Metabolic and endocrine changes induced by chronic heat exposure in broiler chickens: biological and endocrinological variables

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The present study was designed to investigate the effect of chronic heat exposure (32° constant) on plasma metabolites and hormone concentrations in broiler chickens. At 2 and 4 weeks of age, fifty-four male Shaver broiler chickens were allocated to one of three treatments: 22°, *ad lib.* feeding (22AL), 32°, *ad lib.* feeding (32AL) and 22°, pair-feeding with the 32AL group (22PF). Ambient temperature was kept constant at either 22 or 32° for 2 weeks. Plasma glucose, triacylglycerols, phospholipids, non-esterified fatty acids (NEFA), individual amino acids, uric acid, insulin, triiodothyronine (T3), thyroxine, corticosterone were determined. Sensitivity to exogenous insulin was also measured at 7 weeks of age. At 4 and 6 weeks of age, i.e. after 2 weeks at high ambient temperature, fasted 32AL chickens displayed similar concentrations of glucose and triacylglycerols to those of 22AL birds. When fed, 32AL chickens exhibited higher plasma levels of glucose and decreased concentrations of NEFA and amino acids. Feed restriction resulted in intermediate values. Concentrations of all plasma free amino acids were decreased under heat exposure except for aspartic acid, glutamic acid and phenylalanine. At 6 weeks of age, plasma T3 was reduced irrespective of the nutritional state, while plasma corticosterone concentrations were increased in 32AL birds compared with 22AL birds. Heat exposure did not change plasma insulin concentration in either fasted or fed chickens. The 32AL chickens displayed significantly reduced sensitivity to exogenous insulin when fasted, but an enhanced response to insulin when fed, compared with both 22° groups. Such endocrinological changes could stimulate lipid accumulation through increased *de novo* lipogenesis, reduced lipolysis and enhanced amino acid catabolism under chronic heat exposure.

Chronic heat exposure: Endocrinological changes: Plasma metabolites

One of the first consequences of chronic heat exposure is a reduction in feed intake. However, when compared with pair-fed birds exposed to thermoneutrality, heat-exposed chickens exhibit lower growth, decreased feed efficiency and enhanced fat deposition (Geraert *et al.* 1993, 1996).

Few authors (Balnave, 1972; Moss & Balnave, 1978) have considered metabolic changes induced by chronic heat exposure in chickens. Indeed, most studies deal with heat-induced hormonal changes and particularly the endocrine control of thermogenesis. Thyroid function has been thoroughly investigated under hot conditions; plasma triiodothyronine (T3) concentrations appear to decrease while thyroxine (T4) concentrations do not change (Klandorf *et al.* 1981; Sinurat *et al.* 1987) or even increase (Moss & Balnave, 1978). Moreover, deiodinase activity is reduced (Rudas & Pethes, 1984; for review, see May, 1989). Surprisingly, whereas growth hormone (GH) stimulates the conversion of T4 to T3,

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Mitchell & Goddard (1990) reported higher plasma GH in heat-exposed growing chickens. The role of thyrotropin-releasing hormone (TRH) as a GH secretagogue has been analysed also in relation to ambient temperature. Heat-exposed birds displayed a higher plasma GH level shortly after TRH administration but they showed a higher acute elimination rate (Herremans *et al.* 1992). Dietary T4 supplementation or T3 administration were not sufficient to improve growth or even feed intake under hot conditions (May, 1989). Involvement of corticosteroids has been investigated under thermal stress conditions, but not in chronically heat-exposed chickens. In heat-stressed broilers, a sharp increase was followed by a rapid decline in plasma corticosterone (Edens, 1978).

The decreased feed efficiency of heat-exposed broilers suggests changes in nutrient digestion or in their metabolic utilization. The digestibility of energy or metabolizable energy value of a complete diet has often been found to be unchanged (Geraert *et al.* 1992) or even increased (Keshavarz & Fuller, 1980) under chronic heat exposure. However, amino acid digestibility is decreased (Wallis & Balnave, 1984; Zuprizal *et al.* 1993). The effects of elevated environmental temperatures on intestinal absorptive function have been further investigated in poultry. Despite a reduced villus size and a lower jejunal weight, jejunal hexose and amino acid uptakes were enhanced in heat-exposed broilers (Mitchell & Carlisle, 1992). Such changes associated with the circulatory modifications, reduced vascularization of the inner organs and increased peripheral circulation (Wolfenson *et al.* 1981), may lead to the absence of effect of chronic heat exposure on overall digestive abilities.

