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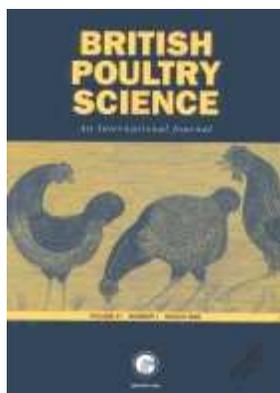
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**Thermoregulatory capacity and muscle membrane integrity are  
compromised in broilers compared with layers at the same age or  
body weight**

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**RUNNING TITLE: Thermoregulation in chicks**

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4 20 **Abstract** 1. The effects of acute heat stress (2 h at 32°C and 75% RH) on body temperature  
5  
6 21 and indices of respiratory thermoregulation and skeletal muscle function were examined in two  
7  
8 22 divergently selected male grandparent lines of broiler and layer-type chickens at two ages (35 and  
9  
10 23 63 d), or at a similar body weight (~2.2 kg).

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12  
13 24 2. The two chicken lines exhibited markedly different base-line blood acid-base and skeletal  
14  
15 25 muscle characteristics. At the same age or liveweight, birds from the broiler line had significantly  
16  
17 26 higher venous blood carbon dioxide tensions associated with lower blood pH. Plasma creatine  
18  
19 27 kinase (CK) activities reflecting muscle membrane damage were also greatly elevated in the  
20  
21 28 broiler line.

22  
23  
24 29 3. Exposure to acute heat stress caused an increase in deep body temperature, panting-induced  
25  
26 30 acid-base disturbances and elevated plasma CK activity in both lines of chicken, an effect that  
27  
28 31 increased with age. The extent of disturbances in acid/base regulation and heat stress-induced  
29  
30 32 myopathy were more pronounced in the broiler than the layer line at the same age or similar  
31  
32 33 liveweights.

33  
34 34 4. It is suggested that genetic selection for high muscle growth in broiler lines has  
35  
36 35 compromised their capacity to respond to an acute thermal challenge, leading to detrimental  
37  
38 36 consequences for muscle function. This reduction in heat tolerance may have important  
39  
40 37 implications for bird welfare and subsequent meat quality.

## 41 38 **INTRODUCTION**

42  
43  
44 39 Programmes of genetic selection in poultry for economically important production traits such as  
45  
46 40 high growth rate, food conversion efficiency and muscle yield have been extremely successful.  
47  
48 41 Intensive commercial selection in broiler chickens aimed at improving weight gain and meat  
49  
50 42 yields have resulted in birds that grow up to 4 times faster than layer strains and exhibit 8-fold  
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3 43 increases in breast muscle growth rate (Griffin and Goddard, 1994). Whilst genetic selection in  
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5 44 broiler chickens has made major advances in the development of production traits, it is  
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7  
8 45 increasingly recognised that these may also be associated with a number of undesirable patho-  
9  
10 46 physiological metabolic derangements (Scheele, 1997). These include respiratory and cardio-  
11  
12 47 pulmonary disorders (Julian, 1993, 1996) and skeletal disease (Thorp, 1994). Selection for high  
13  
14 48 meat yields coupled with rapid growth rates in chickens have also led to an increase in the  
15  
16 49 incidence of pathological features observed in skeletal muscle. Degenerative changes include  
17  
18 50 focal fibre necrosis, hypercontraction, mononuclear cell infiltration, alterations in cell membrane  
19  
20 51 integrity and the loss of myo-cellular constituents (Siller, 1985; Mitchell and Sandercock, 1996;  
21  
22 52 Soike and Bergmann, 1998; Mahon 1999; Cooke *et al.*, 2003). Sandercock and Mitchell (2003,  
23  
24 53 2004) published evidence that such growth-associated or stress-induced myopathies may result  
25  
26 54 from disruption of intracellular calcium homeostasis in muscles of rapidly growing broiler birds  
27  
28 55 exhibiting high metabolic rates (Mitchell, 1999). It has been proposed that the increased  
29  
30 56 incidence of such metabolic disorders in broiler chickens is due to an imbalance between the  
31  
32 57 production and supply of energy and metabolites for maintenance requirements (Savory, 1995;  
33  
34 58 Scheele, 1997). The resulting homeostatic dysregulation then leads to cellular, tissue and organ  
35  
36 59 dysfunction.

