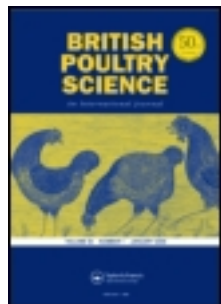


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Wetting of broilers during cold weather transport: A major source of physiological stress?

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demonstrate a reduction in sperm transfer in low mating ratio groups of all ages.

The above results indicate the greater discriminatory power of estimating IPVL-holes or OPVL-sperm compared to percent fertile eggs. The perivitelline assay can also demonstrate differences between the breeding efficiency of flocks under varying management conditions in which there is no apparent difference in fertile eggs but which may manifest differences in fertility when the flock ages or is stressed in some way. The data of the present study and those of other flocks studied was used to develop a prediction equation (%fertility = $6.7 \text{ Ln. GD-IPVL-holes} + 59$; $R^2 = 0.91$) in which flocks showing <85% fertility can be identified with good % accuracy. Further if a standard level of sperm-egg

interaction is established for each strain or flock, extreme mean deviations from normality could indicate a reproductive problem in that flock long before a decline in fertility is detected.

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Wetting of broilers during cold weather transport: a major source of physiological stress?

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Currently more than 700 million broiler chickens are produced in the UK each year. During road transportation of these birds from their rearing sites to the processing plant approximately 0.15% are dead on arrival (DOA). The magnitude of this transport mortality and the exposure of surviving birds to those factors responsible constitute important welfare and production issues. Frequent episodes of greatly elevated DOA occur in an unpredictable manner, which impose further processing problems on a day to day basis. Heterogeneity of the thermal microenvironment on board poultry transport vehicles brought about by inadequate or inappropriate ventilation has been strongly implicated as a cause of mortality (Mitchell *et al*, 1992). The distribution of DOAs within commercial poultry transporters in warm and cold weather coincides precisely with the poorly ventilated 'thermal core' and passive ventilation inlet of the vehicles, respectively (Hunter *et al*, 1997). Post mortem examination and analyses of deep body temperature of birds in transit indicates that many DOAs can be attributed to acute hyperthermia in warm weather and acute hypothermia in the cold. Field studies also suggest that wetting of birds occurs in poultry crates near ventilation inlets, due to the ingress of aerosolised road spray in wet weather. At low air temperatures the combination of wetting which disrupts effective feather insulation and air movement may result in rapid cooling and potentially lethal hypothermia. Whilst the use of physiological response modeling coupled with thermal mapping and the concept of 'Apparent

Equivalent Temperature' have provided the basis for understanding the problem of heat stress in the thermal core of broiler transporters, the question of cold stress at the periodically over-ventilated air inlet has not been previously addressed.

The present study examined the thermoregulatory and physiological responses of slaughter weight broiler chickens at a range of temperatures, with additional air movement and wetting, considered representative of cold weather in the UK and conditions encountered at the passive ventilation inlet of moving commercial poultry transporters. In a controlled climate chamber, groups of 8, 6-week-old broiler chickens were placed in transport crates in a wind tunnel. Ten experiments of 3 h duration were conducted at chamber temperatures (T_c) of -4° , 0° , 4° , 8° and 12°C , 5 were performed dry and 5 with intermittent wetting by fine mist spraying for 1 min every 30 min. An air velocity of 0.7 m/s was maintained throughout each trial, based on measurements of the average speed of air penetrating passively-ventilated vehicles in the field. Rectal temperature (T_r) and blood samples by venepuncture were taken immediately before and after each exposure. In addition 2 birds from each group were surgically implanted under general anaesthetic with miniaturised data logging devices to provide a continuous record of deep body temperature (T_d) and allowed to recover for 3 d before exposure to the experimental treatments. Blood was analysed for plasma concentrations of

the metabolites triglyceride (TG) and non-esterified fatty acids (NEFA).

