Correction of carnitine deficiency in children with recent onset Type 1 diabetes (T1D)

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Background
Carnitine deficiency (CD) has been reported in children at time of T1D diagnosis. By impairing free fatty acid β-oxidation in liver, skeletal muscle, heart, and pancreatic β cells, CD might impair glucose homeostasis in various tissues including β cells thus residual insulin secretion.

Objectives
Evaluate the effects of L-carnitine supplementation during the 1st year following T1D diagnosis
We postulated that improving fatty acid oxidation through L-carnitine supplementation may improve insulin sensitivity and β cells function possibly leading to an increase of remission frequency or magnitude.

Patients and Methods
21 children chosen at random at diagnosis of T1D (age 4-16 years), with positive autoantibodies) received carnitine supplementation (« C+ ») with 100 mg/kg L-carnitine per day. They were compared to 20 untreated children (« C- »). Patients with ketoacidosis at diagnosis were excluded. Compliance was checked by the elevation of plasma carnitine.

Results
1. At diagnosis (within 2 weeks after diagnosis):
C+ and C- groups were comparable for HbA1c, C peptide, pH, age, plasma carnitine, insulin doses.
CD was confirmed with a mean plasma total carnitine at 31.8 µmol/L (normal: 43-65).

2. At 12 months:
HbA1c was 7.1% in C+ and 7.6% in C- (NS) with insulin doses 0,9 U.K.d and 0,8 U.K.d respectively (NS). C peptide was 0,1-1,1 µg/L in C+ and 0,1-0,8 µg/L in C- (NS).
Gained weight was 20% of initial body weight in the two groups (table).

Conclusions
CD at T1D diagnosis is confirmed. CD supplementation at the given doses hardly brought plasma carnitine up to the normal mean, leaving ¼ of children in the low carnitine range. The current protocol of supplementation did not change HbA1c, insulin doses or residual β cell function within the first year of clinical T1D.

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Disclosure statement
none of the authors have conflict of interest to declare.