

Production of glycerol by rat muscles in vivo: demonstration using microdialysis.

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It is generally considered that circulating glycerol is produced mostly by the hydrolysis of adipose tissue triglycerides and, for a small part, by the hydrolysis of circulating triglycerides. However, an hormone-sensitive lipase is present in muscles suggesting that this tissue could also produce glycerol. To test this hypothesis, we infused (3 h) rats ($n = 5$, 24 h fasted) with [6,6- ^2H] glucose and [2- ^{13}C] glycerol. Microdialysis catheters were inserted into subcutaneous abdominal adipose tissue and into hind leg muscles. Blood and dialysate samples, were collected during the third hour to measure isotopic enrichments (IE, MPE) of glucose and glycerol. It had been previously verified in vitro that there was no difference between ^{12}C and ^{13}C glycerol for passing through the dialysis membrane. Glucose IE was the same in muscle ($5.39 \pm 0.32\%$) and adipose tissue ($5.3 \pm 0.31\%$) dialysates samples and in blood ($5.47 \pm 0.24\%$). As expected, glycerol IE was lower in adipose tissue dialysate samples than in blood (2.3 ± 0.3 vs $6.2 \pm 0.2\%$, $P < 0.01$) due to lipolysis in adipose tissue. However, glycerol IE in muscle dialysate samples ($3.3 \pm 0.5\%$), although higher than in adipose tissue ($P < 0.05$), was also lower than in blood ($P < 0.01$).

Conclusion: These results strongly suggest that muscles are an active producer of glycerol in vivo.

Disorder of apolipoprotein AI metabolism in non insulin-dependent diabetes mellitus: a kinetic study. R Frenais, K Ougueram, C Maugeais, P Mahot, T Magot, M Krempf (*Centre de recherche en nutrition*

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A disorder of High Density Lipoprotein (HDL) metabolism could explain the excess of cardiovascular diseases in non insulin-dependent diabetic (NIDDM) subjects. However, kinetic perturbations of this metabolism have not been clearly shown. A 14 h ($^2\text{H}_3$]-leucine primed infusion (10.10^{-6} mol/kg/h) was given to eight NIDDM patients (Hb-A1C = $8.16\% \pm 1.93$) and seven control subjects. A monocompartmental model was used to analyse HDL kinetic data (SAAM II software). For apolipoprotein AI (apo AI) precursor enrichment, it was assumed that the VLDL-apo B100 tracer-to-tracee ratio at plateau, calculated by monoexponential regression, corresponded to the tracer-to-tracee ratio of the leucine precursor pool. Apo AI concentration was lower in NIDDM than in controls (96 ± 12 vs 124 ± 13 mg.dL $^{-1}$, $P < 0.002$). The mean apo AI fractional catabolic rate (FCR) was significantly faster (0.39 ± 0.16 vs 0.21 ± 0.06 day $^{-1}$, $P = 0.032$) and the apo AI absolute production rate (APR) was not significantly greater (16.6 ± 6.1 vs 12.0 ± 4.2 mg.kg $^{-1}$.day $^{-1}$, $P = 0.17$) in NIDDM. Furthermore, HDL-triglycerides level and apo AI/HDL-cholesterol ratio were higher in diabetic patients (respectively 14.9 ± 6.2 vs 4.3 ± 1.5 mg.dL $^{-1}$, $P < 0.004$ and 2.78 ± 0.84 vs 2.31 ± 0.16 , $P = 0.24$). The FCR of apo AI was not correlated with Hb-A1C level, but positively correlated with plasma and HDL-triglycerides levels, and inversely correlated with apo AI concentration.

We conclude that decreases in plasma apo AI and HDL cholesterol concentrations are related to the increase in HDL-apo AI FCR in NIDDM.

Kinetic heterogeneity of non insulin-dependent diabetic patients: stable isotope study. C Maugeais, P Mahot, K Ougueram, M Krempf, T Magot (*Centre*