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43 60 During the course of production, poultry may be exposed to stressful environmental  
44  
45 61 challenges. Chronic thermal stress (high or low environmental temperatures) has been shown to  
46  
47 62 have a detrimental effect upon production efficiency and mortality (Washburn, 1985). Episodes  
48  
49 63 of acute heat or cold stress in broilers may be experienced under commercial production and  
50  
51 64 transport conditions (Webster *et al.*, 1993; Mitchell *et al.*, 1992, 1997; Mitchell and Kettlewell  
52  
53 65 1998). In the case of heat stress, elevated deep body temperature is accompanied by panting in  
54  
55 66 order to increase respiratory evaporative heat loss (Hillman *et al.*, 1985). Panting induces a

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3 67 hypocapnic alkalosis due the increased elimination of carbon dioxide resulting from elevated  
4  
5 68 respiratory minute volume (Wideman *et al.*, 2003; Borges *et al.*, 2004). Acute hyperthermia and  
6  
7  
8 69 respiratory alkalosis are in turn associated with a heat-stress-induced myopathy, a pathology that  
9  
10  
11 70 may underlie subsequent problems with meat quality (Sandercock *et al.*, 2001). Such skeletal  
12  
13 71 muscle damage "in transit" associated with hyperthermia, and reflected in measurement of  
14  
15 72 plasma creatine kinase (CK) activities, has been described in broiler chickens exposed to acute  
16  
17 73 heat stress under both actual and simulated commercial transport conditions (Mitchell *et al.*,  
18  
19  
20 74 1992; Mitchell and Sandercock, 1995 a, b; Sandercock *et al.*, 2001).

21  
22 75 Tolerance of short or long term elevated thermal loads is greater in more traditional and  
23  
24 76 less selected chicken breeds than commercial intensively selected broiler lines (Arad and Marder,  
25  
26  
27 77 1982 a, b, c; Berrong and Washburn, 1998; Zulkifli *et al.*, 1999). A reduced resistance to heat  
28  
29 78 stress in broiler birds may be attributable to a decreased ability to lose heat (MacLeod and  
30  
31  
32 79 Hocking, 1993) or an inappropriately increased heat production during exposure to high thermal  
33  
34 80 loads (Sandercock *et al.*, 1995). Comparative studies that characterise the responses to thermal  
35  
36 81 challenge in broiler chickens and in lines not selected for enhanced growth and feed conversion  
37  
38  
39 82 efficiency at the same age may be confounded by disparities in body size and geometry  
40  
41  
42 83 (Monteith, 1973). To overcome some of the difficulties this presents in interpretation,  
43  
44 84 comparisons can be made at "matched" liveweights. While it is acknowledged that this approach  
45  
46 85 can also present problems in interpretation due to age-dependent developmental differences, it  
47  
48  
49 86 does provide a more acceptable indication of the severity of a thermal challenge between  
50  
51  
52 87 different lines (Zulkifli *et al.*, 1999).

53 88 Elevated plasma CK activity is recognised as a reliable diagnostic marker of muscle  
54  
55 89 damage (Hamburg *et al.*, 1991; Mitchell and Sandercock 1995 a, b). It is indicative of disruptions  
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57  
58 90 in muscle cell membrane (sarcolemma) function and permeability (Mitchell 1999). Previous  
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3 91 studies have demonstrated a strong relationship between the extent of histologically demonstrable  
4  
5 92 muscle fibre pathology and plasma CK activity (Soike and Bergmann 1998; Cooke *et al.* 2003).  
6  
7  
8 93 Thus, baseline absolute CK activity reflects the extent of any idiopathic myopathy present  
9  
10 94 whereas changes following an environmental challenge indicate the degree of stress induced  
11  
12 95 muscle damage.

