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THERMAL CONTROL OF METABOLIC COLD DEFENCE IN PIGEONS *COLUMBA LIVIA*

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Summary

Metabolic rate (MR) and various body temperatures were recorded in pigeons *Columba livia* during gradual lowering of the ambient temperature (T_a) and during cold exposure (–10 °C) for an entire circadian cycle. The aim was to study how changes in MR accorded with the observed body temperature displacements and our knowledge of regional cold sensitivity. When T_a was lowered from 28 to –10 °C, the threefold increase in MR was accompanied by a significant increase in deep-body and spinal cord temperatures. Cold exposure also resulted in a distinct rise in breast skin temperature, whereas the skin temperatures of the neck, back and leg decreased significantly. Thus, during acute cold exposure of pigeons, peripheral thermosensors seem to have the primary effect in mediating metabolic cold defence, while internal thermosensors are apparently of minor importance. By using differential weightings of various skin areas, cold-sensitivity values of between –3.9 and –14.7 W kg^{-1} °C^{-1} were estimated. These values indicate a much higher skin thermosensitivity of birds than has been previously suggested. When cold-exposed for an entire circadian cycle, the pigeons usually maintained a low leg skin temperature. However, during these experiments, the pigeons showed regular distinct increases in leg skin temperature, obviously due to cold-induced vasodilatations (CIVDs). The flushing of cold peripheral blood through the body in connection with these CIVDs resulted in a decrease in deep-body temperature (T_b), to which the pigeons responded with a distinct increase in MR. From the concomitant changes in MR and T_b, a total body cold sensitivity of –8.0 W kg^{-1} °C^{-1} was calculated, a value that is close to the previously published cold-sensitivity value of pigeons. This shows that, at least under some naturally occurring conditions, internal thermosensors will provide a significant error signal in the control of metabolic cold defence. Furthermore, the results of our study show that the relative importance of various thermosensitive sites in the control of metabolic cold defence is contextual.

Key words: body temperature, metabolic rate, cold thermosensitivity, cold-induced vasodilatation, pigeon, *Columba livia*, spinal cord, thermoregulation.

Introduction

The ability of homeotherms to regulate their body temperature precisely is based on a high intrinsic heat production, in addition to a well-developed ability to regulate the heat loss from the body. During cold exposure, homeotherms respond to the increased temperature gradient between the body and its surroundings with a decrease in thermal conductance and an increase in metabolic heat production. The regulation of heat loss is manifested as a general decrease in skin temperature, due to a reduced peripheral blood flow, whereby the temperature gradient between the body surface and the environment is decreased (Feist and White, 1989; Marsh and Dawson, 1989).

Since nearly all parts of the body are sensitive to thermal stimulation, a weighted mean body temperature would seem to represent the regulated variable in homeothermic thermoregulation (Hensel, 1981; Jessen, 1990). Thus, the augmentation of heat production under cold conditions is probably controlled by the combined sensory inputs from a multitude of thermosensors located in the body core as well as in the skin. According to the concept of proportional control in thermoregulation, the increase in heat production will be proportional to the extent to which the body temperature is displaced (Hammel, 1968). This has been demonstrated experimentally for both birds and mammals (Mercer and Simon, 1984; Simon *et al.* 1986). The precise contribution of the central and peripheral thermosensors in providing information on the thermal state of the whole body is unknown. However, it is generally assumed that the central thermosensors are the most important in mediating this information (Hensel, 1981; Mercer and Simon, 1984; Simon *et al.* 1986).

Quantitative assessments of local cold-sensitivity values have been made by comparing the effector responses induced by selective cooling of particular parts of the body. In birds, total body thermosensitivity (TBTS) has been estimated to range from approximately –5 to –20 W kg^{-1} °C^{-1} (Simon *et al.*

1986; Mercer and Hammel, 1993). Whilst the spinal cord is regarded as an important thermosensory area in birds (Rautenberg *et al.* 1972; Inomoto and Simon, 1981; Østnes and Bech, 1992), thermal stimulation of the hypothalamus elicits paradoxical or only weak thermoregulatory effector responses (Rautenberg *et al.* 1972; Simon-Oppermann *et al.* 1978; Mercer and Simon, 1987). From a comparison of the different thermosensitive areas of Pekin ducks *Anas platyrhynchos*, Inomoto and Simon (1981) suggested that deep-body thermosensors, external to the central nervous system (CNS), play the dominant role in the control of body temperature, while the contribution of the skin to TBTS is minor.

Experimentally induced temperature displacements do not necessarily reflect naturally occurring body temperature changes. In this connection, it is interesting to note that any attempt to demonstrate a correlation between normal fluctuations in CNS temperature and thermoregulatory effector activity have failed both in mammals (Abrams and Hammel, 1965; Rawson *et al.* 1965) and in birds (Graf and Necker, 1979). Furthermore, several studies have noted a lack of cold signal inputs from the body core during exposure of homeotherms to low ambient temperatures (Graf and Necker, 1979; Mercer and Hammel, 1989; Hammel, 1990; Østnes and Bech, 1997). Thus, it is still unknown to what extent thermosensors situated inside the body core contribute to the combined error signal that controls metabolic cold defence when homeothermic animals are subjected to an external cold challenge. Information about the actual regional body temperature changes which occur during exposure to low ambient temperatures is therefore needed for an evaluation of the importance of the different thermosensory areas in mediating metabolic cold responses.

The aim of the present study was to describe temperature changes in different parts of the body during cold exposure in pigeons *Columba livia*, with the specific objective of investigating how the changes in metabolic rate accord with the observed body temperature displacements and our knowledge of regional cold sensitivity. Metabolic rate was recorded, together with deep-body temperature, spinal cord temperature and various skin temperatures in two different situations: (a) during gradual lowering of the ambient temperature (T_a) and (b) during long-term (24 h) exposure of pigeons to a low T_a (–10 °C). The concomitant changes in the metabolic rate and the various body temperatures recorded in the first series of experiments led us to conclude that peripheral thermosensors have a primary effect in the initiation and maintenance of metabolic cold defence, while centrally located thermoreceptors are apparently of minor importance. During long-term cold exposure, the pigeons generally maintained a low leg skin temperature indicative of a general peripheral vasoconstriction. However, in some of these experiments, we observed regular distinct increases in leg skin temperature, obviously due to cold-induced vasodilatations (CIVDs). Metabolic responses elicited during these CIVDs were correlated with distinct decreases in deep-body temperature. Thus, the present study will provide evidence that the relative importance of different thermosensitive areas might depend on the type of thermoregulation in which the birds are engaged at any particular time.

