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Nutritional Press Release: DHA & AMD

Invest Ophthalmol Vis Sci. 2014 Mar 28;55(3):2010-9. doi: 10.1167/iops. 14-13916.

Circulating omega-3 Fatty and neovascular age-related macular degeneration.

Merle BM¹, Benlian P, Puche N, Bassols A, Delcourt C, Souied EH, Nutritional AMD Treatment 2 Study Group.

Abstract

PURPOSE:

We assessed the associations of serum, red blood cell membranes (RBCM) and dietary long-chain n-3 polyunsaturated fatty acids (LC-PUFAs) with neovascular age-related macular degeneration (AMD).

CONCLUSIONS:

The RBCM EPA and EPA+DHA, as long term biomarkers of n-3 dietary PUFA status, were associated strongly with neovascular AMD and may represent an objective marker identifying subjects at high risk for neovascular AMD, who may most benefit from nutritional interventions.



Nutritional Press Release: DHA & AMD.

J Nutr. 2013 Apr;143(4):505-11. doi: 10.3945/jn. 112.171033. Epub 2013 Feb 13.

High concentrations of plasma n3 fatty acids are associated with decreased risk for late age-related macular degeneration.

Merle BM¹, Delyfer MN, Korobelnik JF, Rougier MB, Malet F, Féart C, Le Goff M, Peuchant E, Letenneur L, Dartigues JF, Colin J, Barberger-Gateau P, Delcourt C.

Abstract

High dietary intakes of n3 (ω 3) polyunsaturated fatty acids (PUFA) and fish have been consistently associated with a decreased risk for age-related macular degeneration (AMD). We assessed the associations of late AMD with the plasma n3 PUFA, a nutritional biomarker of n3 PUFA status. The Antioxydants Lipides Essentiels Nutrition et Maladies Oculaires (Alienor) Study is a prospective, population-based study on nutrition and age-related eye diseases performed in 963 residents of Bordeaux (France) aged \geq 73 y. Participants had a first eye examination in 2006-2008 and were followed for 31 mo on average. Plasma fatty acids were measured by GC from fasting blood samples collected in 1999-2001. AMD was graded from non-mydratric color retinal photographs at all examinations and spectral domain optical coherence tomography at follow-up. After adjustment for age, gender, smoking, education, physical activity, plasma HDL-cholesterol, plasma triglycerides, CFH Y402H, apoE4, and ARMS2 A69S polymorphisms, and follow-up time, high plasma total n3 PUFA was associated with a reduced risk for late AMD [OR = 0.62 for 1-SD increase (95% CI: 0.44-0.88); P = 0.008]. Associations were similar for plasma 18:3n3 [OR = 0.62 (95% CI: 0.43-0.88); P = 0.008] and n3 long-chain PUFA [OR = 0.65 (95% CI: 0.46-0.92); P = 0.01]. This study gives further support to the potential role of n3 PUFAs in the prevention of late AMD and highlights the necessity of randomized clinical trials to determine more accurately the value of n3 PUFAs as a means of reducing AMD incidence.

TABLE 2 Associations of plasma n3 PUFA with the risk for late AMD (Alienor Study 2006–2011, Bordeaux, France)¹

Plasma n3 PUFA	Without late AMD ²	With late AMD ³	OR (95% CI) ⁴	P value	Fully adjusted OR (95% CI) ⁵	P value
<i>% of total plasma fatty acids</i>						
Total n3 PUFA	4.5 \pm 1.3 ¹	4.0 \pm 1.2	0.64 (0.45, 0.90)	0.01	0.62 (0.44, 0.88)	0.008
18:3n3	0.42 \pm 0.19	0.38 \pm 0.11	0.65 (0.47, 0.89)	0.007	0.62 (0.43, 0.88)	0.008
n3 LC-PUFA ⁶	4.1 \pm 1.3	3.7 \pm 1.1	0.66 (0.47, 0.94)	0.02	0.65 (0.46, 0.92)	0.01
20:5n3	1.1 \pm 0.6	0.95 \pm 0.49	0.71 (0.49, 1.03)	0.07	0.66 (0.44, 1.03)	0.07
22:5n3	0.48 \pm 1.19	0.44 \pm 0.11	0.67 (0.45, 1.00)	0.05	0.65 (0.41, 1.02)	0.06
22:6n3	2.5 \pm 0.8	2.3 \pm 0.8	0.70 (0.49, 0.99)	0.046	0.73 (0.53, 0.99)	0.04

¹ Values are mean \pm SD. AMD, age-related macular degeneration; LC-PUFA, long-chain PUFA.

² n = 1074 eyes/541 participants.

³ n = 96 eyes/64 participants.

⁴ OR for 1-SD increase in plasma n3 PUFA, estimated using generalized estimating equations logistic regression adjusted for age, gender, and follow-up time.

⁵ Fully adjusted OR for 1-SD increase in plasma n3 PUFA, estimated using generalized estimating equations logistic regression adjusted for age, gender, smoking, educational level, physical activity, plasma HDL-cholesterol, plasma TG, CFH Y402H, ApoE4, and ARMS2 A69S polymorphisms, and follow-up time.

⁶ n3 LC-PUFA = 20:5n3+22:5n3+22:6n3.

