

# Thermoregulatory ability of female rats during pregnancy and lactation

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KNECHT, EDWIN A., MARK A. TORAASON, AND GARY L. WRIGHT. *Thermoregulatory ability of female rats during pregnancy and lactation*. *Am. J. Physiol.* 239 (Regulatory Integrative Comp. Physiol. 8): R470-R475, 1980.—Thermoregulatory ability of female rats was examined before pregnancy, during gestation, and during lactation. Thermoregulatory pattern, colonic temperature, evaporative water loss, and survival time were monitored during terminal heating ( $39.5 \pm 0.9^\circ\text{C}$ ) designed to allow prolonged survival (3–4 h) with a sustained thermoregulatory effort. Results confirmed our previously reported observation of decreased thermoregulatory ability in lactating dams, with evidence suggesting thermoregulatory impairment during late gestation. Lactating dams displayed a type III thermoregulatory pattern, and established a rate of evaporative water loss effective for thermostasis at an elevated colonic temperature. However, survival time was significantly decreased compared to nonreproducing females. In contrast, prior heat acclimation tended to increase the survival time of lactating dams. It was concluded that the reduction in thermoregulatory ability observed in lactating dams was related to their inability to maintain a rate of evaporative water loss effective for thermostasis at an elevated colonic temperature.

terminal heat exposure; thermoregulatory pattern; thermostasis; evaporative water loss; colonic temperature; survival time; acclimation

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TEMPERATURE REGULATION in rats exposed to elevated environmental temperatures ( $36\text{--}44^\circ\text{C}$ ) depends primarily on the grooming of saliva onto vascularized body surfaces for evaporative cooling (6, 8, 17). Female rats have been shown to be less tolerant to heat exposure than males (6), and recently we reported a decreased thermoregulatory ability in reproducing female rats exposed to daily short-term heat treatment (14). Exposures were well tolerated during gestation; however, dams became increasingly intolerant to heat exposures during lactation, and maternal deaths at approximately 14 days postpartum were coincident with colonic temperatures exceeding  $42^\circ\text{C}$ . Decreased thermoregulatory ability has been observed following impairment of evaporative water loss by desalivation and dehydration (6, 16), thereby suggesting that the decreased thermoregulatory ability observed in lactating dams may be related to an impairment of evaporative water loss.

The present study examined the ability of female rats to effectively utilize body water for evaporative cooling

during exposure to an elevated environmental temperature before pregnancy, during gestation, and during lactation. Thermoregulatory pattern, evaporative water loss, colonic temperature, and survival time were monitored in females exposed to heating conditions designed to allow prolonged survival with a sustained thermoregulatory effort.

## MATERIALS AND METHODS

**Animals.** Female Sprague-Dawley-derived rats (200 to 225 g) obtained from Charles River Breeding Laboratories were grouped as shown in Table 1. Animals were housed at  $24 \pm 1^\circ\text{C}$ , relative humidity 40–50%, with a 12 h light (0700–1900):12-h dark (1900–0700) photoperiod. Tap water and Purina lab chow were available ad libitum. *Group 1* females served as nonreproducing controls. *Groups 2–5* were cohoused with proven males and the presence of spermatozoa in the vaginal smears denoted *day 0* of gestation. *Groups 2* and *3* were evaluated at mid- and late gestation, respectively. *Groups 4* and *5* were allowed normal delivery and litters were randomly reduced to 10 pups shortly after birth. Litter size and pup weights were recorded at 5-day intervals. To examine the effect of heat acclimation on thermoregulatory ability during lactation, dams from *group 5* were exposed daily during lactation to a 50-min (0900–0950) heat acclimation period (environmental temperature was  $38 \pm 1^\circ\text{C}$ ).

**Thermoregulatory evaluation.** A previous report published from this laboratory (18) has indicated that thermoregulatory ability during exposure to elevated environmental temperatures can best be determined by heating an animal to the point of recognizable thermoregulatory impairment. The point of thermoregulatory breakdown can be determined by monitoring colonic temperature until thermostasis is no longer maintained. Heat exposures were additionally continued beyond thermoregulatory breakdown to determine the relationship between evaporative water loss and colonic temperature during the final stage of hyperthermia. Exposures were terminated when death occurred (cessation of respiratory movement).