The reduced efficiency of heat-exposed chickens could then be due to changes in metabolic utilization of nutrients. Such an eventuality has not been investigated widely. Several studies have analysed the acid–base balance in order to elucidate the mechanisms involved in the respiratory alkalosis observed under heat stress (El Hadi & Sykes, 1980) and, thus, to design nutritional solutions to alleviate acute heat stress, but metabolic changes induced by chronic heat exposure were not studied. The increased fatness often observed in birds chronically exposed to hot conditions suggests diversion of nutrients towards lipid deposition under such conditions. Because carbohydrates represent the main component of poultry feed, the effects of heat exposure on glucose metabolism and its relationship with insulin secretion need to be investigated.

Thus, the present experiment was performed in order to determine the metabolic and endocrinological changes which could explain the reduced growth of chronically-heat-exposed chickens when compared with pair-fed or *ad lib.*-fed birds reared under thermoneutral conditions.

MATERIALS AND METHODS

Animals and breeding

Conditions of breeding and experimental procedure of *ad lib.* and pair-feeding are detailed in Geraert *et al.* (1996). Eighteen birds were assigned randomly to one of three treatments: 32° *ad lib.* feeding (32AL), 22° *ad lib.* feeding (22AL) and 22° pair-feeding based on the feed intake of the heat-exposed group (22PF). Birds from the 22PF group received in a single portion the amount of feed consumed by the 32AL birds. Chickens were exposed at constant ambient temperature between 2 and 4 or 4 and 6 weeks of age.

Measurements

At 4 and 6 weeks of age, blood samples were collected from eighteen fed birds per treatment. At 2 h before sampling, 22PF chickens received an amount of feed equivalent to that consumed by 32AL birds in 2 h. After 2 d, birds were fasted overnight (16 h) and then re-sampled. Blood samples were taken from the wing vein using heparin as an

Table 1. *Plasma concentrations of glucose (mmol/l), triacylglycerols (g/l), phospholipids (g/l) and non-esterified fatty acids (NEFA; μmol/l) in* ad lib.*-fed heat-exposed (32AL),* ad lib.*-fed control-exposed (22AL) and pair-fed control-exposed (22PF) male chickens at 4 weeks of age**

(Means with their standard errors for eighteen chickens per treatment except for amino acids, ten chickens per treatment)

Treatment...	22AL		22PF		32AL		Statistical significance of difference: *P*
	Mean	SE	Mean	SE	Mean	SE	
Glucose							
Fasted	10·77[a]	0·14	10·82[a]	0·10	10·22[b]	0·13	0·002
Fed	12·28[a]	0·19	12·99[ab]	0·21	13·94[b]	0·56	0·007
Triacylglycerols							
Fasted	0·48	0·03	0·45	0·02	0·51	0·02	0·325
Fed	1·42	0·05	1·68	0·07	1·56	0·14	0·158
Phospholipids							
Fasted	2·32	0·05	2·44	0·06	2·35	0·10	0·507
Fed	2·83	0·07	2·78	0·07	2·80	0·14	0·948
NEFA							
Fasted	509	22·3	511	19·2	464	23·0	0·238
Fed	267[a]	13·8	308[a]	14·3	180[b]	10·6	< 0·001
Uric acid							
Fasted	31·5[a]	2·1	17·8[b]	1·7	32·0[a]	2·0	< 0·001
Fed	69·9	3·2	83·5	3·6	73·9	6·6	0·116
3-Methyl histidine							
Fasted	nd	nd	nd	nd	nd	nd	
Fed	0·30	0·02	0·33	0·03	0·29	0·03	0·428
Total amino acids							
Fasted	nd	nd	nd	nd	nd	nd	
Fed	95·08[a]	2·39	107·09[b]	3·24	72·28[c]	3·81	< 0·001

[a, b, c] Mean values in the same horizontal row with different superscript letters were significantly different ($P < 0·05$).

nd, not determined.

* For details of treatments and procedures, see pp. 206–207.

anticoagulant. The samples were immediately chilled and centrifuged. Plasma was separated and stored at −20° until required for assay.

Chemical analyses

Plasma glucose was determined using a glucose analyser (model 2; Beckman Instruments, Palo Alto, CA, USA). Plasma triacylglycerols, phospholipids and non-esterified fatty acids (NEFA) were measured by enzymic methods (Takayama *et al.* 1977; Okabe *et al.* 1980; Fossati & Prencipe, 1982) using the kits provided by BioMerieux SA (Charbonnières-les-Bains, France). Plasma uric acid was measured according to Fossati *et al.* (1980) using kits provided by Sigma Diagnostics (St Louis, MO, USA). Plasma free amino acids and 3-methyl histidine were extracted with sulphosalicylic acid (Geraert *et al.* 1987) and determined using a Biotronik LC5001 autoanalyser.

