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INHIBITING VENTILATORY EVAPORATION PRODUCES AN ADAPTIVE INCREASE IN CUTANEOUS EVAPORATION IN MOURNING DOVES *ZENAIDA MACROURA*

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Summary

We tested the hypothesis that birds can rapidly change the conductance of water vapor at the skin surface in response to a changing need for evaporative heat loss. Mourning doves (*Zenaida macroura*) were placed in a two-compartment chamber separating the head from the rest of the body. The rate of cutaneous evaporation was measured in response to dry ventilatory inflow at three ambient temperatures and in response to vapor-saturated ventilatory inflow at two ambient temperatures. At 35 °C, cutaneous evaporation increased by 72 % when evaporative water loss from the mouth was prevented, but no increase was observed at 45 °C. For both dry and vapor-saturated treatments, cutaneous evaporation increased significantly with increased ambient temperature. Changes in skin temperature made only a minor contribution to any observed increase in cutaneous evaporation. This indicates that *Z. macroura* can effect rapid adjustment of evaporative conductance at the skin in response to acute change in thermoregulatory demand.

Key words: evaporative water loss, cutaneous evaporation, thermoregulation, conductance, bird, mourning dove, *Zenaida macroura*.

Introduction

Homeothermy requires continual adjustment of one or more of five possible heat fluxes (conduction, convection, radiation, evaporation and metabolism), such that the sum of these fluxes remains at or near zero. Metabolism always represents a heat gain of appreciable magnitude for an endotherm. On hot sunny days, when environmental conditions are such that each of the heat fluxes due to conduction, convection and radiation is positive, homeothermy can persist only if evaporative heat flux is sufficient to dissipate all the heat added to the body *via* the other four routes.

Water has a high latent heat of vaporization that is only weakly dependent on the temperature of the water being evaporated (Harrison, 1963). The evaporation of water is therefore a highly endergonic process that is well-suited to the demands of heat dissipation (Calder and Schmidt-Nielsen, 1967; Crawford and Schmidt-Nielsen, 1967; MacMillen and Trost, 1967; Dawson and Bartholomew, 1968; Moldenhauer, 1970; Schleucher et al., 1991). Biologists discriminate two spatially and physiologically distinct routes of evaporative heat loss. Evaporation from the external surface of an animal is variously called ‘cutaneous evaporation’, ‘transepidermal evaporation’ or ‘peripheral evaporation’; that from the pharyngeal or buccal epithelia is usually called ‘respiratory evaporation’ or ‘pulmonary evaporation’. We use ‘cutaneous’ to describe the former evaporative route (although this may include ocular as well as cutaneous evaporation) because the term is well-established. We use ‘ventilatory’ to describe the latter route, so as to encompass evaporation by normal breathing, hyperventilation, panting and gular flutter.

The capacity for thermally significant evaporation from the skin of birds has long been disputed (Menon et al., 1986) because of the lack of avian sweat glands. However, it is now firmly established that many bird species are able to dissipate substantial amounts of heat by cutaneous evaporation of water (Bernstein, 1971a,b; Lasiewski et al., 1971; Dawson, 1982; Marder and Ben-Asher, 1983; Marder et al., 1989). Depending on ambient temperature (T_a), cutaneous evaporation can even account for the majority of evaporation (Webster and King, 1987).

On energetic grounds, it seems logical that cutaneous evaporative water loss could be important in birds, because all modes of ventilatory evaporative water loss entail some gain of heat in the forms of friction in the musculature involved and of the metabolism required to power the muscles. Cutaneous evaporation appears to be a passive diffusional process, wherein the rate at which vapor escapes from the external surface is simply a function of the vapor density gradient and the resistance to diffusion. Considered in this light, cutaneous evaporation would occur at no energetic cost and should, therefore, be favored over ventilatory evaporation.

Such an assessment of these processes is, however, overly simplistic. First, both routes of evaporative heat loss are ultimately passive, diffusional processes. They differ only in the site of the epithelium from which vapor escapes. Also, both

evaporative routes might entail energetic costs. Whereas ventilatory evaporation requires muscular work for the convective flow of air across the normally moist pharyngeal and buccal epithelia, cutaneous evaporation requires the delivery of water to the stratum corneum. The mechanism of such delivery might, in itself, be energetically costly. In addition, the circulatory shunt presumably required for delivery of water to the stratum corneum must involve a convective redistribution of heat within the body. This could have appreciable thermal or physiological consequences.

With the importance of cutaneous evaporation for heat dissipation well-established in many avian species, the question arises whether birds can regulate cutaneous evaporative heat flux. Regulation could be accomplished, for instance, by changing the rate of water delivery to the stratum corneum, the cutaneous resistance to diffusion, or both. Nevertheless, a direct test of the ability of birds to adjust cutaneous evaporation in response to change in heat load is lacking. Directly addressing this question entails experimentally changing the rate of cutaneous evaporation required for thermostasis, while keeping conductive, convective and radiative conditions constant. Increasing the thermostatic need for cutaneous evaporation can be accomplished by decreasing ventilatory evaporation. In this study, we minimized the ability of mourning doves (*Zenaida macroura*) to evaporate water from the pharyngeal and buccal epithelia by placing them in a two-compartment respirometry chamber and sending water-saturated air into the ventilatory compartment (thereby drastically reducing or eliminating the ventilatory vapor pressure gradient), while sending dry air into the cutaneous compartment.