A preliminary summary of the results of this study has been reported elsewhere (Bech and Østnes, 1996).

Materials and methods

Experimental animals

Nine pigeons, with a mean body mass of 414±12 g (S.E.M., range 383–486 g), were used in the study. They were all adult birds (more than 3 years old) that had been kept under constant conditions of ambient temperature (20–22 °C) and illumination (12 h:12 h L:D cycle; lights off at 20:00 h) for more than 2 years prior to the experiments. Food and water were available *ad libitum*, although food was withheld for 16 h prior to the start of each experiment (from 17:00 h on the evening before an experiment) to ensure that the pigeons were post-absorptive.

Temperature measurements

Under halothane anaesthesia, the pigeons were fitted with blind-ending tubes (PP 00, Portex Ltd, UK) placed in the vertebral canal dorsal to the spinal cord using the procedure described previously (Rautenberg *et al.* 1972; Østnes and Bech, 1992). From a common opening made in the first thoracic vertebra, one tube was advanced approximately 5 cm into the cervical region, while a second tube was inserted approximately 4.5 cm into the thoracic region. During the experiments, thin Co–Cu thermocouples (California Fine Wire Company, type 0.005) were inserted into these tubes to enable us to record the cervical (T_{ce}) and the thoracic (T_{th}) spinal cord temperatures. After the experiments were concluded, the exact positions of the two tubes were verified during *post-mortem* examinations. Temperatures in the cervical region were measured at the level of C_6–C_8, while temperatures in the thoracic region were measured at Th_5. The pigeons were also equipped with a blind-ending tube (PP 50) inserted 5 cm into the abdominal cavity through an incision made close to the cloacal opening (Østnes and Bech, 1992). This tube acted as a guide tube for a thermocouple used to measure the deep-body temperature (T_b). The pigeons were allowed at least 14 days to recover before being used in the experiments. At the start of each experiment, thermocouples were also placed on the breast (T_{breast}), back (T_{back}), neck (nape, T_{neck}) and leg (lower part of the tarsus, T_{leg}) to measure the various skin temperatures. Leg skin temperature was measured on the right leg only.

Oxygen consumption measurements

During the experiments, the pigeons were confined in an acrylic metabolic chamber (7 l). This chamber was placed inside a climatic chamber (Heraeus Vötsch, type VEM 03/500). Within the metabolic chamber, the pigeons were exposed to darkness. Rates of oxygen consumption were measured using flow-through respirometry. Dry atmospheric air was sucked through the chamber by an air pump (Miniport) at a rate of approximately 2.1 l min^{-1}. After drying over silica

gel, the actual airflow rates were measured, using a mass flow controller (Bronkhorst high-tech, type 201C-FA), before fractions of the air were directed into an oxygen analyzer (Servomex, type 1100A). The oxygen analyzer was calibrated before each experiment using dry atmospheric air (20.95 % O_2) and pure nitrogen. Any change in the O_2 reading at the end of an experiment was corrected for by assuming a linear drift. The recordings of the O_2 content of the excurrent air (accuracy 0.01 %) were stored, along with the temperature measurements, on a data logger (Squirrel series 1200, Grant Instruments, UK), at 1 min intervals.

Oxygen consumption rates were calculated from the STPD air flow rates and the O_2 content of the excurrent air, using equation 3a of Withers (1977). The equation given by Niimi (1978) was used to correct for wash-out delays. Values of metabolic rates (MR, $W\,kg^{-1}$) were subsequently calculated from the instantaneous oxygen consumption rates, assuming that $1\,ml\,O_2\,g^{-1}\,h^{-1}$ is equal to $5.582\,W\,kg^{-1}$ and a respiratory exchange ratio of 0.79.

Experiments

Two different sets of experiments were performed. In the first (termed acute cold exposure), the pigeons were first exposed to thermoneutral ambient conditions (approximately 28 °C) for 3 h. The temperature inside the metabolic chamber was thereafter lowered to approximately −10 °C, a value that was usually attained within 1.5 h. Each experiment was terminated 3 h after the onset of cooling. To exclude biorhythmic factors, the experiments were always performed between 09:00 and 18:00 h. Data were obtained from seven pigeons. However, in one of these pigeons, only the deep-body and spinal cord temperatures were measured.

In the second set of experiments (termed long-term cold exposure), each experimental run was initiated at approximately 09:00 h (1 h after lights on). The pigeons were first exposed to the thermoneutral T_a for 2–3 h. Thereafter, the temperature in the metabolic chamber was gradually reduced to approximately −10 °C. The pigeons were kept at this cold T_a until the next morning, i.e. the total time of each experiment was approximately 24 h. Data were obtained from six pigeons. However, for two individuals, we failed to obtain data for T_{th} and T_{neck}, respectively.

Calculations and statistics

In the experiments involving acute cold exposure, the average MR and body temperature values measured over the last 60 min before cold exposure commenced were taken to represent the resting values under thermoneutral conditions. Similarly, the average MR and body temperature values calculated for the period 90–150 min after cooling commenced were taken to represent the comparable values under cold conditions. Each pigeon was used 2–5 times in experiments and, on the basis of the results obtained from these experiments, individual mean MR and body temperature values were calculated for each of the seven pigeons.

