

Na-alginate, and for comparative purposes, pectin were fed to faecal bacteria cultured in single-stage chemostats, with pH control (6.5). Substrate (10 g.L⁻¹) was supplied for 15 days, and samples were taken for determinations of β -eliminated products, bacterial counts, isolation of alginate-degrading populations, and measurements of polyuronate depolymerase activities in bacterial cell extracts.

β -eliminated products accumulated in alginate chemostats during the initial 4 days of culture enrichment. Pectine decreased *Fusobacterium* sp populations whereas alginate selected for aerobes microorganisms. Bifidobacterial numbers were increased by alginate and decreased by pectine, however, these effects were not significant.

All alginolytic bacteria isolated among anaerobes catalysed β -elimination reactions.

Bacteria from both cultures exhibited low pectin lyase activities (ca 0.5 μ mol- β -eliminated-products mg-protein⁻¹). Conversely, high alginate lyase activity (ca 9 μ mol- β -eliminated-products mg-protein⁻¹) was specifically detected in alginate chemostats, although it was not present on the first day of culture. Hydrolase activity was high in pectin chemostats (ca 20 μ mol-reducing-ends mg-protein⁻¹), whereas reducing ends produced by cell extracts from alginate containing media corresponded only to bacterial lyase activities.

It is therefore concluded that alginate fermentation by human intestinal bacteria first requires substrate depolymerisation via β -elimination. Induction of alginate lyase and/or enrichment in alginate degrading bacterial populations may explain the latency phase observed prior to fermentation. Bacterial metabolism of β -eliminated products could also explain the lack of correlation between alginate utilization and SCFA formation.

Effect of dietary lipids on fatty acid composition and fertility of fowl semen. E Blesbois, M Lessire, D Hermier (*Inra, station de recherches avicoles, 37380 Nouzilly, France*).

Phospholipids are the major lipids (~ 80%) of spermatozoa (SPZ) and contain ~30% of *n*-6 polyunsaturated fatty acids (FA). The role of these FA on fertility has been investigated in 32 male fowls receiving isolipidic diets containing 5% of either salmon oil (salmon, *n*-6/*n*-3 = 1.1) or corn oil (corn, *n*-6/*n*-3 = 41.6). Analyses were performed on composition of spermatozoa (SPZ) and of seminal plasma (SP) and on fertilizing ability after artificial insemination. SP contained more saturated FA than SPZ, respectively 50% and 40%. SP and SPZ had high amounts of 20:4 *n*-6 (5–9%) and 22:4 *n*-6 (11–21%); these two FA were absent from the diets and partly replaced 18:2 *n*-6 in sperm (only 2–6% in sperm vs 15–46% in the diets). Moreover, when compared to the corn diet, the salmon diet increased significantly the amount of *n*-3 long-chain FA (6.7% vs 2.6%) and decreased the amount of *n*-6 long-chain FA (23.3% vs 33.3%). In parallel, the fertility rate was significantly higher with the salmon diet (96.0% vs 91.5% with the corn diet). Thus the nature of dietary FA may influence the fertilizing ability of fowl semen, probably by modifying the *n*-6/*n*-3 ratio of membrane lipids.

Additive effect of A→G (–3826) variant of the uncoupling protein gene and the Trp64Arg mutation of the β 3-adrenergic receptor gene on weight gain in morbid obesity. K Clément^{1,2}, J Ruiz^{2,4}, AM Cas-sard-Doulcier³, F Bouillaud³, D Ricquier³, A Basdevant¹, B Guy-Grand¹, P Froguel² (¹ *Département de nutrition, Hôtel-Dieu, 75004 Paris*; ² *CNRS EP 10, Institut Pasteur de Lille, 59000 Lille*; ³ *CNRS, 92000 Meudon, France*; ⁴ *Division d'endocrinologie et métabolisme, Lausanne, Switzerland*).

Obesity results from an imbalance between caloric intake and energy expenditure, which is partly genetically determined.

Objectives: We have investigated, using a PCR-RFLP assay, the effects on weight gain of two genetic variants of the uncoupling protein and the β 3-adrenoceptor, two major expressed proteins of the brown adipose tissue (BAT) involved in thermo-genesis.

Subjects: 238 morbidly obese and 91 non-obese Caucasians subjects.

Results: A high prevalence (27%) in French Caucasians of the A→G change variation located in the 5' flanking domain of the UCP gene was observed with no significant difference between morbidly obese patients and non-obese subjects, suggesting that UCP gene is not a major gene for obesity. However, in the population of morbidly obese subjects, the presence of the A→G allelic variant of the UCP gene showed to be an associated factor of high weight gain during adult life (odd-ratio: 1.4, $P = 0.02$). Such an association was previously described for the Trp64Arg mutation of the β 3-AR gene. Furthermore, an additive effect of these two gene variants on weight gain was observed (odd-ratio: 4.95, trend test: $P = 0.05$). The attributable risks for UCP gene and β 3-AR gene variants were respectively 25% and 9%.

Conclusion: These data support the hypothesis of a possible link between energy balance, BAT and weight gain in obese human subjects.

Hyperglycemia, but not hyperinsulinemia, inhibits liver glucose-6 phosphatase activity in rat. L Guignot, G Mithieux (*Inserm U 449, faculté de médecine René-Laënnec, 69372 Lyon cedex 08, France*).

It has recently been reported that liver glucose-6 phosphatase (Glc6Pase) activity is inhibited by 3 h-refeeding in rats. To study the respective roles of hyperglycemia and hyperinsulinemia in this inhibition, we have performed hyperinsulinic euglycemic and hyperglycemic clamps in rats. Five hours after food removal, anesthetized rats were perfuse for 3 h with saline (S), or with insulin at 480 pmol/h (I), or with glucose at 2.4 mmol/h (G1) and 3.9 mmol/h (G2). In group I, the perfusion of 1.29 ± 0.01 mmol/h glucose (mean \pm SEM, $n = 5$) has been required to maintain glycemia at the basal level. Hepatic glucose production (HGP) was assessed by [3 H]glucose dilution and Glc6Pase assayed in homogenates from liver lobes freeze-clamped *in situ*. The results were:

Group	n	Final glycemia mmol/L	Final insulinemia pmol/L	HGP μ mol/kg/min	Glc6Pase U/g wet liver
S	5	8.6 ± 0.5	185 ± 17	77 ± 9	8.1 ± 0.1
I	5	7.7 ± 1.4	$461 \pm 45^*$	$21 \pm 12^*$	7.7 ± 0.6
G1	4	$12.6 \pm 0.1^*$	$567 \pm 47^*$	$27 \pm 2^*$	$6.1 \pm 0.6^\circ$
G2	4	$17.3 \pm 0.9^*$	$876 \pm 68^*$	$0.3 \pm 6^*$	$6.0 \pm 0.4^*$

The data are expressed as mean \pm SEM

$^\circ$, *: different from S value, $P < 0.05$, $P < 0.01$, respectively.