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EPA prevents adipose tissue expansion in mice fed a high fat-high sucrose diet

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► **To cite this version:**

Alexandre Pinel, Jean-Paul Rigaudière, Elodie Pitois, Chrystele Jouve, Béatrice Morio, et al.. EPA prevents adipose tissue expansion in mice fed a high fat-high sucrose diet. 12. European Nutrition Conference FENS 2015, Oct 2015, Berlin, Germany. KARGER, Annals of Nutrition and Metabolism, 67, 601.p, 2015, Annals of Nutrition and Metabolism. hal-01223535

HAL Id: hal-01223535

<https://hal.science/hal-01223535>

Submitted on 19 Nov 2015

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Introduction:

Fat mass development and alterations in adipocyte metabolism contribute to the progression of insulin resistance (IR) and Metabolic Syndrome (MetS). IR is a key element in MetS and results from ectopic fat depots in the liver and skeletal muscle when adipose tissue storage capacity is exceeded. Epidemiological and interventional studies show that n-3 PUFA protect against risk factor for cardiovascular diseases. Nutritional strategies using n-3 PUFA were also proposed to limit adipocyte expansion and metabolic alterations in other tissues which are associated to MetS.

Objectives:

The aim of the present work was to compare the effect of ALA, EPA and DHA on the development of adipose tissue and its metabolism during a high fat-high sucrose (HFHS) challenge.

Materials & Methods :

C57BL6 and Ob/Ob mice were fed a HF (45% energy)-HS (17% energy) diet without or with 1% energy replaced by ALA, EPA or DHA for 16 and 6 weeks respectively. Body composition was evaluated using EchoMRI. Indirect gas calorimetry was performed with the TSE metabolic platform. mRNA and total DNA was isolated using Trizol Reagent from adipose tissue for gene expression (real time qPCR) and cellularity assessment. Leptin and adiponectin plasma levels were determined using A multiplex (BioRad) and ELISA (Marq) assay respectively. The Impact of each n-3 PUFA on the differentiation process of 3T3-L1 adipocytes was also evaluated *in vitro*. Statistical Analyses were performed using an ANOVA and a Fisher Post-Hoc test. (See also the poster from Pinel et al. with other details)

Results:

Table 1. Body composition

Variable	Control	HF	HF-A	HF-E	HF-D
body mass, g (n=18)	27.0 ± 0.4 b	29.6 ± 0.3 a	29.8 ± 0.5 a	29.8 ± 0.5 a	29.2 ± 0.4 a
Body fat mass, % (n=13-17)	9.5 ± 0.4 c	13.4 ± 1.2 a	13.0 ± 1.2 ab	10.3 ± 1.1 bc	13.3 ± 1.1 a
Body lean mass, % (n=13-17)	82.6 ± 0.4 a	79.6 ± 1.2 b	79.5 ± 1.1 b	82.8 ± 1.1 a	79.8 ± 1.0 b
eWAT fat, % BW (n=13-17)	1.3 ± 0.1 b	2.1 ± 0.3 a	2.4 ± 0.4 a	1.3 ± 0.2 b	2.3 ± 0.2 a

Table 2. Metabolic phenotyping

	Control	HF	HF-A	HF-E	HF-D
DE (kJ/gram)	1.60±0.02a	1.49±0.04ab	1.50±0.05ab	1.58±0.05a	1.41±0.04b
locomotor activity	178±12	174±19	139±14	179±19	119±18*
RQ	0.95±0.01a	0.81±0.01b	0.79±0.01b	0.80±0.01b	0.8±0.01b

* individual comparison vs LF, HF and HF-E found a difference a p=0.1

eWAT: epididymal white Adipose Tissue

Table 3. Plasma adipokines concentrations

	CTRL	HF	ALA	EPA	DHA
Leptine (n=8/11)	2.39 ± 0.24 b	2.3 ± 0.53 b	1.82 ± 0.33 b	0.85 ± 0.07 c	5.24 ± 0.55 a
Adiponectine (n=8/11)	9.13 ± 0.55 a	6.14 ± 0.49 b	5.73 ± 0.37 b	5.57 ± 0.31 b	5.68 ± 0.57 b

Leptin and Adiponectin concentration are expressed in ng/ml µg/ml respectively

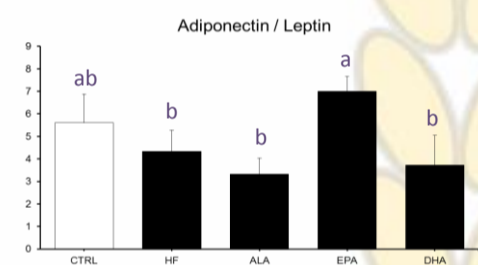


Figure 1. Adipose Tissue cellularity (n=12/15)