In the experiments involving cold exposure for an entire circadian cycle (long-term cold exposure), the pigeons proved to be variable in their response to cold. In particular, a variable number of CIVDs was recorded for each of the individual pigeons. Consequently, for each of the six pigeons for which CIVDs were recorded, we calculated the mean values for all the measured parameters based on the number of CIVDs recorded for that particular individual. For each episode of CIVD, the 'pre-CIVD' levels recorded for the deep-body, spinal cord and skin temperatures, as well as the MR, were calculated as the mean value for the last 5 min prior to any observed change in leg skin temperature. The maximum value of MR reached during a CIVD was calculated as the maximum value of a 3 min running average of the instantaneous oxygen consumption. The corresponding values of all measured body temperatures were calculated as the mean values for the same 3 min interval. From the changes in T_b and MR, we calculated values of ΔT_b and ΔMR. From these values, a total body thermosensitivity (TBTS), which is defined as the change in metabolic heat production in response to a decrease in body temperature of 1 °C (expressed in $W\,kg^{-1}\,°C^{-1}$), could be calculated as $\Delta MR/\Delta T_b$ (Mercer and Simon, 1984; Simon *et al.* 1986). In addition, TBTS was assessed from the slope of a linear regression line (least-squares regression method) of ΔMR against ΔT_b (including the data for all individuals), in which the regression line was forced through the origin.

All data are presented as means ± S.E.M., N denotes the number of birds investigated. A paired t-test was used for comparisons of two groups of mean values, while more than two groups of mean values were compared using a one-way analysis of variance (ANOVA) followed by the Newman–Keuls test for pairwise multiple comparisons. Overall differences were considered significant when $P<0.05$. All statistical analyses were performed using SigmaStat software (version 1.0, Jandel Scientific, Germany).

Results

Acute cold exposure

The mean MR measured under thermoneutral ambient conditions (28.1±0.2 °C) was $4.71\pm0.19\,W\,kg^{-1}$ (N=7). Mean T_b, T_{ce} and T_{th} at thermoneutrality were 40.8±0.1 °C, 40.3±0.1 °C and 40.5±0.1 °C (N=7), respectively. Significant differences were found between these values ($F_{2,18}$=4.54, $P<0.05$). However, a pairwise multiple comparison revealed a significant difference only between T_b and T_{ce}. Mean T_{breast}, T_{back} and T_{neck} values measured at thermoneutrality were 40.1±0.2 °C, 39.8±0.1 °C and 39.6±0.2 °C (N=6), respectively. No significant differences existed between the temperatures of the various feathered skin areas at thermoneutrality. Mean T_{leg} at thermoneutrality was 32.0±0.7 °C (N=6).

The pigeons responded to the lowering of T_a with an immediate increase in MR (Fig. 1). At a T_a of –9.8±0.7 °C, MR reached a new stable level of $14.10\pm0.63\,W\,kg^{-1}$ (N=7). This represents a threefold increase in MR compared with the level measured at thermoneutrality. The increase in MR was accompanied by a concomitant increase in T_b as well as in the

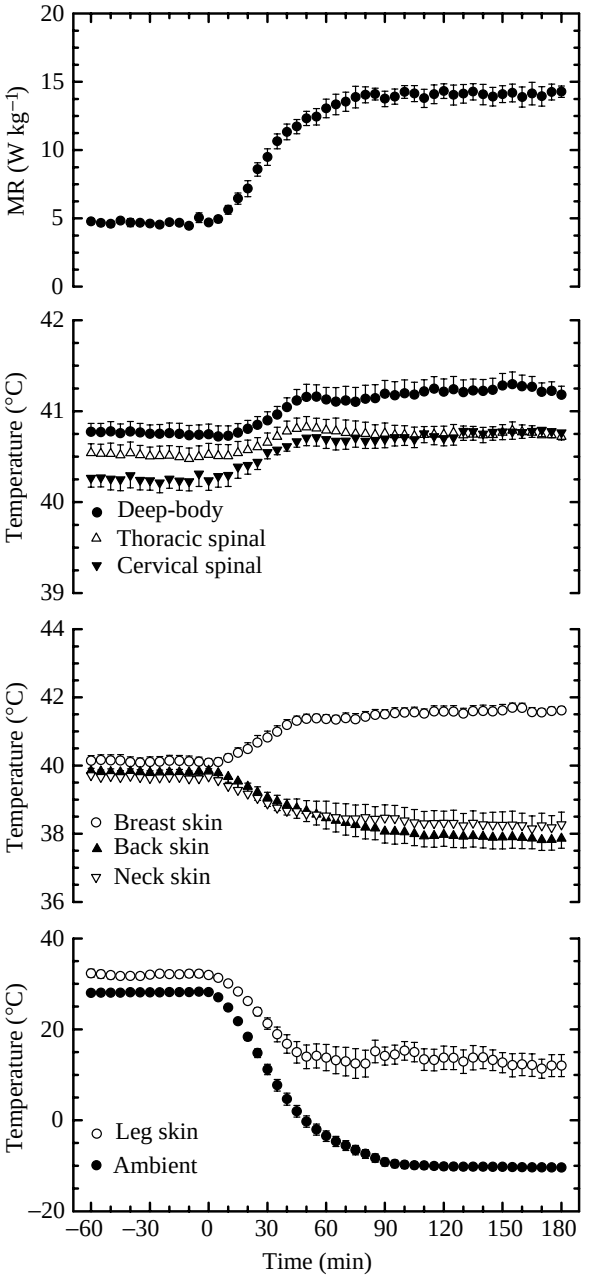

Fig. 1. Metabolic rate (MR) and various body temperatures measured in pigeons during the transition from thermoneutral to cold ambient temperatures. Cooling began at time zero. Values are means ± S.E.M., *N*=6. Some error bars are smaller than the symbols.

two spinal cord temperatures measured (Fig. 1). As would be expected, T_{back}, T_{neck} and T_{leg} decreased during the transition from thermoneutral to cold ambient conditions. In contrast, T_{breast} increased during cold exposure.

