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The effect of He-O_2 exposure on metabolic rate, thermoregulation and thermal conductance during normothermia and daily torpor

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Abstract Recently it was proposed that the low metabolic rate during torpor may be better explained by the reduction of thermal conductance than the drop of body temperature or metabolic inhibition. We tested this hypothesis by simultaneously measuring body temperature and metabolic rate as a function of ambient temperature in both torpid and normothermic stripe-faced dunnarts, *Sminthopsis macroura* (Marsupialia; approx. 25 g body mass), exposed to either air or He-O_2 (21% oxygen in helium) atmospheres. He-O_2 exposure increases the thermal conductance of homeothermic mammals by about twofold in comparison to an air atmosphere without apparent side-effects. Normothermic *S. macroura* exposed to He-O_2 increased resting metabolic rate by about twofold in comparison to that in air because of the twofold increase in apparent thermal conductance. Torpid *S. macroura* exposed to He-O_2 at ambient temperatures above the set-point for body temperature showed a completely different metabolic response. In contrast to normothermic individuals, torpid individuals significantly decreased or maintained a similar metabolic rate as those in air although the apparent thermal conductance in He-O_2 was slightly raised. Moreover, the metabolic rate during torpor was only a fraction of that of normothermic individuals although the apparent thermal conductance differed only marginally between normothermia and torpor. Our study shows that a low thermal conductance is not the reason for the low metabolic rates during torpor. It suggests that interrelations between metabolic rate and body temperature of torpid endotherms above the set-point for body temperature differ fundamentally from those of normothermic and homeothermic endotherms.

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Key words Daily torpor · Metabolic rate reduction · Thermoregulation · He-O_2 atmosphere · *Sminthopsis macroura*

Abbreviations T_a ambient temperature · T_b body temperature · *BMR* basal metabolic rate · *C* apparent thermal conductance · *He-O_2* 21% oxygen in helium · *MR* metabolic rate · *MSe* mean square-error · *RMR* resting metabolic rate · *TMR* metabolic rate during torpor · ΔT, difference T_b-T_a · *TNZ* thermoneutral zone · T_{set} set-point for body temperature · $\dot{V}O_2$ rate of oxygen consumption

Introduction

Daily torpor is characterised by substantial drop in MR, T_b and other physiological functions (Wang 1989; Geiser and Ruf 1995). The most widely held view in the literature and the one generally presented in physiological text books is that the reduction in MR during torpor (TMR) below the BMR is caused by the fall in T_b (Snapp and Heller 1981; Geiser 1988), because the Q_{10} for steady-state MR of many species is between 2 and 3 which is characteristic of many biological reactions. Since during entry into torpor and at high T_b during torpor TMRs below those predicted by temperature effects have been observed, it was concluded that metabolic inhibition, in addition to temperature effects, may cause the low TMR (Malan 1986, 1993; Geiser 1988, 1993; Storey and Storey 1990; Nicol et al. 1992; Guppy et al. 1994).

The use of Q_{10} for describing the fall in TMR in endotherms was recently challenged (Snyder and Nestler 1990; Heldmaier and Ruf 1992). Since *C* is often lower in torpid than in normothermic individuals, it was proposed that low *C* during torpor, together with the small difference between T_b and T_a (ΔT) during torpor, may be responsible for the low TMR rather

than, or in addition to, the fall of T_b or inhibition of MR (Snyder and Nestler 1990).

To test this hypothesis, we simultaneously measured MR, T_b and T_a during normothermia and torpor in both air (about 21% oxygen in nitrogen) and He-O_2 (21% oxygen in helium) atmospheres. Helium, similar to nitrogen, is an inert carrier gas. However, the thermal conductance of homeothermic mammals in He-O_2 is about twofold higher in comparison to that in air (Rosenmann and Morrison 1974; Dawson et al. 1986; Hallam and Dawson 1993). Other effects on respiratory physiology are not apparent (Brice and Welch 1983). The experimental animal was the stripe-faced dunnart, *Sminthopsis macroura*, a small nocturnal marsupial which readily displays daily torpor over a range of T_a (Geiser and Baudinette 1985; Song et al. 1995).