The Table describes the pattern of T_r changes and T_d rates of change (ROC) at each environment. In dry conditions the greatest decrease in T_r was observed at the lowest T_e (-4°C) but this was not significantly different from the decrease in T_r observed at $T_e = 12^{\circ}\text{C}$. The imposition of wetting, however, induced marked hypothermia at each T_e . When compared to their dry counterparts, wetted birds displayed an additional decrease in T_r of 2.1°C at $T_e = 12^{\circ}\text{C}$ ($P < 0.005$). This additional wetting induced hypothermia was observed at each T_e and a maximum and potentially lethal decrease of $14.2 \pm 5.47^{\circ}\text{C}$ ($P < 0.005$) was recorded at $T_e = -4^{\circ}\text{C}$. The relationship between the change in T_r and T_e in wet conditions was described by the equation $y = -0.692x + 10.39$ ($P < 0.001$). Examination of the T_d profiles revealed a parallel response of ROC to T_r . ROC was low at all dry T_e though comparable to 12° and 8°C wet. With decreasing T_e ROC increased proportionally to a maximum of $-1.03 \pm 1.52^{\circ}\text{C}/10 \text{ min}$. 24°C has been reported as the lethal body temperature for fowl with increased mortality below 32°C (Freeman, 1971). Clearly the birds exposed to 0° or -4°C experienced decreases in T_r to around or below 32°C and may expect to achieve those temperatures (assuming a uniform ROC) within approximately 2 h. Further examination of T_d at $T_e = -4^{\circ}\text{C}$ with wetting, however, showed a biphasic profile where T_d gradually dropped by 7°C over 90 min before a further rapid decline of 11.5°C over the remaining 90 min. Similar response patterns were seen at $T_e = 0^{\circ}$ and 4°C suggesting some thermoregulatory failure around 1.5 h. Because the average time spent by broilers in transit is estimated at 2.7 h (Warriss *et al*, 1990), in poor weather birds in vulnerable locations experience potentially lethal T_r on routine journeys. The plasma concentration of the metabolites TG and NEFA were observed to fall and increase respectively over the 3-h experimental period (TG -22.9% , $P < 0.005$; NEFA $+41.9\%$, $P < 0.005$) with no differences between wet and dry treat-

ments. This suggests that catabolism of TG to NEFA was occurring at a faster rate than mobilisation of stored energy in adipose tissue, yet the requirement for this energy was a consequence of inanition rather than thermoregulatory demand. It is possible that glycogen was being recruited as the primary source of metabolic energy for thermogenesis but was exhausted, while fat catabolism provided additional energy as reserves became depleted.

The present study has demonstrated that the transport of broilers in well ventilated vehicles can be done safely at ambient temperatures down to -4°C in dry conditions. However, if wetting occurs at the ventilation inlet, temperatures as high as 8°C will induce moderate hypothermia. It is clear that the combination of profuse wetting and air movement conspire to compromise feathering insulation and increase evaporative and convective cooling to a degree that broiler chickens cannot compensate by physiological regulation and metabolic thermogenesis. The management practice of food withdrawal up to 6 h prior to transport may exacerbate the situation by depriving the birds of a readily available source of metabolic energy for thermogenesis, thus accelerating their susceptibility to acute hypothermia. Reappraisal of vehicle ventilation design must be undertaken to restrict the ingress of water at ventilation inlets yet retain thermal homogeneity throughout the bio-load. In this way, focal mortality in these locations can be reduced thus improving standards of welfare and productivity.

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Table. Changes in rectal temperature (ΔT_r) and rate of change of deep body temperature (ROC) over a range of environmental challenges with and without wetting (Values represent mean ± 1 standard deviation)

Temperature	No wetting		Wetting	
	$\Delta T_r (^{\circ}\text{C})$	ROC ($^{\circ}\text{C}/10 \text{ min}$)	$\Delta T_r (^{\circ}\text{C})$	ROC ($^{\circ}\text{C}/10 \text{ min}$)
-4°C	-1.21 ± 0.73	-0.07 ± 0.80	-14.19 ± 5.37	-1.03 ± 0.15
0°C	-1.01 ± 0.45	-0.02 ± 0.88	-9.74 ± 5.38	-0.66 ± 0.15
4°C	-0.76 ± 0.61	-0.03 ± 0.93	-6.74 ± 2.27	-0.28 ± 0.10
8°C	-0.85 ± 0.59	-0.07 ± 0.49	-4.40 ± 3.25	-0.09 ± 0.11
12°C	-0.96 ± 0.88	-0.02 ± 0.72	-3.03 ± 1.75	-0.02 ± 0.09