The increases in T_b, T_{ce} and T_{breast} measured during the transition from thermoneutral to cold ambient conditions were statistically significant (paired *t*-test; Fig. 2), whereas T_{th} did not increase significantly (t=2.32, P=0.056). The decreases in T_{back} and T_{neck} were statistically significant (Fig. 2). Leg skin temperature declined to a mean value of 13.8±2.3 °C during

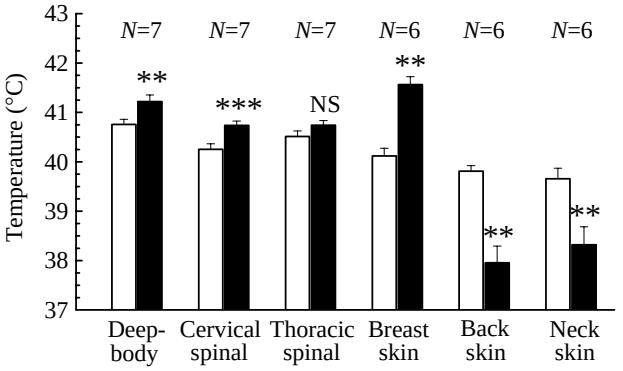

Fig. 2. Various body temperatures measured in pigeons exposed to thermoneutral (approximately 28 °C; open bars) and cold (approximately −10 °C; black bars) conditions. Bars represent the mean temperatures + S.E.M. measured during the last 60 min period at thermoneutral conditions and during the first 60 min period after a new steady-state had become established in the cold conditions. ***P<0.001; **P<0.01; NS, not significantly different from the thermoneutral value.

cold exposure. The change in T_{leg} were highly significant (t=7.81, P<0.001).

Long-term cold exposure

An example of the periodically occurring CIVDs during an experiment is shown in Fig. 3. Each CIVD was paralleled by a concomitant decrease in deep-body (T_b) and spinal cord temperatures, as well as by an increase in MR (Fig. 3). Of the nine pigeons initially tested, only six showed distinct CIVDs during long-term cold exposure. The remaining three pigeons never showed any CIVDs, despite being tested in several experiments. In addition, a great variation was noted in the number of CIVDs, both between pigeons and between experimental runs (Table 1).

Details of two CIVDs, reflecting this variation in response, are shown in Fig. 4. Clear differences existed in the duration of the temperature increase of the legs, indicating differences in the duration of the vasodilatation. From the examples shown in Fig. 4, it is obvious that vasodilatations in the legs caused distinct decreases in T_b, which in turn caused the metabolic heat production to increase.

The flushing of the legs during the CIVDs caused T_{leg} to increase from 8.3±1.1 to 26.0±2.8 °C (t=8.39, P<0.001). The concomitant decrease in T_b was from 40.2±0.1 to 39.6±0.2 °C (t=17.7, P<0.0001, Fig. 5). This caused the MR to increase from 12.39±0.76 to 16.68±0.51 W kg^{-1} (t=9.28, P<0.001). Spinal cord temperatures decreased significantly (Fig. 5) from 39.9±0.1 to 39.6±0.1 °C (t=4.24, P<0.01) for T_{ce} and from 39.5±0.2 to 39.2±0.2 °C (t=3.19, P<0.05) for T_{th}. In contrast T_{breast}, T_{back} and T_{neck} did not change significantly (Fig. 5), although there was a tendency for T_{back} to decrease (t=2.44, P=0.059). The mean skin temperatures before the CIVDs were 40.2±0.2 °C for the breast, 35.2±0.5 °C for the back and 36.8±0.5 °C for the neck.

The increase in MR as a direct response to a transient

Table 1. *Changes in deep-body temperature (ΔT_b) and metabolic rate (ΔMR) and the resultant total-body cold sensitivity (TBTS) during periods of cold-induced vasodilatation (CIVD) in pigeons*

Individual	*n*	*x*	Body mass (g)	ΔT_b (°C)	ΔMR (W kg^{-1})	TBTS (W kg^{-1} °C^{-1})
0606	1	9	414	–0.68±0.04	3.91±0.56	–5.69±0.68
1000	1	2	383	–0.51	5.92	–11.93
1446	2	9	389	–0.64±0.11	3.91±0.38	–6.96±0.81
1473	2	5	486	–0.52±0.09	5.23±1.14	–10.46±2.40
1586	3	10	441	–0.59±0.05	4.07±0.34	–7.20±0.65
12474	3	3	388	–0.48±0.03	2.70±0.30	–5.70±0.98
Mean	(*N*=6)		416±16	–0.57±0.03	4.29±0.46	–7.99±1.06

n, number of experimental nights; *x*, number of CIVDs on which TBTS is based.
Mean values ± S.E.M. are presented whenever the number of measurements exceeds two; otherwise, only the mean value is given.

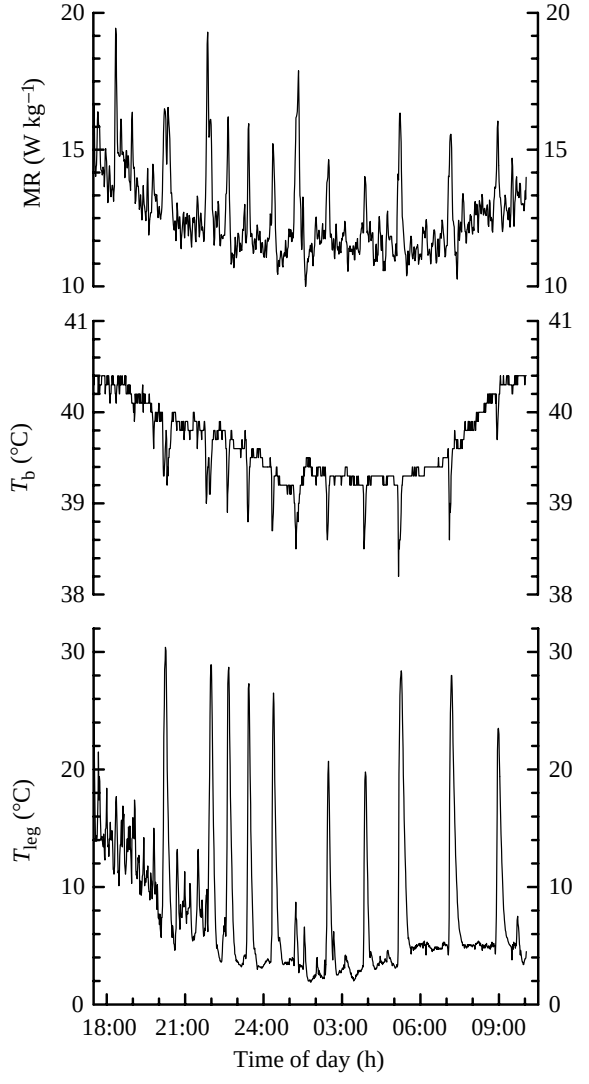

Fig. 3. Metabolic rate (MR), deep-body temperature (T_b) and leg skin temperature (T_{leg}) in a pigeon during long-term cold exposure (approximately –10 °C) as a function of time of day. Cold exposure began at 12:00 h.