13  
14  
15 96 The objectives of the present study were to characterise the effects of acute heat stress (HS)  
16  
17 97 on physiological indices of thermoregulatory capacity and muscle membrane integrity in  
18  
19 98 divergently selected pure male lines of chickens (meat and layer type) at the same age and very  
20  
21 99 different body weights or at two different ages but the same body weight. The birds were  
22  
23 100 subjected to a thermal load whose duration and magnitude was representative of that known to  
24  
25 101 induce symptoms of heat stress in chickens during commercial transportation (Mitchell and  
26  
27 102 Kettlewell, 1998). Blood  $p\text{CO}_2$  and pH were measured to assess the extent of respiratory  
28  
29 103 thermoregulatory effort and plasma CK activity was used as a tissue specific indicator of skeletal  
30  
31 104 muscle damage.

## 32 33 34 35 36 105 **MATERIALS AND METHODS**

### 37 38 39 106 **Animals and husbandry**

40  
41 107 The birds used in the study were pedigreed male chicks from a male great grandparent broiler  
42  
43 108 (B), and White Leghorn layer (L) line obtained from two commercial breeding companies.  
44  
45 109 Ninety-six birds of both lines were reared together from 1-d-old in 12 pens (3.6 m<sup>2</sup>) on wood  
46  
47 110 shaving litter (8 birds per line per pen). Room temperatures of 18-20°C were maintained by  
48  
49 111 controlled ventilation and heating. Birds were provided with *ad libitum* access to a commercial  
50  
51 112 broiler diet and water and were subjected to a 14h light: 10h dark photoperiod for the duration of  
52  
53 113 the experiment. All birds were beak trimmed at 14 d of age and light intensities reduced to < 5  
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3 114 lux to minimise possible outbreaks of bird-to-bird pecking (Kjaer and Vestergaard, 1999). A  
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5 115 second batch of 1-d-old broiler chicks (n = 48) was obtained 10 weeks after the arrival of the  
6  
7  
8 116 initial consignment of White Leghorn birds to allow for the comparison of the two lines at the  
9  
10 117 same liveweight. These birds were reared as previously described and comparisons of the birds'  
11  
12 118 responses to acute HS at approximately the same liveweight were carried out in the B- and L-line  
13  
14 119 respectively at 35 and 105 d of age. All experiments were performed with authorisation of the  
15  
16  
17 120 UK Home Office and with the approval of the Roslin Institute Ethical Committee.

### 121 **Heat stress protocol**

122 At 35 and 63 d of age (same age) and at 35 (B) and 105 (L) d (same liveweight), 24 birds from  
123 both lines were transferred into 4 or 6 commercial transport containers (0.7 × 0.5 × 0.3 m). Half  
124 the containers were placed in a controlled climate chamber at 32°C/75% relative humidity (RH)  
125 for a period of 2 h. The remaining containers were placed in an adjacent identical climate  
126 chamber at 21°C/50% RH (thermoneutral) for the same duration. The experiment was repeated  
127 with 12 L at 105 d of age when they were of comparable body weight to the second batch of 12 B  
128 at 35d. Feed was withdrawn from the birds 4 prior to the heat stress experiments

129 Rectal temperatures and blood samples were taken from all birds immediately before ( $T_0$ )  
130 and after ( $T_1$ ) the heat stress exposure period using a thermistor probe (Model 612-849; RS  
131 Components Ltd., Northants, UK) inserted 5 cm into the bird's rectum. Blood samples were  
132 obtained at the same time as rectal temperature by superficial venepuncture (brachial vein) and  
133 transferred into 5-ml blood collection tubes containing Li-heparin anti-coagulant (50 IU ml<sup>-1</sup>) and  
134 immediately chilled on ice. Blood  $p\text{CO}_2$  and pH were measured within 2 minutes of sample  
135 collection using an automated blood gas analyser (Model 238 pH/Blood Gas Analyser, CIBA-  
136 Corning, Halstead, UK) with body temperature compensation. Plasma samples for CK

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3 137 determination were obtained by centrifugation of whole blood at 1500 g for 5 minutes and stored  
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6 138 at  $-20^{\circ}\text{C}$  pending enzyme analysis. The activity of CK was assessed using a commercial kit  
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8 139 (Biotrol CK Monoreactif, Alpha Laboratories, Hants, UK) modified for use with a multi-well  
9  
10 140 plate spectrophotometer (MR 5000, Dynatech Laboratories, West Sussex UK) as previously  
11  
12 141 described (Mitchell *et al.*, 1999).