Material and methods

Eight laboratory-bred adult male *S. macroura* were used. Animals were maintained individually in cages (30 × 22 × 14 cm) containing nest-boxes. Bedding consisted of sawdust and shredded paper which was renewed once a week when cages were washed. The photoperiod throughout the experiment was 12L:12D (lights on 0600–1800 hours) and the T_a was 20 ± 2 °C. A mixture of canned dog food and macerated cat food pellets supplemented with calcium and vitamins was supplied daily. *Tenebrio* larvae were provided occasionally and water was available *ad libitum*.

MR was measured as $\dot{V}O_2$. Animals were placed into 0.5-l respiratory chambers within a temperature-controlled cabinet (± 0.5 °C). Four channels, three animal channels and one reference channel, were scanned with solenoid valves. Each channel was read for 3 min (i.e. the $\dot{V}O_2$ of each individual was measured every 12 min in comparison to outside air). The flow rate (about 250 ml · min^{-1}) of dry gas through the respiratory chambers was controlled with rotameters (7908, Aarlborg, New York, USA), measured with a mass flowmeter (FMA-5606, Omega, Stamford, USA). A correction factor was applied to the He-O_2 flow rates because of the different physical properties of this gas. Oxygen content of gas leaving the respiratory chamber and in the reference chamber was consecutively measured with a single channel oxygen analyser (Ametek Applied Electrochemistry S-3A/1, Pittsburgh) fitted with a high resolution output board (80335SE).

Temperature-sensitive transmitters (Model X-M, Mini-mitter, Sunriver accuracy ± 0.1 °C) were calibrated to the nearest 0.1 °C against a calibrated precision mercury thermometer (R.6578, Dobros, Australia) in a water bath between 5 and 40 °C. The transmitters were implanted intraperitoneally under Halothane anaesthesia. At least seven days were allowed for recovery from the surgery. The transmitter signal from each individual was received with a ferrite rod antenna that was multiplexed to a receiver and transformed to a square-wave signal after subtraction of background noise. T_a in the respiratory chamber was measured to the nearest 0.1 °C by a thermocouple inserted about 1 cm into the metabolic chamber. Thermocouple output was amplified by a digital thermometer (Omega DP116). The T_b and T_a of each channel were also determined every 12 min together with MR.

Analog outputs from the flowmeter, oxygen analyser, transmitter receiver and digital thermometer were interfaced to a personal computer via a 14 Bit A/D card. Data acquisition and processing were performed with software written by B. Lovegrove, T. Ruf and G. Körtner.

For determination of TMR, animals were kept in the respiratory chambers from late afternoon for about 1 day at constant T_as ranging from 17.2 to 19.0 °C (mean 18.0 ± 0.7 °C; referred to as T_a 18 °C), which is close to the T_a where the species defends its T_b during torpor (T_{set}), and at T_as ranging from 22.0 to 24.7 °C (mean 23.2 ± 0.7 °C; referred to as T_a 23 °C), which is well above the set-point for T_b for this species during torpor. Animals were either exposed to air throughout the measurement or they were initially exposed to an air atmosphere which during the night was exchanged with a He-O_2 atmosphere (Fig. 1). RMR in both atmospheres was measured when animals were resting in the late afternoon or after they had aroused from torpor on the following day. RMR was also determined during about 6-h measurements during the day time at constant T_a. Measurements of BMR were carried out in air atmosphere between 0930 and 1700 hours after animals had been in the chambers for at least 2 h; T_a was increased progressively from about 25 to 36 °C in about 1.5-°C temperature increments lasting for at least 2 h each. Body weight was recorded before and after each experiment. Food and water were not available during measurements.

