

in part by a higher secretion of *trans*-C18:1 (+63 g/day), C20:5 (+1 g/day), C22:6 (+2 g/day) and other C20-C22 fatty acids (+28 g/day).

Unprotected FO addition to dairy cow diet (R infusion) was shown to decrease the milk protein content, to decrease sharply the milk fat content and to increase the secretion of *trans*-C18:1, that arose from changes in rumen ecosystem and was probably the cause of the collapse of mammary lipogenesis. Protected FO addition (D infusion) would decrease the milk fat content and increase the *n*-3 polyunsaturated fatty acid secretion.

**Heavy sustained exercise-induced changes in body water compartments.** P Ritz<sup>1</sup>, N Fellmann<sup>2</sup>, G Pickering<sup>2</sup>, B Beaufrère<sup>1</sup>, J Coudert<sup>2</sup> (<sup>1</sup> *Laboratoire de nutrition humaine*; <sup>2</sup> *Laboratoire de biologie et de physiologie du sport, CRNH-Auvergne, 63000 Clermont-Ferrand, France*).

Calculation of lean body mass (LBM) from total body water (TBW) measurements assumes a known hydration coefficient for LBM. An increase in plasma volume (PV) is established during prolonged and repeated exercise and could be the consequence of either a fluid shift from intracellular and/or interstitial compartments or of TBW retention. In the latter case hydration of LBM would change. The aim of this study was to measure changes in body water compartments during a 7-day endurance raid and their consequences on LBM estimates.

Nine subjects ( $42.1 \pm 7.8$  year, mean  $\pm$  SD) engaged in a triathlon of 595 km and 13.100 m cumulative gain in altitude. PV (Evans Blue dye dilution), TBW (<sup>18</sup>O dilution) and extracellular water (ECW, Bromide dilution) were measured before (C) and after (R, day + 1) the raid. Daily changes in TBW (BIA, 100 kHz), ECW (BIA 5 kHz) and PV (from changes in haemoglobin and

hematocrit) were also assessed. Although the weights of the subjects remained stable, ( $68.4 \pm 6.5$  kg day 1,  $68.1 \pm 6.8$  kg day 7), an inflation of all body water compartments was observed; between C and R, increases were  $+22 \pm 11\%$  (PV,  $P < 0.001$ ),  $+4.1 \pm 2.0$  L (TBW,  $P < 0.001$ ) and  $+2.0 \pm 1.3$  L (ECW,  $P < 0.001$ ). PV, TBW and ECW significantly increased from day 1 to day 4 then plateaued till day 7-8.

In conclusion, heavy sustained exercise induces a body water retention, in all compartments including intracellular water. If hydration of LBM had been kept constant, this would result in an increase of LBM by 5.7 kg and an equivalent loss in fat mass, which is energetically impossible. In circumstances prevailing in this study changes in body composition cannot be estimated from changes in TBW.

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**Binding of anti lipoprotein lipase immunoglobulins to chylomicrons in autoimmune type I hyperlipidemia.** V Pruneta<sup>1</sup>, P Moulin<sup>1</sup>, F Labrousse<sup>2</sup>, P Bondon<sup>3</sup>, G Ponsin<sup>1</sup>, F Berthezène<sup>2</sup> (<sup>1</sup> *Laboratoire de métabolisme des lipides*; <sup>2</sup> *Service d'endocrinologie et des maladies de la nutrition*; <sup>3</sup> *Laboratoire de biochimie, Hôpital de l'Antiquaille, 69005 Lyon, France*).

Secondary hyperchylomicronemia are mostly induced by diabetes mellitus and/or environmental factors such as excess of alcohol or carbohydrate intake. We describe a rare case of secondary hyperchylomicronemia induced by an autoimmune disease in a 35-year-old woman who presented a severe and intermittent type I hypertriglyceridemia (TG: 2–60 mmol/L). Treatment by fresh plasma exchange, strict dietary therapy and administration of fibrates or *n*-3 fatty acids were unable to maintain a consistent remission. Because of an history of familial and

personal autoimmunity, we introduced an immunosuppressive therapy (azathioprine + corticosteroids). A consistent long term and stable remission was induced with only two relapses which occurred when the treatment was reduced and was put under control by reintroduction of the immunosuppressors.

