



HAL
open science

Transcriptionnal and epigenetic signatures of adaptive increased resistance to diet-induced obesity by dietary alleviation of malprogramming by maternal obesity during gestation

Linda L. Attig, A. Vigé, M. Karmimi, Aurore A. Beauger, M.S. Gross, A. Athias, C. Gallou-Kabani, Anne Gabory, P. Gamber, J.P. Jais, et al.

► To cite this version:

Linda L. Attig, A. Vigé, M. Karmimi, Aurore A. Beauger, M.S. Gross, et al.. Transcriptionnal and epigenetic signatures of adaptive increased resistance to diet-induced obesity by dietary alleviation of malprogramming by maternal obesity during gestation. Colloque SF-DOHaD, Nov 2012, Paris, France. Cambridge University Press, Journal of Developmental Origins of Health and Disease, 4 (Supplement 1), 2013, Founding meeting of SF-DOHaD. hal-02749244

HAL Id: hal-02749244

<https://hal.inrae.fr/hal-02749244>

Submitted on 3 Jun 2020

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.



Société Francophone
pour la recherche et l'éducation
sur les Origines Développementales,
Environnementales et Epigénétiques
de la Santé et des Maladies



8-9 Novembre 2012
Cnrs, PARIS
3 Rue Michel-Ange
75016 Paris

SF-DOHaD

Colloque Fondateur
Recherche Education Communication



Journal of Developmental Origins of Health and Disease
Volume 4, Supplement 1, March 2013
Proceedings of the founding meeting of SF-DOHaD
8-9 November 2012
Paris, France

Guest Editors: Marie-Aline Charles, Isabelle Le Hucrou-Luron and Claudine Junien

Publication of this supplement was supported by the Société Francophone pour la recherche et l'éducation sur les Origines Développementales, Environnementales et Epigénétiques de la Santé et des Maladies.

Abstracts of oral and poster presentations from the founding meeting of SF-DOHaD included in this supplement have been assessed by the Scientific Committee. They were reviewed by Editor-in-Chief of the Journal prior to publication, and may be cited.

With the support of:



aviesan

alliance nationale
pour les sciences de la vie et de la santé

Instituts
thématiques **Inserm**

Institut national
de la santé et de la recherche médicale

INRA



LaSalle★

UNIVERSITÉ DE LA SALLE

3. Artig L, et al. Submitted.
4. Lin S, et al. *Int J Obes Relat Metab Disord.* 2000; 24, 639–646.

Email: claudine.junien@jouy.inra.fr

POSTER N°61

Transcriptional and epigenetic signatures of adaptive increased resistance to diet-induced obesity by dietary alleviation of malprogramming by maternal obesity during gestation

L. Artig^{1,2}, A. Vigé³, M. Karimi⁴, A. Beauger^{1,2}, M.-S. Gross³, A. Athias³, C. Gallou-Kabani³, A. Gabory^{1,2}, P. Gamber⁶, T. Ekstrom⁴, J.-P. Jais⁷ and C. Junien^{1,2,3}

¹INRA, UMR1198 Biologie du Développement et Reproduction, Jouy-en-Josas, France; ²ENVA, Maison Alfort, France;

³INSERM U781, Paris, France; ⁴Laboratory for Medical Epigenetics, Karolinska Institutet, Stockholm, Sweden; ⁵IFR100 Santé-STIC, Plateau Laboratoire de Biochimie Médicale, Dijon, France; ⁶Service de Biostatistique et Informatique Médicale, Université Paris Descartes, Paris, France

Maternal obesity and type 2 diabetes (T2D) at conception and during gestation promote the development of obesity and diabetes in adulthood.¹ However, very few studies have considered whether and how appropriate nutrition could alleviate this malprogramming. An important proportion of inbred animals develop resistance to the obesogenic effects of a high-fat diet (HFD), regardless of the species, the window and mechanisms at stake.² In a previous study, we showed that despite maternal obesity and T2D, a control diet (CD) during the periconceptional/gestation/lactation period led to a pronounced sex-specific shift from susceptibility to resistance to a HFD in the female offspring.³ The aim of this study was to determine the molecular mechanisms of resistance and susceptibility, and how a CD could alleviate the effects of maternal obesity and T2D on the fetus and increase resistance. Despite being similarly lean (resistant) or obese (sensitive), F2 and F1 mice clearly differed in several aspects of their metabolism, with F2 mice presenting obvious features of 'adaptation' on the HFD. Expression data using a custom-built mouse microchip for the liver and quantitative RT-PCR for muscle and adipose tissue highlighted that adaptive processes in F2 mice were associated in the liver with an enhancement of pathways protecting against steatosis, the recruitment of unexpected neurotransmission-related genes and the modulation of a vast imprinted gene network. In the adipose tissue, adipogenesis and lipid storage were also modified in F2 mice. Global DNA methylation and several histone marks assessed using LUMA technique and western blot analysis, as well as the expression of 15 genes encoding chromatin-modifying enzymes, supported the response and adaptation to HFD, in a generation- and tissue-specific manner.⁴ Thus, improvements in the nutrition of obese and

diabetic women during pregnancy would be an efficient management strategy, with lower risks than current strategies on the basis of weight loss or nutrient supplementation, which may have a negative impact on fetal programming.

Key words: developmental programming, DOHaD, epigenetics, fatty liver, hepatocellular carcinoma, high-fat diet, metabolic syndrome, nutrition, obesity, sexual dimorphism, type 2 diabetes

Statement of interest: Authors report no conflict of interest.

References

1. Shankar K, et al. *Am J Physiol Regul Integr Comp Physiol.* 2008; 294, R528-R538.
2. Koza RA, et al. *PLoS Genet.* 2006; 2, e81.
3. Gallou-Kabani C, et al. *Am J Physiol Endocrinol Metab.* 2007; 292, E1095–E1100.
4. Artig L, et al. Revised.

Email: claudine.junien@jouy.inra.fr

TOWARD DIETARY AND THERAPEUTIC RECOMMENDATIONS?

10 – L. Najar and D. Linton

Long-term consequences of prematurity, oxidative stress, noxious neonatal environmental and diet-induced obesity on metabolic and cardiovascular disorders are examined. Regarding the programming of appetite, an assessment of neonatal weight loss with child weight at 1 and 3 years was presented. In view of the importance of dietary supplementation for optimal development, authors provide insight into the impact of prenatal vitamin D on cord blood level. Intake of bioactive molecules (docosahexaenoic acid and arachidonic acid) from milk during development of premature newborn was examined.

ORAL N°52

Long-term consequences of neonatal pain and analgesia on growth, behavior and corticotroph axis in rat

I. Dutriez-Casteloot, V. Bor-Robin, V. Emmanuelli, A. Tavernier, J. Demetz, S. Auriens, J. Maillet, B. Pusniak, A. Dickes-Coopman, V. Montel, E. Denhez, S. Deloof, D. Vieau, L. Storme and V. Debarge

EA4489, Environnement périnatal et croissance, Université Lille Nord de France, Lille, France

Previous data reported that a noxious neonatal environment alters brain development and is responsible for diseases in adulthood (metabolic syndrome, obesity).¹ With advances in medical care over the last two decades, the number of newborn infants exposed to chronic pain has considerably increased,^{2,3} and this has led to the augmentation of the use of opioid to induce analgesia.⁴ However, the long-term effects of perinatal