

Effect or selenium supplementation on clinical manifestations and plasma biochemical parameters in streptozotocindiabetic rats mildly balanced by insulin

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The nature of dietary fatty acids affects the glycemic and insulinemic responses to carbohydrates in healthy subjects. JL Joannic ^{1,2}, S Auboiron ^{1,2}, J Raison ¹, A Basdevant ¹, F Bornet ², B Guy-Grand ¹ (¹ Internal Medicine and Nutrition, Hôtel-Dieu; ² Éridania Béghin-Say, 75008 Paris, France)

The effect of a mixed meal composed of different kinds of carbohydrates and fats on postprandial plasma concentrations of glucose, insulin, free fatty acids and triglycerides was investigated in eight young normolipidemic men $(24 \pm 1 \text{ year}, \text{ body mass})$ index (BMI) $21.5 \pm 0.8 \text{ kg/m}^2$). Three hours after a standardized breakfast (300 kcal, 18% fats, 70% carbohydrates, 12% proteins), the subjects ingested four test meals (1 200 kcal, 50% fats, 38% carbohydrates, 12% proteins) in 30 min on separate days in random order according to a Latin square design.

The meals contained two kinds of carbohydrates: instant mashed potatoes (high glycemic index 70–90%) or rice (low glycemic index 50–55%) and two mixtures of vegetable oils, with either a high monounsaturated/polyunsaturated fatty acids n-6 ratio (M), or a low one (P). Proteins, saturated and polyunsaturated fatty acids n-3 were comparable in all meals. The plasmatic parameters were measured every 30 min during 3 h after the beginning of the test meal.

During the postprandial kinetic, the glycemic response was significantly lower with rice-P than potato-M or rice-M (P < 0.01) after 30 min. The insulinemic response was

lower for rice-P than with potatoes-M (P < 0.05). At 90 min, the average insulin level was similar for rice-P and rice-M and significantly lower than potato-P or potato-M (P < 0.005). No significant differences were found between meals in free fatty acid or triglyceride plasma levels.

In conclusion: i) The insulin response was significantly different between rice and instant mashed potatoes only when carbohydrates are associated with a polyunsaturated rich meal. ii) The polyunsaturated rich meals decreased the insulin response to the two kinds of carbohydrates. The same tendency was observed for the glycemic response.

Thus, the postprandial plasma concentrations of glucose and insulin are influenced by the nature of dietary fatty acids present in the meal.

Effect or selenium supplementation on clinical manifestations and plasma biochemical parameters in streptozotocindiabetic rats mildly balanced by insulin. C Douillet ¹, M Accominoti ², M Bost ³, F Borson-Chazot ⁴, M Ciavatti ¹ (¹ Inserm 63, 69675 Bron; ² Hôpital Édouart-Herriot, 69008; ³ Trace Element Institute for UNESCO, 69008; ⁴ Hôpital de l'Antiquaille, 69005 Lyon, France)

Seventy-six Sprague-Dawley rats were used in this study. Twelve rats were used as control (group C). Diabetes was induced in 64 rats by iv injection of streptozotocin (30 mg/kg). All rats with glycemia levels > 2.5 g were considered diabetics. All rats received a purified diet (in calories: 30% lipids, 15% proteins, 55% glucides). Three groups of 16 diabetic rats each were supplemented with selenium (Se): a Se-rich yeast (group DSel), or selenomethionine (group DSm) or selenomethionine + vitamin E (group DSmE). The supplementation of Se in all groups corresponded to $0.99 \ \mu mol \ Se/100 \ g$ of diet and for vitamin E to $0.145 \ \mu mol/100 \ g$. Sixteen diabetic rats were not supplemented (group D). All the diabetic rats were treated by insulin.

After 24 weeks the weight gain in group C was 33% and only 15% in group D (P < 0.05 vs group C), but when the rats were supplemented with selenium the increase was higher and not significantly different from group C. Mortality was null in group C, and from 6/14, 4/14, 6/14 and 3/16, respectively, in groups D, DSel, DSm and DSmE.

Plasma selenium levels were significantly increased in all Se-supplemented diabetic groups compared to group C. Glycemia was significantly increased in the diabetic groups compared to group C (P < 0.0005), but it tended to be lower in the Se-supplemented groups compared to group D. The same effect of selenomethionine was observed with glycosylated Hb. Plasma lipid levels (cholesterol, phospholipids, triglycerides) were increased in the diabetic groups compared to group C, but a large decrease in triglycerides (TG) was observed in groups DSm and DSmE compared to group D after 9 and 24 weeks of the diet (P < 0.01, P <0.05, respectively).

The ratio vitamin E/TG did not change in the diabetic groups compared to group C, except in group DSmE, where it was significantly increased compared to all the other groups (P < 0.01 vs C, D, DSel, P < 0.05 vs DSm). In group DSmE, TBARS and conjugated diene levels were significantly decreased after 24 weeks compared to group D (P < 0.01). These parameters increased in all the other diabetic groups after 24 weeks but more weakly in the Sesupplemented group compared to group D. Moreover, plasma fatty acid changes were modulated in diabetic rats by selenomethionine and more efficiently by selenomethionine + vitamin E where the overcorrection of of Δ 9- and Δ 6-desaturases was reduced.

These results indicate that selenium and more efficiently Se + vitamin E tend to normalize the glycemic balance and to correct several plasma abnormalities observed in diabetic rats (increase of TG and peroxides), which represent risk factors for the cardiovascular complications associated with this pathology.

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Long chain polyunsaturated fatty acid (LCPUFA) assay before and after orthotopic liver transplantation (OLT) in children with extrahepatic biliary atresia (EBA). M Chambon ¹, A Lapillonne ², V Chirouze ³, V Mamoux ¹, O Boillot ⁴, M Lievre ⁵, A Lachaux ¹ (¹ Hépatograstroentérologie et nutrition pédiatriques; ² Néonatologie, hôpi-

Age (months)	Linoleic acid	AA	DHA
1.5–4	9.45 ± 1.78	16.47 ± 0.66	4.71 ± 1.82
8–11	7.54 ± 2.44	15.35 ± 1.55	2.32 ± 0.42**
After PN	10.43 ± 3.24**	15.06 ± 0.80	3.42 ± 1.10**
6 months post-OLT	10.30 ± 1.54	19.52 ± 1.03**	3.44 ± 0.63**
1 year post-OLT	11.30 ± 1.77	19.18 ± 0.77*	4.19 ± 0.95*

Laboratory data (% of total fatty acids, means ± SD).

* P < 0.01: 8-11 vs 1 year post-OLT; ** P < 0.05: 1.5 - 4 vs 8 - 11, 8 - 11 vs after PN, 8 - 11 vs 6 months post-OLT.