RMR was determined from the mean of the lowest consecutive $\dot{V}O_2$ values and their corresponding T_b and T_a values observed over at least 36 min in normothermic, resting individuals below the TNZ. BMR was determined as the mean of the lowest $\dot{V}O_2$ measurements of normothermic individuals over at least 36 min within the TNZ and their corresponding T_b and T_a values. Torpor was defined as MR < 75% of RMR at the same T_a and a T_b below 30 °C. TMR, T_b and T_a during torpor were obtained by calculating the mean of the consecutive lowest $\dot{V}O_2$ values and their corresponding T_b and T_a values measured over at least 36 min. Only steady-state values from torpor bouts that lasted longer than 2 h were taken into account. A linear decrease of body weight throughout each experiment was assumed for calculation of mass-specific MR. The body mass did not differ between atmospheres and the mean body mass for all measurements of MR was 24.7 ± 2.2 g.

The mass-specific C was calculated using the equation:

$$C = \frac{MR}{T_b - T_a}.$$

Q_{10} was calculated using the equation:

$$Q_{10} = \left(\frac{MR_2}{MR_1}\right) \exp\left(\frac{10}{T_{b_2} - T_{b_1}}\right).$$

Data are presented as mean ± 1 standard deviation of the number of individuals (n) measured. Differences between means were determined by initially testing the F-ratio for group variances and then applying a t-test for either equal or unequal variances as appropriate. Two-way ANOVA was applied to test for effects of T_a and atmosphere. Linear regressions were fitted using the method of least squares (Zar 1984). Differences between regressions were initially determined by testing the MSe by ANOVA. If the MSe was indistinguishable by ANOVA, differences in slope were tested using a t-test (Zar 1984).

Results

Pronounced daily fluctuations of MR and T_b were observed in *Sminthopsis macroura* in both air and He-O_2 atmosphere as shown for one individual in Fig. 1. A period of rest in the afternoon with relatively low T_b and RMR (Fig. 1A; around 1700 hours) was followed by a period of activity after lights off as indicated by the increase in MR and T_b. The MR during activity in air was about twice the RMR (Fig. 1A). The animal entered torpor before midnight, but the change from air to He-O_2 at midnight induced a brief period of arousal