Materials and methods

Adult mourning doves (*Zenaida macroura* Linnaeus) of undetermined sex were captured in late January 1998 in Tempe, Arizona, USA, and subsequently housed in a temperature-controlled room on the campus of Arizona State University. The room was maintained at 30 °C under a 12 h:12 h L:D photoperiod. For 15 min prior to and following full illumination, a low-output light source was turned on to graduate the artificial day/night transitions. Birds were housed in pairs in metal mesh cages (61 cm×42 cm×61 cm) and given food and water *ad libitum*.

Measurements were made using a 10.9 l, two-compartment, respirometry chamber (28.6 cm×20.4 cm×18.7 cm), constructed on five sides of aluminum and on the sixth of Plexiglas to allow constant monitoring of the subject. An aluminum partition separated the ventilatory volume (3583 ml) from the cutaneous volume (7327 ml). An 8 cm×8 cm opening in the aluminum partition was spanned with latex sheeting (Semantodontics Dental Dam), into which a hole was cut to accommodate the neck of the experimental subject. The size of the hole was such that the neck very slightly stretched the latex, thus sealing the ventilatory volume from the cutaneous volume while allowing unimpeded breathing. An aluminum stock placed above the latex sheet prevented the head from being pulled through the latex. Unless the subject struggled, the stock did not restrain it. If a subject struggled for more than a few seconds, the trial was terminated and the data were discarded. Subjects stood in a natural posture, with the feet on a stainless-steel grid that allowed for the passage of excreta into a bath of non-volatile mineral oil, precluding the contribution of excreta to measurements of vapor density.

The respirometry chamber was placed in a temperature-controlled room that also served to isolate the subjects sonically. Measurements were made in total darkness, and subjects were monitored under infrared light using a CCD camera (Magnavox 18MC205T). Separate ventilatory- and cutaneous-chamber ambient temperatures were continuously measured using copper-constantan thermocouples.

With the subject in place, the ventilatory portion of the respirometry chamber was sealed from the cutaneous portion. Each portion was equipped with its own influx and efflux ports. The influx ports were fitted with diffusion plates to facilitate mixing of chamber air.

Measurements were made at 35, 45 and 50 °C for trials in which dry, CO_2-free influent was sent to both chambers (‘dry’ trials). At 35 and 45 °C, measurements were also made using vapor-saturated, CO_2-free ventilatory influent and dry, CO_2-free cutaneous influent (‘wet’ trials). We deemed 50 °C wet trials to be too dangerous for the subjects, but we collected data for 50 °C dry trials to test whether rates of evaporation during 45 °C wet trials represented absolute maxima.

Influent air was scrubbed of CO_2 and dried by an air purifier (Puregas CDA1) before being sent through rotameters (Omega FL3405ST, calibrated against a soap-film flowmeter). For the dry trials, air exiting the rotameters was sent through 3.2 mm i.d. tubing (Li-Cor Bev-A-Line) directly to the chamber inlets. During wet trials, air exiting the rotameters was diverted to a sealed, plastic cylinder (206 cm, 7.6 cm i.d.) serving as a hydration chamber. The air line entering the hydration chamber branched to terminate in several porous aquarium aerators at the floor of the hydration chamber. These were used to increase the number and decrease the size of bubbles introduced into the water column. Air was bubbled through a 165 cm column of distilled water before exiting the hydration chamber, and the water was continuously circulated through an external metal coil immersed in a water bath equipped with a temperature controller (Cole-Parmer Dyna·Sense, model 2156). Both the hydration chamber and the water bath were placed in the temperature-controlled room housing the test chambers to ensure that influent air was vapor-saturated at the ambient temperatures encountered in the experiment. In case hydration chamber temperature exceeded ventilatory chamber temperature, air exiting the hydration chamber was sent to a glass vessel to collect any condensed water before being sent to the ventilatory chamber. Preliminary measurements were made on wet-trial influent, using a dew-point hygrometer (EG&G, model 911), yielding dew-point temperatures equalling ventilatory-chamber ambient temperatures. These measurements, and the appearance of condensate in the glass

vessel, leave us confident that wet trials were conducted with vapor-saturated ventilatory influent. Since the rotameters were upstream of the hydration chamber, ventilatory airflow was corrected to reflect the addition of vapor to the airstream.