Terminal heat exposures and evaporative water loss determinations were conducted in an open-flow system similar to that described by Hainsworth (7). Females were placed in a 2.8-liter glass chamber located inside a 480-liter incubator maintained at  $39.5 \pm 0.9^\circ\text{C}$ . Room air

TABLE 1. Grouping of female rats for determination of thermoregulatory ability

Group	n	Thermoregulatory Evaluation	Body Wt, g
1. Nonreproducing females	7	12 wk of age	237 ± 7
Pregnant dams			
2. Midgestation	6	day 11 of gestation*	260 ± 7
3. Late gestation	8	day 20 of gestation†	325 ± 14
4. Lactating dams	5	21 days postpartum	332 ± 8
5. Heat-acclimated lactating dams	6	21 days postpartum	323 ± 12

Values are means ± SE. \* Two dams were evaluated on days 9 and 12, respectively. † One dam was evaluated on day 19.

was pumped (8 l/min) through columns of Drierite desiccant located inside the incubator, thereby drying the air and equilibrating its temperature. The air was then introduced into the bottom of the animal chamber, equipped with a 2-cm mesh wire floor over 2.5 cm of mineral oil that prevented evaporation from urine and feces. Water evaporated from the animal was collected for weighing by passing the chamber air through a series of Drierite columns. Samples were obtained at 15-min intervals and evaporative water loss calculated on a body weight basis. Colonic temperature was monitored during heat exposure by a thermistor probe inserted 5–6 cm through the rectum and taped to the tail.

**Data analysis.** Colonic heating curves were analyzed by segment line construction as described by Wright et al. (18). The resting temperature ( $T_r$ ) at the beginning of heat exposure, the equilibrium temperature ( $T_{eq}$ ) corresponding to the colonic temperature at the first inflection point of the curve, the thermoregulatory breakdown temperature ( $T_{bk}$ ) corresponding to the second inflection point, and the lethal temperature ( $T_l$ ) were determined, as well as the time from  $T_r$  to  $T_{eq}$  (segment 1),  $T_{eq}$  to  $T_{bk}$  (segment 2), and  $T_{bk}$  to  $T_l$  (segment 3).

Females were classified into one of three thermoregulatory types, based on their colonic heating pattern (18). Type I females exhibited a linear heating pattern from  $T_r$  to  $T_l$ . Type II subjects showed an initial rapid rate of colonic temperature elevation that preceded a relatively well-defined convexity of the curve, resulting in a secondary rate of heating, which was maintained to the lethal temperature. Type III animals were identified by a three-stage heating pattern. The first two segments of the curve were similar to that in type II curves except that the decrease in the rate of heating during the second segment was more well defined and was followed by a dramatic elevation of colonic temperature (segment 3) to the lethal temperature.

Significance of differences between data in groups 1–4 was determined by Dunnett's test (Table 2). Groups 4 and 5 were compared by *t* test and Cochran's test where preliminary analyses indicated that variances were not homogeneous (15). Data presented in Table 3 and Fig. 3 were analyzed for significant differences using *t* test and Dunnett's test, respectively. In all analyses, the level of significance chosen was  $P < 0.05$ . To aid in the interpretation of nonsignificant differences,  $\beta$  (type II error) probabilities were calculated (5). Data are presented throughout the text as mean ± SE.

## RESULTS

Segmental analysis of colonic heating curves revealed a type III thermoregulatory response to terminal heat exposure in all groups with the exception of the type II thermoregulatory pattern observed in three dams during late gestation (Table 2). Compared to nonreproducing females, survival time was significantly decreased in lactating dams, and in type II dams, but not type III ( $\beta_{60 \text{ min}} = 0.13$ ),<sup>1</sup> during late gestation. Heat acclimation tended to increase the survival time of lactating dams compared to nonacclimated lactating dams, although differences were not statistically significant ( $\beta_{60 \text{ min}} = 0.84$ ). Segment 1 and 3 heating times were similar ( $\beta_{15 \text{ min}} = 0.20$ ) between control and experimental groups, except for the significant increase in segment 1 heating time observed in type II dams during late gestation. In contrast, segment 2 heating time tended to differ markedly ( $\beta_{30 \text{ min}} = 0.79$ ) between control and experimental groups and was highly correlated ( $P < 0.0001$ ) with survival time in all groups (Fig. 1).