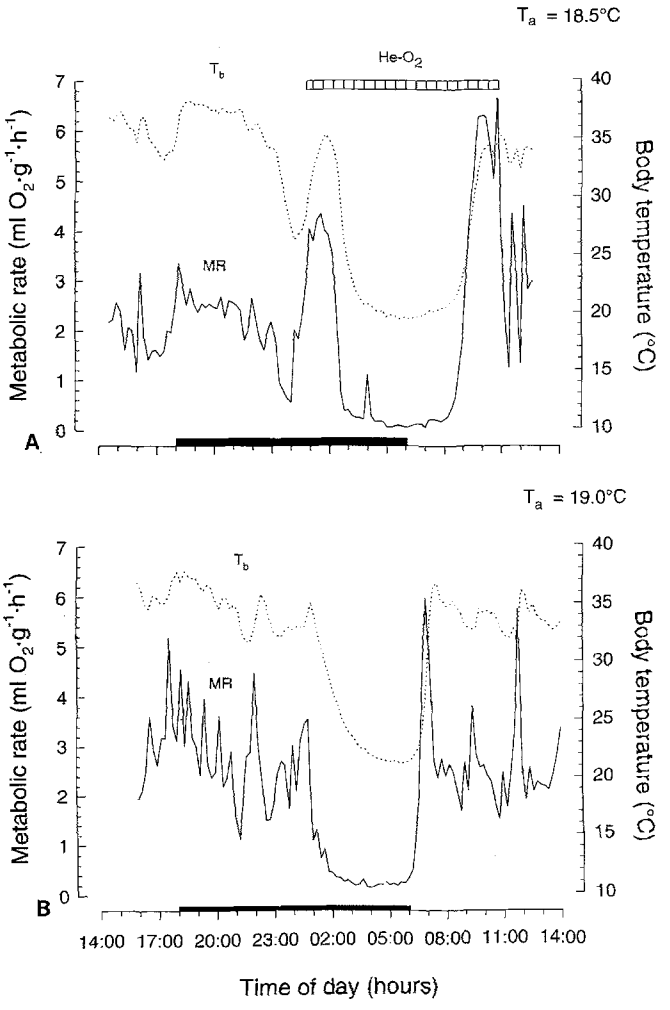

Fig. 1 Daily fluctuations in metabolic rate and body temperature of *Sminthopsis macroura* Sm22 in air and He-O_2 at ambient temperatures (T_a) of 19.0 and 18.5 °C. In **A** the animal was kept in air atmosphere from the beginning of the measurement until midnight; exposure to He-O_2 lasted from midnight until 1050 hours, when air was reintroduced. In **B** air atmosphere was maintained throughout the measurement. The *black bars* represent the dark phase

followed by re-entry into torpor as shown by the dramatic drop in MR and T_b (Fig. 1A). Torpor in He-O_2 lasted for about 7 h and was followed by spontaneous arousal after 0800 hours. The daily pattern of MR and T_b of the same individual in air were similar to that in He-O_2 (Fig. 1B). Activity after lights-off was followed by entry into torpor at about 0100 hours. Torpor in air lasted for about 6 h and spontaneous arousal occurred at about 0630 hours (Fig. 1B). While the overall torpor pattern in He-O_2 (Fig. 1A) was similar to that in air (Fig. 1B), the TMR was somewhat lower in He-O_2 and T_b was 1.8 °C lower. This difference was greater than that which could have been caused by the 0.5 °C difference in T_a in an air atmosphere. Another difference between the two atmospheres was observed during arousal. While the arousal peak in air was brief (about 1 h) and overall rewarming was relatively fast ($0.24\,°C \cdot min^{-1}$) (Fig. 1B), exposure to He-O_2 resulted in a much wider arousal overshoot (over 2 h) and the rewarming process was much slower ($0.10\,°C \cdot min^{-1}$) (Fig. 2A). Since the maximum MR in both atmospheres was similar it is likely that maximum heat production during arousal was reached in both atmospheres, but the greater C and heat loss in He-O_2 resulted in a longer arousal overshoot.

Steady-state T_b and MR in all normothermic and torpid *S. macroura* changed with T_a (Fig. 2). Normothermic individuals lowered their T_b from 34.5 ± 0.5 °C ($n = 6$) in the TNZ to about 32.5 °C at T_a 18 °C in both air and He-O_2 atmospheres. MR increased from BMR ($0.87 \pm 0.04\,l \cdot g^{-1} \cdot h^{-1}$, $n = 6$) to RMR at T_a 18 °C of $2.27 \pm 0.24\,ml \cdot g^{-1} \cdot h^{-1}$ (2.6-fold; $n = 7$) in air and $4.47 \pm 0.57\,ml \cdot g^{-1} \cdot h^{-1}$ (5.1-fold; $n = 7$) in He-O_2. Torpid individuals reduced their T_b with T_a in both atmospheres and ΔT was similar during torpor and in