Effluent from both test chambers was sent to a capacitance hygrometer (Thunder Scientific PC2101) before passing through a desiccant (Drierite) and then into an infrared CO_2 analyzer (Li-Cor, model LI6252). The CO_2 analyzer was used only to supplement our visual assessment of quiescence in the subjects; data were collected when subjects appeared to stand still and when the CO_2 analyzer indicated a relatively flat and low-level output. A glass vessel was interposed between the ventilatory chamber and the hygrometer to collect condensate from the saturated, ventilatory effluent during wet trials. This protected the hygrometer from water that would have condensed because gas analysis was conducted in a room much cooler than the test chambers.

We calibrated the hygrometer by passing vapor-saturated air at 23 °C through a thermocouple-equipped copper coil immersed in calcium chloride brines of various concentrations and cooled to slurry with frozen CO_2. Emerging air was brought to room temperature and sent to the hygrometer. Hygrometric readings were thus measurements of vapor density, not relative humidity. The CO_2 analyzer was calibrated daily using pure nitrogen and a CO_2/N_2 mixture of known composition as zero and span gases, respectively.

We tried unsuccessfully to monitor core temperature (Minimitter transmitter implant) and cloacal temperature (thermocouple probe); both procedures proved too injurious to the birds. Skin temperature was measured continuously during 45 and 50 °C trials using a 40 AWG copper-constantan thermocouple soldered to a rectangle of copper foil (approximately 0.25 cm^2) attached using cyanoacrylic glue to the ventral apterium immediately posterior to the sternum. Unfortunately, we did not develop the technique for cutaneous thermometry until all but one of the 35 °C trials were completed.

The order of treatment (dry or wet) for any one temperature was randomized. No bird was subjected to more than one trial in a single day. Most trials were conducted at a particular temperature before proceeding to the next temperature, because the test room and the water in the hydration chamber required hours for equilibration following a change of temperature.

Subjects were placed in the thermally equilibrated respirometry chamber and monitored for at least 30 min before any data were collected. Transient and infrequent twitches were permitted but, in general, data were collected after the subject had been still for at least 8 min (the time for 99 % chamber-air turnover, after Lasiewski et al., 1966).

Data for temperatures (air, skin and water), vapor density and CO_2 content were sampled every second and averaged every 60 s while being recorded on a datalogger (Campbell Scientific CR23X). The data were continuously monitored on a computer running data-acquisition software (Campbell Scientific PC208W). Effluent from either test chamber was serially shunted to the gas analysis system by solenoid valves. Ventilatory measurements were thus temporally displaced (by approximately 5 min) from cutaneous measurements, although all measurement sequences were made during the same near-steady state with respect to CO_2 and vapor density.

Statistical analyses

All data were subjected to paired *t*-tests to determine the significance of differences between treatments, with $P<0.05$ being considered significant. Each *t*-test was conducted twice: once with all available data, and once with outliers removed. An outlier was defined, using a two-sample *t*-test, as any datum differing at the 0.01 significance level from the remaining data taken as a group. In all but one of the *t*-tests, removal of outliers made no difference to the significance. However, in comparing evaporative conductance (g_v) between 45 and 50 °C dry trials, removal of outliers indicated a change from no significance ($P>0.082$) to significance ($P<0.014$).

Although less frequently reported in the literature than resistance, we report values for conductance to indicate ease of evaporative flux. For any given skin temperature (T_s), conductance varies directly with flux density, whose variance is assumed to be normally distributed. Therefore, variance in conductance (and not its reciprocal, resistance) is normally distributed, and we retain the power of the parametric, paired *t*-test, which assumes normality.

All values are presented as means ± the standard error of the mean (S.E.M.). All values for *P* are results of paired *t*-tests.

Results

Evaporative water loss

At an ambient temperature (T_a) of 35 °C, cutaneous evaporation underwent a significant ($P<0.006$) increase of 72 % between dry trials and wet trials (Fig. 1). At T_a=45 °C,

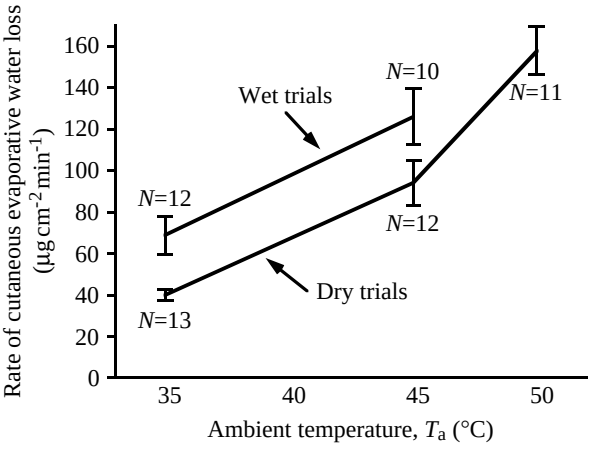

Fig. 1. Cutaneous evaporative flux-density at three ambient temperatures. Values are means ± S.E.M. The difference in cutaneous evaporation at T_a=35 °C between wet and dry trials is significant ($P<0.006$).