Resting temperature was significantly reduced during mid- and late gestation (type II) compared to nonreproducing controls (Table 2). Lactating dams displayed a significant elevation in equilibrium temperature, thermoregulatory breakdown temperature, and lethal temperature. The mean temperature recorded during segment 2 was significantly reduced during gestation and significantly increased during lactation. Heat-acclimated lactating dams displayed a significantly elevated resting temperature and a significantly decreased lethal temperature compared to group 4 lactating dams.

A comparison of colonic temperature and evaporative water loss during terminal heating demonstrated that water evaporation increased rapidly in all groups, peaked shortly after  $T_{eq}$  was observed, and remained relatively constant during segment 2 (Fig. 2). Nonreproducing females and dams in midgestation displayed similar ( $\beta_{4 \text{ g} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}} = 0.10$ ) and relatively constant rates of evaporative water loss during segment 2 (Table 2). The rate of water evaporation declined during segment 2 among type III dams in late gestation, however, the mean rate of evaporative water loss during segment 2 did not differ significantly ( $\beta_{4 \text{ g} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}} = 0.20$ ) from that of nonreproducing females. Type II dams in late gestation demonstrated a limited ability to establish a constant rate of water evaporation during segment 2. Peak values were comparable to those observed in type III dams during late gestation, but evaporation declined rapidly thereafter. Lactating dams displayed a significantly higher mean rate of evaporative water loss during segment 2 compared to nonreproducing females (Table 2). A large initial peak of water evaporation was followed by a rapid decline; however, a second peak was observed near the end of segment 2. In contrast, heat-acclimated lactating dams displayed a relatively constant rate of water evaporation during segment 2. In all groups, evaporative water loss declined rapidly during segment 3, reaching a final value of 5–9 g · kg<sup>-1</sup> · h<sup>-1</sup>.

Evaporative water loss did not differ significantly be-

<sup>1</sup> The subscript indicates the true mean difference detectable at the corresponding  $\beta$  value.

TABLE 2. Data resulting from segmental analysis of colonic heating curves

Group	Thermo-regulatory Type	n	Survival Time, min	Duration of Heating Segments, min			Colonic Temperatures, °C					EWL <sub>Seg 2</sub> † g·kg <sup>-1</sup> ·h <sup>-1</sup>
				1	2	3	T <sub>r</sub>	T <sub>eq</sub>	T <sub>bk</sub>	T <sub>1</sub>	T <sub>Seg 2</sub> *	
1. Nonreproducing females	III	7	223.3±10.0	24.9±1.6	150.9±10.9	47.6±1.5	38.2±0.2	40.6±0.2	41.2±0.2	44.5±0.2	40.6±0.1	22.9±0.9
Pregnant dams												
2. Midgestation	III	6	272.5±19.1	29.5±2.6	200.5±21.0	42.5±6.2	37.6±0.1‡	40.2±0.2	41.1±0.3	44.6±0.1	40.2±0.1‡	24.8±0.7
3. Late gestation	II	3	135.0±10.5‡	41.0±8.2‡	94.0±2.7	—	37.3±0.2‡	40.7±0.3	—	44.4±0.1	—	—
	III	5	211.2±8.2	29.4±1.5	132.6±10.1	49.2±2.6	37.9±0.2	40.1±0.2	40.9±0.2	44.4±0.2	40.2±0.1‡	23.3±0.9
4. Lactating dams	III	5	159.0±24.1‡	28.2±2.4	96.2±23.6	34.6±3.4	38.5±0.1	41.8±0.2‡	42.1±0.2‡	45.1±0.1‡	41.5±0.1‡	31.9±1.4‡
5. Heat-acclimated lactating dams	III	6	250.5±42.0	24.0±3.6	188.5±44.5	38.0±4.8	39.1±0.2§	41.7±0.2	42.4±0.2	44.9±0.2§	41.3±0.1	30.5±1.2

Data are presented as mean ± SE. \* Colonic temperature recorded during segment 2. † Rate of evaporative water loss during segment 2. ‡ Significantly different from group 1 (P < 0.05). § Significantly different from group 4 (P < 0.05).