Plasma T3 and T4 were determined by radioimmunoassay using a commercial kit (CIS-ORIS Industries, Gif-sur-Yvette, France). Plasma insulin was measured by radio-immunoassay using a guinea-pig antiporcine insulin serum (Ab 27-6) with chicken insulin as standard according to Simon & Rosselin (1978). Plasma corticosterone was measured by radioimmunoassay using rabbit antibodies (Etches, 1976).

Table 2. *Plasma concentrations of glucose (mmol/l), triacylglycerols (g/l), phospholipids (g/l) and non-esterified fatty acids (NEFA; μmol/l) in* ad lib.*-fed heat-exposed (32AL),* ad lib.*-fed control-exposed (22AL) and pair-fed control-exposed (22PF) male chickens at 6 weeks of age**

(Means with their standard errors for eighteen chickens per treatment except for amino acids, ten chickens per treatment)

Treatment...	22AL		22PF		32AL		Statistical significance of difference: *P*
	Mean	SE	Mean	SE	Mean	SE	
Glucose							
Fasted	10·54[a]	0·13	11·41[b]	0·21	10·30[a]	0·18	< 0·001
Fed	12·55[a]	0·08	13·07[ab]	0·18	13·40[b]	0·21	0·003
Triacylglycerols							
Fasted	0·27	0·01	0·25	0·01	0·26	0·02	0·719
Fed	1·28[ab]	0·08	1·35[a]	0·05	1·05[b]	0·09	0·011
Phospholipids							
Fasted	2·08[a]	0·07	2·23[ab]	0·06	2·40[b]	0·11	< 0·001
Fed	2·46[a]	0·11	2·48[a]	0·08	2·92[b]	0·11	0·005
NEFA							
Fasted	595[a]	23·2	638[a]	31·3	483[b]	31·8	< 0·001
Fed	511[a]	31·2	579[a]	27·5	351[b]	23·0	< 0·001
Uric acid							
Fasted	27·1[ab]	1·8	25·0[a]	1·7	35·3[b]	2·9	< 0·001
Fed	73·0	4·9	64·9	3·3	70·4	4·1	0·388
3-Methyl histidine							
Fasted	nd	nd	nd	nd	nd	nd	
Fed	0·34	0·02	0·37	0·03	0·43	0·05	0·22
Total amino acids							
Fasted	nd	nd	nd	nd	nd	nd	
Fed	90·94[a]	1·70	95·79[a]	2·54	75·49[b]	3·13	< 0·001

[a, b] Mean values in the same horizontal row with different superscript letters were significantly different ($P < 0·05$).

nd, not determined.

* For details of treatments and procedures, see pp. 206–207

Sensitivity to exogenous insulin

At 7 weeks of age, forty-eight fed birds were injected intramuscularly (thigh muscle) with saline (9 g NaCl/l) or bovine insulin (Endopancrine 10 monopic; Organon, Saint Denis, France; 20 μg/kg body weight). After 90 min blood samples were taken from the wing vein and plasma glucose measured. After 2 d the same test was performed in chickens fasted overnight.

Statistical analyses

Results are presented as means with their standard errors. Homogeneity of variance between treatments was determined by Bartlett's test, data were analysed using ANOVA and means compared by Tukey's test. All analyses were performed using Systat software (Systat Inc., USA).

RESULTS

Metabolic variables

At 4 weeks of age, fasted 32AL chickens exhibited similar plasma concentrations of triacylglycerols, phospholipids, NEFA and uric acid to those of 22AL birds (Table 1). The only difference was a lower plasma glucose level: 10·22 *v.* 10·77 mmol/l for 32AL and 22AL

Table 3. *Plasma concentrations of free amino acids (mg/l) in* ad lib.*-fed heat-exposed (32AL),* ad lib.*-fed control-exposed (22AL) and pair-fed control-exposed (22PF) male chickens at 4 weeks of age**

(Means with their standard errors for ten chickens per treatment)

