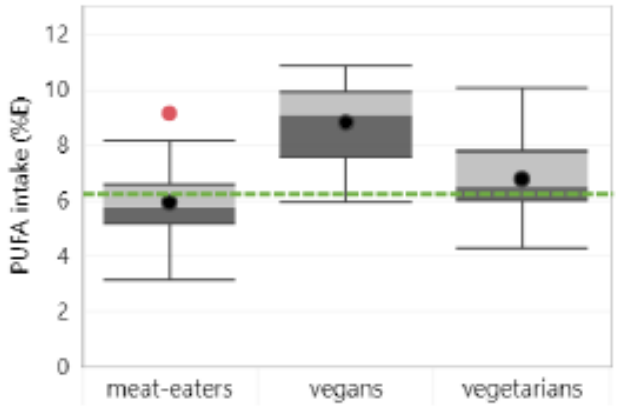
Statuts sanguins OMEGA-3

Santé des populations végétariennes

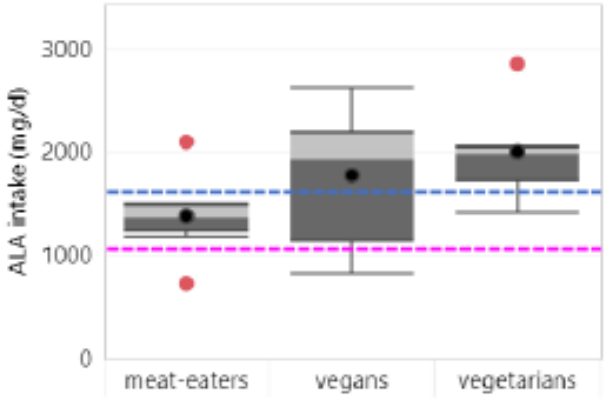
Statuts sanguins

ALA :
Vegan & Végétariens > Omni

EPA + DHA :
Omni > Végétariens > Vegan



(c)



(d)

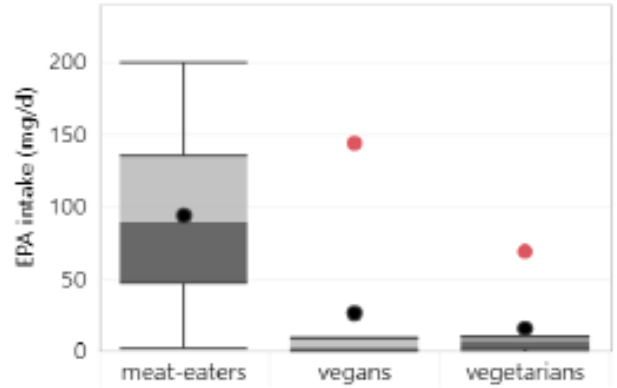


Figure 2. Box
tile range (IQ
(a) protein—d
(b) fiber—blu
(c) polyunsatu
tribution rang
for men and w

Twenty-two studies reported on fatty acid status. Because of differences in methodologies used to measure fatty acid status, it was not possible to calculate overall means of fatty acid status but we describe the findings of individual studies that compared fatty acid status of dietary patterns. Six studies compared total PUFA status between diet groups. Most studies showed significantly higher PUFA status in vegetarians (3/3 studies) and vegans (3/5 studies) compared to meat-eaters. For ALA status, as reported in 11 studies, there was a significantly higher status in vegans (4/9 studies) and vegetarians (3/8 studies) compared to meat-eaters. Thirteen studies reported on EPA and/or DHA status, most of which reported lower EPA and DHA status in vegetarians (5/7 and 7/7 studies) and vegans (7/8 and 8/9 studies) compared to meat-eaters. Vegans also mostly had lower EPA and DHA status than vegetarians (5/6 and 5/6 studies).

Neufingerl et Eilender, 2022 (DOI: 10.3390/nu14010029)

Royaume-Uni

Études observationnelles

Statut sanguin DHA

Santé des populations végétariennes

Consommations

ALA (vegan / Omni):
♂ → x 2 ; ♀ → idem

AL (vegan / Omni):
♂ → x 2-3 ; ♀ → x 2

EPA + DHA : absence

Table 1
Intakes (g/d) of polyunsaturated fatty acids in 20 UK vegan and 20 age-matched omnivores based on 3-d duplicate diet analyses (data from Roshanai and Sanders [8]).

Fatty acids	Male		Female	
	Vegan	Omnivore	Vegan	Omnivore
Linoleic (18:2n-6; LA)	28.5 ± 5.32[a]	10.2 ± 1.63	21.9 ± 3.53[a]	9.0 ± 1.07
Arachidonic acid (20:4n-6; AA)	ND[a]	0.72 ± 0.31	ND[a]	0.08 ± 0.02
Linolenic acid (18:3n-3; ALA)	1.8 ± 0.37[b]	1.0 ± 0.017	1.2 ± 0.22	1.1 ± 0.15
Eicosapentaenoic acid (20:5n-3; EPA)	ND[a]	0.02 ± 0.05	ND[a]	0.09 ± 0.02
Docosapentaenoic acid (22:5n-3; DPA)	ND[a]	0.09 ± 0.03	ND[a]	0.10 ± 0.02
Docosahexaenoic acid (22:6n-3; DHA)	ND[a]	0.42 ± 0.23	ND[a]	0.04 ± 0.02

Mean ± SEM for 10 males and 10 females in each group.
ND not detected.

[a] $P < 0.01$, two-sample t-test compared with corresponding value for same gender.
[b] $P < 0.05$, two-sample t-test compared with corresponding value for same gender.

Table 2
Fatty acid intakes (g/d) in UK vegan men compared with omnivores who consumed both meat and fish in the UK estimated from 7-d weighed food records determined between 2000 and 2003.

	Vegans[a]	Omnivores[b]	Difference (95% CI)
Saturated fatty acids	6.5 ± 0.25	12.6 ± 0.31	−6.1 (−7.1 to −5.1)[c]
Monounsaturated fatty acids	11.1 ± 0.50	11.5 ± 0.20	0.4 (−1.3 to 0.5)
Linoleic acid (18:2n-6; LA)	24.8 ± 1.82	12.0 ± 0.44	12.8 (10.2 to 15.5)[c]
Linolenic acid (18:3n-3; ALA)	2.2 ± 0.16	1.3 ± 0.05	0.9 (0.6 to 1.2)[c]
Eicosapentaenoic acid (20:5n-3; EPA)	ND	0.2 ± 0.02	−0.2 (−0.16 to−0.23)[c]
Docosahexaenoic acid (22:6n-3; DHA)	ND	0.3 ± 0.03	−0.3(−0.23 to −0.37)[c]

Mean ± SEM.
ND not detected.

[a] $n = 57$ – previously unpublished data.
[b] $n = 138$ data from Ref. [9].
[c] $P < 0.001$, two-sample t-test.