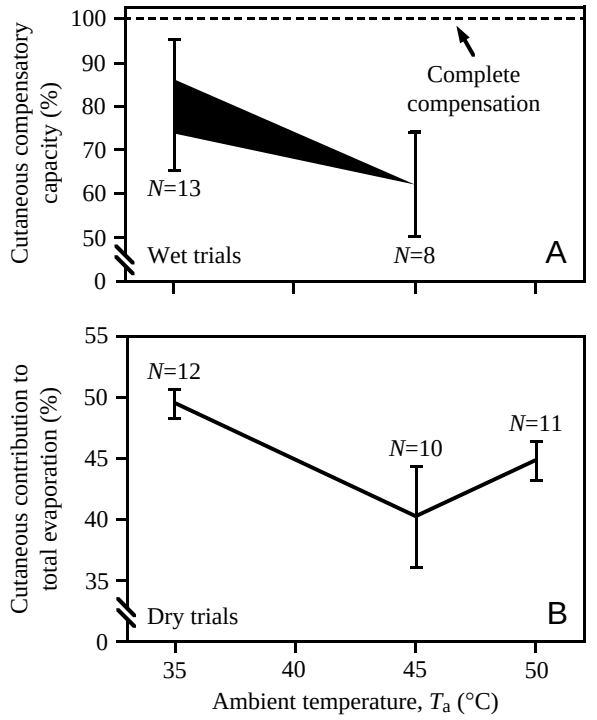

Fig. 2. (A) Cutaneous compensatory capacity. Wet-trial cutaneous evaporation expressed as a percentage of dry-trial total evaporation indicates the degree of compensation, *via* increased cutaneous evaporation, for the elimination of a ventilatory contribution to water loss during wet trials. The range of means at T_a=35 °C reflects the possibility that ventilatory evaporation was not completely eliminated at that ambient temperature (see text). (B) Cutaneous contribution to total evaporation. Dry-trial cutaneous evaporation expressed as a percentage of dry-trial total evaporation indicates normal partitioning of ventilatory and cutaneous components of evaporation. Values are means ± S.E.M.

however, there was no significant increase from dry trials to wet trials (P>0.057). Ambient temperature strongly affected cutaneous evaporation (Fig. 1). For dry trials, it increased by 135 % (P<0.002) from T_a=35 °C to T_a=45 °C. Similarly, it increased by 68 % (P<0.004) from T_a=45 °C to T_a=50 °C. For wet trials, cutaneous evaporation increased by 83 % (P<0.004) from T_a=35 °C to T_a=45 °C.

In dry trials, the effect of T_a on total evaporation was just as strong. At T_a=35 °C, total evaporation was 10.72±1.16 mg min^{-1}. At T_a=45 °C, this increased by 99 % to 21.30±1.69 mg min^{-1} (P<0.0005), and a further 61 % increase occurred at T_a=50 °C when total evaporation reached 34.34±2.58 mg min^{-1} (P<0.007).

The relative contribution of cutaneous evaporation to total evaporation in dry trials changed only slightly with changes in T_a, accounting for 49.6±1.2 % of the total at T_a=35 °C, 40.3±4.3 % at T_a=45 °C and 44.9±1.6 % at T_a=50 °C. Only values for T_a=35 °C and T_a=50 °C differed significantly (P<0.012, Fig. 2B).

Cutaneous compensatory capacity (CCC) was calculated as the wet-trial cutaneous evaporation divided by the dry-trial

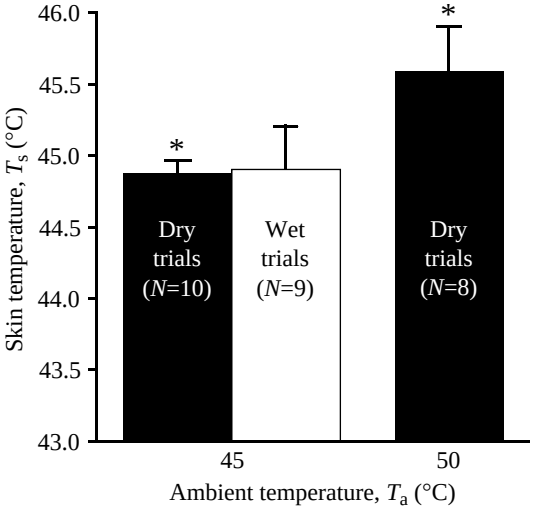

Fig. 3. Effects of ambient temperature on skin temperature. Values are means + S.E.M. Asterisks indicate significantly different values (P<0.002).

total evaporation and expressed as a percentage (Fig. 2A). Defined in this way, CCC represents the degree to which a bird can increase its cutaneous evaporation to make up for a decrease in ventilatory evaporation. At T_a=35 °C, CCC was between 74.0±8.3 % and 86.4±9.4 % (see explanation in Discussion). At T_a=45 °C, the CCC fell to 62.5±12.1 %, which is a decrease of between 15.5 % (P<0.007) and 27.7 % (P<0.0008) compared with the range at T_a=35 °C.