TABLE 3. Effect of maternal heat acclimation on indices of lactation

Group	Days Postpartum														
	1			6			11			16			21		
	Mat wt	Litter size	Pup wt	Mat wt	Litter size	Pup wt	Mat wt	Litter size	Pup wt	Mat wt	Litter size	Pup wt	Mat wt	Litter size	Pup wt
4. Lactating dams	286	10.0	6.0	307	10.0	12.5	326	10.0	21.9	329	9.8	29.7	339	9.8	39.8
	±8	±0.0	±0.2	±8	±0.0	±0.3	±7	±0.0	±0.6	±7	±0.2	±0.9	±8	±0.2	±0.9
5. Heat-acclimated lactating dams	283	10.0	6.3	288	10.0	11.7	298*	10.0	17.1*	313	10.0	24.4*	321	10.0	33.7*
	±5	±0.0	±0.1	±8	±0.0	±0.3	±7	±0.0	±0.9	±9	±0.0	±1.3	±8	±0.0	±1.7

Data are presented as mean ± SE. Mat wt (maternal body wt) and pup wt in grams. \* Significantly different from group 4 (P < 0.05).

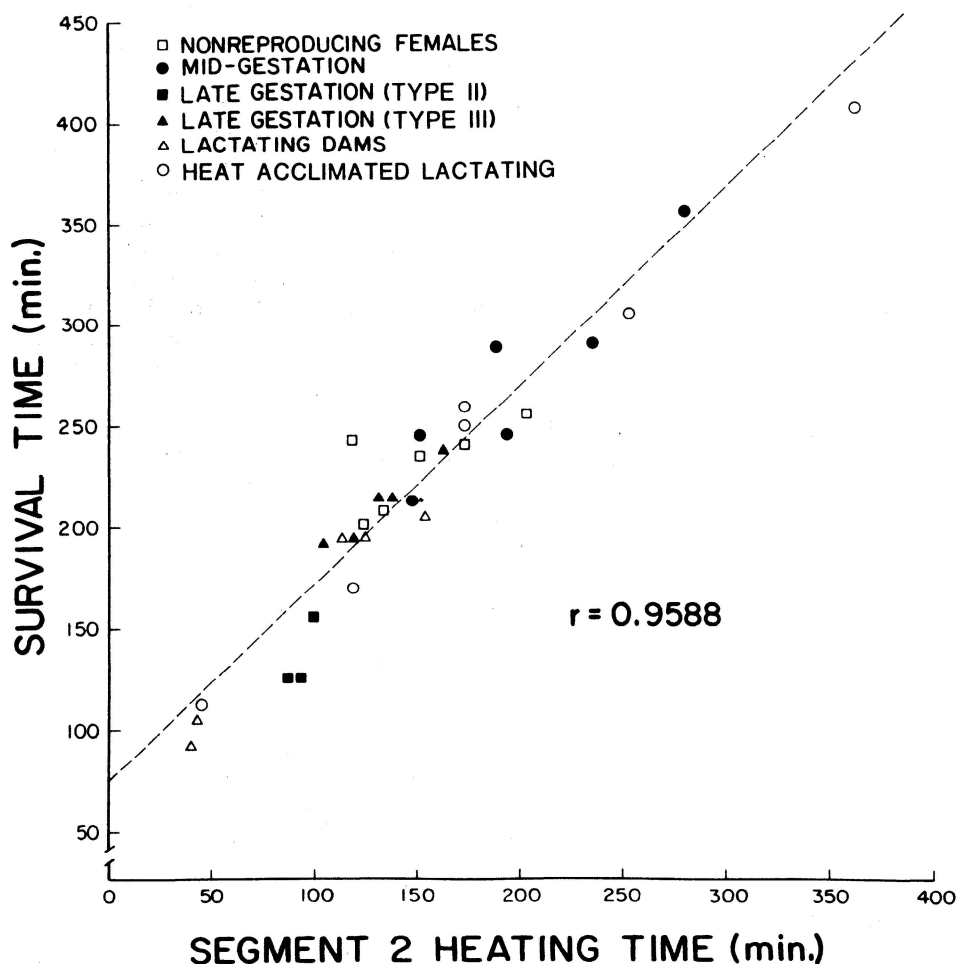


FIG. 1. Correlation between segment 2 heating time and survival time of female rats in different reproductive states. Least squares regression line corresponding to correlation is shown.

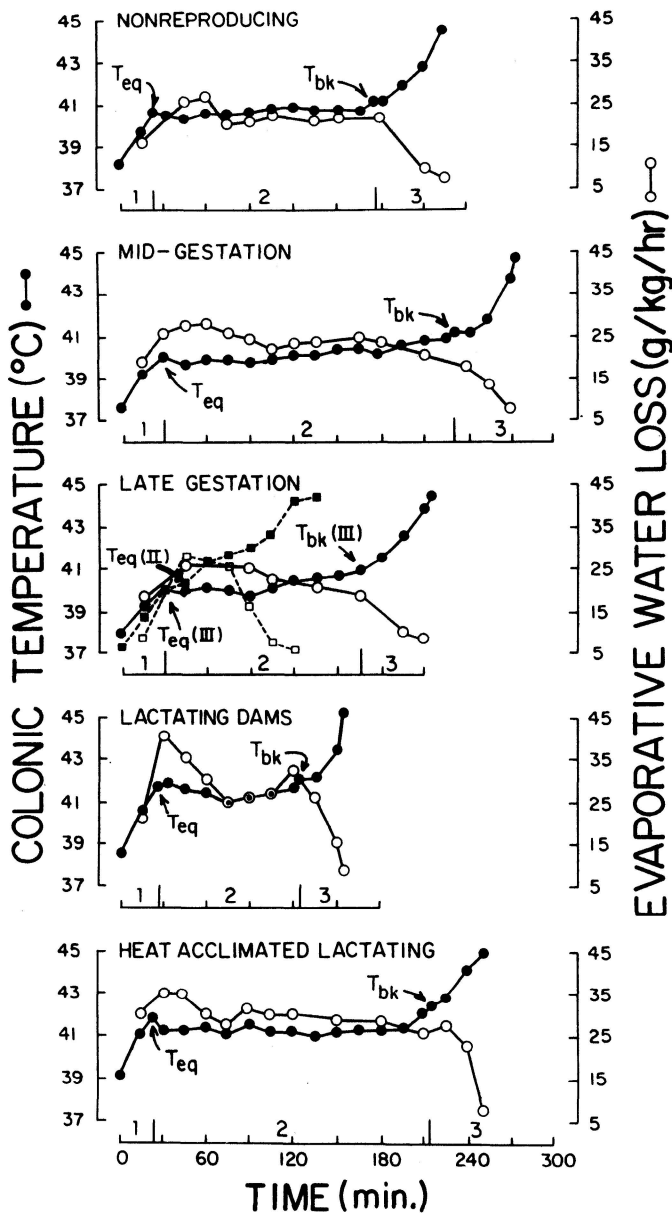


FIG. 2. Evaporative water loss and colonic temperature response of female rats to terminal heat exposure in different reproductive states. Heating segments are shown on time axis. Type II dams in late gestation are presented by squares.