Sanders, 2009 (DOI:10.1016/j.plefa.2009.05.013)

Fabien BADARIOTTI
1 mars 2024

Royaume-Uni

Études observationnelles

Statut sanguin DHA

Santé des populations végétariennes

Table 3
Proportions of docosahexaenoic acid (22:6n-3) in red blood cell (RBC), platelet, and plasma lipid fractions in vegans and vegetarians compared with omnivores (control)

Study	Ref.	Country	Gender	Lipid fraction	Vegan n		Vegetarian n		Control n	
Sanders et al. (1978)	15	UK	M+F	RBC lipids	18	1.9±0.23[a]		–	18	5.8±0.38
Melchert et al. (1987)	17	Germany	M	Plasma PC		–	40	0.98±0.15[a]	38	2.85±0.08
Melchert et al. (1987)	17	Germany	F	Plasma PC		–	62	1.37 0.15[a]	70	2.25±0.11
Miller et al. (1988)	13	UK	M	Plasma PL		–	18	2.69±0.24[a]	19	4.65±0.28
Phinney et al. (1990)	19	USA	M+F	Plasma PL		–	25	3.19±0.29	100	3.59±0.11
Sanders and Roshanai (1992)	14	UK	M+F	Platelet PL	20	0.8±0.07[a]		–	20	2.1±0.10
Reddy et al. (1994)	22	UK	F	Plasma PL		–	21	1.2±0.09[a]	22	2.26±0.19
Lee et al. (2000)	21	Hong Kong	M+F	Serum		–	60	1.7 ±0.32[c]	133	3.4±0.19
Rosell et al. (2005)	16	UK	M	Serum	232	0.7±0.05[a]	231	1.16±0.05[a]	196	1.69±0.05
Mann et al. (2006)	20	Australia	M	Plasma	18	2.0±0.09[b]	43	2.2±0.11[b]	60	3.3±0.10
Kornsteiner et al. (2008)	18	Austria	M+F	RBC PE	37	1.59±0.09[a]	25	2.35±0.15[c]	23	3.18±0.23

Mean±SEM.
PC, phosphatidyl cholilne; PE, phosphatidyl ethanolamine; PL, phospholipids.
[a] $P<0.001$ compared with control.
[b] $P<0.01$ compared with control.
[c] $P<0.05$ compared with control.

Statut sanguin DHA
Omni > Végétariens > Vegan

Poisson maigre

Répercussions sanitaires ?

Conso. d'ALA pourrait être suffisante pour couvrir les besoins physiologiques ?

In conclusion, the relatively lower intake of linoleic acid and the presence of preformed DHA in the diet of omnivores explain the relatively higher proportion of DHA in blood and tissue lipids compared with vegetarians. In the absence of convincing evidence for the deleterious effects resulting from the lack of DHA from the diet of vegetarians, it must be concluded that needs for omega-3 fatty acids can be met by dietary ALA. Further research is needed to ascertain whether the lower DHA level and its partial replacement by DPA-n-6 are of any pathophysiological consequence.

Sanders, 2009 (DOI:10.1016/j.plefa.2009.05.013)

Grande-Bretagne

Études observationnelles

Santé Cardiaque

Santé des populations végétariennes

Le risque de maladie cardiaque ischémique est plus faible chez les végétariens

Risk of hospitalization or death from ischemic heart disease among British vegetarians and nonvegetarians: results from the EPIC-Oxford coho

Frances

ABSTRA
Backgro ferences etarians
Objectiv etarian d
Design: Scotland tion into consume Incident tal recor measuren to IHD estimate
Results: cases (1 nonveget (95% CI: (95% CI [−3.3 (9 risk (HR which w not diffe IHD risk
Conclusi lower IH in non-H
Clin Nut

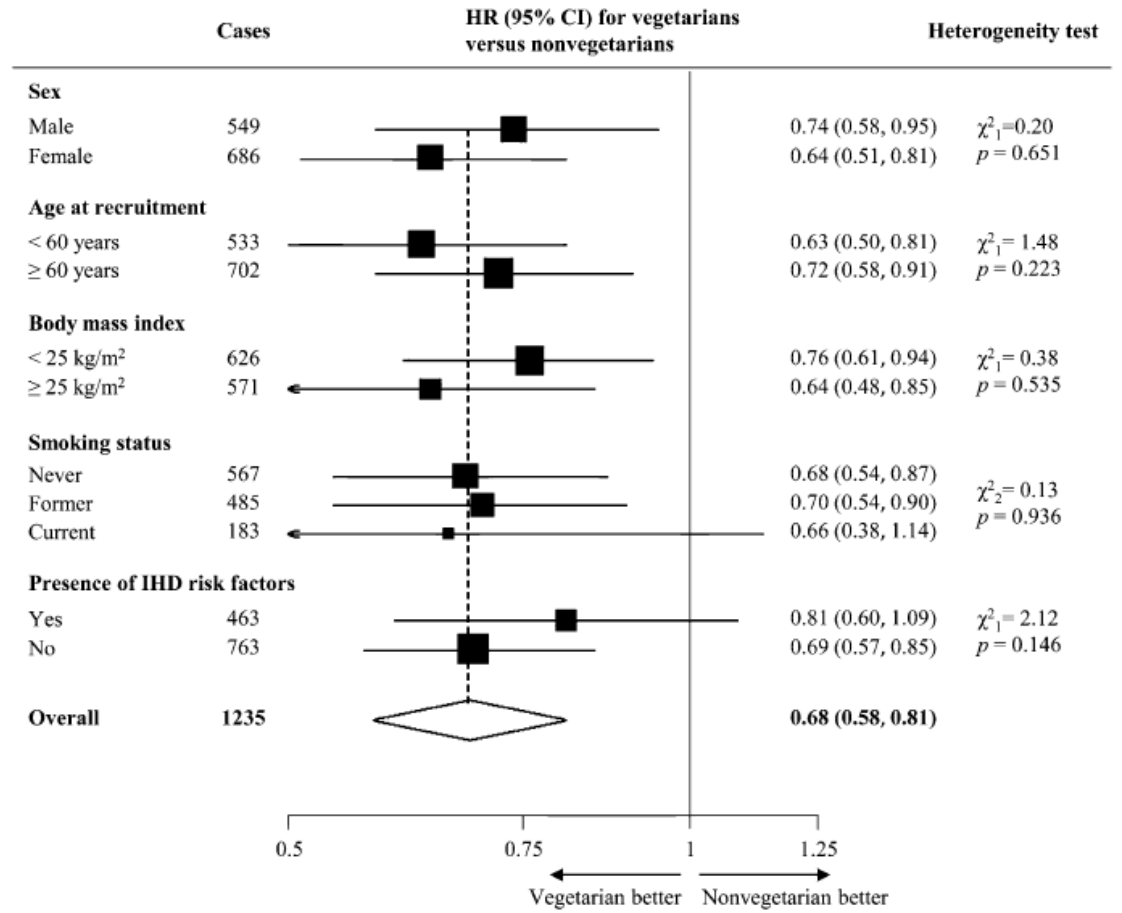


FIGURE 1. Risk of IHD in vegetarians and in nonvegetarians within certain subgroups in the EPIC-Oxford study. Presence of IHD risk factors includes at least one of the following: hypertension, hyperlipidemia, or diabetes. All analyses were stratified by sex, method of recruitment, and region of residence and were adjusted for age, smoking, alcohol, physical activity, educational level, and Townsend Deprivation Index and the use of oral contraceptives or hormone therapy for menopause in women. Tests of heterogeneity were performed by using data for all participants by adding a vegetarian status × sex, age, BMI, smoking, or IHD risk factor group interaction term to the model, as appropriate. EPIC, European Prospective Investigation into Cancer and Nutrition; IHD, ischemic heart disease.

INTROI

Comp (ie, thos

In summary, this analysis showed that British vegetarians have a lower risk of hospitalization for or death from IHD than do comparable nonvegetarians. A substantial proportion of the difference in risk was probably mediated through the effect of a vegetarian diet and lifestyle on non-HDL-cholesterol concentrations and systolic blood pressure, which supports the important role of diet in the prevention of IHD.

ssures (6–9), and a lower risk of diabetes than do non- s (10). As a result of these differences, the risk of de- ardiovascular disease in vegetarians would be expected r. Several studies have investigated the effect of a veg- t on the risk of fatal ischemic heart disease (IHD)[4]. The om a collaborative analysis of 5 prospective studies

[7] Abbreviations used: EPIC, European Prospective Investigation into Cancer and Nutrition; HES, Hospital Episode Statistics; ICD, International Classification of Diseases; IHD, ischemic heart disease; MI, myocardial infarction; MINAP, Myocardial Ischemia National Audit Project; NHS, National Health Service; SMR, Scottish Morbidity Records.

