Massey University Institute of Food Nutrition & Human Health Te Kunenga ki Pūrehuroa

Adaptation to an MCFA-Rich Diet: Effect on Gastric Tolerance, the Capacity for MCFA Oxidation, and Performance while Ingesting Exogenous Carbohydrate and Structured Oils during Endurance Exercise.

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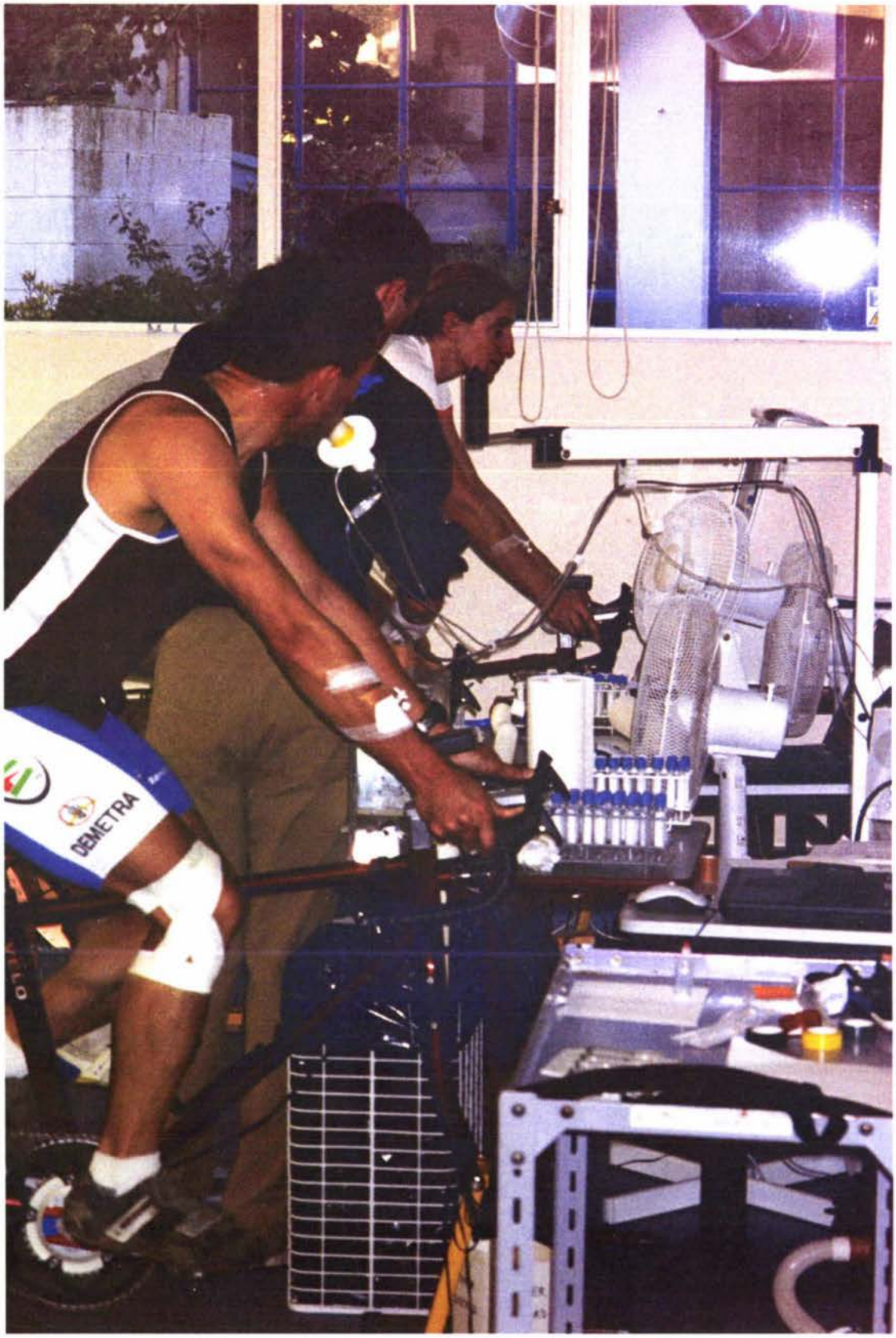
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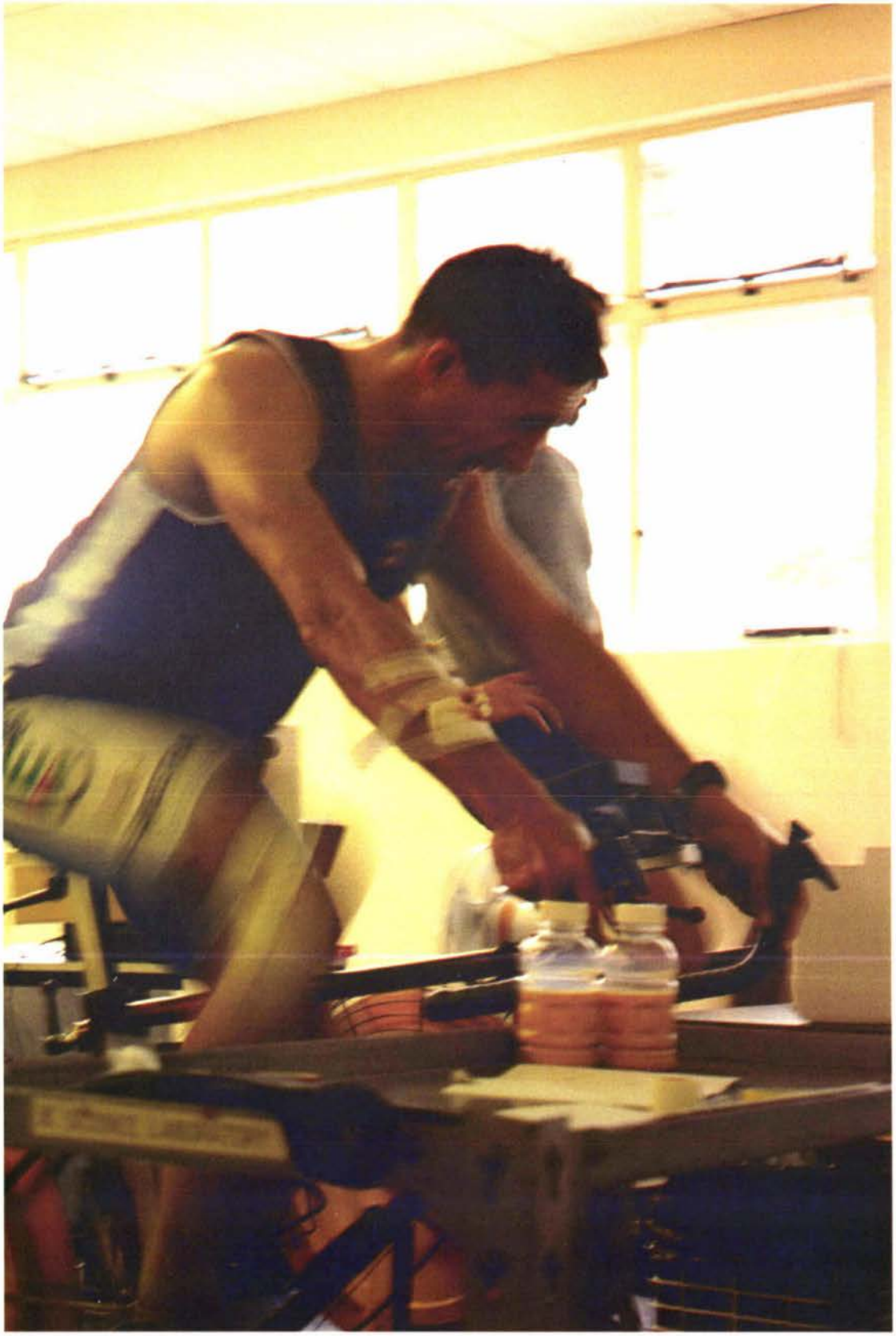