Treatment...	22AL		22PF		32AL		Statistical significance of difference: *P*
	Mean	SE	Mean	SE	Mean	SE	
Asp	9·8	1·0	12·8	1·3	13·0	1·4	0·131
Thr	107·4[a]	7·5	80·1[b]	3·3	69·8[b]	6·3	< 0·001
Ser	73·8[a]	2·4	90·0[b]	4·6	59·1[c]	4·2	< 0·001
Asn	42·4[a]	4·6	53·9[a]	4·6	23·7[b]	5·2	0·001
Glu	30·3	1·3	30·8	1·2	32·7	1·9	0·498
Gln	162·1[a]	7·7	185·1[a]	5·2	121·9[b]	8·5	< 0·001
Pro	70·4[a]	3·2	91·7[b]	4·8	61·3[a]	3·3	< 0·001
Gly	44·3[a]	1·4	57·4[b]	2·5	37·2[c]	2·2	< 0·001
Ala	58·0[a]	2·1	72·0[b]	3·9	54·1[a]	4·7	0·005
Val	31·8[a]	1·3	41·3[b]	2·1	25·4[c]	1·4	< 0·001
Cys	22·4[a]	0·7	22·6[a]	1·4	15·2[b]	0·9	< 0·001
Met	9·1[a]	0·4	10·3[a]	0·7	6·0[b]	0·4	< 0·001
Ileu	17·5[a]	0·7	21·9[b]	1·0	12·2[c]	0·8	< 0·001
Leu	42·6[a]	1·5	53·7[b]	2·6	30·9[c]	1·7	< 0·001
Tyr	30·2[a]	1·4	28·2[a]	1·5	18·9[b]	2·4	< 0·001
Phe	24·4	0·8	23·8	0·5	22·5	1·1	0·296
Lys	71·9[a]	4·2	59·9[a]	3·4	34·8[b]	4·2	< 0·001
His	21·1[a]	1·0	26·8[b]	1·2	13·7[c]	1·4	< 0·001
Arg	81·5[a]	4·0	108·8[b]	6·5	76·9[a]	5·0	< 0·001

[a, b, c] Mean values in the same horizontal row with different superscript letters were significantly different ($P < 0·05$).

* For details of treatments and procedures, see pp. 206–207.

respectively ($P = 0·002$). When fed, 32 AL chickens displayed higher plasma glucose levels (13·94 *v.* 12.28 mmol/l), lower plasma NEFA and total free amino acid concentrations than 22AL birds. At 22° feed restriction decreased plasma uric acid concentrations in the fasting state and increased plasma free amino acid contents when fed.

At 6 weeks of age, after 2 weeks of heat exposure, fasted 32AL chickens exhibited similar plasma levels of glucose and triacylglycerols but increased concentrations of phospholipids and uric acid than 22AL birds (Table 2). When fed, heat-exposed chickens displayed higher glucose and phospholipid concentrations, while the plasma levels of NEFA and amino acids were decreased compared with those of 22AL chickens. The 22PF chickens often exhibited intermediate values (Table 2).

The free amino acid profiles are shown in Tables 3 and 4. The decreased total amino acid concentration of 32AL birds compared with that of 22AL birds was due to a decrease in all amino acids except aspartic acid, glutamic acid and phenylalanine at 4 weeks of age. Similar results were obtained at 6 weeks of age, but the decreases in plasma threonine, valine, cystine, lysine and arginine were not significant at $P < 0·05$. Fig. 1(*a* and *b*) shows the difference in the plasma amino acid concentrations of heat-exposed and pair-fed control chickens compared with those of 22AL birds. Feed restriction at thermoneutrality did not reduce plasma total free amino acid concentrations. Some amino acids were even increased, e.g. serine and alanine. Chronic heat exposure significantly decreased S amino acid levels (mg/l plasma): 30·8 for 32AL, 33·1 for 22PF and 33·7 for 22AL at 6 weeks of age. The

Table 4. *Plasma concentrations of free amino acids (mg/l) in* ad lib.*-fed heat-exposed (32AL), ad lib.-fed control-exposed (22AL) and pair-fed control-exposed (22PF) male chickens at 6 weeks of age**

(Means with their standard errors for ten chickens per treatment)