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First published online January 30, 2013; doi: 10.3945/ajcn.112.044073.

Crowe *et al*. 2013 (DOI: 10.3945/ajcn.112.044073)

Fabien BADARIOTTI
1 mars 2024

Etats-Unis

Études observationnelles

Santé Cardiovasculaire

Santé des populations végétariennes

Risque cardio-vasculaire plus faible chez les végétariens

Significatif uniquement chez les hommes

Orlich *et al.*,2013 (DOI:10.1001/jamainternmed.2013.6473)

Original Investigation

Vegetarian Dietary Patterns and Mortality in Adventist Health Study 2

Michael J. Orlich, MD; Pramil N. Singh, DrPH; Joan Sabaté, MD, DrPH; Karen Jaceldo-Siegl, DrPH; Jing Fan, MS; Synnove Knutsen, MD, PhD; W. Lawrence Beeson, DrPH; Gary E. Fraser, MBChB, PhD

Table 1. Comparison of Vegetarian With Nonvegetarian Dietary Patterns With Respect to All-Cause and Cause-Specific Mortality From a Cox Proportional Hazards Regression Model Among Participants in the Adventist Health Study 2, 2002-2009

	Deaths, Hazard Ratio (95% CI)				
Characteristic	All-Cause	Ischemic Heart Disease	Cardiovascular Disease	Cancer	Other
All (N = 73 308), No. of deaths[a,b]	2560	372	987	706	867
Vegetarian	0.88 (0.80-0.97)	0.81 (0.64-1.02)	0.87 (0.75-1.01)	0.92 (0.78-1.08)	0.85 (0.73-0.99)
Nonvegetarian	1 [Reference]	1 [Reference]	1 [Reference]	1 [Reference]	1 [Reference]
Men (n = 25 105), No. of deaths[a]	1031	169	390	273	368
Vegetarian	0.82 (0.72-0.94)	0.71 (0.51-1.00)	0.71 (0.57-0.90)	1.02 (0.78-1.32)	0.83 (0.66-1.04)
Nonvegetarian	1 [Reference]	1 [Reference]	1 [Reference]	1 [Reference]	1 [Reference]
Women (n = 48 203), No. of deaths[a,c]	1529	203	597	433	499
Vegetarian	0.93 (0.82-1.05)	0.88 (0.65-1.20)	0.99 (0.83-1.18)	0.87 (0.71-1.07)	0.88 (0.72-1.08)
Nonvegetarian	1 [Reference]	1 [Reference]	1 [Reference]	1 [Reference]	1 [Reference]

[a] Adjusted by age (ie, attained age as time variable), race (black, nonblack), smoking (current smoker, quit <1 year, quit 1-4 years, quit 5-9 years, quit 10-19 years, quit 20-29 years, quit ≥30 years, and never smoked), exercise (none, ≤20 min/wk, 21-60 min/wk, 61-150 min/wk, and ≥151 min/wk), personal income (≤$20 000/y, >$20 000-$50 000/y, >$50 000-$100 000/y, and >$100 000/y), educational level (up to high school graduate, trade school/some college/associate degree, bachelor degree, and graduate degree), marital status (married/common-law and single/widowed/divorced/separated), alcohol (nondrinker, rare drinker [<1.5 servings/mo], monthly drinker [1.5 to <4 servings/mo], weekly drinker [4 to <28 servings/mo], and daily drinker [≥28 servings/mo]), region (West, Northwest, Mountain, Midwest, East, and South), and sleep (≤4 h/night, 5-8 h/night, and ≥9 h/night).

[b] Also adjusted by sex (male and female), menopause (in women) (premenopausal [including perimenopausal], postmenopausal), and hormone therapy (in postmenopausal women) (not taking hormone therapy, taking hormone therapy).

[c] Also adjusted by menopause (premenopausal [including perimenopausal], postmenopausal) and hormone therapy (in postmenopausal women) (not taking hormone therapy, taking hormone therapy).

detected for cardiovascular mortality, noncardiovascular noncancer mortality, renal mortality, and endocrine mortality. Associations in men were larger and more often significant than were those in women.

CONCLUSIONS AND RELEVANCE Vegetarian diets are associated with lower all-cause mortality and with some reductions in cause-specific mortality. Results appeared to be more robust in males. These favorable associations should be considered carefully by those offering dietary guidance.

Etats-Unis

Études observationnelles

Santé Cardiovasculaire

Santé des populations végétariennes

Significatif notamment chez les vegan

Seulement chez les hommes

Pas de piste explicative solide

Original Investigation

Vegetarian Dietary Patterns and Mortality in Adventist Health Study 2

Michael J. Orlich, MD; Pramil N. Singh, DrPH; Joan Sabaté, MD, DrPH; Karen Jaceldo-Siegl, DrPH; Jing Fan, MS; Synnove Knutsen, MD, PhD; W. Lawrence Beeson, DrPH; Gary E. Fraser, MBChB, PhD

Table 4. Associations of Dietary Patterns With All-Cause and Cause-Specific Mortality From a Cox Proportional Hazards Regression Model Among Participants in the Adventist Health Study 2, 2002-2009

	Deaths, Hazard Ratio (95% CI)				
Characteristic	All-Cause	Ischemic Heart Disease	Cardiovascular Disease	Cancer	Other
All (N = 73 308), No. of deaths[a,b]	2560	372	987	706	867
Vegetarian					
Vegan	0.85 (0.73-1.01)	0.90 (0.60-1.33)	0.91 (0.71-1.16)	0.92 (0.68-1.24)	0.74 (0.56-0.99)
Lacto-ovo	0.91 (0.82-1.00)	0.82 (0.62-1.06)	0.90 (0.76-1.06)	0.90 (0.75-1.09)	0.91 (0.77-1.07)
Pesco	0.81 (0.69-0.94)	0.65 (0.43-0.97)	0.80 (0.62-1.03)	0.94 (0.72-1.22)	0.71 (0.54-0.94)
Semi	0.92 (0.75-1.13)	0.92 (0.57-1.51)	0.85 (0.63-1.16)	0.94 (0.66-1.35)	0.99 (0.72-1.36)
Nonvegetarian	1 [Reference]	1 [Reference]	1 [Reference]	1 [Reference]	1 [Reference]
Men (n = 25 105), No. of deaths[a]	1031	169	390	273	368
Vegetarian					
Vegan	0.72 (0.56-0.92)	0.45 (0.21-0.94)	0.58 (0.38-0.89)	0.81 (0.48-1.36)	0.81 (0.53-1.22)
Lacto-ovo	0.86 (0.74-1.01)	0.76 (0.52-1.12)	0.77 (0.59-0.99)	1.01 (0.75-1.37)	0.89 (0.69-1.15)
Pesco	0.73 (0.57-0.93)	0.77 (0.45-1.30)	0.66 (0.44-0.98)	1.10 (0.73-1.67)	0.60 (0.39-0.93)
Semi	0.93 (0.68-1.26)	0.73 (0.33-1.60)	0.75 (0.43-1.32)	1.15 (0.65-2.03)	1.03 (0.62-1.71)
Nonvegetarian	1 [Reference]	1 [Reference]	1 [Reference]	1 [Reference]	1 [Reference]
Women (n = 48 203), No. of deaths[a,c]	1529	203	597	433	499
Vegetarian					
Vegan	0.97 (0.78-1.20)	1.39 (0.87-2.24)	1.18 (0.88-1.60)	0.99 (0.69-1.44)	0.70 (0.47-1.05)
Lacto-ovo	0.94 (0.83-1.07)	0.85 (0.59-1.22)	0.99 (0.81-1.22)	0.85 (0.67-1.09)	0.93 (0.75-1.17)
Pesco	0.88 (0.72-1.07)	0.51 (0.26-0.99)	0.90 (0.66-1.23)	0.86 (0.61-1.21)	0.81 (0.58-1.15)
Semi	0.92 (0.70-1.22)	1.09 (0.60-1.98)	0.93 (0.64-1.34)	0.85 (0.56-1.30)	0.97 (0.64-1.47)
Nonvegetarian	1 [Reference]	1 [Reference]	1 [Reference]	1 [Reference]	1 [Reference]