### 142 **Statistical analyses**

143 The first experiment (comparing two lines at two ages) was a  $2^4$  factorial design (Line  $\times$   
144 Treatment  $\times$  Time  $\times$  Age). Blocking factors were pen within crate and bird as a split-plot effect.  
145 The second experiment (comparing two lines at the same body weight) was a  $2^3$  factorial design  
146 (Line  $\times$  Treatment  $\times$  Time) with the same blocking factors. Standard analysis of variance  
147 methods were used to assess the treatment effects using Genstat ([http://www.vsn-](http://www.vsn-intl.com/genstat/)  
148 [intl.com/genstat/](http://www.vsn-intl.com/genstat/)). For analysis at the same age,  $p_v\text{CO}_2$  and plasma CK were transformed by  
149 taking natural logarithms to obtain residual errors that were normally distributed.

## 150 **RESULTS**

151 The thermal conditions experienced by birds of the two lines were similar at the two ages for both  
152 control (C) and acute heat stress (HS) treatments. Mean ambient temperatures ( $^{\circ}\text{C}$ ) and RH  
153 respectively were  $22.5^{\circ}\text{C}/29.1\%$  RH at 35 d;  $21.3^{\circ}\text{C}/30.5\%$  RH at 63 d for C and  $32.5^{\circ}\text{C}/67.1$   
154  $\%$  RH at 35 d;  $32.1^{\circ}\text{C}/75.5\%$  RH at 63 d for HS. For birds compared at the same liveweight,  
155 mean values for C and HS treatments respectively were  $21.9^{\circ}\text{C}/29\%$  RH and  $32.3^{\circ}\text{C}/71.3\%$  RH.

### 156 **Comparisons at the same age**

157 Differences in liveweight were observed between the two lines at the two ages ( $P<0.001$ ). Mean  
158 body weights of B at 35 and 63 d, respectively, were 2.27 and 5.58 kg compared with 0.41 and  
159 0.98 kg in L (sed 0.027). Both lines exhibited significant ( $P<0.001$ ) increases in liveweight with

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3 160 age of 2.5 and a 2.4-fold respectively from 35 to 63 d of age in the B- and L-line. There were no  
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5 161 differences in liveweight between the control and HS groups in any of the treatment comparisons.  
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8 162 Mean trait values before and after HS in control and treated birds of both lines at the two  
9  
10 163 ages are presented in Table 1.

Table 1 near here

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13 164 *Rectal temperature*

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15 165 The change in rectal temperature in response to HS was significantly greater in the B-line  
16  
17 166 compared to the L-line leading to a Line  $\times$  Treatment  $\times$  Time interaction ( $P < 0.001$ ). In the B-line,  
18  
19 167 mean HS-induced changes in body temperature were +1.7 °C greater than in controls at the same  
20  
21 168 age, at both ages compared with 0.5 and 0.2 °C respectively at 35 and 63 d in the L-line (Table  
22  
23 169 1). Average rectal temperature was greater at 63 than 35 d ( $P < 0.001$ ).  
24  
25  
26

27 170 *Venous blood carbon dioxide ( $p_vCO_2$ )*

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29  
30 171 The 4-way interaction of Line  $\times$  Age  $\times$  Treatment  $\times$  Time was highly significant ( $P < 0.001$ ) for  
31  
32 172  $p_vCO_2$ . Basal  $p_vCO_2$  was greater ( $P < 0.001$ ) in the B-line compared to the L-line at 35 and 63 d.  
33  
34 173 Changes in  $p_vCO_2$  in response to HS challenge in the B-line at both ages, and in L-line at 63 d  
35  
36 174 were significantly ( $P < 0.001$ ) lower than in the controls (Table 1).  
37  
38