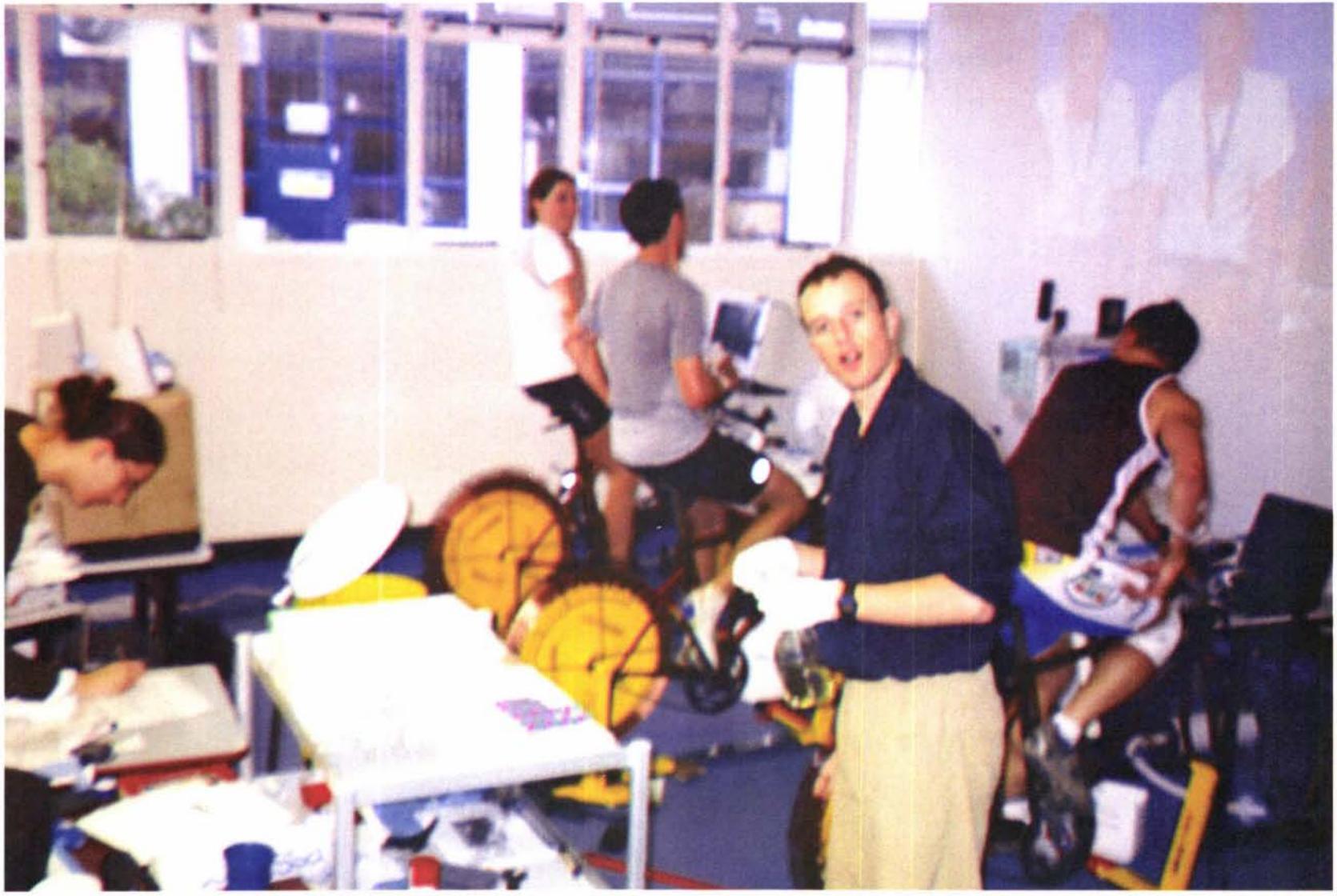

Abstract

Introduction: Elevating the availability of fatty-acids to the muscle can potentially benefit endurance exercise performance by reducing intramuscular-glycogen utilisation. Digestion of triglycerides containing long-chain fatty acids (LCFAs) is slow, and fatty acids must pass through the carnitine palmityl transferase (CPT) transport system to enter the mitochondria, which potentially limits fat oxidation during prolonged-heavy exercise. Conversely, medium-chain triglycerides (MCTs) are rapidly digested and their constituent fatty acids (MCFAs) by-pass the CPT transport system. Ingestion of MCFAs may therefore supply mitochondrial acetyl-CoA, potentially reducing the requirement for glycolytic flux during exercise. However, studies comparing carbohydrate (CHO) with CHO-containing MCFA-rich exercise supplements have revealed inconsistent results, probably because of the variation in gastrointestinal (GI) distress suffered by participants associated with MCT ingestion.