[a] Adjusted by age (ie, attained age as time variable), race (black, nonblack), smoking (current smoker, quit <1 year, quit 1-4 years, quit 5-9 years, quit 10-19 years, quit 20-29 years, quit ≥30 years, and never smoked), exercise (none, ≤20 min/week, 21-60 min/week, 61-150 min/week, and ≥151 min/week), personal income (≤$20 000/y, >$20 000-$50 000/y, >$50 000-$100 000/y, and >$100 000/y), educational level (up to high school graduate, trade school/some college/associate degree, bachelor degree, and graduate degree), marital status (married/common-law and single/widowed/divorced/separated), alcohol (nondrinker, rare drinker [<1.5 servings/mo], monthly drinker [1.5 to <4 servings/mo], weekly drinker [4 to <28 servings/mo], and daily drinker [≥28 servings/mo]), region (West, Northwest, Mountain, Midwest, East, and South), and sleep (≤4 h/night, 5-8 h/night, and ≥9 h/night).

[b] Also adjusted by sex (male and female), menopause (in women) (premenopausal [including perimenopausal], postmenopausal), and hormone therapy (in postmenopausal women) (not taking hormone therapy, taking hormone therapy).

[c] Also adjusted by menopause (premenopausal [including perimenopausal], postmenopausal) and hormone therapy (postmenopausal women) (not taking hormone therapy, taking hormone therapy).

Poisson gras

Poisson maigre

Orlich *et al.*,2013 (DOI:10.1001/jamainternmed.2013.6473)

Recommandations d'apports - ONAV-

population végétarienne générale (enfant > 6 mois, adulte)

α-linolenic acid interconversion is sufficient as a source of longer chain ω-3 polyunsaturated fatty acids in humans: An opinion

Graham C. Burdge

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Abstract

α-linolenic acid (αLNA) conversion into the functionally important ω-3 polyunsaturated fatty acids (PUFA), eicosapentaenoic acid (EPA), and docosahexaenoic acid (DHA), has been regarded as inadequate for meeting nutritional requirements for these PUFA. This view is based on findings of small αLNA supplementation trials and stable isotope tracer studies that have been interpreted as indicating human capacity for EPA and, in particular, DHA synthesis is limited. The purpose of this review is to re-evaluate this interpretation. Markedly differing study designs, inconsistent findings and lack of trial replication preclude robust consensus regarding the nutritional adequacy of αLNA as a source of EPC and DHA. The conclusion that αLNA conversion in humans is constrained is inaccurate because it presupposes the existence of an unspecified, higher level of metabolic activity. Since capacity for EPA and DHA synthesis is the product of evolution it may be argued that the levels of EPA and DHA it maintains are nutritionally appropriate. Dietary and supra-dietary EPA plus DHA intakes confer health benefits. Paradoxically, such health benefits are also found amongst vegetarians who do not consume EPA and DHA, and for whom αLNA conversion is the primary source of ω-3 PUFA. Since there are no reported adverse effects on health or cognitive development of diets that exclude EPA and DHA, their synthesis from αLNA appears to be nutritionally adequate. This is consistent with the dietary essentiality of αLNA and has implications for developing sustainable nutritional recommendations for ω-3 PUFA.

1. **Pour les enfa**

L'examen des
recommander
végétariennes
d'affirmer qu'il
partiellement, v

Nous conseillons cependant d'avoir une attention particulière sur l'apport en ALA qu'il convient de favoriser à travers la consommation régulière d'huile de colza, de cameline, de lin, de graines de lin moulues et autres bonnes sources alimentaires d'oméga 3.

Burdge, 2022 (DOI: 10.1002/lipd.12355)

Asie du Sud

Statuts dans le sang et l'artère du cordon

Personnes végétariennes enceintes et/ou allaitantes

ω-6 à longue chaine (DPA) remplace partiellement son équivalent ω-3 à longue chaine (DHA)

