

Low density lipoprotein quality and discordance with apolipoprotein B in intensively controlled Type 1 diabetes: Any relationship with nutrition?

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Type 1 diabetes (T1D) is partly characterised by a higher prevalence of cardiovascular disease (CVD). Despite low density lipoprotein cholesterol (LDL-C) being a commonly treated target, apolipoprotein B (Apo B) has been shown to be a superior predictor of CVD and discordance between these two markers may predispose patients to altered risk⁽¹⁾. The distribution of LDL-C also contributes to these risks, with LDL III & IV fractions possessing greater atherogenic potential⁽²⁾. Few studies have investigated LDL-C quality and it's discordance with Apo B in relation to the nutritional intake of patients with intensively controlled Type 1 diabetes. The aim of this study was to address this dearth of research.

Following ethical approval and informed consent 28 patients (32 % male; 68 % female) (mean age 48 ± 15) were asked to complete a food frequency questionnaire (FFQ), donate a sample of blood and allow the authors access to their medical records to determine HbA_{1c}. The initial FFQ responses were processed using FETA software. The blood sample was analysed for LDL-C, constituent sub-fractions and Apo B. All data were interrogated using descriptive statistics. Dichotomous dependent variables pertaining to LDL-C and Apo B were compared using McNemar's test and correlations between dietary variables were determined with Spearman's rho test.

Significant differences were shown between LDL-C categories when compared to Apo B ($p = 0.039$) and the majority of patients (46.4 %) presented LDL-C >2.0 mmol/L and Apo B >80 mg/dL (Fig. 1). Although not discordant, these findings still suggest an increased risk according to recommendations⁽³⁾. Closer inspection of results revealed that individuals with raised LDL-C typically had an abundance of LDL I & II fractions which may somewhat reduce this risk (Fig. 2). Spearman's correlation applied to the whole population produced no relationship between diet and LDL-C or Apo B; however, when focussing on the predominant 'at risk' cluster significant and strong relationships between LDL-C and total carbohydrate ($R^2 = 0.835$; $p < 0.001$) and sucrose ($R^2 = 0.758$; $p = 0.003$) were found. No hypoglycaemia data were collected and the authors tentatively speculate that these relationships may be a consequence of its treatment. In the light of the small sample size a further more comprehensive investigation with an appropriately powered sample would be beneficial.

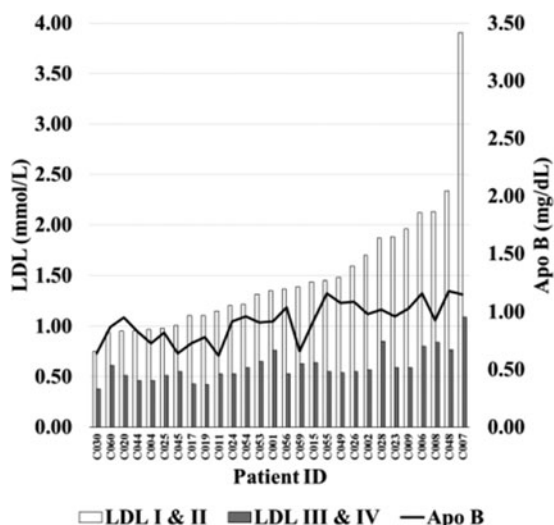


Fig. 1. Patient LDL-C and Apo B in relation to recommendations (Cluster HbA_{1c} (mmol/mol) shown on columns).

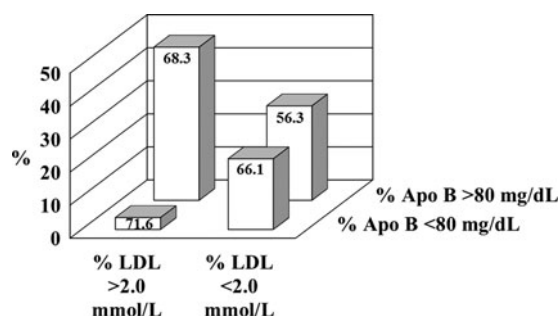


Fig. 2. Individual patient LDL subfractions and Apo B.

1. Otvos J *et al.* (2011) *J Clin Lipidol* 5, 105–113.
2. Vergès B (2009) *Diabetes & Metabolism* 35, 353–60.
3. Catapano AL *et al.* (2016) *Eur Heart J* 37, 2999–3058.