39 175 *Venous blood pH ( $pH_v$ )*

40  
41 176 The 4-way interaction of Line  $\times$  Age  $\times$  Treatment  $\times$  Time was significant ( $P < 0.01$ ) for  $pH_v$ . Base-  
42  
43 177 line  $pH_v$  was similar in B- and L-lines at 63 and 35 d of age. The increase in  $pH_v$  following  
44  
45 178 exposure to HS was greater ( $P < 0.001$ ) in the B- compared with L-line birds at both ages and in  
46  
47 179 the L-line birds at 63 ( $P < 0.001$ ) compared with 35 d (not significant).  
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51 180 *Plasma creatine kinase (CK) activity*  
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3 181 Plasma CK activities were significantly higher in B- compared with L-line before and after HS at  
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6 182 both ages (Line  $\times$  Age  $\times$  Time interaction  $P<0.01$ ). Exposure to HS at 35 and 63 d respectively  
7  
8 183 was associated with increases of 28 and 25% in CK in B-line and 12% and 24% in L-line birds.

#### 10 184 **Comparisons at the same liveweight**

11  
12 185 Body weights of B were slightly greater than L (2.17 vs. 1.93, sed 0.036 kg;  $P<0.001$ ) at the time  
13  
14  
15 186 of the experiment when they were respectively 35 and 105 d old. Responses to HS in the two  
16  
17 187 lines are presented in Table 2.

Table 2 near here

#### 20 188 *Rectal temperature*

21  
22 189 Acute heat stress increased rectal temperatures in both lines ( $P<0.001$ ). The extent of the change  
23  
24 190 was significantly greater in B-line than in the L-line birds, reflecting a mean increase in rectal  
25  
26  
27 191 temperature compared with controls of +1.55 °C in the B-line and +1.16 °C in the L-line  
28  
29 192 (Treatment  $\times$  Line  $\times$  Time interaction  $P<0.05$ ).

#### 32 193 *Venous blood carbon dioxide ( $p_vCO_2$ )*

33  
34 194 The Treatment  $\times$  Line  $\times$  Time interaction for  $p_vCO_2$  was significant ( $P<0.001$ ). Baseline  $p_vCO_2$   
35  
36 195 measurements were greater in B-line and increased in the control whereas  $p_vCO_2$  declined in L-  
37  
38 196 line control and in both lines subjected to HS. Exposure to acute heat stress reduced blood  $p_vCO_2$   
39  
40 197 in both lines and the extent of the reduction was much greater in the B-line compared to the L-  
41  
42 198 line.

#### 46 199 *Venous blood pH ( $pH_v$ )*

47  
48 200 Heat stress induced a greater increase in  $pH_v$  in the broilers than in the layers (Treatment  $\times$  Line  
49  
50 201 interaction  $P<0.05$ ) and the change was greater under HS than control conditions (Treatment  $\times$   
51  
52 202 Time  $P<0.001$ ).

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3 204 *Plasma creatine kinase (CK) activity*  
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5 205 Plasma CK activities were significantly ( $P<0.001$ ) different between the two lines at the same  
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8 206 live weight with the B-line birds exhibiting an average 1.8-fold greater plasma CK activity than  
9  
10 207 the L-line. Hyperthermia-associated CK activity was 19 and 18 % higher respectively in the B-  
11  
12 208 line and L-line compared with the controls (Treatment  $\times$  Time interaction  $P<0.001$ )  
13