Purpose: To investigate whether 2-weeks of dietary adaptation to MCFA-rich supplements reduces the severity of gastrointestinal (GI) distress, or increases the rate of MCFA oxidation during endurance exercise. A decrease in ratings of GI distress, or an increase in MCFA oxidation was anticipated to lead to performance benefits.

Method: Nine well-trained male endurance cyclists participated in a double-blind, pseudo-randomised, triple-crossover protocol. Participants were 37 ± 7.26 years, 81.36 ± 7.67 kg, training at least 8-10 h per week and riding competitively. Mean $\dot{V}O_2$max and peak power output (PPO) were 4.84 ± 0.46 L·min^{-1} and 357.33 ± 20.55 W respectively. The effects of a 2-week MCFA-rich diet + ^{13}C-enriched MCFA+CHO exercise supplement (MC-MC) on GI distress, MCFA-oxidation rate and sprint performance variables were compared against a 2-week LCFA-rich diet with either: (a) a ^{13}C-enriched MCFA+CHO exercise supplement (LC-MC), or (b) a CHO-only supplement (LC-CHO). Dietary and exercise MCFA-rich supplements were consumed in the form of randomised-structured triacylglycerols made with a 3:1 molar ratio of MC- and LCFAs randomly esterified to glycerol backbones. Participants followed a controlled training regime whilst on the diets.

The performance test consisted of a 3-h ride at 50% PPO followed by 10 maximal sprints. At rest and every 20-min throughout the ride, participant ratings of GI and exertion sensations were recorded, followed by external respiratory-gas analysis, collection of a breath sample for breath ^{13}C-enrichment analysis, a venous blood sample and ingestion of a supplement.

Similarly, after sprints 1, 4, 7 and 10 participants recorded their GI ratings followed by a blood sample.

Results: Peak MCFA-oxidation rates were 0.38 $g \cdot min^{-1}$ (95% CI 0.31-0.47) and 0.43 $g \cdot min^{-1}$ (0.30-0.61, p-value = 0.21) in the MC-MC and LC-MC conditions respectively, but there was no evidence for CHO sparing following MCFA adaptation. Participant ratings of GI distress decreased slightly during exercise with 2-weeks of a diet high in MCFAs relative to LCFAs. Ratings of reflux, bloatedness, nausea, and urge to vomit were, respectively, 1.34 (0.88-3.14), 1.03 (0.74-2.27), 0.81 (0.62-1.69) and 0.93 (0.64-2.45) scale units lower in the MC-MC condition relative to LC-MC. The attenuation in GI distress corresponded with a tendency toward increased sprint mean power, which was 3.4% (± 5.9%, 0.25) higher in the MC-MC condition relative to LC-MC. However, sprint mean power was still lower in both the MC-MC (6.8% ± 2.8%, <0.0001) and LC-MC (10.4% ± 5.5%, 0.0004) conditions relative to LC-CHO.

Mechanism covariate analysis illustrated a negative effect of the GI distress marker nausea on sprint performance. For every 1 unit increase in nausea for the MC-MC and LC-MC conditions, sprint power decreased by 6 W (± 3.8, 0.004) relative to LC-CHO.

Conclusion: No clear metabolic adaptation was evident with high dietary MCFA relative to LCFA. In addition, MCFA-rich exercise supplements caused a decrement in performance relative to CHO ingestion in both MC-MC and LC-MC conditions, suggesting that light-moderate GI distress still causes substantial performance detriments. There was little evidence to support the ingestion of randomised structured triglycerides high in MCFA with the intention of enhancing endurance performance.

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