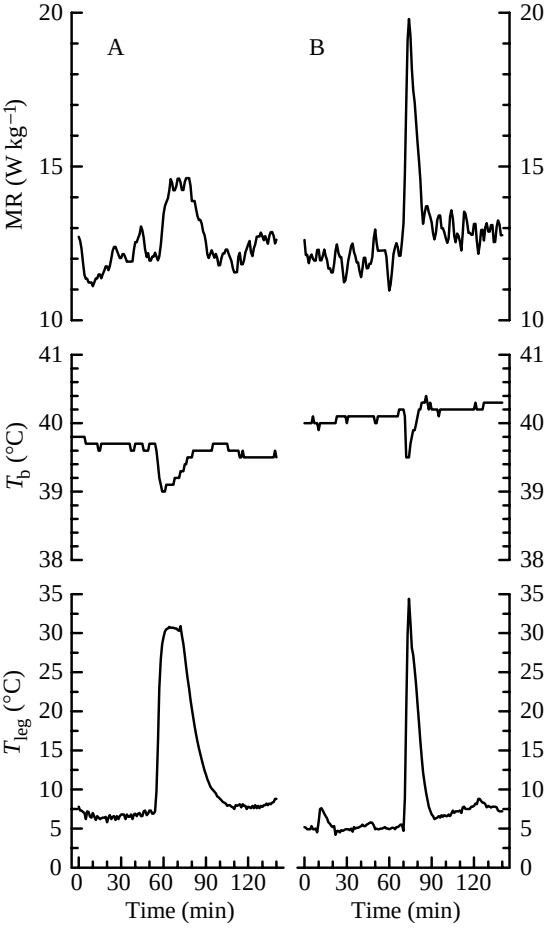

Fig. 4. (A,B) Two examples of the changes in metabolic rate (MR), deep-body temperature (T_b) and leg skin temperature (T_{leg}) in pigeons during episodes of cold-induced vasodilatation during long-term cold exposure (approximately –10 °C).

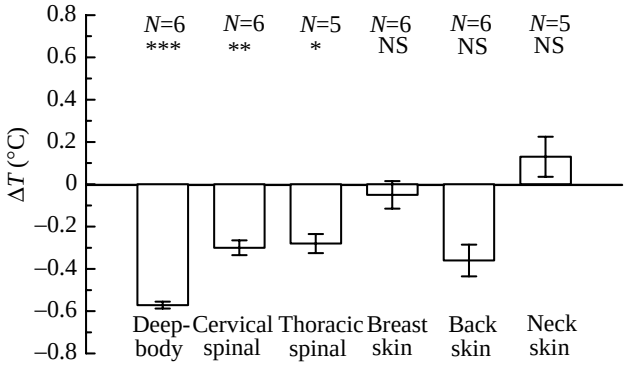

Fig. 5. Mean changes (± S.E.M.) in deep-body, spinal cord and skin temperatures during episodes of cold-induced vasodilatation during long-term cold exposure (approximately −10 °C) in pigeons. *N* is the number of birds. ***P<0.001; **; P<0.01; *P<0.05; NS, not significantly different from zero.

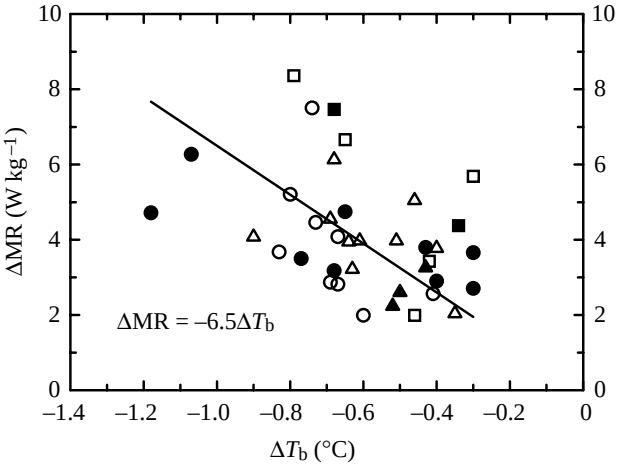

Fig. 6. Change in metabolic rate (ΔMR) as a function of change in deep-body temperature (ΔT_b) during periodic cold-induced vasodilatations (CIVDs) in pigeons during 24 h of cold exposure (approximately −10 °C). The regression line is forced through zero. Each point represent the value from a single episode of CIVD. Based on data from six individuals, as indicated by the different symbols.

lowering of T_b allowed us to calculate the total-body cold thermosensitivity (TBTS). Values of TBTS for the six pigeons could be calculated from 2, 3, 5, 9, 9 and 10 occurrences of CIVDs, respectively (Table 1). The individual TBTS values ranged from −5.7 to −11.9 W kg^{-1} °C^{-1}, with a mean value of −8.0 W kg^{-1} °C^{-1}. A one-way ANOVA on the individual means revealed a significant difference in TBTS between the pigeons ($F_{5,32}$=2.61, P<0.02), but a subsequent Newman–Keuls test failed to reveal any pairwise differences, probably because of the low number of CIVDs obtained from pigeon 1000 (Table 1). Fig. 6 shows all the individual TBTS measurements, the combined equation being $\Delta MR = -6.5\Delta T_b$ (r^2=0.87, P<0.0001) when the linear regression line was forced through origin. The lower slope of this regression line (–6.5 W kg^{-1} °C^{-1}, Fig. 6) compared with the overall mean based on the individual values (–8.0 W kg^{-1} °C^{-1}, Table 1) reflects the uneven contributions to the regression line from the different individuals.