14  
15 209 **DISCUSSION**  
16

17 210 Exposure to acute heat stress of the magnitude and duration used in this study produced a  
18  
19 211 significant hyperthermia in both lines at both ages. The extent of the hyperthermia was much  
20  
21 212 greater in the more rapidly growing B-line than the L-line, irrespective of age or liveweight. The  
22  
23 213 disparities in extent of the increase in deep body temperature at the same age are most likely to be  
24  
25 214 attributable to differences in body size and geometry and their effects upon heat exchange  
26  
27 215 (Monteith, 1973; Hillman *et al.*, 1985). These findings are consistent with a previous report in  
28  
29 216 which slower growing “dwarf line” broilers exhibited greater heat stress resistance than more  
30  
31 217 rapidly growing lines, a characteristic that was attributed to differences in body size (Deeb and  
32  
33 218 Cahaner 2001). In the present study in birds of the same liveweight, exposure to HS induced a  
34  
35 219 greater degree of hyperthermia in the B-line compared to the L-line. The differences in the degree  
36  
37 220 of induced hyperthermia may reflect variations in thermotolerance in the two lines, possibly  
38  
39 221 attributable to differing efficiencies of heat loss mechanisms (Sandercock *et al.*, 1995). Deep  
40  
41 222 body temperature represents the balance between heat production and heat loss (Monteith, 1973;  
42  
43 223 Hillman *et al.*, 1985) and changes when one exceeds the other, such as during acute heat stress  
44  
45 224 (Lin *et al.*, 2004). Relative differences in hyperthermia or “heat storage” may result from a  
46  
47 225 reduced capacity for heat loss, an increase in heat production, or a combination of both.  
48  
49 226 Temperature regulation by panting carries a high metabolic cost, which has been shown to  
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3 227 increase heat production and may have an influence upon thermoregulatory efficiency in birds  
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5 228 (Whittow, 1976). It has previously been demonstrated that modern broiler birds have a relatively  
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8 229 high body-weight corrected heat production associated with hyperthermia and panting compared  
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11 230 to their genetic predecessors (Sandercock *et al.*, 1995).

12  
13 231 It has long been established that profound respiratory alkalosis is induced in broiler  
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15 232 chickens by heat stress (Hillman *et al.*, 1985; Teeter *et al.*, 1985). Examination of the effects of  
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17 233 HS on these indices revealed differences in the extent of panting-induced hypocapnic alkalosis  
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19  
20 234 that were negatively correlated with differential rectal temperatures in the two lines at the same  
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22 235 age. These findings may be explained, at least in part, by the influence of body size and geometry  
23  
24 236 upon heat transfer and therefore core body temperature disturbances and the resultant  
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26  
27 237 thermoregulatory demands. In the body-weight matched birds there was a marked difference in  
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29 238 the extent of the HS-induced acid-base and blood gas disturbances. These results indicate that the  
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31 239 thermoregulatory respiratory effort was much greater in the broiler line, resulting in elevations in  
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34 240 total respiratory ventilation rates. From the measurements made in this study it is not possible to  
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36 241 determine precisely the contributions of higher respiratory frequencies and greater relative tidal  
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38  
39 242 volumes in the B-line. The key finding is that the broiler line experiences a greater degree of  
40  
41 243 hyperthermia despite an apparently increased thermoregulatory effort in terms of panting when  
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43 244 compared to the layer line even at the same body weight. We hypothesise that the heat loss  
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45 245 mechanism is less efficient in the broiler birds than in the layers, or, is associated with a higher  
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47  
48 246 heat production that leads to increased heat storage, acute hyperthermia and more marked heat  
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50  
51 247 stress.

52  
53 248 Interestingly, the two lines used in this study also exhibited markedly different baseline  
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55 249 blood  $p_v\text{CO}_2$ . The different magnitude of the decline in  $p_v\text{CO}_2$  between the lines may be  
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57  
58 250 attributable to differences in body weight specific metabolic rate. This would be consistent with  
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3 251 the findings of Korte *et al.*, (1999), who reported that selection for rapid growth and a high  
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5 252 metabolic rate in broilers results in a high basal  $p_v\text{CO}_2$ . Alternatively broilers may have an  
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8 253 inadequate control of  $\text{CO}_2$  elimination and regulation of blood gas and acid-base status. Olkowski  
9  
10 254 *et al.*, (1999) reported that  $p_v\text{CO}_2$  was higher in broiler birds than age matched (35 d) White  
11  
12 255 Leghorn chickens, that  $p_v\text{CO}_2$  increased with age in the broilers and was accompanied by a  
13  
14 256 tendency towards increasing acidosis. Scheele *et al.*, (2005) have recently described elevated  
15  
16 257  $p_v\text{CO}_2$  and lower  $\text{pH}_v$  in native breeds compared to modern selected lines and found a positive  
17  
18 258 correlation between a predisposition to hypercapnic acidosis and the incidence of ascites  
19  
20 259 syndrome precursors in young birds. Examination of baseline  $\text{pH}_v$  values in the different lines in  
21  
22 260 the current study revealed similar mean values for  $\text{pH}_v$  ( $7.38 \pm 0.03$ ) in the L-line at 35, 63 and 105  
23  
24 261 d and 35 d in B-line, despite line disparities in baseline  $p_v\text{CO}_2$ . The generally higher blood  $p\text{CO}_2$   
25  
26 262 and a trend towards lower  $\text{pH}$  in rapidly growing broilers may predispose the birds to a number of  
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28 263 other pathologies including the ascites syndrome (Olkowski *et al.*, 1999; Scheele *et al.*, 2003;  
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30 264 Wideman *et al.*, 2003).