Treatment...	22AL		22PF		32AL		Statistical significance of difference: *P*
	Mean	SE	Mean	SE	Mean	SE	
Asp	10·4	0·9	12·2	1·1	15·1	1·1	0·261
Thr	89·5	6·2	75·8	6·7	73·7	6·0	0·154
Ser	70·0[a]	2·9	93·2[b]	3·3	66·8[a]	3·4	< 0·001
Asn	37·4[ab]	2·5	47·4[a]	2·5	23·7[b]	4·8	0·005
Glu	32·5	1·1	30·6	1·5	34·0	2·7	0·480
Gln	127·3[a]	3·7	125·9[a]	3·1	90·7[b]	5·4	< 0·001
Pro	84·6[ab]	4·0	86·3[a]	3·0	70·7[b]	5·2	0·029
Gly	51·6[ab]	1·5	62·7[a]	2·1	44·9[b]	2·5	< 0·001
Ala	65·8[a]	2·0	81·1[b]	3·5	45·9[c]	3·3	< 0·001
Val	35·3	1·5	34·0	1·4	31·7	1·1	0·167
Cys	24·0	1·0	23·5	1·3	22·6	1·4	0·690
Met	9·7[a]	0·5	9·6[ab]	0·5	8·2[b]	0·4	0·048
Ileu	18·6[a]	0·7	17·5[a]	0·7	14·9[b]	0·7	0·002
Leu	45·7[a]	1·4	44·0[a]	1·7	34·5[b]	1·5	< 0·001
Tyr	28·6[a]	1·5	31·9[a]	2·0	19·0[b]	1·4	< 0·001
Phe	22·3	0·5	19·9	0·4	20·7	1·0	0·063
Lys	54·3	2·7	50·4	3·8	43·0	5·4	0·131
His	21·3[a]	0·9	24·0[a]	0·8	13·5[b]	0·9	< 0·001
Arg	81·2	5·2	87·9	2·5	72·9	3·6	0·062

[a, b, c] Values on the same line with different superscripts were significantly different ($P < 0·05$).

difference was even greater at 4 weeks of age and reached −34% in heat-exposed chickens compared with those maintained at thermoneutrality (Table 3). Branched-chain amino acids were also decreased in 32AL compared with 22AL chickens: 68·5 *v.* 91·9 and 81·1 *v.* 99·6 mg/l plasma at 4 and 6 weeks of age respectively.

Hormones

Fasted and fed 4-week-old 32AL chickens exhibited lower plasma T3 concentrations than 22AL birds (Table 5). Conversely, plasma T4 was increased in the fasting state and unchanged in fed 32AL chickens compared with 22AL birds. Feed restriction had no effect on plasma T4 and T3 irrespective of the nutritional state.

At 6 weeks of age, plasma insulin and T3 were similar in fasted 32AL and 22AL chickens, while T4 was decreased and corticosterone significantly increased in heat-exposed birds (Table 6). In fed 32AL chickens, plasma T3 and T4 were decreased while plasma corticosterone concentration was greatly increased compared with 22AL or 22PF birds. Chronic heat exposure did not affect plasma insulin concentrations of fed birds (Table 6).

Sensitivity to insulin

Plasma glucose concentrations after saline or insulin administration at 7 weeks of age are shown in Table 7. In fasted birds the initial plasma glucose levels were similar irrespective of the temperature or the feeding level at 22°. There was a decrease in glycaemia after insulin administration (5·39, 6·11 and 4·33 mol/l in 22AL, 22PF and 32AL birds respectively). Thus, heat-exposed birds showed a lower response to insulin injection when

