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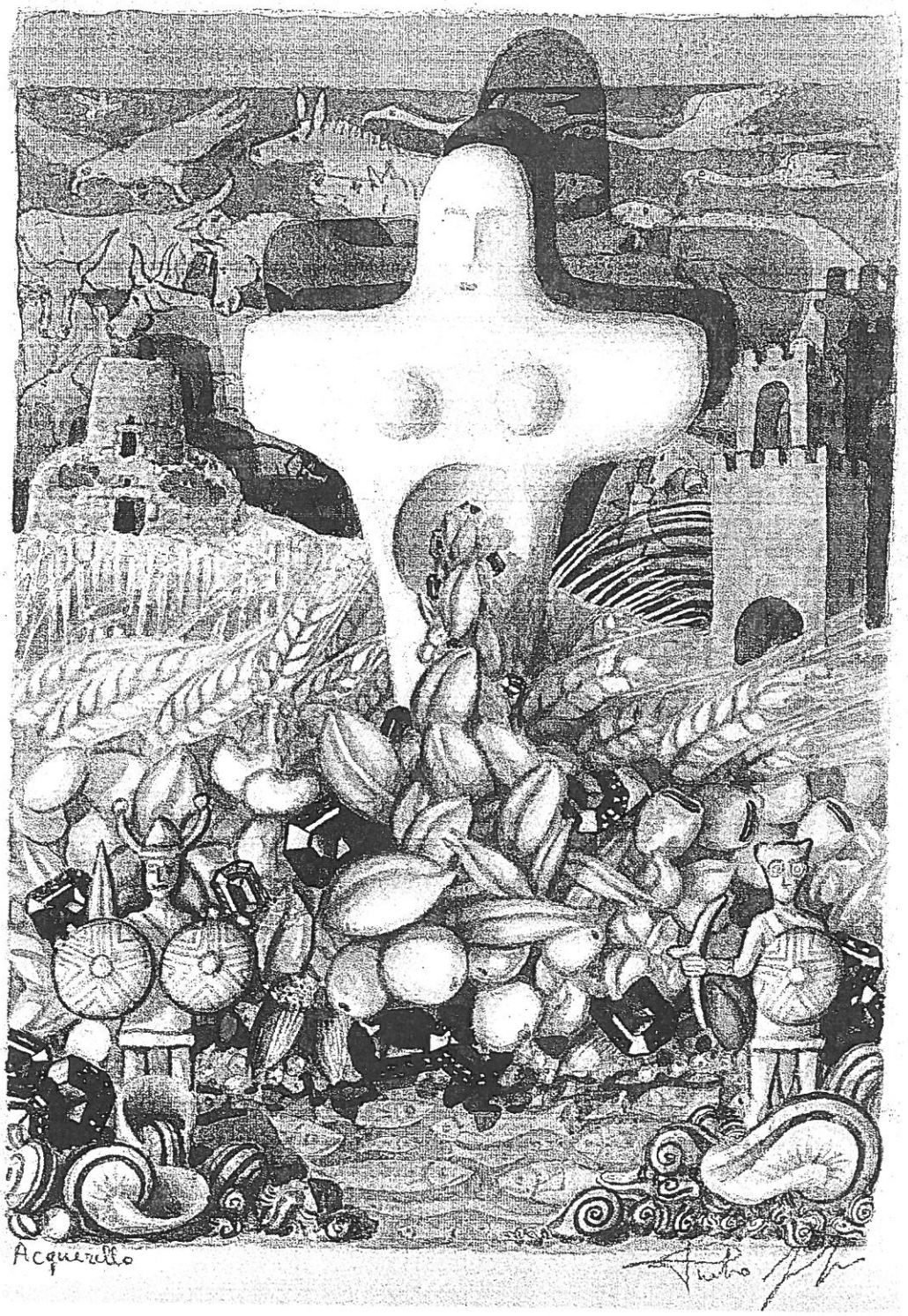
Published in:
Congress Proceedings

Publication date:
2009

Document version
Publisher's PDF, also known as Version of record

Host:
Sezione di Produzioni Animali
Faculty of Veterinary Medicine
University of Sassari

