

## Liver damage caused by maternal protein malnutrition are not recovered by restoring the normal diet in adult life

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Growth restriction in utero is associated with the development of obesity, hypertension, and diabetes in animal models. The current understanding is that intrauterine deprivation programs the individual for a deprived environment, and that such programming is maladaptive in a nondeprived environment. The liver plays an essential role in metabolism regulation.

The aim of this work was study the effect of protein malnutrition during gestation and lactation on liver function offspring. And consider whether these changes can be reversed by refeeding with normal protein diet.

Pregnant Wistar rats of three months of age who were fed a diet containing 8% of proteins (M), malnourished group (M) or 20% control group (C). The male offspring of mothers M, after weaning, were fed diet 8% P (MM) or control diet (reversed group-MC). In addition, male offspring from mothers C were feed with Diet C (CC). At day 60 post-birth the rats were slaughtered, bled by cardiac puncture and the liver was dissected. Body weights and liver were lower in the MM. MC recovered body weight but have greater liver. Serum levels of protein, albumin, triglycerides and cholesterol in MM were lower compared to the CC and MC. GOT-GPT transaminases,  $\gamma$ -Glutamyl transferase and alkaline-phosphatase were higher in MM. MC presented high values of GPT and  $\gamma$ -Glutamyl transferase. MM hepatic content of glycogen and proteins were lower than CC, but TG and Col were much higher. MC did not reverse cholesterol levels, remain high. Histological studies showed MC and MM with alterations in nuclear morphology with apoptosis increased.

**Conclusion:** The lack of protein during development compromises the integrity of the structural and functional liver, manifested in adulthood and not revert until the time studied.

### Biography

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