TABLE 3 Associations of plasma n3 PUFA with the risk of late atrophic and neovascular AMD (Alienor Study, 2006–2011, Bordeaux, France)¹

Plasma n3 PUFA	Late atrophic AMD ²		Late neovascular AMD ³	
	OR ⁴ (95% CI)	P value	OR (95% CI)	P value
Total n3 PUFA	0.50 (0.29, 0.85)	0.01	0.64 (0.41, 1.01)	0.06
18:3n3	0.59 (0.36, 0.97)	0.04	0.63 (0.40, 0.97)	0.04
n3 LC-PUFA ⁵	0.52 (0.31, 0.88)	0.02	0.67 (0.43, 1.05)	0.08
20:5n3	0.35 (0.16, 0.75)	0.007	0.85 (0.54, 1.36)	0.51
22:5n3	0.59 (0.33, 1.05)	0.07	0.56 (0.31, 1.00)	0.05
22:6n3	0.70 (0.45, 1.10)	0.12	0.68 (0.46, 1.02)	0.06

¹ AMD, age-related macular degeneration; LC-PUFA, long-chain PUFA.

² OR were calculated for total (n = 1119 eyes, 565 participants)/participants with late atrophic AMD (n = 45 eyes, 30 participants).

³ OR were calculated for total (n = 1125 eyes, 568 participants)/participants with late neovascular AMD (n = 51 eyes, 34 participants).

⁴ OR for 1-SD increase in plasma n3 PUFA, estimated using generalized estimating equations logistic regression adjusted for age, gender, smoking, educational level, physical activity, plasma HDL-cholesterol, plasma TGs, CFH Y402H, ApoE4 and ARMS2 A69S polymorphisms, and follow-up time.

⁵ n3 LC-PUFA = 20:5n3+22:5n3+22:6n3.

Nutritional Press Release: DHA & AMD.

Am J Ophthalmol. 2014 Nov;158(5): 1071-78. doi: 10.1016/j.ajo.2014.07.036. Epub 2014 Aug 1.

Omega-3 supplementation combined with anti-vascular endothelial growth factor lowers vitreal levels of vascular endothelial growth factor in wet age-related macular degeneration.

Rezende FA¹, Lapalme E², Qian CX², Smith LE³, San Giovanni JP⁴, Sapehia P².

Abstract

PURPOSE:

To determine the influence of omega-3 supplementation on vitreous vascular endothelial growth factor A (VEGF-A) levels in patients with exudative age-related macular degeneration (wet AMD) receiving intravitreal anti-VEGF treatment.

DESIGN:

Prospective, randomized, open-label, single-center, clinical trial, consecutive interventional case series.

METHODS:

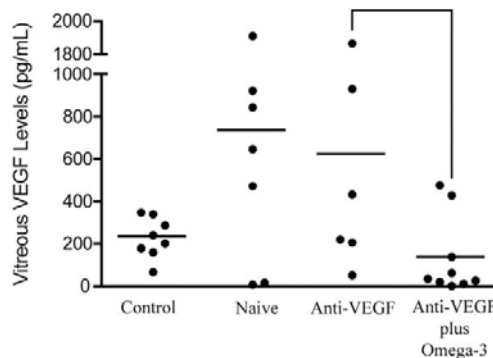
The study included 3 cohorts with wet AMD and a control group with epiretinal membrane or macular hole. Twenty wet AMD patients being treated with anti-VEGF were randomized to daily supplementation of antioxidants, zinc, and carotenoids with omega-3 fatty acids (docosahexaenoic acid and eicosapentaenoic acid; group 1, n = 10) or without omega-3 fatty acids (group 2, n = 10). They were compared with an anti-VEGF treatment-naïve wet AMD group (group 3, n = 10) and an epiretinal membrane or macular hole group (group 4, n = 10). Primary outcome was vitreal VEGF-A levels (at the time of anti-VEGF injection). Secondary outcomes were plasma VEGF-A and central foveal thickness. Patients with new submacular hemorrhage or any other treatment within 3 months were excluded. Final analyses included 9, 6, 7, and 8 patients in groups 1 through 4, respectively.

RESULTS:

Patients receiving omega-3s (group 1) had significantly lower levels of vitreal VEGF-A (141.11 ± 61.89 pg/mL) when compared with group 2 (626.09 ± 279.27 pg/mL; $P = .036$) and group 3 (735.48 ± 216.43 pg/mL; $P = .013$), but similar levels to group 4 (235.81 ± 33.99 pg/mL; $P = .215$). All groups showed similar values for plasma VEGF-A and central foveal thickness measurements.

CONCLUSIONS:

This study demonstrated that omega-3 supplementation combined with anti-VEGF treatment is associated with decreased vitreal VEGF-A levels in wet AMD patients.



Graph showing omega-3 supplementation and vitreous vascular endothelial growth factor (VEGF) A concentrations in wet age-related macular degeneration and control patients. Concentrations of vitreous VEGF-A, demonstrating that group 1 (anti-VEGF plus omega-3; n=9) had significantly lower levels than group 2 (anti-VEGF alone; n=6; $P=.0360$) and group 3 (treatment naïve, starting on anti-VEGF; n = 7; $P = .0139$). Data also demonstrate that group 1 (anti-VEGF plus omega-3) and group 4 (control; n=8) had similar vitreous VEGF levels ($P = .2153$) and that group 3 (treatment naïve) had significantly higher vitreous VEGF levels than group 4 (control; $P = .0387$). Group 2 (anti-VEGF alone) and group 3 (treatment naïve), both not taking omega-3 supplementation, had similar vitreous VEGF levels ($P = .7582$, t test).

Ces études récentes montrent l'intérêt d'une supplémentation en acides gras polyinsaturés longues chaînes Oméga-3, dont le DHA triglycérides dans les maladies dégénératives



DHA - Zinc : Pour maintenir une fonction cognitive et cérébrale normale.

DHA - Zinc : Pour maintenir une fonction visuelle normale.

Zinc : Pour contribuer à un métabolisme normal des glucides, des acides gras, de la vitamine A.

Sélénium - Zinc - Vit E : Pour protéger les cellules du stress oxydant.

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