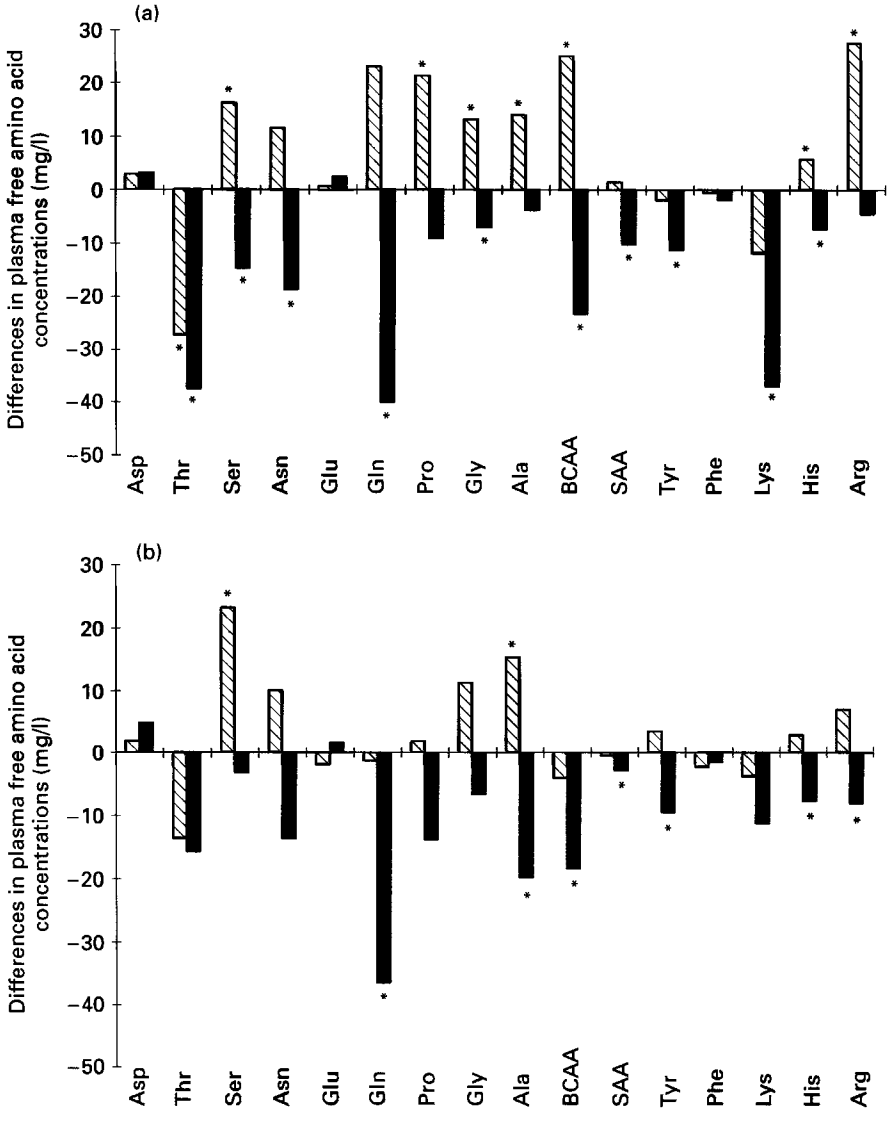

Fig. 1. Differences in plasma free amino acid concentrations between *ad lib.*-fed heat-exposed (32AL) and *ad lib.*-fed control-exposed (22AL) chickens (■), and between pair-fed control-exposed (22PF) and *ad lib.*-fed control-exposed (22AL) chickens (▨) at (*a*) 4 weeks and (*b*) 6 weeks of age. For details of treatments and procedures, see pp. 206–207. BCAA, branched-chain amino acids; SAA, S amino acids. Differences between treatments were significantly different. * $P < 0{\cdot}05$.

fasting. When fed, the initial plasma glucose level was higher in 32AL chickens compared with both 22° groups. The decrease in plasma glucose concentration was less marked than that observed in fasted chickens (2·61, 1·17 and 3·78 mmol/l in 22AL, 22PF and 32AL birds respectively). Chickens exposed to hot conditions thus demonstrated an enhanced response to exogenous insulin when fed.

Table 5. *Plasma concentrations of insulin, triiodothyronine (T3), thyroxine (T4) and corticosterone in* ad lib.*-fed heat-exposed (32AL),* ad lib.*-fed control-exposed (22AL) and pair-fed control-exposed (22PF) male chickens at 4 weeks of age**

(Means with their standard errors for fourteen chickens per treatment)

Treatment...	22AL		22PF		32AL		Statistical significance of difference: *P*
	Mean	SE	Mean	SE	Mean	SE	
Insulin (μU/ml)							
Fasted	nd		nd		nd		
Fed	nd		nd		nd		
T3 (nmol/l)							
Fasted	2·30[a]	0·18	2·22[a]	0·13	1·30[b]	0·28	0·002
Fed	4·77[a]	0·29	4·63[a]	0·19	2·12[b]	0·17	< 0·001
T4 (nmol/l)							
Fasted	25·21[a]	2·01	25·52[a]	1·93	34·48[b]	2·78	0·01
Fed	7·81	0·56	8·07	0·59	7·54	1·06	0·892
Corticosterone (ng/ml)							
Fasted	nd		nd		nd		
Fed	nd		nd		nd		

[a, b] Mean values in the same horizontal row with different superscript letters were significantly different ($P < 0·05$).

nd, not determined.

* For details of treatments and procedures, see pp. 206–207.

Table 6. *Plasma concentrations of insulin, triiodothyronine (T3), thyroxine (T4) and corticosterone in* ad lib.*-fed heat-exposed (32AL),* ad lib.*-fed control-exposed (22AL) and pair-fed control-exposed (22PF) male chickens at 6 weeks of age**

(Means with their standard errors for fourteen chickens per treatment)

Treatment...	22AL		22PF		32AL		Statistical significance of difference: *P*
	Mean	SE	Mean	SE	Mean	SE	
Insulin (μU/ml)							
Fasted	9·10[a]	2·30	22·10[b]	4·28	14·44[a]	3·58	0·04
Fed	45·44	4·35	55·60	5·10	44·10	8·02	0·357
T3 (nmol/l)							
Fasted	1·60	0·10	1·43	0·08	1·44	0·10	0·387
Fed	3·51[a]	0·27	3·59[a]	0·22	1·47[b]	0·12	< 0·001
T4 (nmol/l)							
Fasted	21·61[a]	0·82	18·38[b]	0·86	18·56[b]	0·86	0·016
Fed	13·23[a]	0·50	13·08[a]	0·53	9·64[b]	0·50	< 0·001
Corticosterone (ng/ml)							
Fasted	1·46[a]	0·19	2·16[b]	0·35	2·32[b]	0·37	< 0·001
Fed	0·45[a]	0·07	0·47[a]	0·07	1·86[b]	0·33	< 0·001

[a, b] Mean values in the same horizontal row with different superscript letters were significantly different ($P < 0·05$).