Postheparin plasma LPL activity was decreased in the patient and was normalized by the treatment (1.9 vs 5.9  $\mu\text{mol/h/mL}$  controls:  $7.2 \pm 2.2 \mu\text{mol/h/mL}$ ). Hepatic lipase activity (HL) remained normal (6.1 vs 6.8  $\mu\text{mol/h/mL}$ ; controls:  $6.9 \pm 2 \mu\text{mol/h/mL}$ ). To demonstrate a direct interaction between an autoantibody and LPL, we studied by Western blot, the binding of the patient's immunoglobulins to nitrocellulose-bound human LPL. A specific binding of IgG to LPL was observed only with the chylomicrons of the patient. No binding of IgG was found in other patients lipoproteins. No signal was obtained either with patient's chylomicrons obtained in the postprandial state while she was under immunosuppressive therapy or with those from diabetic patients with type V hyperchylomicronemia. A competitive inhibition of the IgG-LPL binding was obtained with porcine pancreatic lipase, a protein that shares a partial homology with human LPL. In vitro, proteins extracted from patients chylomicrons induced a dose-dependent specific inhibition of control postheparin plasma LPL activity.

These data demonstrate that an autoantibody can specifically inhibit LPL activity and induce hyperchylomicronemia. Additionally, they suggest that in vivo, LPL is bound on the surface of chylomicrons. However, it is likely that the inhibition of LPL activity is dependent upon a direct interaction of the antibody with LPL bound at the arterial wall.

**Contribution of hepatic lipogenesis and plasma free fatty acids reesterification to hepatic triglycerides secretion in healthy subjects.** F Diraison, C Pachiardi, M Beylot (*Laboratoire de physiopathologie métabolique et rénale, Faculté de médecine René-Laënnec, 69373 Lyon cedex 08 et CRNH de Lyon, Hôpital Edouard-Herriot, 69472 Lyon cedex, France*).

Triglycerides (TG) secreted by liver can be synthesized with fatty acids derived from hepatic lipogenesis or from plasma free fatty acids (FFA) uptake (reesterification). The contribution of these two sources of fatty acids was measured in four healthy subjects (TG =  $0.86 \pm 0.02 \text{ mmol/L}$ , mean  $\pm$  SEM) in the post-absorptive period. Lipogenesis was calculated using the measurement of deuterium incorporation in TG-palmitate molecules from deuterated water (loading dose of 3 g/kg of estimated total body water). FFA reesterification was calculated using the kinetic of the incorporation in TG of intravenously infused [ $1-^{13}\text{C}$ ]palmitate during 4 h. The fractional (k) and absolute ( $R_t$ ) turnover rate and the half-life ( $t_{1/2}$ ) of TG were calculated using the kinetic of decay (6 h) of  $^{13}\text{C}$  in the palmitate of TG after the end of [ $1-^{13}\text{C}$ ]palmitate infusion. The enrichments were measured using isotopic ratio mass spectrometry (deuterium in plasma water,  $^{13}\text{C}$  in FFA-palmitate and TG-palmitate) or organic mass spectrometry (deuterium in TG-palmitate). Results were  $2.91 \pm 0.34 \text{ h}$ ,  $0.226 \pm 0.017 \text{ h}^{-1}$  and  $0.16 \pm 0.01 \text{ mmol/kg/min}$  respectively for  $t_{1/2}$ , k and  $R_t$ . These values agreed with those of other studies. The contribution of FFA reesterification to this TG plasma pool was  $0.1 \pm 0.01 \text{ h}^{-1}$ , that is to say  $44 \pm 3\%$  of TG  $R_t$ . The lipogenesis contribution to this  $R_t$  was only  $3.5 \pm 0.5\%$ . These results confirm that hepatic lipogenesis, contrary to reesterification, is a minor pathway in healthy subjects. However, these two metabolic pathways represent only 50% of secreted TG. The remaining 50% could come from either