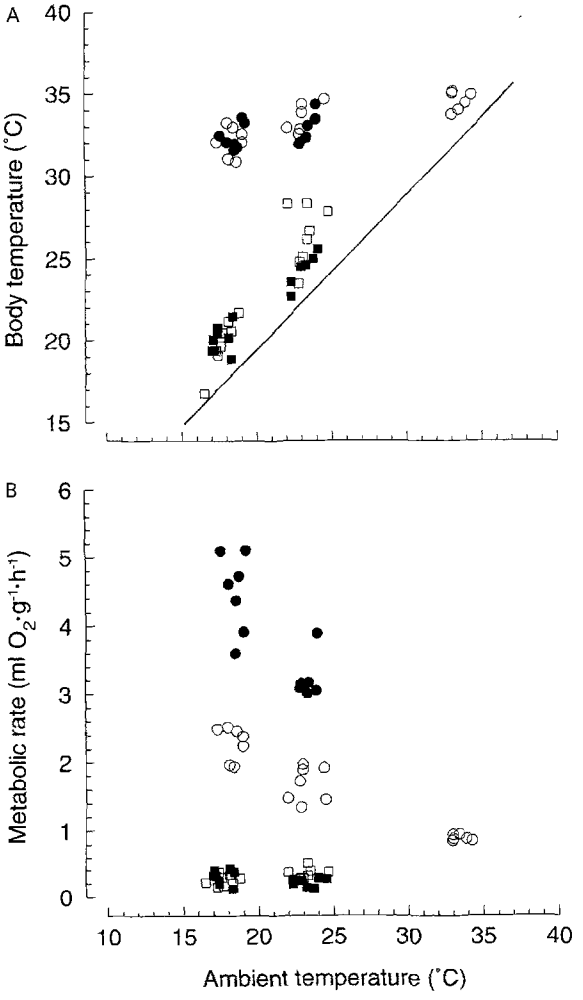

Fig. 2A,B Body temperatures (**A**) and metabolic rate (**B**) of normothermic (*circles*) and torpid (*squares*) *Sminthopsis macroura* as a function of ambient temperature in air (*open symbols*) and He-O_2 (*solid symbols*) atmospheres. The constructed diagonal line in **A** represents $T_b = T_a$

animals within the TNZ. The fall in TMR was more pronounced in He-O_2 than in air at T_a 23 °C. In contrast, at T_a 18 °C TMR was similar in the two atmospheres. TMR dropped to 38% (air) and 23% (He-O_2) of BMR at T_a 23 °C and to 28% (air) and 32% (He-O_2) of BMR at T_a 18 °C.

Mean physiological variables of normothermic individuals at the two T_as tested are summarised in Fig. 3. T_a, T_b and ΔT at 18 °C (T_a 18.3 ± 0.6 °C air: T_a 18.5 ± 0.6 °C He-O_2) were indistinguishable, while RMR and C were about twofold higher in He-O_2 than in air ($P < 0.0001$). At T_a 23 °C (T_a23.2 ± 0.9 °C air; T_a 23.4 ± 0.5 °C He-O_2), T_a and T_b were also indistinguishable, but ΔT was lower in He-O_2 than in air ($P < 0.02$) while both RMR and C were again about twofold higher in He-O_2 than in air ($P < 0.0001$).

The responses of physiological variables of torpid individuals to exposure to He-O_2 were completely different to those of normothermic individuals (Fig. 4). At T_a 23 °C (T_a 23.2 ± 0.7 °C air; T_a 23.3 ± 0.9 °C He-O_2),

