gliadin antibodies (IgA-AGA) levels were also measured. An annual follow-up will take place to evaluate the SAT as a predictive tool for developing CD in the future. We would like to present the first-year screening results.

In the SAT a solution of lactulose (L), mannitol (M) and sucrose was ingested and the L/M ratio was measured in 5-h urine. A ratio > 0.089 was considered aberrant. 126 first-degree relatives of biopsy-proven coeliacs and 84 controls were investigated. The SAT was performed and in 80 relatives IgA-AGA levels were also measured. Relatives with an aberrant L/M ratio or IgA-AGA level were biopsied. For statistical analysis the Mann-Whitney *U*-test was used. A *p*-value < 0.05 was considered significant.

L/M ratios were significantly higher in the relatives group (mean ± SEM: 0.092 ± 0.006) than in the controls (0.039 ± 0.003) ($p < 0.001$). In the relatives group 45/126 (36%) had an aberrant L/M ratio, in the controls 3/84 (4%). So far, 34 relatives with an aberrant L/M ratio have been biopsied: 5 had (sub)total villous atrophy and 8 infiltrative lesions (Marsh classification: 1–2/4). In 2/80 relatives an elevated level of IgA-AGA was found: one had an aberrant L/M ratio and total villous atrophy in biopsy, the other had a normal L/M ratio and infiltrative lesions in biopsy. Three relatives with (sub)total villous atrophy were IgA-AGA-negative.

Conclusions: Relatives of coeliacs have higher L/M ratios than controls as measured with the SAT. An aberrant L/M ratio may be related to constitutional factors, a low-grade immunological reaction to gluten or latent CD. IgA-AGA analyses were not useful in screening for CD. A follow-up study will be performed to investigate whether an aberrant SAT, without histological changes at first biopsy, is a predictive tool for the development of clinical CD in the future.

Influence of medium-chain and structured triglycerides on the mononuclear phagocyte system in postoperative patients. J.W. Kruimel [1], A.H.J. Naber [1], J.W.M. Veh[illegible] [1], E. Koenders [2], J.A. van der Vliet [3], F.G.M. Buskens [3], F.H.M. Corstens [2], J.B.M.J. Jansen [1]. *Departments of [1] Gastroenterology, [2] Nuclear Medicine and [3] Surgery, University Hospital, Nijmegen, Netherlands.*

We studied the effects of parenteral infusion of structured triglycerides (STG) and an emulsion containing 50% medium-chain and 50% long-chain triglycerides (MCT/LCT) on the mononuclear phagocyte system (MPS) in postoperative patients, measuring 99m-technetium-sulfur colloid (TSC) clearance. TSC clearance is known to be reduced after administration of long-chain triglyceride emulsions. Thirteen well-nourished patients underwent elective implantation of an aortic prosthesis and were treated with total parenteral nutrition (TPN) for the first 5 days postoperatively. Amino acids were supplied in a dose of 0.2 g N/kg/day; $\frac{1}{3}$ of the non-protein energy was given as a lipid emulsion (STG or MCT/LCT) and $\frac{2}{3}$ as carbohydrates. The lipid emulsion was infused in 6 h, daily; one group received the STG emulsion ($n = 6$) and the other group received the MCT/LCT emulsion ($n = 7$). TSC clearance was measured before the start of TPN and after 5 days of TPN. A bolus of 12–20 MBq TSC was injected intravenously. At 1.5, 3, 4.5, 6, 9, 12, 15, 30 and 60 min after injection, venous blood samples were drawn and radioactivity was measured. The two-tail probation test was used. Before the start of TPN, TSC clearance in the STG group was 192 ± 66 ml/min ($n = 6$) and in the MCT/LCT group 202 ± 34 ml/min ($n = 7$) (n.s.). Two patients did not complete the study. After 5 days of TPN, TSC clearance was 206 ± 62 ml/min ($n = 6$) in the STG group and 183 ± 94 ml/min ($n = 5$) (n.s.) in the MCT/LCT group. The change in TSC clearance, during 5 days of TPN, was $+14 \pm 74$ ml/min (n.s.) in the STG group and -20 ± 57 ml/min (n.s.) in the MCT/LCT group. There was no significant difference between the change in TSC clearance in the STG group and the MCT/LCT group.