* For details of treatments and procedures, see pp. 206–207.

DISCUSSION

Unlike mammals, heat-exposed chickens show decreased growth, even when compared with pair-fed birds maintained at thermoneutrality (Geraert *et al.* 1996). Moreover, the reduced feed efficiency reported in poultry under hot conditions is not found in growing

Table 7. *Sensitivity to exogenous insulin: plasma glucose (mmol/l) 90 min after intramuscular injection of exogenous insulin (20 μg/kg) or saline (9 g NaCl/l) solutions in* ad lib.*-fed heat-exposed (32AL),* ad lib.*-fed control-exposed (22AL) and pair-fed control-exposed (22PF) 48-d-old male chickens**

(Means with their standard errors for eight chickens per treatment)

Treatment...	22AL		22PF		32AL		Statistical significance of difference: *P*
	Mean	SE	Mean	SE	Mean	SE	
Fed							
Saline	12·67^{bc}	0·22	12·47^{b}	0·40	13·56^{c}	0·43	< 0·001
Insulin	10·09^{a}	0·26	11·43^{b}	0·28	9·83^{a}	0·30	
Fasted							
Saline	10·37^{c}	0·18	10·77^{c}	0·24	10·29^{c}	0·22	< 0·001
Insulin	4·99^{ab}	0·20	4·67^{a}	0·39	5·99^{b}	0·39	

^{a,b,c} Mean values within the same nutritional state with different superscript letters were significantly different ($P < 0·05$).

piglets (Rinaldo & Le Dividich, 1991). Such a reduced growth performance which is independent of feed intake needs to be explained. Furthermore, in chickens, chronic heat exposure enhanced fatness and decreased protein retention (Geraert *et al.* 1996) which suggests changes in nutrient utilization and in the hormonal control of anabolic and catabolic mechanisms.

In birds, the lipids deposited are mainly synthesized by *de novo* lipogenesis from carbohydrates which are the primary source of dietary energy. While limited to single point measurements, plasma glucose determined in either fasted or fed states or after exogenous insulin administration could help to understand glucose utilization. Unlike adult hamsters (Chayoth & Cassuto, 1971*a*), adult rats (Christon *et al.* 1984) and growing piglets (Rinaldo & Le Dividich, 1991), heat-exposed chickens exhibited similar fasting glycaemia compared with 22AL and, conversely, increased plasma glucose concentrations when fed compared with those at thermoneutrality. Such changes could reveal modifications in glucose metabolism. Indeed, Chayoth & Cassuto (1971*b*) report an increase in glucose utilization through glycogen synthesis in heat-exposed hamsters and argued that such a mechanism could lower heat production and help the animal to get rid of the extra energy. The reduced sensitivity to insulin, also observed in rats (Chayoth *et al.* 1984), while not associated with reduced basal glycaemia, could stimulate lipid deposition. Indeed, genetic or VMH-lesioned obese rodents often present a basal insulin resistance (Bray & York, 1979). The reduced peripheral uptake of glucose by the muscles might lead to an enhanced glucose supply to hepatocytes, which increases lipid synthesis. However, sensitivity to exogenous insulin and glucose tolerance tests should be further investigated to understand better the control of glucose utilization.

The enhanced fatness observed under chronic heat exposure (Aïn Baziz *et al.* 1993; Geraert *et al.* 1996) was not associated with increased plasma triacylglycerol concentrations. Indeed, lower triacylglycerolaemia was observed in fed 6-week-old 32AL chickens which might indicate an enhanced uptake of lipids by peripheral adipose tissues. Rinaldo & Le Dividich (1991) found that heat-exposed pigs contained less total body lipids but lipoprotein lipase (*EC* 3.1.1.34) activity was increased in the internal adipose tissue, while there was no change in the activity measured in the subcutaneous and cardiac adipose tissues. Like genetically-fat chickens (Leclercq *et al.* 1984), 6-week-old heat-exposed

chickens exhibited higher plasma phospholipids than control birds. In poultry the major site of lipogenesis is the liver, and HDL, rich in phospholipids, are the main lipoproteins synthesized. Hermier *et al.* (1984) and Leclercq *et al.* (1988) demonstrated significant relationships between plasma phospholipids, HDL secretion and fatness in genetically-fat and lean lines of chickens. Moreover, plasma phospholipids measured at 4 weeks of age were not affected by heat exposure as body lipid deposition was not affected by heat exposure at 4 weeks of age (H. Aïn Baziz, P. A. Geraert, J. C. F. Padilha & S. Guillaumin, unpublished results).