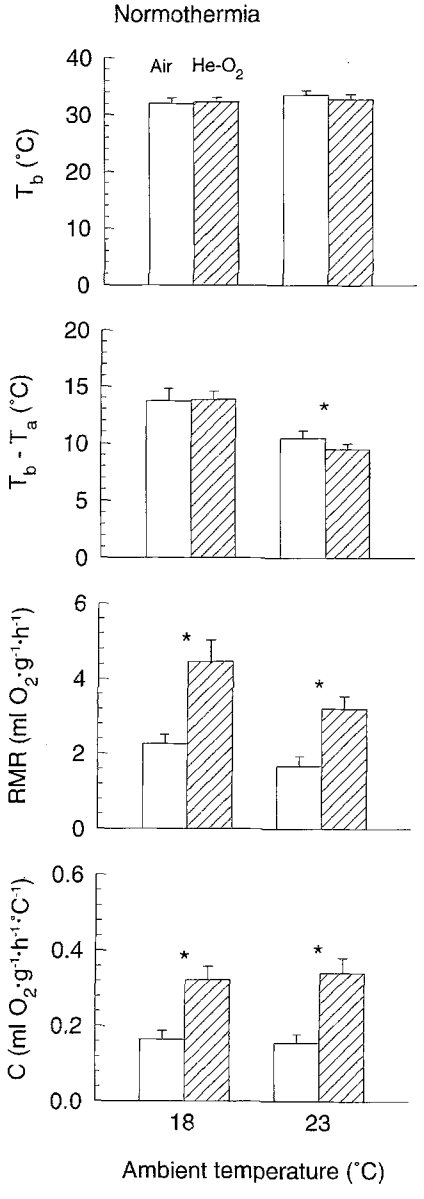

Fig. 3 Summary of means of physiological variables in normothermic *Sminthopsis macroura* at ambient temperatures (T_a) of 18 °C (18.0 ± 0.7 °C) and 23 °C (23.2 ± 0.7 °C) in air (*open bars*) and He-O_2 (*hatched bars*). Significant differences (*) between air and He-O_2 were observed for metabolic rate (RMR, $P < 0.0001$) and apparent thermal conductance (C, $P < 0.0001$) at both T_as and for the difference between T_b and T_a (ΔT, $P < 0.02$) at T_a 23 °C (t-test)

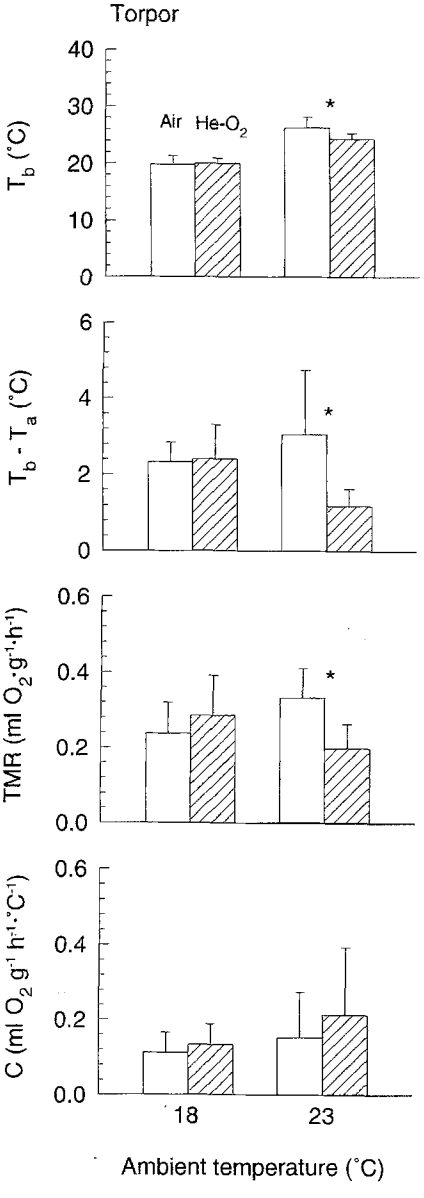

Fig. 4 Summary of means of physiological variables in torpid *Sminthopsis macroura* at ambient temperatures (T_a) of 18 °C (18.0 ± 0.7 °C) and 23 °C (23.2 ± 0.7 °C) in air (*open bars*) and He-O_2 (*hatched bars*). Significant differences (*) between air and He-O_2 were observed for body temperature (T_b, $P < 0.05$), the difference between T_b and T_a (ΔT, $P < 0.02$), and metabolic rate (TMR, $P < 0.01$) at T_a 23 °C (t-test)

T_b, ΔT, and TMR were significantly lower ($P < 0.05$) in He-O_2 than in air, while C was slightly elevated and T_a was indistinguishable. At T_a 18 °C (T_a 17.7 ± 0.7 °C air; T_a 17.6 ± 0.6 °C He-O_2), neither TMR nor C differed significantly between He-O_2 and air, and T_a, ΔT and T_b were also indistinguishable ($P > 0.3$). It is likely that this was caused by the slight increase of TMR in some individuals in He-O_2 in comparison to that at T_a 23 °C (Fig. 2). This increase indicates the onset of physiological regulation of T_b in He-O_2 at T_a 18 °C. When these individuals were excluded, the mean TMR at T_a 18 °C in He-O_2 was 71% of that in air; however, the difference was not significant. While TMR at the same T_a was only a fraction of RMR (about 5–20%) in both atmospheres, the C of torpid animals in both atmospheres ranged from 40 to 94% of that in normothermic individuals. T_b was the only physiological variable of torpid individuals that was significantly affected by T_a ($P < 0.001$).