Conclusions: In contrast to long-chain triglyceride emulsions, lipid emulsions containing medium-chain triglycerides do not suppress the functioning of the mononuclear phagocyte system. In this respect, there is no difference between the effects of a physical mixture of medium-chain and long-chain triglycerides and a structured triglyceride emulsion.

Dietary sulfate might inhibit colonic methane production in man. A. Tangerman [1], W. Wesseling [2], M.B. Katan [2], F.M. Nagengast [1]. *[1] Department of Gastroenterology, University Hospital, Nijmegen; [2] Agricultural University, Wageningen, Netherlands.*

Hydrogen produced during colonic fermentation is consumed by methanogens to form methane or by sulphate-reducing bacteria (SRB) to form toxic hydrogen sulphide (H_2S). Impairment of colonocyte nutrition by H_2S and involvement of H_2S in the pathogenesis of ulcerative colitis have been postulated. It is controversial whether methanogens outcompete SRB for H_2 consumption. Problems in quantitating sulphide in faeces have hampered research in this field. The aim of the present study was to establish the role of SRB in the human colon in relation to that of methanogens.

In a cross-over study, 12 normal subjects with methanogenic activity (CH_4-producers) and 8 subjects without (CH_4-non-producers) received a 1-week diet rich (S-rich period) or poor (S-poor period) in dietary sulphate. Both periods were separated by a wash-out of 1 week. At the end of each period, faeces was sampled and faecal sulphide concentrations (free, bound and total) were measured by means of gas chromatography. No differences in total faecal sulphide levels were found between the S-rich (1.79 ± 1.15 mmol/kg dry weight, mean ± SD) and the S-poor period (1.96 ± 1.61 mmol/kg). The CH_4-producers and non-producers had also similar sulphide levels. Nearly all sulphide was bound to the insoluble particulate faeces fraction. During the S-rich period a significant reduction in breath methane was observed in the CH_4-group. Of the 12 CH_4-producers, 4 became non-producers, 3 showed a strong reduction and 5 remained unchanged. This reduction was accompanied by a reduced CH_4 production in the corresponding faecal samples during in vitro faecal incubation experiments. In vitro faecal incubation with 20 mM Na_2SO_4 showed a huge increase in free sulphide concentrations, reaching levels of 30 mmol/kg dry weight, in both the S-rich and S-poor samples. This increase was even more

pronounced in the CH_4-producers, compared to the non-producers. In the CH_4-producers, this increase in sulphide was accompanied by a decrease in CH_4 production.

Conclusions: Although a sulphate-rich diet did not result in elevated faecal sulphide levels, high sulphate levels might stimulate SRB activity at the cost of methanogens, as also shown by in vitro incubation experiments. This study also showed that SRB activity is not confined to CH_4-non-producers, but is at least equally active in CH_4-producers.

Effect of insulin on pancreatico-biliary secretion during hyper- and normoglycaemic conditions. M. Coenraad, W.F. Lam, H.A.J. Gielkens, I. Biemond, C.B.H.W. Lamers, A.A.M. Masclee. *Department of Gastroenterology-Hepatology, University Hospital, Leiden, Netherlands.*

Acute hyperglycaemia is known to inhibit gastrointestinal motility and secretion. In non-diabetic subjects acute hyperglycaemia stimulates endogenous insulin release. It is not known, however, whether hyperinsulinaemia contributes to the inhibitory effect of hyperglycaemia on gastrointestinal function. Therefore, we have investigated the effects of hyperinsulinaemia (aimed at 200 mU/l) both during normoglycaemic clamping (blood glucose 5 mmol/l, plasma insulin 200 mU/l; hyperinsulinaemic normoglycaemic clamp; HI) and during hyperglycaemic clamping (blood glucose 15 mmol/l, plasma insulin 200 mU/l; hyperglycaemic hyperinsulinaemic clamp; HG) on pancreatico-biliary secretion in healthy subjects and compared them with the results obtained during normoglycaemia (NG). Nine healthy subjects (age 22–52 yr) were studied on 3 separate occasions during HI, HG and NG clamping. Pancreatico-biliary secretion was measured with a 4-lumen tube in 15-min portions under basal conditions (60 min) and during cholecystokinin infusion (CCK 0.25 IDU·$kg^{-1}\cdot h^{-1}$) for 60 min. Plasma CCK and pancreatic polypeptide (PP) levels were determined at regular intervals.