tween nonreproducing females and dams in mid- or late gestation with regard to maximal rate ( $\beta_{8 \text{ g} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}} = 0.18$ ) or the colonic temperature ( $\beta_{0.8^\circ\text{C}} = 0.15$ ) at which the peak occurred (Fig. 3). In contrast, lactating dams showed a significantly higher peak rate of water loss ( $41.2 \pm 2.2 \text{ g} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$ ) at a significantly higher colonic temperature ( $41.6 \pm 0.2^\circ\text{C}$ ) compared to nonreproducing controls ( $26.5 \pm 1.4 \text{ g} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$ ;  $40.3 \pm 0.2^\circ\text{C}$ ). Heat-acclimated lactating dams ( $38.2 \pm 3.6 \text{ g} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$ ;  $41.3 \pm 0.4^\circ\text{C}$ ) did not differ significantly ( $\beta_{12 \text{ g} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}} = 0.22$ ;  $\beta_{1.4^\circ\text{C}} = 0.22$ ) from lactating dams, however, evaporative water loss tended to be greater in heat-acclimated dams at lower ( $39\text{--}40^\circ\text{C}$ ) and higher ( $43\text{--}44^\circ\text{C}$ ) colonic temperatures.

Maternal body weights increased consistently in groups 4 and 5 during lactation (Table 3). Heat acclimation had no significant effect ( $\beta_{35 \text{ g}} = 0.15$ ) on maternal

body weight, except at 11 day postpartum. Mean pup weights increased consistently in both lactating groups, but were significantly lower in the heat-acclimated group at 11, 16, and 21 days postpartum. However, maternal heat acclimation during lactation had no effect on pup survival, indicating that heat acclimation had a negligible effect on lactation.

DISCUSSION

The thermoregulatory ability of female rats was markedly influenced by reproductive status. Results confirmed our previously reported observation of decreased thermoregulatory ability in lactating dams (14), with evidence suggesting a thermoregulatory impairment during late gestation. Survival time was highly correlated with segment 2 heating time in all groups. As recently discussed by Wright et al. (18), this indicates that differences in survival time are primarily related to alterations in active thermoregulatory processes rather than passive factors affecting heat gain or loss.

Evaporative water loss was clearly related to the pattern of colonic heating in all groups. Evaporative water loss increased during segment 1 and peaked shortly following the establishment of  $T_{eq}$ . A subsequent balance of heat gain from the environment, endogenous heat production, and heat loss by the animal was established during segment 2, indicating that thermostasis was achieved at a higher colonic temperature by an increase in evaporative water loss. Evaporative cooling by the rat during active temperature regulation in hot environments ( $36\text{--}44^\circ\text{C}$ ) depends primarily upon voluntary grooming of saliva onto vascularized surfaces (6, 8, 17). Salivary water loss is replenished by concomitant water loss from plasma and other tissues, producing intravascular and intracellular dehydration (9). Although the physiological factors limiting evaporative water loss dur-

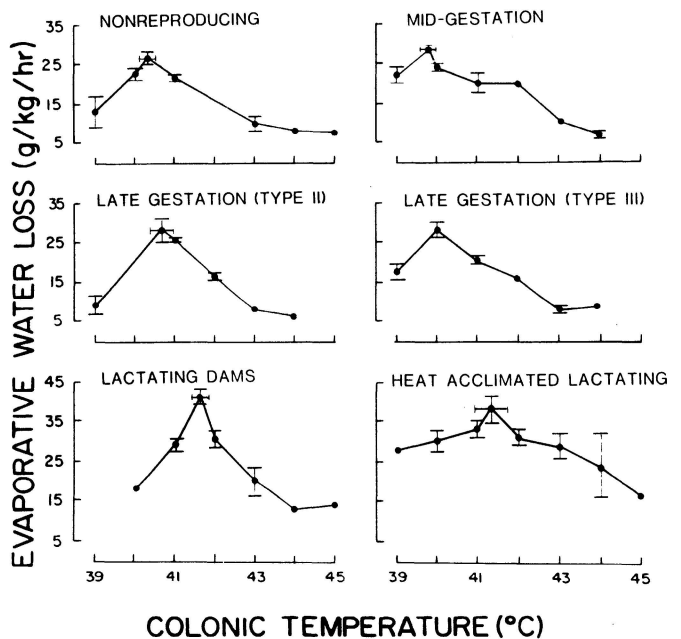


FIG. 3. Rate of evaporative water loss as a function of colonic temperature in female rats exposed to terminal heating. Data are presented as mean  $\pm$  SE.

ing heat exposure are not fully known, it appears that increasing hypovolemia and hypertonicity result in cardiovascular shock and increasing hyperthermia (10, 16). Failure of the heat-dissipating mechanism thereby results in virtually unregulated elevation of colonic temperature and death during segment 3.