Skin temperature

Mean skin temperatures (T_s) are reported in Fig. 3. For T_a=45 °C (the only trials for which T_s was measured for both ventilatory-influent treatments), no significant change (P>0.23) in T_s occurred between dry (44.9±0.094 °C) and wet (44.9±0.30 °C) trials. In dry trials for which T_s was measured, values for T_s increased significantly (P<0.002) from 44.9±0.094 °C at T_a=45 °C to 45.6±0.32 °C at T_a=50 °C. The single animal for which T_s was measured at T_a=35 °C had a dry-trial T_s of 40.3 °C and a wet-trial T_s of 43.0 °C.

Evaporative conductance

The conductance of water vapor (g_v) is defined as the ratio of evaporative flux density (mass of water per unit surface area of skin per unit time, g m^{-2} s^{-1}) to vapor-density gradient (difference in absolute humidity between skin and air, g m^{-3}). Therefore, by cancellation, g_v takes units of m s^{-1}. This is in keeping with the fact that g_v is the reciprocal of resistance to water-vapor diffusion (r_v), which is usually expressed in s m^{-1}.

The calculation of g_v requires knowledge of T_s and, therefore, values of g_v are reported only for the 45 and 50 °C trials (Fig. 4). At T_a=45 °C, there was no significant change (P>0.058) in g_v between dry trials (245±30 μm s^{-1}) and wet trials (316±37 μm s^{-1}). Among dry trials, g_v increased by 71 % (P<0.014) from 245±30 μm s^{-1} at T_a=45 °C to 420±8.0 μm s^{-1} at T_a=50 °C.

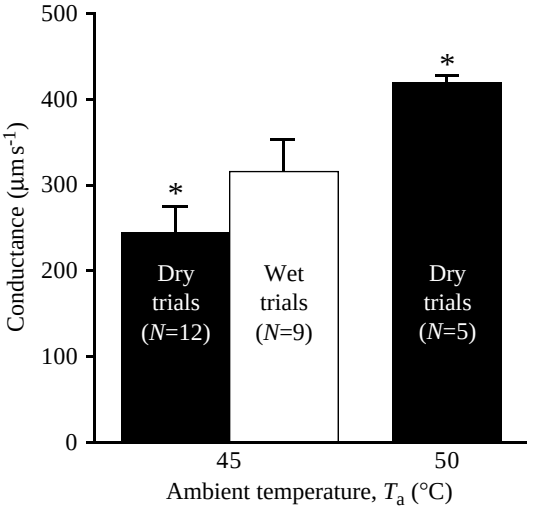

Fig. 4. Effects of ambient temperature on evaporative conductance. Values are means + S.E.M. Asterisks indicate significantly different values (P<0.014).

Discussion

Our data clearly indicate a substantial increase in the rate of cutaneous evaporation in mourning doves when ambient temperature is 35 °C and ventilatory evaporation is greatly reduced compared with cutaneous evaporation at the same ambient temperature but with uninhibited ventilatory evaporation. However, there are two peculiarities of the 35 °C trials that must be addressed: the absence of skin temperature measurements and the possibility that ventilatory evaporation was not eliminated during wet trials.

Possibility of ventilatory evaporation during 35 °C wet trials

While the incurrent air during wet trials had a dew-point temperature of 35 °C, this may have been insufficient to eliminate the ventilatory vapor-density gradient. If portions of the pharyngeal epithelium had temperatures in excess of 35 °C during these wet trials (and therefore vapor densities exceeding that of the influent), then some ventilatory evaporation could have occurred. Since condensation from the excurrent air precluded ventilatory hygrometry during wet trials, we were unable to measure how much ventilatory evaporation (if any) was occurring. We assumed that the birds attained a maximal lung temperature of 40 °C. This would allow for ventilatory evaporation across an 11.5 μg ml^{-1} vapor-density gradient (the saturation vapor density of 40 °C air minus that of 35 °C air). Thus, cutaneous compensatory capacity at T_a=35 °C was calculated as a range from 74 % (no ventilatory evaporation) to 86 % (ventilatory evaporation across an 11.5 μg ml^{-1} vapor-density gradient), as shown in Fig. 2A. Our estimate of 40 °C for lung temperature is based on measurements of body temperature of columbiforms at an ambient temperature of approximately 35 °C (Lasiewski and Seymour, 1972; Dawson and Bennet, 1973; Webster and King, 1987; Withers and Williams, 1990; Prinzinger et al., 1991) coupled with the assumption that lung temperature is at least slightly reduced, by evaporation, compared with body temperature. A regression (Schmidt-Nielsen et al., 1970) of exhaled air temperature on T_a for pigeons yields an exhaled air temperature slightly above 35 °C at T_a=35 °C. If this is typical of columbiforms, then the low end of the range (74 %) may be the most realistic estimate of cutaneous compensatory capacity.