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36 265 Differences in plasma CK activities resulting from efflux of the enzyme from muscle in  
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38 266 birds of different sizes or lines are unlikely to be attributable to different volumes of distribution  
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40 267 as similar mean body-weight specific plasma volumes have been reported in both broiler and  
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42 268 layer-type chickens (at ages used in this study), constituting approximately 5-6% liveweight  
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44 269 (Sturkie, 1976; Yahav *et al.*, 1997).

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48 270 At matched ages and body weights the B-line exhibited much more extensive idiopathic  
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50 271 myopathy than the L-line, the baseline CK activities averaging 2.4 and 3.4 fold higher in the  
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52 272 broilers respectively at 35 and 63 d of age and 1.8 fold greater at similar weights. It has been  
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54 273 proposed that this pathology is a direct consequence of selection for high growth rate from  
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56 274 comparisons of fast and slow growing lines of broilers and turkeys (Mitchell and Sandercock  
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3 275 1994, 1995b; Hocking *et al.*, 1998; Sandercock *et al.*, 2001). Soike and Bergmann (1998) have  
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6 276 suggested also that selection for high growth rate and muscle yield have adverse effects upon  
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8 277 structural, metabolic and functional parameters in poultry skeletal muscle. In the current age-  
9  
10 278 matched comparisons the proportional increase in plasma CK activity following HS was higher in  
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12 279 the B-line than in the L-line at 35 d and was similar at 63 d of age. The post-stress absolute  
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14 280 activities, however, at 35 and 63 d respectively were 3.6 and 2.7 fold greater compared with pre-  
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16 281 stress values in the B- than in the L-line. In the body weight matched groups the heat stress  
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18 282 induced proportional increase in plasma CK activity was similar in the two lines whereas the  
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20 283 absolute values were about 2-fold greater in the B- compared with the L-line. It is concluded that  
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22 284 acute heat stress induces further muscle damage in rapidly growing broiler chickens, which  
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24 285 already exhibit a substantial degree of spontaneous or idiopathic myopathy. An increased  
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26 286 susceptibility to heat stress-induced myopathy in rapidly growing broiler lines has been proposed  
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28 287 previously (Mitchell and Sandercock, 1995b).  
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34 288 In summary, we have shown that there are major differences in thermoregulatory and  
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36 289 respiratory responses to heat stress in lines of domestic fowl selected for either greater  
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38 290 reproductive or meat traits. The two lines differ in their ability to cope with exposure to an acute  
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40 291 thermal challenge. The present study showed that the fast-growing commercial broiler lines were  
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42 292 less able to withstand an acute heat stress challenge than a slower growing line at the same  
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44 293 liveweight. The broilers developed a more profound hypocapnic alkalosis during thermal stress  
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46 294 despite exhibiting higher resting  $p_v\text{CO}_2$  and a lower blood  $pH_v$  than the L-lines at thermoneutral  
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48 295 temperatures. Base-line variation in plasma CK activities suggests that disruption of muscle  
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50 296 sarcolemmal integrity is much greater in the broiler line and that their skeletal muscle exhibits an  
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52 297 enhanced sensitivity to acute thermal challenge associated with a greater degree of hyperthermia  
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54 298 and disturbances in acid-base and blood gas status. It is proposed that genetic selection for  
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3 299 improved growth and muscle yields in broiler chickens has had a detrimental effect upon  
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6 300 thermotolerance and deleterious consequences for muscle function. This may have implications  
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8 301 both for meat quality and bird welfare by limiting the capacity of broiler chickens to respond to  
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10 302 an acute thermal challenge.  
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**Table 1.** Mean rectal temperature, venous blood  $p\text{CO}_2$  and pH, and plasma creatine kinase activity (CK) in divergently selected male-line broiler (B) and White Leghorn layer (L) lines of chickens under control (C) and acute heat stress (HS) conditions at two different ages (35 and 63 d). T0 is pre-treatment sample and T1 post-treatment sample.