The decreased survival time of lactating dams is consistent with observations previously reported by this laboratory (14). However, it appeared that the decreased survival time is not attributable to an inability of lactating dams to establish a rate of water loss effective for evaporative cooling as previously suggested, but rather to the inability to sustain an adequate evaporative cooling effort. Evaporative water loss increased with 15 min of heat exposure (Fig. 2), and the rate of evaporative water loss at a colonic temperature of 40°C was similar to that observed in nonreproducing controls (Fig. 3). However, thermostasis was established at a higher colonic temperature, but at a correspondingly greater rate of evaporative water loss compared to nonreproducing females, suggesting that decreased survival time was due to an accelerated rate of body water depletion for evaporative cooling in response to elevated heat load. An increase in metabolic rate has been observed during lactation (2) suggesting the endogenous heat load imposed on lactating dams may exceed that of nonreproducing females. Furthermore, several published reports (6, 11, 12, 17) have demonstrated that the colonic temperature achieved during thermostasis is positively correlated to the environmental temperature and thus the thermal load on the animal. Hence, the combined metabolic and environmental heat load may be greater in lactating dams, thus explaining the increased colonic temperature and rate of body water depletion for evaporative cooling during segment 2 thermostasis. Biochemical and cellular alterations have been observed at or above 41.5°C (1, 3, 4), and a colonic temperature of 41.5°C has been suggested as an upper limit for unimpaired physiological function in rats (18). The occurrence of colonic temperatures exceeding 41.5°C in lactating dams during segment 2 suggests that heat-induced tissue damage may have also contributed to the decreased thermoregulatory ability of these animals.

Heat acclimation tended to increase the survival time of lactating dams. However, the rate of evaporative water loss and the colonic temperature during thermostasis (segment 2) were similar in lactating and heat-acclimated lactating dams (Table 2, Fig. 2), indicating that the increased survival time observed in heat-acclimated

dams did not involve a reduction in the heat load or rate of body water depletion. Although the maximal rate of evaporative water loss observed in heat-acclimated lactating dams did not differ significantly from that observed in lactating dams of *group 4* and occurred at approximately the same colonic temperature (Fig. 3), the rate of evaporative water loss tended to be elevated at lower colonic temperatures, thereby suggesting a more advantageous initiation of evaporative water loss in heat-acclimated dams. Recently, heat acclimation has been shown to result in a preferential conservation of plasma and extracellular volume at the expense of intracellular fluid volume during heat exposure, thereby maintaining the cutaneous circulation necessary for heat dissipation (13). This suggests that heat acclimation increased the survival time of lactating dams by also evoking a more effective regulation of body water for prolonged evaporative cooling.

The decreased survival time observed in pregnant dams during late gestation is attributed to the inability of three dams (type II) to establish a rate of evaporative water loss effective for thermostasis at an elevated colonic temperature. However, the fact that the five remaining dams examined during late gestation demonstrated a thermoregulatory response similar to controls suggests that the impairment was confined to a portion of the group which, for unexplained reasons, was less resistant than the majority of dams in late gestation. The mechanism responsible for the alternative thermoregulatory response to heat exposure exhibited by the type II dams may be similar to that in lactating dams, but may also be related to uncontrolled factors such as litter size and the proximity of labor and delivery.

In summary, thermoregulatory ability was reduced in reproducing female rats during lactation and, to some degree, during late gestation. The thermoregulatory impairment of lactating dams was related to their inability to sustain an adequate rate of evaporative water loss effective for thermostasis at an elevated colonic temperature. Alterations in metabolic heat production, plasma volume regulation, and other factors associated with pregnancy and lactation may contribute to this phenomenon.