Absence of empirical data for skin temperature during 35 °C trials

Except for one bird, skin temperatures were not measured during 35 °C trials. This precludes calculation of g_v at T_a=35 °C and allows for the proposal that the 72 % increase in cutaneous evaporation in response to curtailment of ventilatory evaporation is due to a change in skin temperature rather than to a change in conductance of water vapor. Indeed, this proposal seems appealing in the light of the fact that cutaneous evaporation in 45 °C wet trials was not significantly increased over that of 45 °C dry trials. Several lines of evidence exist, however, to suggest that most of the increase in cutaneous evaporation during wet trials at 35 °C must have been due to a substantial shift in g_v.

First, the single dove for which T_s was measured during the 35 °C dry trial had a skin temperature of 40.3 °C. Although this unique measurement carries little statistical weight, a dry-trial T_s of 40.3 °C is unlikely to be atypically high. Mourning doves kept at 4 °C overnight have been shown to maintain skin temperatures of approximately 39 °C (Bartholomew and Dawson, 1954). The same study demonstrated that skin temperature is independent of ambient temperature (remaining between 39 and 40 °C) up to approximately 30 °C. Above this, skin temperature rises slightly. It is unlikely, therefore, that the doves in the present study, held by a restraint in a chamber at an ambient temperature of 35 °C, had skin temperatures below 39 °C during dry trials.

Second, at least three independent studies of columbiforms (Bartholomew and Dawson, 1954; Randall, 1943; von Saalfeld, 1936) have shown that panting does not occur until body temperature reaches a threshold of between 42 and 43 °C. In the present study, doves were monitored continuously, and no panting occurred during any 35 °C trials. Thus, it is reasonable to assume that body temperature was less than 43 °C throughout these trials. Since, in the absence of irradiance, body temperature must exceed skin temperature when body temperature is higher than ambient temperature, wet-trial skin temperatures could not have exceeded 43 °C and were probably considerably lower. The overall range of skin temperature at an ambient temperature of 35 °C (wet and dry trials combined) was almost certainly, therefore, within the 39–43 °C range (Fig. 5).

Change in cutaneous evaporation decoupled from change in skin temperature

The range of probable skin temperatures (39–43 °C) is much too narrow to account for the observed increases in cutaneous evaporation between dry trials and wet trials, assuming a constant evaporative conductance. An analysis of superlative scenarios reveals why.

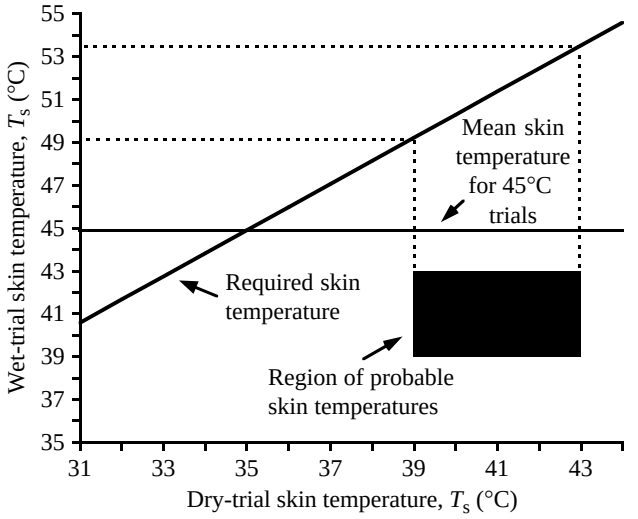

Fig. 5. Wet-trial skin temperatures necessary for observed evaporation if conductance did not change. Values along the curve were calculated from data for evaporation at T_a=35 °C (N=12) assuming the dry-trial skin temperatures indicated on the abscissa. The filled region indicates the boundaries of probable skin temperatures (see text). Dashed lines indicate the range of required wet-trial skin temperatures assuming that dry-trial skin temperatures were between 39 and 43 °C.

Assuming that skin temperature for 35 °C wet trials is 43 °C (above which panting would occur), then evaporative conductance can be calculated as:

$$g_{v35} = \frac{CEWL_{35W}}{\rho'_v(43\,°C)}\,, \qquad (1)$$

where g_{v35} is the evaporative conductance at T_a=35 °C, $CEWL_{35W}$ is the wet-trial, cutaneous evaporative water loss at T_a=35 °C and ρ'_v(43 °C) is the saturation vapor density of air at 43 °C. The latter equals the vapor-density gradient, since the influent is dry. Assuming that g_v at T_a=35 °C is the same for dry trials and wet trials, then the dry-trial skin temperature necessary for the increase in cutaneous evaporation to depend entirely on a change in skin temperature is:

$$\begin{aligned} T_{sD} &= T_D(\Delta\rho_v) \\ &= T_D\left(\frac{CEWL_{35D}}{g_{v35}}\right), \end{aligned} \qquad (2)$$