Discussion

Acute cold exposure

The mean MR (4.71 W kg^{-1}) value recorded under thermoneutral ambient conditions falls within the range of previous measurements of the resting MR in pigeons (4.62–6.13 W kg^{-1}; Bennett and Harvey, 1987). This indicates that the birds were in a non-stressed condition prior to the cold exposure period. Significant local temperature differences were found inside the body core at thermoneutrality. Such local temperature differences in the body core have been described previously. They can be ascribed to differences in the specific metabolic rates of various organs and the temperature of the blood flowing through them (Jessen, 1990).

Previous studies, aimed at quantifying the relative importance of thermal inputs from various thermosensitive areas, have suggested that the thermosensors within the body core play a predominant role in the control of metabolic cold defence of homeothermic animals (Inomoto and Simon, 1981; Mercer and Simon, 1984; Simon *et al.* 1986). In those studies, the thermosensitivity was determined mainly by experimental cooling of specific parts of the body core, using heat exchangers. Our aim was to investigate whether the thermal information derived from the spinal cord, or the deep-body tissue external to the CNS, contributes to the combined error signal that controls metabolic cold defence during exposure to low T_a values in pigeons. As expected, lowering of the T_a induced a near-linear increase in the MR of the pigeons (Fig. 1). However, this increase was not accompanied by a concomitant decrease in the temperatures measured within the body core. In fact, the increase in MR was accompanied by an increase in T_b as well as in spinal cord temperature. Thus, the increase in MR was positively correlated with the temperature changes within the body core. This is in agreement with the results presented by Graf and Necker (1979), but is contrary to the clear negative correlation found by experimental cooling of either the spinal cord (Rautenberg *et al.* 1972; Hammel *et al.* 1976; Inomoto and Simon, 1981) or the whole-body core (Helfmann *et al.* 1981; Inomoto and Simon, 1981; Mercer, 1989) in birds. Our results indicate that the increase in MR occurs despite the absence of any apparent error signals from thermosensors within the body core. Hence, afferent cold signals from thermosensors in the body core do not appear to be important in either initiating or sustaining the elevated rate of heat production during acute cold exposure in pigeons. This points to the peripheral thermosensors as being the most important feedback elements in the control of metabolic cold defence.

As deep-body tissue has been found to be highly sensitive to temperature changes, one would expect to find that even a small rise in T_b would have a strong inhibitory effect on MR

(Nagel *et al.* 1986; Hammel, 1990). Do temperature increases, such as those seen in the present study, exert an inhibitory effect on MR? An increase in T_b during acute cold exposure is not unique to pigeons. An increase in T_b has also been observed during exposure to low T_a in Pekin ducklings (Østnes and Bech, 1997) and in several mammals, such as dogs *Canis familiaris* (Hammel *et al.* 1958), rats *Rattus norvegicus* (Székely *et al.* 1994), nine-banded armadillos *Dasypus novemcinctus* (Johansen, 1961; Mercer and Hammel, 1989) and humans (Timbal *et al.* 1976). In the armadillo, cold exposure was found to be accompanied by such large increases in heat production that T_b rose by 1–3 °C (Johansen, 1961; Mercer and Hammel, 1989). Since such appreciable increases in T_b during cold exposure are difficult to reconcile with the principle of proportional control in thermoregulation, Jessen (1990) has suggested that this phenomenon could be ascribed to a reduction in the inner body core. However, Mercer and Hammel (1989) showed that this was not the case in the armadillo, in which the total heat content showed a distinct increase during cold exposure. To explain the unexpected responses of the armadillo to low T_a, Hammel (1990) suggested that the contradictory feedback signals expected to originate from an elevated core temperature could be overridden by autofacilitation of the neurones that innervate the shivering muscles by means of a positive feedback loop. Another possible explanation is that the elevated T_b levels found during cold exposure in homeotherms represent an increase in the preferred deep-body temperature (‘set-point’), instead of a sustained error signal used to counteract the thermal input from the peripheral thermosensors. The adaptive value of maintaining an elevated T_b is difficult to explain from an energetic point of view. However, because physiological rate functions are temperature-dependent (Q_{10} effect), an increase in T_b might improve the efficiency of the metabolic system. It could also be argued that the initial increase in the T_b of the pigeons resulted from a dynamic overshoot of cold sensors during the transition from thermoneutral to cold conditions (Kuhnen and Jessen, 1988). However, since T_b was maintained at an elevated level throughout the entire cold-exposure period (Fig. 1), it would appear that the increase in T_b was regulated. This is in agreement with the findings for the armadillo (Mercer and Hammel, 1989). It should, however, be mentioned that results such as those presented here do not necessarily predict what the birds will do under natural conditions, and there are also data suggesting that pigeons may not elevate their T_b during acute cold exposure (Rautenberg, 1969).

Since the core temperatures of the pigeons were positively correlated with MR during acute cold exposure, it is tempting to suggest that the cold-induced increase in heat production was entirely a result of a fall in the temperature of the body surface. T_{back}, T_{neck} and T_{leg} all decreased significantly during the transition from thermoneutral to cold conditions (Fig. 2). In contrast, T_{breast} increased by a mean value of 1.4 °C. The pectoral muscles constitute approximately 20 % of the body mass of a pigeon and are assumed to play the most important