Unlike adult rats and growing pigs (Christon *et al.* 1984; Christon, 1988), heat-exposed birds exhibited a decreased plasma NEFA concentration, suggesting reduced lipid mobilization or increased lipid oxidation. The enhanced fatness under chronic heat exposure in chickens, therefore, might be due more to reduced lipolysis than to increased lipogenesis. Further studies should be undertaken to investigate hepatic lipogenesis, peripheral uptake and recycling.

The reduced protein accretion observed in heat-exposed chickens (Geraert *et al.* 1996) suggests changes in protein synthesis and/or degradation. Few studies have investigated the effect of ambient temperature on protein metabolism. Only low temperatures during the first days of life have been considered in chickens (Aoyagi *et al.* 1988). The enhanced plasma uric acid concentration in fasted heat-exposed chickens compared with 22PF birds suggests increased protein catabolism. This hypothesis is supported by the fact that fed 32AL chickens exhibited similar uric acid and 3-methylhistidine concentrations to those of their pair-fed counterparts, whereas their plasma amino acid concentrations were significantly lower than those of 22AL or 22PF birds. Moreover, all amino acids except aspartic acid, glutamic acid and phenylalanine were reduced in 32AL chickens. McNaughton *et al.* (1978) also observed increased plasma glutamic acid concentrations in heat-exposed chickens at 4 weeks of age. An increase in plasma aspartic acid and glutamic acid concentrations might be the expression of increased amino acid catabolism. These amino acids are the main acceptors of the amino groups by transamination through ketoglutarate and oxaloacetate. Moreover, the changes in plasma amino acid profiles could also have depressive effects on protein synthesis. Another interesting question concerns the animal's needs for the different amino acids under heat exposure, which may differ from those at thermoneutrality. Finally, the effect of heat exposure on S amino acids was greater in young chickens, probably in relation to feather growth.

Heat-induced changes in endocrine profiles might explain the enhanced lipid deposition and reduced protein accretion observed in growing chickens. A specific effect of heat on thyroid hormones was observed, which was independent of feed intake. There was a significant reduction in plasma T3 concentrations while plasma T4 concentrations did not decrease as much or even remained unchanged. Reduced deiodinase activity (Mitchell & Goddard, 1990) could explain such a discrepancy. The elevated level of total T4 in 4-week-old heat-exposed birds was reported also by Moss & Balnave (1978) and could be related to a reduction in its utilization. In older heat-exposed chickens, plasma T4 concentrations decreased in the fed state. There is evidence that altered thyroid hormone metabolism may play a role in enhanced fatness in birds (Leclercq *et al.* 1988) and in the development of obesity in mammals (Bray & York, 1979). Genetically-fat chickens exhibited similar or even higher plasma T4 and lower plasma T3 concentrations. Using dietary T3 supplementation, Leclercq *et al.* (1988) estimated that 17% of the difference in fatness between genotypes could be due to difference in plasma T3 titres.

Corticosteroid administration has been shown to decrease body-weight gain, to increase protein catabolism and to enhance lipid deposition (Decuypere & Buyse, 1988). Sensitivity to insulin was decreased also in corticosterone-treated chickens (Taouis *et al.* 1993). The

high plasma level of corticosterone as well as the reduced plasma T3 concentrations could stimulate diversion of nutrients towards lipid deposition. The increased fatness induced by corticosterone administration in chickens was associated also with a reduction of plasma NEFA concentrations, as observed in heat-exposed chickens.

Chronic heat exposure in chickens resulted in a significant modification of the hormonal control of metabolism. Indeed, reduced plasma T3, enhanced plasma corticosterone and basal insulin resistance could contribute to the diversion of nutrients towards lipid deposition. However, such enhanced lipid accretion would not necessarily be due to increased lipogenesis as plasma triacylglycerol concentrations were lower. Enhanced uptake through increased lipoprotein lipase activity or decreased lipid turnover could explain the greater fatness. Finally, the endocrine changes could also sustain changes in protein metabolism under heat exposure. Reduced protein deposition might result from reduced protein synthesis or enhanced catabolic rate. Further investigations are required into the metabolic pathways involved either in lipid or protein metabolism.

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