Trait	Treatment	35 d				63 d				Sed <sup>1</sup>	Sed <sup>2</sup>
		Broiler		Layer		Broiler		Layer			
		T0	T1	T0	T1	T0	T1	T0	T1		
Rectal temperature (°C)	C	41.3	41.9	41.2	41.5	41.3	42.1	41.5	41.9	0.15	0.16
	HS	41.3	43.6	41.3	42.0	41.3	43.8	41.4	42.1		
Plasma $p_v\text{CO}_2$ (ln mm Hg)	C	3.79	3.83	3.58	3.49	3.89	3.50	3.52	3.43	0.050	0.052
	HS	<i>44.2<sup>3</sup></i>	<i>45.9</i>	<i>35.8</i>	<i>32.6</i>	<i>48.9</i>	<i>33.1</i>	<i>33.8</i>	<i>30.8</i>		
Plasma pH <sub>v</sub>	C	7.39	7.38	7.38	7.36	7.30	7.45	7.37	7.43	0.018	0.019
	HS	7.32	7.51	7.38	7.39	7.35	7.56	7.39	7.51		
Plasma CK (ln IU l <sup>-1</sup> )	C	6.34	6.76	5.47	5.61	6.84	6.98	5.63	5.99	0.056	0.057
	HS	<i>567<sup>3</sup></i>	<i>860</i>	<i>236</i>	<i>274</i>	<i>932</i>	<i>1073</i>	<i>278</i>	<i>399</i>		
		<i>6.65</i>	<i>7.00</i>	<i>5.45</i>	<i>5.73</i>	<i>6.88</i>	<i>7.20</i>	<i>5.64</i>	<i>6.21</i>		
		<i>774<sup>3</sup></i>	<i>1099</i>	<i>232</i>	<i>307</i>	<i>972</i>	<i>1339</i>	<i>280</i>	<i>496</i>		

488 <sup>1</sup> Standard error of a difference between means across rows (Line, Time and Age).

489 <sup>2</sup> Standard error of a difference between two means within a column (C vs. HS).

490 <sup>3</sup> Back transformed values in italics.

**Table 2.** Mean rectal temperature, venous blood  $p\text{CO}_2$  and pH, and plasma creatine kinase activity (CK) in a divergently selected male broiler and layer lines under control (C) and acute heat stress (HS) conditions at the same approximate live weight (~2.2 kg). T0 is pre-treatment sample and T1 post-treatment sample

Trait	Treatment	Broiler (35 d)		Layer (105 d)		Sed <sup>1</sup>	Sed <sup>2</sup>
		T0	T1	T0	T1		
Rectal temperature (°C)	C	41.5	41.7	41.5	41.8	0.11	0.10
	HS	41.3	43.2	41.4	42.9		
Plasma $p_v\text{CO}_2$ (mm Hg)	C	40.4	44.8	37.6	33.1	1.47	1.49
	HS	44.1	27.9	37.0	27.9		
Plasma pH <sub>v</sub>	C	7.37	7.41	7.39	7.41	0.016	0.017
	HS	7.35	7.51	7.38	7.46		
Plasma CK (IU l <sup>-1</sup> )	C	782	878	428	503	45.2	32.6
	HS	798	1046	437	597		

<sup>1</sup> Standard error of a difference between means across rows (Line vs. Time).

<sup>2</sup> Standard error of a difference between two means within a column (C vs. HS).