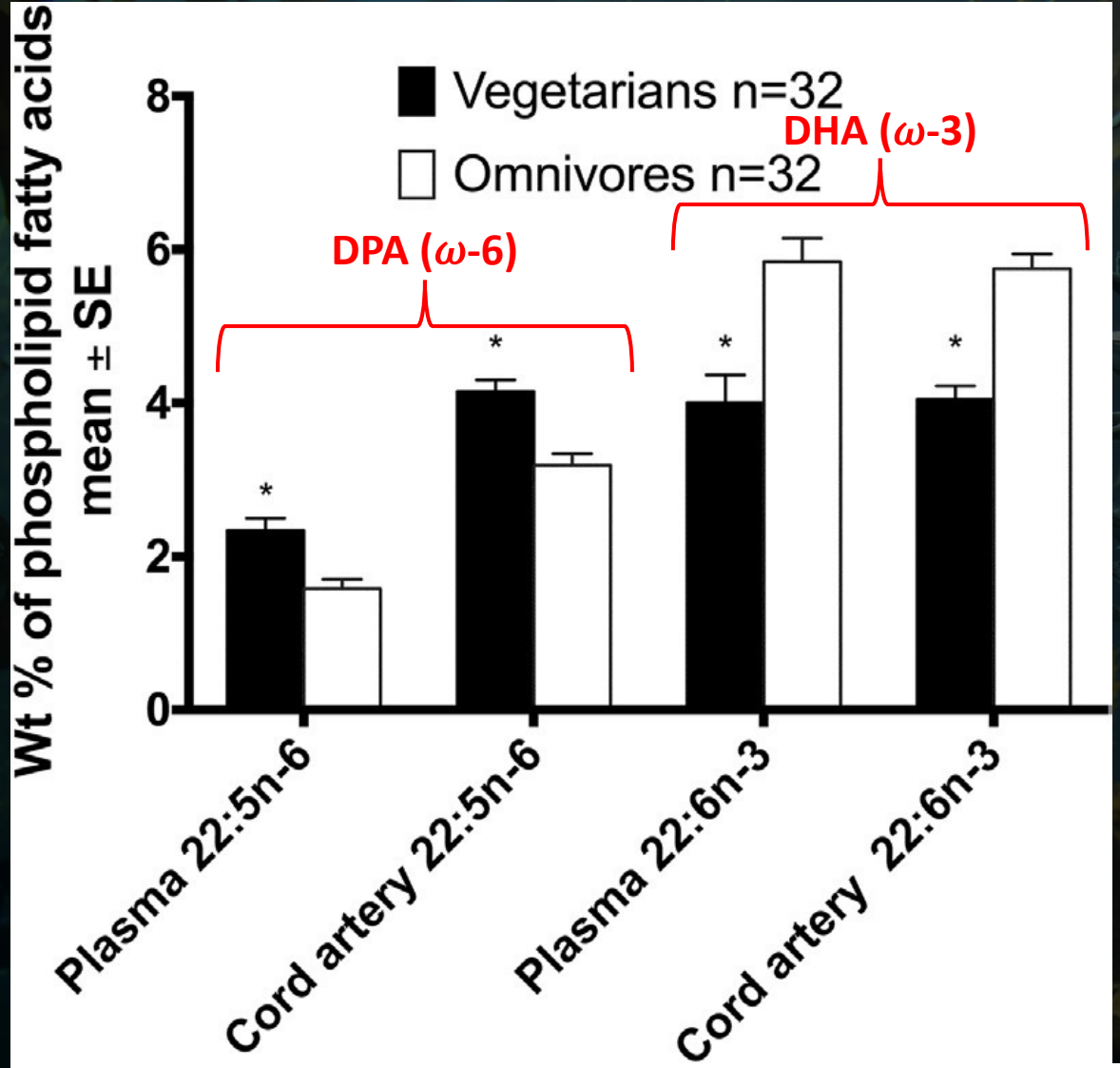

FIGURE 37.3 Differences in the proportions of docosahexaenoic acid (22:6n-3) and omega-6 docosapentaenoic acid (22:5n-6) in cord blood and cord artery phospholipids obtained at delivery from vegetarians compared with omnivores. *Asterisk* denotes $P<.01$ for comparisons between groups using a *t*-test. *Data from Reddy, S., Sanders, T.A., Obeid, O., 1994. The influence of maternal vegetarian diet on essential fatty acid status of the newborn. Eur. J. Clin. Nutr. 48, 358–368.*

Fabien BADARIOTTI
1 mars 2024

Sanders, 2017 (DOI:10.1016/B978-0-12-803968-7.00037-X)

Royaume-Uni

Teneurs dans le
LAIT MATERNEL

Personnes végétariennes enceintes et/ou allaitantes

Teneur significativement plus faible en DHA & plus forte en ALA et AL

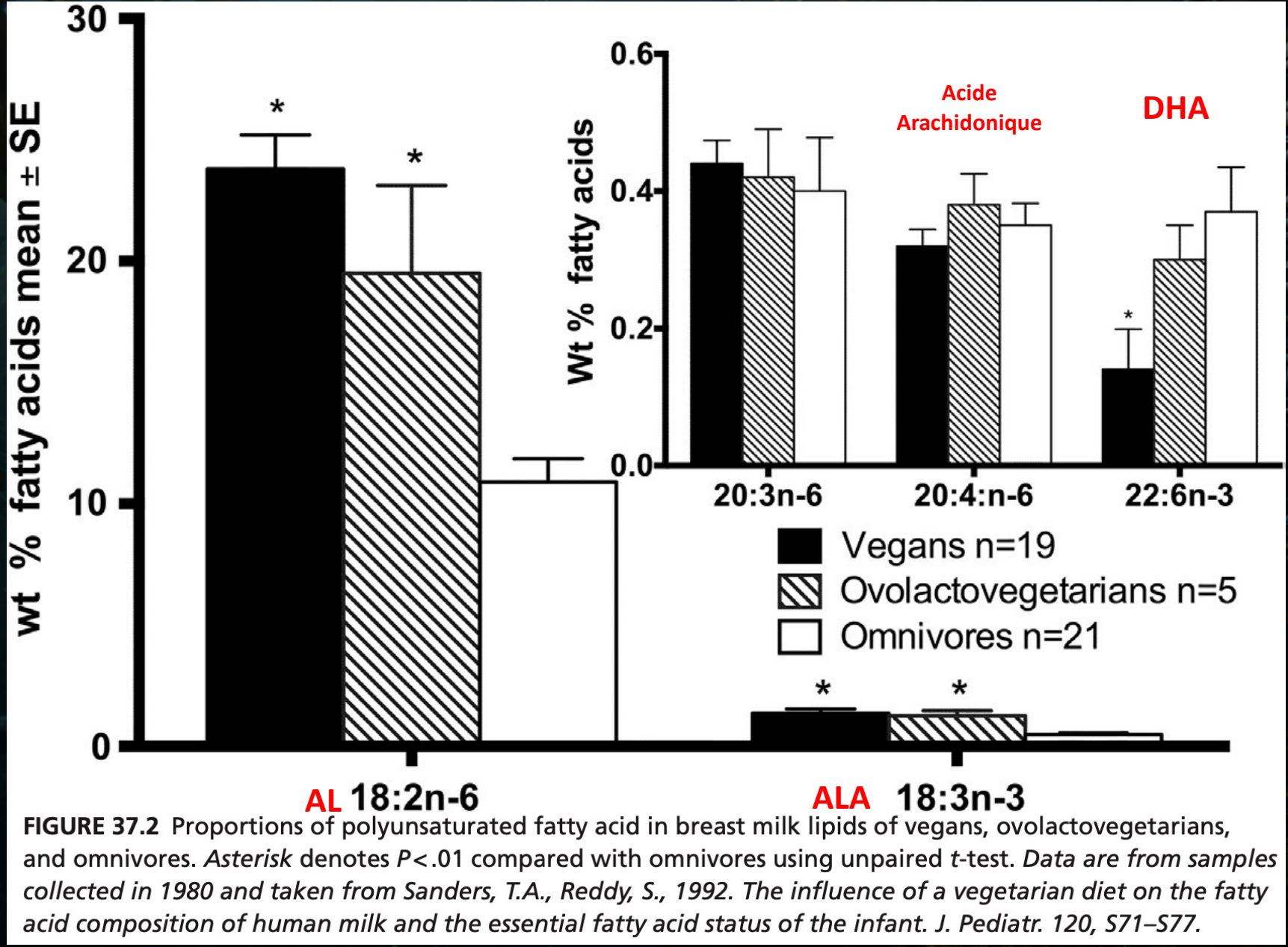

FIGURE 37.2 Proportions of polyunsaturated fatty acid in breast milk lipids of vegans, ovolactovegetarians, and omnivores. *Asterisk* denotes $P < .01$ compared with omnivores using unpaired *t*-test. *Data are from samples collected in 1980 and taken from Sanders, T.A., Reddy, S., 1992. The influence of a vegetarian diet on the fatty acid composition of human milk and the essential fatty acid status of the infant. J. Pediatr. 120, S71–S77.*

Conséquences sur la santé du bébé ?