where T_{sD} is the dry-trial skin temperature, $\Delta\rho_v$ is the vapor-density gradient, $T_D(\Delta\rho_v)$ is the dew-point temperature for a vapor density equal to $\Delta\rho_v$, and $CEWL_{35D}$ is the dry-trial, cutaneous evaporative water loss at T_a=35 °C. The calculation predicts a dry-trial skin temperature of 33 °C when ambient temperature is 35 °C. This is not credible for two reasons. First, a T_s of 33 °C would mean that doves are undergoing a full 10 °C shift in skin temperature (with no change in T_a) to compensate for a reduction in ventilatory evaporation. Furthermore, this 10 °C shift would occur without any attempt to shed heat by panting. Second, a T_s of 33 °C is lower than any skin temperature reported by Bartholomew and Dawson (1954), even for mourning doves exposed to ambient temperatures as low as 3 °C.

It is also possible, given a dry-trial T_s, to calculate the wet-trial skin temperature required for the increase in cutaneous evaporation to be due entirely to a change in skin temperature (based solely on data for T_a=35 °C). Fig. 5 shows that none of the required temperatures falls within the region of likelihood, based on the absence of panting.

Finally, although it is possible for an animal to have a skin temperature below both ambient temperature and body temperature, maintaining such a skin temperature would involve both of the following: (1) that the animal is gaining heat across the thermal gradient from environment to skin, and (2) that the animal is evaporating water cutaneously at a rate sufficient to maintain both the thermal gradient from environment to skin and the thermal gradient from skin to body core. This is, at best, an unlikely scenario at T_a=35 °C.

Components of water-vapor conductance

A change in evaporative conductance need not be entirely due to a change in skin conductance, because g_v is a measure of total water-vapor conductance. Total conductance is a combination of constituent conductances at the skin, at the plumage and at the boundary layer. While depth of plumage was not measured, we observed no change in the appearance of the plumage of any animal, whether from dry trial to wet trial at any given ambient temperature or between different ambient temperatures. Moreover, Webster et al. (1985) made direct measurements of constituent water-vapor resistances in pigeons and found boundary-layer resistance to be negligible compared with those at the skin and plumage. In addition, for ambient temperatures between 10 and 40 °C, they showed that plumage resistance to water-vapor diffusion is only approximately 5–20 % of total vapor resistance. This means that vapor conductance at the skin constrains g_v over this range of ambient temperatures, and that plumage conductance probably only becomes important when skin conductance is high (i.e. at high rather than moderate ambient temperatures).

Relative effects of skin temperature and evaporative conductance on cutaneous evaporation

Skin temperatures were measured for all 45 and 50 °C trials, which enabled us to make empirical evaluations of the relative contributions made by changes in g_v and T_s to the significant increase in cutaneous evaporation found between the 45 and 50 °C dry trials. For all dry trials, influent vapor density, ρ_v, was zero, so the vapor-density gradient, $\Delta\rho_v$, was just the saturation vapor density of air at skin temperature, $\rho'_v(T_s)$. The change in cutaneous evaporation between 45 °C dry trials and 50 °C dry trials can therefore be calculated as:

$$\begin{aligned} \Delta CEWL &= CEWL_{50} - CEWL_{45} \\ &= (\Delta\rho_{v50} g_{v50}) - (\Delta\rho_{v45} g_{v45}) \\ &= [\rho'_v(T_{s50}) g_{v50}] - [\rho'_v(T_{s45}) g_{v45}]\,, \end{aligned} \qquad (3)$$

where symbols are defined as before, with numbers in subscripts indicating the ambient temperature of the trial. Using individual values for Δg_v and ΔT_s (i.e. measured differences, within individual birds, in values for g_v and T_s, between 45 and 50 °C trials), we calculated two predicted values for $\Delta CEWL$, for comparison with empirical values for $\Delta CEWL$. One predicted value ($\Delta CEWL_1$) assumed no change in g_v (i.e. $g_{v50}=g_{v45}$); the other ($\Delta CEWL_2$) assumed no change in T_s (i.e. $T_{s50}=T_{s45}$). This gives:

$$\begin{aligned}\Delta CEWL_1 &= [\rho'_v(T_{s50})g_{v50}] - [\rho'_v(T_{s45})g_{v45}]\\ &= [\rho'_v(T_{s50})g_{v45}] - [\rho'_v(T_{s45})g_{v45}]\\ &= g_{v45}[\rho'_v(T_{s50}) - \rho'_v(T_{s45})] \qquad (4)\end{aligned}$$

and

$$\begin{aligned}\Delta CEWL_2 &= [\rho'_v(T_{s50})g_{v50}] - [\rho'_v(T_{s45})g_{v45}]\\ &= [\rho'_v(T_{s45})g_{v50}] - [\rho'_v(T_{s45})g_{v45}]\\ &= \rho'_v(T_{s45})[(g_{v50} - g_{v45})]\,. \qquad (5)\end{aligned}$$