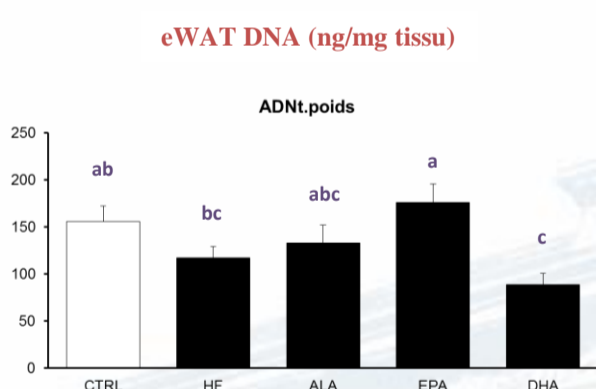


Figure 2. Adipose Tissue mRNA level (n=5/6)

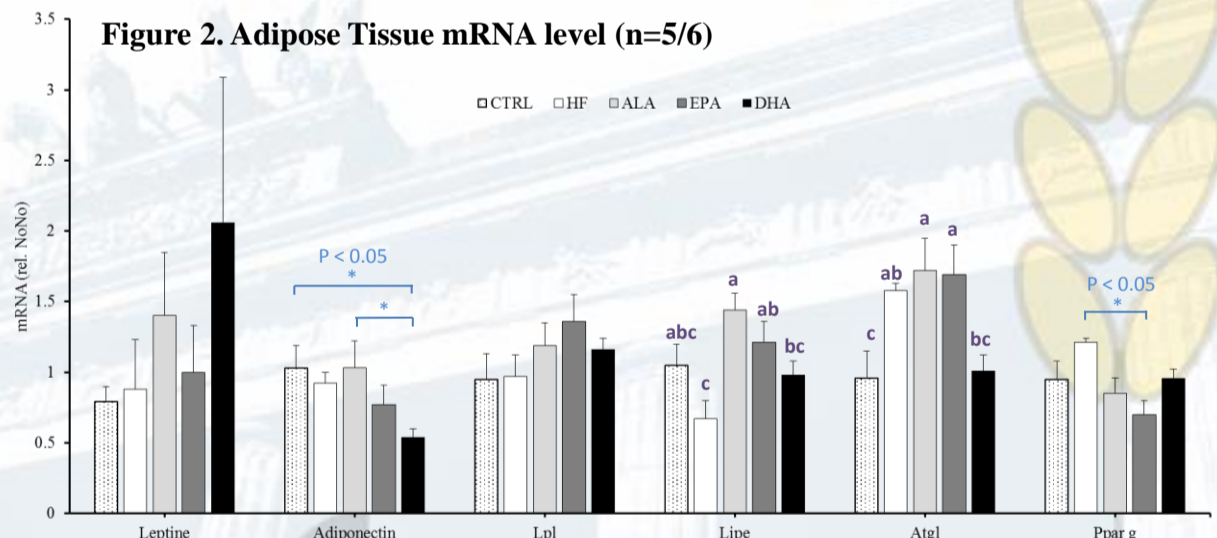
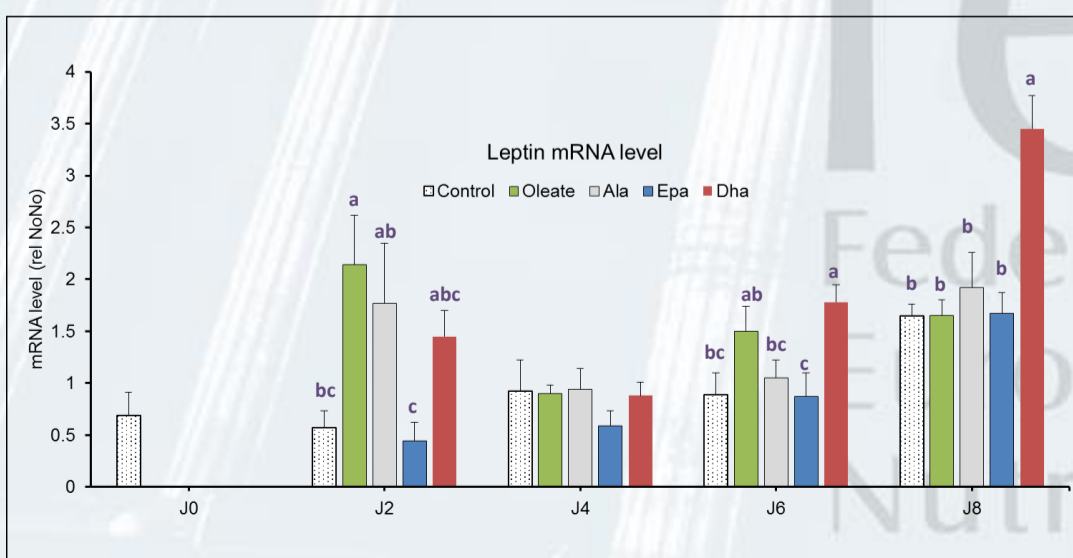


Figure 3. Kinetic of leptin expression in 3T3-L1 adipocytes with FA treatments (n=5)



Conclusion :

Long chain omega 3 fatty acids have distinct effects on adipose tissue cellularity and adipokine expression. Surprisingly, DHA increased leptin and reduced adiponectin expression. By contrast with ALA and DHA, EPA decreased adipose tissue PPARg mRNA level and prevent the increase in plasma leptin. Reduced adipocyte size could explain these preventive effects.