Sanders, 2017 (DOI:10.1016/B978-0-12-803968-7.00037-X)

Fabien BADARIOTTI
1 mars 2024

Espagne

Apports alimentaires
(VS femme omnivore)

Personnes végétariennes enceintes et/ou allaitantes

Table 5. Diet survey: five-day dietary record. Daily nutrients intake of the omnivore human milk donors with full-term infants (Donors) and vegetarian/vegan lactating mothers (Veg), compared with recommended daily intakes.

n = 9 n = 11

		Donors (n = 92)	Veg (n = 20)	p Value	Recommendations [a]	
					EFSA (PRI/AI *)	IOM (RDA/AI *)
	Myristic acid C14:0 (g)	2.86 (0.14)	1.28 (0.26)	<0.001		
	Palmitic acid C16:0 (g)	16.52 (0.50)	8.40 (0.67)	<0.001		
	Palmitoleic acid C16:1 n7 (g)	1.55 (0.06)	0.42 (0.06)	<0.001		
	Stearic acid C18:0 (g)	7.07 (0.23)	3.53 (0.33)	<0.001		
	Oleic acid C18:1n9c (g)	40.58 (1.16)	36.40 (2.35)	0.106		
AL	Linoleic acid C18:2n6c (g)	13.45 (0.51)	18.25 (1.28)	<0.001		
ALA	Linolenic acid C18:3n3 (g)	1.58 (0.07)	2.24 (0.30)	0.016		
EPA	Eicosapentaenoic acid C20:5n3 (g)	0.15 (0.02)	0.04 (0.02)	<0.001		
DPA ω-3	Docosapentaenoic acid C22:5n3 (g)	0.08 (0.03)	0.00 (0.00)	<0.001		
DHA	Docosahexaenoic acid C22:6n3 (g)	0.38 (0.03)	0.11 (0.03)	<0.001	+0.10–0.20 *[d]	
	EPA + DHA (g)	0.53 (0.04)	0.14 (0.04)	<0.001	0.25 *	
	Folate food + folic acid (B_9) (µg)	473.22 (20.87)	668.15 (46.65)	<0.001	500 [f]	500 [g]
	Cobalamin (B_{12}) (µg)	6.92 (0.28)	258.40 (53.44)	0.096	5 *	2.8

+ 75 % / France (INCA3 : 0,30 g) ←

- apports plus faibles en EPA, DPA (ω-3) et DHA

- apports plus élevés en AL et ALA

[a] Recommended daily intake. Adequate intake is presented with an asterisk (*) and PRI/RDA (i.e., Population Reference Intake for the EFSA values, and Recommended Dietary Allowance for the IOM values) in ordinary type.

[d] In addition to the combined intakes of EPA and DHA of 0.25 g/day.

Ureta-Velasco *et al*. 2023 (DOI: 10.3390 nu15081855)

Espagne

Apports alimentaires
(VS femme omnivore)

Personnes végétariennes enceintes et/ou allaitantes

Même niveau de « déséquilibre »
(cf. →)
/ apports recommandés

Table 6. Prevalence of inadequate intakes of specific nutrients in omnivore human milk donors with full-term infants (Donors) and in vegetarian/vegan lactating mothers (Veg) [1].

Nutrient	H-AR * [50]	Donors (n = 92), n (%)	Veg (n = 20), n (%)	p Value
Thiamine (B_1), mg	1.2	5 (5.4%)	0 (0.0%)	0.286
Riboflavin (B_2), mg	1.7	13 (14.1%)	9 (45%)	0.002
Niacin (B_3), mg	13	0 (0.0%)	0 (0.0%)	-
Pantothenic acid (B_5), mg	5.6	22 (23.9%)	6 (30%)	0.568
Pyridoxine (B_6), mg	1.4	1 (1.1%)	0 (0.0%)	0.640
Biotin (B_7), µg	36	34 (37.0%)	7 (35.0%)	0.869
Folate food + folic acid (B_9), µg	380 (DFE)	36 (39.1%)	0 (0.0%)	<0.001
Cobalamin (B_{12}), µg	2.4	0 (0.0%)	5 (25.0%)	<0.001
Vitamin C, mg	145	34 (37.0%)	4 (20.0%)	0.147
Vitamin A, µg RAE	1020	32 (34.8%)	6 (30.0%)	0.682
Vitamin D, µg	10	81 (88.04)	15 (75.00)	0.131
Vitamin E, mg	16	45 (48.9%)	5 (25.0%)	0.051
Iodine, µg	209	40 (43.5%)	8 (40.0%)	0.776
Calcium, mg	860 (19–30 y) 750 (31–50 y)	6 (6.5%)	9 (45.0%)	<0.001
Phosphorous, mg	580	0 (0.0%)	0 (0.0%)	-
Selenium, µg	59	1 (1.1%)	0 (0.0%)	0.640

[1] The number and percentage of women with inadequate intakes of each nutrient (below harmonized average requirements) are presented in each group. * The H-AR, the harmonized average requirement, was proposed by Allen et al., (2020) [50], after they selected values from the standards set by EFSA (for Europe) and the IOM (for the United States and Canada), giving priority to those published most recently. Abbreviations: DFE, dietary folate equivalents; RAE, retinol activity equivalents; and y, years.

Ureta-Velasco *et al*. 2023 (DOI: 10.3390 nu15081855)

Espagne

Statuts
sanguins
(VS femme omnivore)

Personnes végétariennes enceintes et/ou allaitantes

Statuts
EPA & DHA
plus faibles (÷ 2-3)

Table 9. Erythrocytes and plasma fatty acid composition (g/100 g of total fat) of the omnivore human milk donors with full-term infants (Donors) and vegetarian/vegan lactating mothers (Veg).

Fatty Acid (%)		Common Name	Donors (n = 92)	Veg (n = 20)	p Value
ERYTHROCYTES					
n-6 Polyunsaturated Fatty Acids (*n*-6 PUFAs)					
C18:2 (n6)	AL	Linoleic	8.12 (0.14)	9.24 (0.31)	0.002
C20:3 (n6)		Dihomo-γ-linolenic	0.99 (0.05)	1.57 (0.14)	<0.001
C20:4 (n6)		Arachidonic	24.30 (0.32)	24.91 (0.75)	0.130
n-3 Polyunsaturated Fatty Acids (*n*-3 PUFAs)					
C20:5 (n3)	EPA	Eicosapentaenoic	0.12 (0.02)	0.00 (0.00)	0.009
C22:5 (n3)		Docosapentaenoic	0.73 (0.03)	0.66 (0.04)	0.151
C22:6 (n3)	DHA	Docosahexaenoic	2.90 (0.12)	1.72 (0.32)	<0.001
PLASMA					
n-6 Polyunsaturated Fatty Acids (*n*-6 PUFAs)					
C18:2 (n6)	AL	Linoleic	39.20 (0.46)	41.28 (1.42)	0.390
C20:3 (n6)		Dihomo-γ-linolenic	1.11 (0.06)	1.43 (0.18)	0.057
C20:4 (n6)		Arachidonic (ARA)	11.30 (0.33)	10.56 (0.71)	0.533
n-3 Polyunsaturated Fatty Acids (*n*-3 PUFAs)					
C20:5 (n3)	EPA	Eicosapentaenoic (EPA)	0.24 (0.03)	0.08 (0.02)	0.012
C22:6 (n3)	DHA	Docosahexaenoic (DHA)	0.83 (0.06)	0.37 (0.07)	<0.001

Ureta-Velasco *et al*. 2023 (DOI: 10.3390 nu15081855)

Espagne

Teneurs dans le LAIT MATERNEL
(VS femme omnivore)

Personnes végétariennes enceintes et/ou allaitantes

Table 12. Human milk fatty acid methyl esters (FAMEs) composition (g/100 g of total fat) of the omnivore human milk donors with full-term infants (Donors) and vegetarian/vegan mothers (Veg).