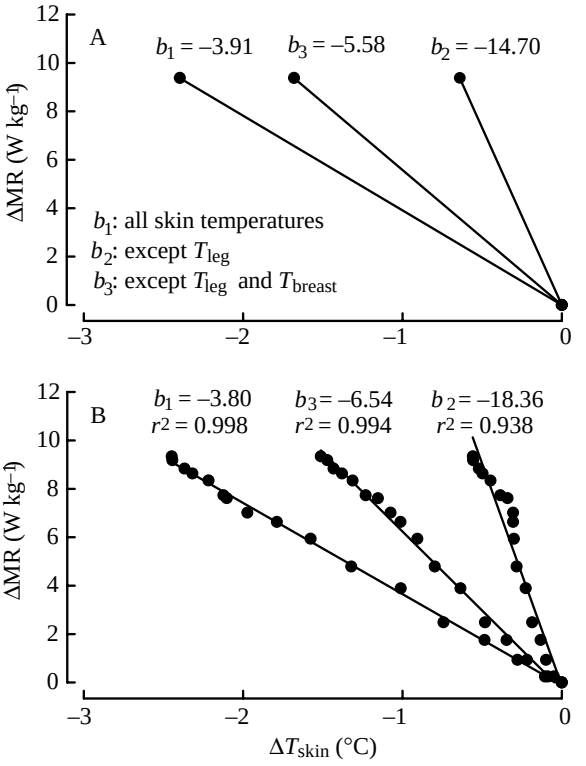

Fig. 7. Relationship between changes in skin temperature (ΔT_{skin}) and changes in metabolic rate (ΔMR) induced by cold exposure (approximately –10 °C) of pigeons (N=6). The slope of the regression lines provide an estimate of skin thermosensitivity based on either the steady-state conditions (A) or the dynamic phase (B). For the dynamic phase, the data points represent the mean values for ΔMR and ΔT_{skin} recorded at 5 min intervals during the transition from thermoneutral to cold conditions. Linear regression lines were calculated according to the equation $\Delta MR = a + b\Delta T_{skin}$, with different weightings for the various skin areas: (1) $0.4T_{back}+0.3T_{breast}+0.2T_{neck}+0.1T_{leg}$; (2) $0.444T_{back}+0.333T_{breast}+0.222T_{neck}$; and (3) $0.667T_{back}+0.333T_{neck}$. The slopes of the regression lines are denoted b_1, b_2 and b_3, respectively. See text for further explanation.

role, quantitatively, in shivering thermogenesis (Nomoto and Nomoto-Kozawa, 1985). The increase in T_{breast} must thus be ascribed to the enhanced heat production of the underlying pectoral muscles. If one adopts the hypothesis that the elevated core temperatures found during cold exposure in pigeons did not exert an inhibitory effect on the MR, it should be possible to estimate skin thermosensitivity solely on the basis of changes in MR and skin temperatures. Sensitivity values were calculated both on the basis of the steady-state conditions, i.e. the mean MR and skin temperature values measured at thermoneutrality and values recorded when the new steady state had been established in the cold, and conditions during the dynamic phase, i.e. during the transition from thermoneutral to cold conditions (Fig. 7).

An exact determination of skin thermosensitivity is difficult to achieve, because of the wide local differences in skin temperature found during cold exposure. In an attempt to calculate skin thermosensitivity, therefore, the different skin

areas were assigned different weightings; $0.4T_{back}$, $0.3T_{breast}$, $0.2T_{neck}$ and $0.1T_{leg}$ (see also Inomoto and Simon, 1981; Rautenberg, 1983). A mean skin thermosensitivity corresponding to $-3.91\,W\,kg^{-1}\,°C^{-1}$ was calculated on the basis of the steady-state conditions (Fig. 7A). This estimate includes the temperature changes recorded in both the feathered and the unfeathered skin areas. However, Necker (1977) has shown that, while the feathered skin areas of pigeons are highly sensitive to thermal stimulation, cooling of the naked skin of the legs and the beak is incapable of eliciting shivering. If, as indicated by Necker's (1977) results, thermal information from unfeathered skin areas does not contribute to the combined error signal, then the thermosensitivity of the skin will be underestimated by including data for those areas. A sensitivity of $-14.70\,W\,kg^{-1}\,°C^{-1}$ was calculated, based solely on the temperature changes recorded for the feathered skin areas (Fig. 7A). However, this estimate includes the T_{breast} values, which showed a distinct increase during cold exposure. By assuming, as for the core temperatures, that an elevated T_{breast} value does not have any influence on MR, a new sensitivity value was calculated, based entirely on the data for those feathered skin areas that actually showed a fall in temperature. This yielded an estimated thermosensitivity for the feathered skin areas corresponding to $-5.58\,W\,kg^{-1}\,°C^{-1}$, a value that is very close to the previous estimate of TBTS for pigeons (approximately $-6\,W\,kg^{-1}\,°C^{-1}$; Simon *et al.* 1986). The calculated regression slopes, based on the data for the dynamic phase, yielded slightly different estimates for the skin thermosensitivity (Fig. 7B). More interestingly, however, for each of the three slopes, there was a very high degree of linearity between the changes in MR and skin temperature ($P<0.0001$ for each of the slopes). This indicates that the relative contribution of the different skin areas to the combined error signal did not change during the transition from thermoneutral to cold ambient conditions. Furthermore, the high degree of linearity points to an action of peripheral thermosensors which is in accordance with the proportional model of body temperature control.

It is difficult to decide which of the above values provides the best estimate of the skin thermosensitivity of pigeons. However, each of the estimated values is much higher than previously estimated skin thermosensitivity values in birds, namely $-0.6\,W\,kg^{-1}\,°C^{-1}$ for Pekin ducks (Inomoto and Simon, 1981) and $-1.7\,W\,kg^{-1}\,°C^{-1}$ for pigeons (Rautenberg, 1971; Simon *et al.* 1986). In particular, if the thermal information provided from the naked skin areas does not contribute to the combined error signal, then feathered skin areas may turn out to be very sensitive to temperature changes. Such high skin thermosensitivity values as those obtained in the present study could explain why birds are able to fine-tune their metabolic responses to an external cold challenge despite the absence of cold signal inputs from thermosensors in the body core.

Long-term cold exposure

Regularly occurring CIVDs, shown as distinct increases in skin temperature, were originally described in human hands by Lewis (1930) as the classical 'hunting reaction'. Subsequently, CIVDs have been described from a variety of birds (Midtgård, 1989) and mammals (Henshaw, 1986) including humans (Krogh *et al.* 1960; Livingstone *et al.* 1978). The classical 'hunting reaction' consists of a primary vasoconstriction followed by a secondary vasodilatation, with a subsequent chain of alternating vasoconstrictions and vasodilatations (Lewis, 1930; Livingstone *et al.* 1978). The transient rises in leg skin temperature of the pigeons, such as those shown in Fig. 4 and which, in the present paper, are referred to as CIVDs, are caused primarily by a large increase in the blood flow through the arteriovenous anastomoses (AVAs). The AVAs, which are known to be widely distributed in the leg skin of birds (Grant and Bland, 1931; Midtgård, 1980; Johansen and Bech, 1983), act as low-resistance vascular shunts that allow high rates of peripheral blood flow, for example during periodic CIVDs (Midtgård, 1989).