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## REFERENCES

1. BRAUER, R. W., R. W. BALAM, H. C. BOND, H. W. CARROLL, J. W. GRISHAM, AND R. L. PESSOTTI. Reversible and irreversible changes in liver at temperatures approaching critical upper level. *Federation Proc.* 22: 724-728, 1963.
2. BRODY, S., J. RIGGS, K. KAUFMAN, AND V. HERRING. Energy metabolism levels during gestation, lactation and post-lactation rest. *Univ. MO Agric. Exp. Stn. Res. Bull.* 281, 1938.
3. BUCHSBAUM, R., J. PERCHERSKY, AND D. MCKIBBEN. *Effects of Heat Stress on Cellular Structure and Function*. Wright-Patterson Air Force Base, OH: Aerospace Med. Res. Lab., 1970. (AMRL-TR-70-1)
4. BURGER, F. J., AND F. A. FUHRMAN. Evidence of injury to tissues after hyperthermia. *Am. J. Physiol.* 206: 1062-1064, 1964.
5. DIXON, W. J., AND F. J. MASSEY, JR. *Introduction to Statistical Analysis* (3rd ed.). New York: McGraw, 1969, p. 75-94.
6. HAINSWORTH, F. R. Saliva spreading, activity, and body temperature regulation in the rat. *Am. J. Physiol.* 212: 1288-1292, 1967.
7. HAINSWORTH, F. R. Evaporative water loss from rats in the heat. *Am. J. Physiol.* 214: 979-982, 1968.
8. HAINSWORTH, F. R., AND A. N. EPSTEIN. Severe impairment of heat-induced saliva-spreading in rats recovered from lateral hypothalamic lesions. *Science* 153: 1255-1257, 1966.
9. HAINSWORTH, F. R., E. M. STRICKER, AND A. N. EPSTEIN. Water metabolism of rats in the heat: dehydration and drinking. *Am. J. Physiol.* 214: 983-989, 1968.
10. HARRISON, M. H., R. J. EDWARDS, AND P. A. FENNESSY. Intravas-

- cular volume and tonicity as factors in the regulation of body temperature. *J. Appl. Physiol.: Respirat. Environ. Exercise Physiol.* 44: 69-75, 1978.
11. HELLSTROM, B. Heat vasodilatation of the rat tail. *Can. J. Physiol. Pharmacol.* 53: 202-206, 1975.
  12. HERRINGTON, L. P. The heat regulation of small laboratory animals at various environmental temperatures. *Am. J. Physiol.* 129: 123-139, 1940.
  13. HOROWITZ, M., S. SAMUELOFF, AND J. H. ADLER. Acute dehydration: body water distribution in acclimated and nonacclimated *Psammomys obesus*. *J. Appl. Physiol.: Respirat. Environ. Exercise Physiol.* 44: 585-588, 1978.
  14. KNECHT, E. A., G. L. WRIGHT, AND M. A. TORAASON. Periodic, short-term heat exposure and reproductive function in male and female rats *Can. J. Physiol. Pharmacol.* 56: 747-753, 1978.
  15. SNEDECOR, G. W., AND W. G. COCHRAN. *Statistical Methods*. Ames, IA: Iowa State Univ. Press, 1967.
  16. STRICKER, E. M., AND F. R. HAINSWORTH. Evaporative cooling in the rat: effects of dehydration. *Can. J. Physiol. Pharmacol.* 48: 18-27, 1970.
  17. STRICKER, E. M., AND F. R. HAINSWORTH. Evaporative cooling in the rat: interaction with heat loss from the tail. *Q. J. Exp. Physiol.* 56: 231-241, 1971.
  18. WRIGHT, G., E. KNECHT, AND D. WASSERMAN. Colonic heating patterns and the variation of thermal resistance among rats. *J. Appl. Physiol.: Respirat. Environ. Exercise Physiol.* 43: 59-64, 1977.