Results ($p^* < 0.05$)		NG	HG	HI
Basal	Trypsin (U/h)	24± 5	7± 2 *	8± 3 *
	Bilirubin (μmol/h)	13± 4	5± 2 *	3± 1 *
CCK	Trypsin (U/h)	39±10	20± 7 *	55±14
	Bilirubin (μmol/h)	64±22	29±14 *	40± 7

Plasma CCK levels were not significantly different between the 3 experiments. Plasma PP secretion during hyperglycaemia (0.2±0.06 nM·120 min) and hyperinsulinaemia (0.8± 0.2 nM·120 min) were significantly ($p < 0.05$) reduced compared to normoglycaemia (1.5±0.2 nM·120 min).

Conclusions: Hyperglycaemia inhibits basal and CCK-stimulated pancreatico-biliary secretion. Hyperinsulinaemia inhibits basal but does not affect CCK-stimulated pancreatic-biliary secretion. Hyperinsulinaemia therefore contributes to the inhibitory effect of hyperglycaemia on basal pancreatico-biliary secretion. The impaired PP secretion points to vagal cholinergic inhibition during both hyperglycaemia and hyperinsulinaemia.

Improved diagnosis of low intestinal lactase activity by the ^{13}C-lactose breath test by a shift in the rate-limiting step due to exercise. F. Stellaard, H.A. Koetse, H. Elzinga, C.M.A. Bijleveld, H.S.A. Heymans, R.J. Vonk. *Beatrix Children's Hospital, University Hospital, Groningen, Netherlands.*

$^{13}CO_2$ breath tests for the clinical diagnosis of metabolic or intestinal impairment are normally performed under standardized resting conditions. After administration of a ^{13}C-enriched substrate, $^{13}CO_2$ abundance is measured in the breath, expressing the contribution of the substrate to total energy metabolism. Experience with the ^{13}C-lactose breath test in young children for diagnosing low intestinal lactase activity indicates a 61% sensitivity of the $^{13}CO_2$ breath test when compared with the actual lactase activity measurement in intestinal biopsy material (HA Koetse et al. $^{13}CO_2/H_2$ breath test for detection of lactose malabsorption in children using naturally enriched ^{13}C-lactose. In press, 1995). A prerequisite for the accurate performance of a $^{13}CO_2$ breath test for the diagnosis of nutrient malabsorption is that intestinal enzymatic hydrolysis or the absorption process under investigation represents the rate-limiting step in the overall process leading to $^{13}CO_2$ production. Oxidation of absorbed hydrolytic products (monosacharides, free fatty acids) is supposedly much faster than the intestinal process. However, this assumption may be questioned when the breath test is performed at rest by applying relatively large dosages of substrate, since the metabolic demand for substrate may then be lower than that supplied by the intestine, even when intestinal function is impaired. This phenomenon was demonstrated by one adult subject who could not be detected as a malabsorber of 80 g lactose when applying the naturally enriched ^{13}C-lactose breath test at rest (4 h cumulative % dose value of 16.6% vs. [mean±SD] 19.6±3.8% for 6 controls), although the simultaneously performed H_2 breath test gave a clear-cut positive result (max. H_2 excretion within the 4-h period: 34.6 vs. 4.0±2.6 μmol/min for the controls). Repeated performance of the breath tests at an increased activity level of 50 W revealed a similar excretion of H_2 (22.6 vs. 5.0±3.7 μmol/min), whereas the $^{13}CO_2$ breath test now revealed a positive test result: 4 h cumulative % dose value of 20.5 vs. 66.1±6.2%. Thus, in contrast to the controls, the subject of interest was not able to increase utilization of the lactose substrate during exercise due to the insufficient influx of substrate to meet the increased metabolic demand.

Conclusions: These data support the hypothesis that the combination of a high dose of substrate and a low level of intestinal enzyme activity may result in false-negative $^{13}CO_2$ breath test results due to a sufficient substrate influx to maintain a normal contribution to the total energy metabolism at rest. By increasing the metabolic demand the shortage of substrate becomes more evident.