Fatty Acid (%)	Common Name	Donors (n = 88)	Veg (n = 20)	p Value	Reference Values	
					European [88] [1]	World [89] [2]
		n-6 Polyunsaturated Fatty Acids (*n*-6 PUFAs)				
C18:2 (n6)	Linoleic (LA)	15.29 (0.39)	20.02 (1.15)	<0.001	14.00 ± 4.95	15.7 ± 7.15
C20:2 (n6)	Eicosadienoic	0.27 (0.01)	0.32 (0.02)	0.063	0.26 ± 0.07	0.37 ± 0.19
C20:3 (n6)	Dihomo-γ-linolenic	0.33 (0.01)	0.36 (0.04)	0.652	0.31 ± 0.09	0.37 ± 0.18
C20:4 (n6)	Arachidonic (AA)	0.55 (0.02)	0.46 (0.03)	0.012	0.44 ± 0.12	0.50 ± 0.25
		n-3 Polyunsaturated Fatty Acids (*n*-3 PUFAs)				
C18:3 (n3)	Linolenic (ALA)	0.52 (0.02)	0.61 (0.05)	0.044	0.94 ± 0.55	1.11 ± 1.05
C22:5 (n3)	Docosapentaenoic (DPA)	0.08 (0.01)	0.04 (0.01)	<0.001		
C22:6 (n3)	Docosahexaenoic (DHA)	0.33 (0.02)	0.15 (0.04)	<0.001	0.34 ± 0.35	0.37 ± 0.31

Variables are presented as means (standard error of the mean). [1] Data are presented as the mean ± standard deviation, from 223 lactating mothers at a lactation stage of 120 ± 5 days. [2] Data of the mature milk are presented as the mean ± standard deviation. Abbreviations: SCFAs, short-chain fatty acids; MCFAs, medium-chain fatty acids; LCFAs, long-chain fatty acids; and VLCFAs, very-long-chain fatty acids.

Teneurs significativement plus fortes en ALA, AL et AA & plus faibles en DHA et DPA (ω-3)

Conséquences sur la santé du bébé ?

Ureta-Velasco *et al*. 2023 (DOI: 10.3390 nu15081855)

Royaume-Uni

Développement neurocognitif de l'enfant

Personnes végétariennes enceintes et/ou allaitantes

Développement cognitif normal de l'enfant (nourrit par le lait maternel d'une végétarienne)

nutrients MDPI

Article

Vegetarian Diet during Pregnancy Is Not Associated with Poorer Cognitive Performance in Children at Age 6–7 Years

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Received: 3 October 2019; Accep

Abstract: Compared with o
nutrients required for ne
impact of vegetarianism dur
of this study was to investig

Table 2. Serum fatty acid concentrations in early and late pregnancy vegetarians and omnivores.

	Fatty Acid Concentration (μg/mL)					
	Early Pregnancy			Late Pregnancy		
	Vegetarians (n = 32)	Omnivores (n = 967)	*p*	Vegetarians (n = 59)	Omnivores (n = 1703)	*p*
14:0	4.8 (3.7, 7.5)	6.0 (4.6, 7.8)	0.07	3.2 (2.0, 4.9)	3.2 (2.2, 4.5)	0.40
16:0	489.5 (420.9, 594.0)	536.8 (458.9, 639.0)	0.06	462.5 (381.5, 569.4)	514.8 (399.6, 631.8)	0.04
16:1n-7	7.1 (5.6, 11.6)	9.3 (6.9, 12.8)	0.05	5.8 (3.3, 8.9)	5.2 (3.3, 8.4)	0.63
18:0	185.5 (161.1, 219.9)	204.6 (171.9, 238.2)	0.02	136.4 (97.5, 169.2)	143.9 (111.2, 178.8)	0.07
18:1n-9	158.9 (132.6, 194.1)	177.9 (148.9, 212.6)	0.08	148.9 (110.4, 192.1)	170.2 (130.3, 210.3)	0.01
18:1n-7	28.8 (24.5, 31.4)	29.9 (24.2, 35.8)	0.19	17.7 (12.7, 21.9)	20.3 (13.9, 26.0)	0.007
AL 18:2n-6	367.2 (317.2, 428.9)	368.5 (312.0, 435.5)	0.97	328.3 (248.2, 387.0)	333.6 (258.7, 408.3)	0.60
18:3n-6	1.4 (1.0, 2.4)	1.7 (1.3, 2.4)	0.07	0.0 (0.0, 0.0) †	0.0 (0.0, 0.0) †	0.19
18:3n-3	5.9 (4.0, 8.9)	6.1 (4.5, 8.1)	0.53	3.7 (2.7, 5.2)	4.0 (3.0, 5.6)	0.21
20:0	3.9 (2.5, 5.6)	4.2 (3.1, 5.6)	0.30	2.1 (0.0, 3.1)	2.3 (0.0, 3.2)	0.36
20:1n-9	3.7 (3.1, 5.0)	3.8 (3.0, 4.8)	0.91	1.4 (0.0, 2.3)	2.0 (0.0, 2.7)	0.007
20:2n-6	7.6 (5.3, 9.3)	8.0 (6.6, 9.8)	0.30	4.9 (3.7, 6.1)	5.1 (3.9, 6.5)	0.26
20:3n-6	58.2 (49.7, 79.2)	67.7 (53.7, 83.9)	0.13	52.4 (38.2, 64.8)	53.6 (39.6, 68.6)	0.26
20:4n-6	143.9 (134.8, 170.1)	165.8 (135.5, 200.8)	0.02	100.9 (72.9, 122.7)	108.4 (83.8, 139.0)	0.03
22:0	2.1 (0.9, 2.9)	1.8 (1.1, 2.7)	0.60	0.0 (0.0, 0.0) †	0.0 (0.0, 0.0) †	0.47
20:4n-3	3.1 (2.3, 5.5)	4.2 (2.9, 5.9)	0.04	0.0 (0.0, 2.3)	1.8 (0.0, 2.9)	0.02
EPA 20:5n-3	8.4 (6.1, 10.4)	14.0 (10.4, 18.8)	<0.001	3.5 (2.0, 4.6)	5.3 (3.7, 7.8)	<0.001
22:4n-6	7.4 (5.0, 9.2)	6.9 (5.2, 8.6)	0.51	3.4 (2.4, 4.4)	3.3 (2.4, 4.3)	0.74
22:5n-6	7.1 (6.0, 10.4)	6.2 (4.5, 8.3)	0.01	5.2 (3.9, 7.3)	4.3 (3.2, 6.0)	0.001
24:1n-9	4.2 (1.8, 5.3)	3.2 (2.1, 5.1)	0.63	0.0 (0.0, 0.0) †	0.0 (0.0, 0.0) †	0.18
22:5n-3	13.3 (10.6, 16.9)	15.2 (12.3, 19.6)	0.01	5.1 (3.6, 6.5)	6.6 (4.8, 8.8)	<0.001
DHA 22:6n-3	57.8 (50.2, 62.9)	82.6 (66.4, 102.7)	<0.001	38.0 (27.9, 48.9)	54.3 (41.2, 72.3)	<0.001

Values are median (IQR); *p* values were determined using the Mann-Whitney U Test. † Undetectable in more than 75% of samples.

5. Conclusions

In conclusion, the findings of this study suggest that consuming a vegetarian diet during pregnancy does not adversely affect the neurocognitive development of the children, provided maternal concentrations of nutrients that are required for neurological development are within usual ranges.

vegetarian diet during pregnancy was not associated with poorer neurocognitive development of the children in this study.

Crozier *et al.*, 2019; (DOI: 10.3390/nu11123029)

Recommandations d'apports - FAO -

- enfants
- personnes enceintes et/ou allaitantes

Fats and fatty acids in human nutrition
Report of an expert consultation

FAO FOOD AND NUTRITION PAPER 91

TABLE 2.2
Recommended dietary intakes for total fat and fatty acid intake: Infants (0-24 months) and Children (2-18 years)

Fat/FA	Age Group	Measure	Numeric Amount	Level of Evidence
n-3 PUFA				
ALA	0-6 mo	AI	0.2-0.3%E[b]	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[b]	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)[f]	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

(Explanations of the abbreviations are found in

[a] Simell *et al.*, 2009

[f] For infants 6-12 mo, the proposed fat intake a concern over increased obesity rates and the re infancy (WHO 2006).