Congress Proceedings



13 th
Congress of the

European

Society of

Veterinary and

Comparative

Nutrition

Co-organiser:
Dipartimento di Produzioni Animali,
Epidemiologia ed Ecologia
Faculty of Veterinary Medicine
University of Turin

October, the 15-17 2009 - Oristano, Sardinia (Italy)

Foetal protein-malnutrition in mink causes changes in F_2 progeny

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Introduction: Malnutrition during foetal life can induce permanent metabolic changes in offspring, due to metabolic and structural adaptation to the available nutrient supply in order to maximize the outcome. These adaptations might result in permanent changes in the offspring, but depend on when the malnutrition occurs, i.e., during certain sensitive periods of foetal development (Lucas, 1991). Epigenetic modification and consequences are not necessarily limited to first-generation (F_1) offspring, but can be passed on to subsequent generations (Pinheiro *et al.*, 2008). Our objectives were to investigate if low protein supply during foetal life affects the quantitative metabolism and expression of key hepatic gluconeogenic and glycolytic enzymes in yearling F_1 generation (F_1) mink dams, despite adequate feeding postnatally and furthermore, if possible changes can be transmitted to the F_2 generation offspring (F_2).

Materials and Methods: Forty-eight F_1 yearling female mink of the standard black genotype were used, 24 of which had been exposed to low protein supply (14% of metabolizable energy – ME – from protein; FL) and 24 to adequate protein supply (29% of ME from protein; FA) during the last 17.9 ± 3.6 days of foetal life. The dams had been given adequate protein supply from birth onwards. Forty F_1 dams were used to study reproductive performance and to record kit body weight (BW) at birth of the F_2 offspring and until 28 days of age. Sixteen F_1 animals, eight dams from each *in utero* treatment, were used in balance and respiration experiments in the first and last thirds of true gestation, and after parturition the F_1 dams and their F_2 offspring (six kits in each litter) were measured in the second and fourth weeks of lactation. Blood was drawn once a week from the F_1 dams for hormone analyses. Eight F_1 dams were euthanized in late gestation for tissue collection from dams and foetuses for determination of gene expression of key hepatic gluconeogenic and glycolytic enzymes.

Results and Discussion: The quantitative metabolic traits of the F_1 dams were unaffected by protein supply during foetal life, but ME intake (2082 vs 1816 kJ/kg$^{0.75}$) was significantly ($p = 0.02$) higher, and the retained energy (247 vs 4 kJ/kg$^{0.75}$) tended ($p = 0.10$) to be higher among FL than FA during the fourth week of lactation. The F_2 offspring of FL dams had significantly ($p = 0.003$) higher birth weight than those of FA dams (12.2 vs 11.1 g). Plasma concentrations of leptin, insulin and IGF-1 were not affected by *in utero* protein supply but insulin ($p = 0.09$) and IGF-1 ($p = 0.1$) tended to be lower among FL than FA F_1 dams. No significant differences in the relative abundances of G-6-Pase, Fru-1,6-P_2ase, PKM_2 and PEPCK mRNA in hepatic tissue between FL and FA dams were found. Despite this, the relative abundance of PKM_2 mRNA (3.1 vs 13.7) was significantly ($p = 0.007$) lower, and that of Fru-1,6-P_2ase mRNA (2.9 vs 5.6) tended ($p = 0.08$) to be lower in hepatic tissue of F_2 foetuses of FL than FA F_1 dams.

Conclusion: Our study confirms that epigenetic changes in hepatic enzymes affecting glucose homeostasis can be transmitted from the F_1 to the F_2 generation in mink exposed to foetal life protein restriction. A possible cause of the higher birth weights of FL offspring might be maternal gestation hyperglycemia which, however, remains to be confirmed.

References:

Lucas A (1991) *Ciba Foundation Symposium 156, 38-55.*
Pinheiro AR, Salvucci IDM, Aguila MB & Mandarim-de-Lacerda CA (2008) *Clin Sci* 114, 381-392