The mean change in evaporation assuming no change in conductance (from equation 4) was only 12 % of the observed change; the mean change in evaporation assuming no change in skin temperature (from equation 5) was 88 % of the observed change. This means that most of the increase in cutaneous evaporation between the 45 and 50 °C dry trials was caused by an increase in g_v rather than an increase in T_s, despite the fact that both g_v and T_s changed significantly between those ambient temperatures. The change of less than 1 °C in T_s from T_a=45 °C to T_a=50 °C was significant because variance was low. However, despite the steeply increasing relationship between saturation vapor density and ambient temperature, a temperature increase from 44.7 to 45.6 °C allows only a 4.4 % increase (Flatau et al., 1992) in saturation vapor density (from approximately 64 μg ml^{-1} to approximately 67 μg ml^{-1}). Since the influent was dry for all trials, the vapor-density gradient was simply the saturation vapor density of air at a temperature equal to skin temperature, and any increase in cutaneous evaporation that was driven wholly by an increase in T_s must have been directly proportional to the increase in the vapor-density gradient, which was relatively small. In contrast, mean dry-trial conductance increased by approximately 48 % from T_a=45 °C to T_a=50 °C, thereby accounting for the overwhelming majority of the 49 % increase in cutaneous evaporation.

In the present investigation, the overall range of mean values for cutaneous evaporation is from 40 μg cm^{-2} min^{-1} (at T_a=35 °C) to 158 μg cm^{-2} min^{-1} (at T_a=50 °C). This is an increase of 295 %. Our highest individual value for T_s was 46.6 °C at T_a=50 °C. The vapor density gradient at this skin temperature is 70.3 μg ml^{-1}, which is a 295 % increase from a gradient of 17.8 μg ml^{-1}. The dew-point temperature of air containing 17.8 μg ml^{-1} of water vapor, and therefore the dry-air T_s required for that gradient, is 19.6 °C. Birds with such low skin temperatures at T_a=35 °C would have been noticeably cool to the touch. Thus, it is quite unlikely that changes observed in cutaneous evaporation with changes in T_a are largely due to changes in T_s.

Despite the change in conductance across ambient temperatures, g_v did not increase at T_a=45 °C from dry trials to wet trials. This is somewhat surprising, because the values for g_v at T_a=50 °C clearly indicate that g_v was not maximized at T_a=45 °C, despite the need (based on observations of panting) for doves at that T_a to shed more heat during wet trials than they actually did. A definitive explanation must await elucidation of the mechanism by which birds are able to adjust g_v to acute changes in T_a.

Mechanisms for adjusting water-vapor conductance

Previous studies by other investigators have examined possible ways in which birds can adjust cutaneous evaporation to meet changing needs for heat loss. Menon et al. (1988) observed that the increase in cutaneous evaporation of zebra finches (*Poephila guttata*) from nestling to adult could be explained by a comparative abundance of lipid bodies (vacuoles and multigranular bodies) in nestling epidermis. This finding is bolstered by the results of a separate study (Menon et al., 1989) on zebra finches, in which deprivation of water caused both an increase in intercellular deposition of the contents of multigranular bodies in adult epidermis and a change in composition of epidermal lipids, with a concomitant decrease in cutaneous evaporation. Rehydration served to reverse both effects of dehydration. These studies are, however, concerned with comparatively long-term changes (ontogeny or acclimation) rather than acute changes in cutaneous evaporation, as observed in mourning doves.

Marder and Raber (1989) elicited very large changes in skin resistance to water-vapor diffusion in pigeons. Oral administration of a β-receptor blocker (propranolol) caused an increase in cutaneous evaporation *via* a global decrease in skin resistance. Similarly, intradermal injection of propranolol caused a local reduction in skin resistance. Changes took effect within 1–5 min of injection. Marder and Raber (1989) suggest that endogenous chemical transmitters, whether neural or humoral, control avian cutaneous evaporation by reversing the vasoconstrictive effect of tonic stimulation of β-receptors in the cutaneous smooth vasomusculature.

While long-term changes in g_v might be effected by changes in structure and function of epidermal lipids and multigranular bodies (Elias and Menon, 1991; Menon et al., 1991, 1996), short-term control of g_v might be exercised by displacing the constraint on diffusion of water. In this way, evaporative conductance would usually be constrained by the rate of delivery of water to the epidermis, a physiological property of the animal dependent on the current state of cutaneous vasoconstriction, which is under neural and hormonal control. During prolonged heat-stress, g_v would be constrained by conductance of water vapor at the skin surface, an anatomical property of the epidermis, under acclimatory control.

Concluding comments

In conclusion, this study and others (e.g. Marder and Ben-

Asher, 1983; Webster and King, 1987) demonstrate that cutaneous evaporation is an important means of thermoregulatory heat dissipation in birds. Our results provide the first evidence that birds are able adaptively to adjust their rates of cutaneous evaporation. This previously unappreciated capacity for physiological adjustment of cutaneous water-vapor conductance represents an expansion of the known set of thermoregulatory strategies used by birds.

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