Cold-induced vasodilatations are generally claimed to occur widely in birds, but have actually only been formally described for a very limited number of species. The occurrence of CIVDs in the legs of birds was first described by Grant and Bland (1931) after immersion of the feet of domestic hens *Gallus domesticus* in iced water. Subsequently, the phenomenon has only been studied in the southern giant petrel *Macronectes giganteus* (Johansen and Millard, 1973, 1974; Murrish and Guard, 1977). In spite of a large body of literature on body temperature regulation in pigeons, studies have so far failed to describe CIVDs in pigeons. Graf (1980), for example, merely described a continuous vasoconstriction in the legs during the night phase when pigeons were experimentally exposed to T_a values below thermoneutrality. The reason for the absence of CIVDs could be that the pigeons were exposed to only moderately cold T_a values; 12 °C in the study by Graf (1980). In other studies, cold exposure of the pigeons was apparently too brief (e.g. Saarela *et al.* 1984). Thus, although a vast number of studies exist that deal with thermoregulation in pigeons, there have been no previous descriptions of CIVDs such as those described in the present study (Figs 3, 4).

CIVDs in pigeons were generally of short duration, with the 20 min of elevated T_{leg} depicted in Fig. 4B representing a typical CIVD. In contrast, the CIVD shown in Fig. 4A, lasting for approximately 50 min, was atypically long-lasting. The number of CIVDs recorded for the individual pigeons was likewise variable (Table 1), ranging from a single incident during a 24 h experiment, to nine incidents during a single night. There seem to be no previous quantitative data reported for birds with which we could compare these values. Also, we have no explanation as to why some individuals (three out of the original nine birds tested) never showed any CIVDs, despite being repeatedly tested. Such individual differences could be caused by individual differences in the bird's previous thermal experience. For example, Rautenberg (1969) found that cold-acclimated pigeons had a higher foot temperature than controls when exposed to low T_a values. However, all the nine pigeons used in the present study came from the same laboratory stock and had experienced similar conditions of

photoperiod and T_a for at least 2 years prior to the experiments. Those pigeons that did not employ CIVD as a cold-defence mechanism experienced a continuous vasoconstriction of their legs and apparently coped as well as did the pigeons using CIVD.

The CIVDs clearly resulted in a metabolic response (Figs 3, 4). Which thermosensors are involved in mediating the metabolic response? As mentioned above, Necker (1977), in his study on the skin thermosensitivity in pigeons, demonstrated that, in contrast to the feathered parts of the pigeon, the leg skin had only a negligible thermosensitivity. Thus, the large changes noted in T_{leg} are unlikely to have had any influence on the magnitude of the metabolic response observed during the CIVD. If anything, a large increase in T_{leg} would be expected to have counteracted the metabolic response. Since there were only minor, and non-significant, changes in the other skin temperatures measured during the CIVDs (Fig. 5), it seems that the increase in heat production must have been entirely due to stimulation of internal thermosensors by cold blood returning from the legs. Our study seems to be the first to describe the involvement of internal cold sensors in thermoregulation during 'natural' conditions in birds. Steen and Steen (1965), however, did find that cold-immersion of the legs of the great black-backed gull *Larus marinus* caused a cessation of panting. Although the authors claimed that this was proof of cold sensitivity of the leg skin, their results, as was also pointed out by Necker (1977), could have been due to secondary effects of central body cooling by cold peripheral blood; a situation that bears a similarity to that described in the present study on pigeons.

On the basis of the concomitant changes in MR and T_b recorded in connection with the CIVDs, we calculated a mean TBTS value of $-8.0\,W\,kg^{-1}\,°C^{-1}$ (Table 1). This value is slightly higher than previously reported, experimentally based, estimates of TBTS in the pigeon, of approximately $-5.5\,W\,kg^{-1}\,°C^{-1}$ (Rautenberg and Necker, 1975; Simon *et al.* 1986). Our TBTS value for the pigeon, however, lies within the range of values reported for other species of birds. For example, by experimental cooling of the body core of the Pekin duck, Inomoto and Simon (1981) found values of TBTS between −5 and $-6\,W\,kg^{-1}\,°C^{-1}$. Similar values have been reported for the Adelie penguin *Pygoscelis adeliae* (Simon *et al.* 1976). Somewhat higher values of TBTS have been found for the domestic goose *Anser anser*, for which Helfmann *et al.* (1981) reported a value of $-12.5\,W\,kg^{-1}\,°C^{-1}$, and for the common eider *Somateria mollissima*, for which values between −10.7 and $-20.5\,W\,kg^{-1}\,°C^{-1}$ have been reported (Mercer, 1989; Mercer and Hammel, 1993). Hence, the results of the present study have demonstrated a natural situation which points to an action of internal thermosensors that is in accordance with that previously described by experimental cooling of the body core.

Relative weighting of local thermosensitivities

This study shows that the rise in metabolic heat production during acute cold exposure of pigeons is driven mainly by peripheral thermosensors and that centrally located thermosensors are apparently of little informative use under such circumstances. However, as exemplified by the CIVDs, internal thermosensors can, at least under some natural situations, provide a significant error signal in the control of metabolic cold defence. From these results, it follows that quantification of the cold sensitivity of a specific part of the body does not necessarily imply that feedback signals from that part of the body are involved in mediating the metabolic responses to an external cold challenge. A very important issue to take into consideration is the type of thermoregulation in which the birds are engaged at any particular time. Hence, while cold signal inputs from peripheral thermosensors are apparently of the utmost importance in initiating and maintaining long-term steady-state shivering, internal thermosensors would seem to be of primary importance in adjusting any sudden core temperature changes. Furthermore, our results indicate that the total body thermosensitivity cannot necessarily be calculated as the sum of many local cold sensitivity values. On the contrary, the relative importance of various thermosensitive sites in the control of metabolic cold defence appears to be contextual.

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