

**Table I.** (JF Gabbarou, PA Geraert)

	R+				R-			
	Control mean	DFF mean	DFF + PPL mean	RSD	Control mean	DFF mean	DFF + PPL mean	RSD
Food intake (g/day)	125 <sup>a</sup>	58 <sup>c</sup>	54 <sup>c</sup>	8.8	75 <sup>b</sup>	47 <sup>c</sup>	38 <sup>c</sup>	5.9
Heat production <sup>a</sup>	409 <sup>a</sup>	399 <sup>a</sup>	361 <sup>b</sup>	9.7	286 <sup>c</sup>	303 <sup>c</sup>	294 <sup>c</sup>	7.5
Respiratory quotient	0.94 <sup>a</sup>	0.82 <sup>b</sup>	0.80 <sup>b</sup>	0.01	0.93 <sup>a</sup>	0.84 <sup>b</sup>	0.81 <sup>b</sup>	0.01
Diet -induced thermogenesis (% EMI) <sup>a</sup>	23.6 <sup>a</sup>	44.4 <sup>b</sup>	37.3 <sup>b</sup>	5.7	10.3 <sup>c</sup>	23.2 <sup>a</sup>	24.8 <sup>a</sup>	8.9

<sup>a</sup> Heat production (HP) in kJ/kg<sup>0.75</sup>.d. Diet-induced thermogenesis = (fed HP – fasted HP) / metabolisable energy intake. Mean values with different letters were significantly different ( $p < 0.05$ ). RSD: residual standard deviation.

### **$\beta$ -adrenergic and serotonergic control of diet-induced thermogenesis in birds.**

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Two experimental lines have been divergently selected for high (R+) or low (R-) residual feed intake [Bordas *et al* (1992) *Br Poult Sci* 33, 741-754]. Observed feed intake differed by 40% in males for the same bodyweight, and by 20% in females for the same bodyweight and the same egg-mass produced.

Energy balance measurements showed that basal metabolic rate did not differ between lines while diet-induced thermogenesis (DIT) or heat increment appeared significantly increased in R+ males compared to R- males: +84% if expressed as the difference between fed and fasted heat production [Geraert *et al* (1992) *Proc Nutr Soc* 51, 86A]. The energy balance of the females appeared similar, divergence in heat increment reached +133%.

Recent works in mammals suggest that part of the DIT is under sympathetic or  $\beta$ -adrenergic control. In the R+ and R- experimental lines, oral administration of DL-propranolol, a non-specific  $\beta$ -blocking agent (PPL, 5 mg/kg) reduced heat production in R+ birds but not in R-. Central control of DIT has also been investigated using D-fenfluramine, a 5-HT agonist (DFF, 10 mg/kg), well known for its thermogenic and thermolytic properties (table I). Combined stimulation of thermogenesis by fenfluramine and  $\beta$ -adrenergic

blocking effect of propranolol (DFF+PPL) was also studied in females of both lines (table I).

The anorectic effect of DFF seems more important in the R+ line. Indeed, their feed intake was reduced to the level of the R- genotype. While the thermogenic effect of DFF did not differ between lines, propranolol significantly reduced heat production only in R+ hens (-9.5%) without any effect in the R-birds. The divergence in diet-induced thermogenesis between genotypes is partly under sympathetic control, while serotonin (5-HT) is more involved in the regulation of feed intake than thermogenic abilities.

## **V. Genetic-nutrition interactions**

### **Biosynthesis of arachidonic acid (20:4n-6) in the liver of obese Zucker rats.**

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The genetically obese Zucker rat is characterized by a lower level of arachidonic acid (20:4n-6), in hepatic lipids (expressed as a percentage of total fatty acids), than in the lean animal, in agreement with a partial inhibition of  $\Delta 6$  and mainly  $\Delta 5$  microsomal desaturation [Blond *et al* (1989) *Lipids* 24, 389-395; Guesnet *et al* (1990) *Lipids* 25, 517-522]. The aim of this study was to investigate whether treatment with a hypocholesterolemic drug (simvastatin, which stimulates fatty-acid