[b] The amounts are expressed as %E in order to en referring to infants of brea is conversion assumes that h of breast milk to meet half

no specific data from long t ption is that children also be

CONCLUSIONS AND RECOMMENDATIONS FOR N-3 POLYUNSATURATED FATTY ACID INTAKE

The available evidence indicates that 0.5-0.6%E alpha-linolenic acid (ALA) per day corresponds to the prevention of deficiency symptoms. The total n-3 fatty acid intake can range between 0.5–2%E whereas the minimum dietary requirement of ALA (>0.5%E) for adults prevents deficiency symptoms. The higher value 2%E (ALA) plus n-3 long-chain polyunsaturated fatty acids (LCPUFA) eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) (AMDR 0.250 g–2.0 g) can be part of a healthy diet. Whilst ALA may have individual properties in its own right, there is evidence that the n-3 LCPUFA may contribute to the prevention of CHD and possibly other degenerative diseases of aging. For adult males and non-pregnant/non-lactating adult females 0.250 g/day of EPA plus DHA is recommended, with insufficient evidence to set a specific minimum intake of either EPA or DHA alone; both should be consumed. For adult pregnant and lactating females, the minimum intake for optimal adult health and fetal and infant development is 0.3 g/d EPA+DHA, of which at least 0.2 g/d should be DHA.

FAO, 2010 (ISBN:978-92-5-106733-8)

Likewise, the precise balance between the omega-3 PUFAs EPA and DHA for optimal health is unclear, although both seem to be important. DHA is vital for early life visual and neural development. The principal roles of the plant omega-3 PUFA ALA are in controlling conversion of LA to AA and in acting as a substrate for synthesis of EPA.

Djuricic et Calder, 2021 (DOI: 10.1146annurev-pharmtox-051921-090208)

Recommandations d'apports chez végétarien·nes - Saunders *et al.* -

Clinical focus

Omega-3 polyunsaturated fatty acids and vegetarian diets

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Vegetarians have a lower overall risk of common chronic diseases, possibly due to a lower saturated fat and cholesterol intake than non-vegetarians.[1] However, vegetarians (and those who eat minimal amounts of oily fish) may be at a disadvantage where intake of essential fatty acids (EFAs) is concerned, and this could potentially counteract some health benefits of the vegetarian diet. In this article, we review EFA intake and status of vegetarians and consider whether current intakes in this population are sufficient to achieve and maintain optimal health. We also explore the potential benefits of adding supplemental sources of docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA) derived from microalgae, and make practical suggestions for optimising EFA status in vegetarians.

Functional and biological aspects of EFAs

Fats in foods and the body contain saturated, monounsaturated and polyunsaturated fatty acids (PUFAs), the latter comprising omega-6 (n-6) and omega-3 (n-3) families. There are two EFAs: linoleic acid (LA), the parent of the n-6 fatty acid family; and α-linolenic acid (ALA), the parent of the n-3 fatty acid family. EFAs cannot be synthesised by the body and therefore must be supplied by the diet. LA and ALA can be converted by enzymes into long-chain PUFAs.[2] LA is a precursor of arachidonic acid (AA), and ALA is a precursor of EPA, DHA and docosapentaenoic acid (DPA), with stearidonic acid (SDA) an intermediate in the pathway. The long-chain PUFAs are not technically "essential" because they can be produced endogenously, but they can become essential if insufficient precursor is available for their production.

AA and EPA act as substrates for eicosanoids (prostaglandins, thromboxanes, leukotrienes and prostacyclins) that regulate inflammation, platelet aggregation and blood clotting, blood vessel contraction and dilation, muscle contraction and relaxation, immune responses and regulation of hormone secretion. Eicosanoids from n-3 PUFA (3-series) have opposing effects to those from n-6 PUFA (2-series). Eicosanoids from AA are very potent and overproduction is associated with increased risk of disease (heart disease, cancer, diabetes, osteoporosis, and immune and inflammatory disorders).[2-4] Eicosanoids from EPA are less potent and have anti-inflammatory properties that assist in preventing coronary heart disease, hypertension, autoimmune diseases, arthritis and several cancers.[2-4] Extremely powerful mediators called protectins (derived from DHA) and resolvins (derived from DHA and EPA) help protect against and resolve inflammation.[5] Long-chain n-3 PUFAs also favourably affect cell membranes, enhancing intracellular signalling processes and gene expression. DHA is particularly abundant in the cerebral cortex, retina, testes and semen.[2,6,7]

LA and ALA share the same pathway and enzymes for conversion to long-chain PUFAs. An excess of LA, common in Western diets, can suppress conversion of ALA to EPA and DHA and increase production of AA. This in

Summary

- While intakes of the omega-3 fatty acid α-linolenic acid (ALA) are similar in vegetarians and non-vegetarians, intakes of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) are low in vegetarians and virtually absent in vegans.
- Plasma, blood and tissue levels of EPA and DHA are lower in vegetarians than in non-vegetarians, although the clinical significance of this is unknown. Vegetarians do not exhibit clinical signs of DHA deficiency, but further research is required to ascertain whether levels observed in vegetarians are sufficient to support optimal health.
- ALA is endogenously converted to EPA and DHA, but the process is slow and inefficient and is affected by genetics, sex, age and dietary composition. Vegetarians can take practical steps to optimise conversion of ALA to EPA and DHA, including reducing intake of linoleic acid.
- There are no official separate recommendations for intake of fatty acids by vegetarians. However, we suggest that vegetarians double the current adequate intake of ALA if no direct sources of EPA and DHA are consumed.
- Vegetarians with increased needs or reduced conversion ability may receive some advantage from DHA and EPA supplements derived from microalgae. A supplement of 200–300 mg/day of DHA and EPA is suggested for those with increased needs, such as pregnant and lactating women, and those with reduced conversion ability, such as older people or those who have chronic disease (eg, diabetes).

Abbreviations

AA	arachidonic acid
AI	adequate intake
ALA	α-linolenic acid
DHA	docosahexaenoic acid
DPA	docosapentaenoic acid
EFA	essential fatty acid
EPA	eicosapentaenoic acid
LA	linoleic acid
n-3	omega-3
n-6	omega-6
PUFA	polyunsaturated fatty acid
SDA	stearidonic acid

- **Conso. EPA/DHA**
apports très faibles à nuls
- **Statuts EPA/DHA**
plus faibles MAIS absence de répercussions cliniques
- **Conversion ALA → EPA → DHA**
↘ la conso. d'AL
- **Conso. ALA**
X 2 (/reco pour OMNI)
- **Personnes à besoin spé.**
Complément EPA+DHA (200-300 mg/j)

Saunders *et al.*, 2012 (DOI:10.5694/mjao11.11507)

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Recommandations d'apports - ONAV-

- enfants < 6 mois
- personnes enceintes et/ou allaitantes

Quid des personnes âgées ?

2. Pour les enfants jusqu'à l'âge de 6 mois, les femmes enceintes et allaitantes, et les adultes consommant peu d'ALA

Par principe de précaution, il peut être conseillé de se complémenter en DHA à partir d'extraits de microalgues [11]. Les recommandations de l'Anses étant de 250 mg/jour, il ne semble pas utile de recourir à une dose plus importante [12].

Il faut noter que le coût d'une telle supplémentation en DHA n'est pas négligeable (à la différence de celle en vitamine B_{12} par exemple).

Oméga-3 & végétalisation de l'alimentation

Fabien BADARIOTTI
1 mars 2024

Oméga-3 & végétalisation de l'alimentation