Discussion

Our study shows that He-O_2 exposure elicits an increase in MR of normothermic individuals to defend their normothermic T_b against an increased C and thus heat loss. In contrast, torpid individuals, at least in the T_a range above T_{set}, are able to reduce their MR and T_b further in He-O_2 than in air, although C in He-O_2 is slightly raised. This suggests that (i) a low C is not the reason for the low TMR, and (ii) interrelations between MR and T_b of torpid individuals above T_{set} differ fundamentally from those of normothermic individuals.

What are the likely reasons for the differences between normothermic and torpid animals in response of physiological variables to He-O_2? Normothermic *S. macroura* maintain T_b below the TNZ by proportional thermoregulation (Geiser and Baudinette 1985). Exposure to He-O_2 in normothermic individuals is equivalent to exposure to a T_a that is about 20 °C lower than the measured T_a and, as observed in previous studies, the increased heat-loss caused by the greater C resulted in a predicted proportional increase of heat production (i.e. RMR) for maintenance of a constant T_b (Rosenmann and Morrison 1974; Dawson et al. 1986; Hallam and Dawson 1993).

The entirely different response of TMR in torpid animals to He-O_2 exposure shows that at T_as above T_{set}, the T_b is not the result of proportional thermoregulation. This further supports the findings of Heller et al. (1977) that during entry into torpor (i.e. at high T_bs) the T_{set} remains well below the actual hypothalamic temperature. A thermoregulatory increase in TMR is only elicited when either (i) the hypothalamic temperature is experimentally lowered to T_{set} (Heller et al. 1977; Florant and Heller 1977), or (ii) when T_a is low enough so that T_b can actually reach the readjusted T_{set} during torpor (Heller and Colliver 1974). Therefore, if T_a is above T_{set} during torpor no proportional thermoregulation should be detected and the lack of thermoregulatory increase in TMR during exposure to a more conductive medium observed here clearly supports this interpretation. Thus, animals during undisturbed steady-state torpor above T_{set} are thermoregulatory conformers and the increased heat loss during exposure to a more conductive medium should result in a drop in T_b, a drop in ΔT, and a drop in TMR as observed in the present study. Since TMR differed between atmospheres only at 23 °C, but not at 18 °C, it appears that some torpid animals in He-O_2 at 18 °C already had commenced to thermoregulate.

The lower TMR in He-O_2 than in air at T_a 23 °C could be due to a number of factors. Since the drop in TMR is paralleled by a drop in T_b, a temperature effect on TMR may appear to be a plausible explanation. Detailed measurements in air suggest that the steady-state TMR in *S. macroura* is largely a function of T_b (Song et al. 1995). When T_b was regressed against TMR and BMR correlations were observed in both air ($r^2 = 0.84$; $P < 0.001$) and He-O_2 ($r^2 = 0.77$; $P < 0.001$) supporting this interpretation. However, the Q_{10} between BMR and TMR at both T_as was between 2.2 and 3.2 in air and between 2.2 and 4.2 in He-O_2 (3.0–4.2 if individuals with somewhat elevated TMR in He-O_2 at T_a 18 °C were excluded). It is unlikely that temperature effects alone account for all of the pronounced drop in TMR in the He-O_2 atmosphere because the reduction in TMR from air to He-O_2 was accompanied by only a 2 °C drop in T_b. To explain the entire reduction in TMR by a Q_{10} of 3, a drop in T_b by 4–5 °C would be required.

In contrast, ΔT showed a reduction from air to He-O_2 that appears to be similar in magnitude to that of TMR (Fig. 4). This appears to support the argument that the TMR is a linear function of ΔT as during normothermia, MR is actively downregulated, and the drop in T_b is a consequence of, and not the reason for, the low TMR (Heldmaier and Ruf 1992). However, when more closely scrutinised, ΔT of *S. macroura* was not correlated with the combined TMR and BMR values in either air ($P > 0.07$) or He-O_2 ($P > 0.2$) atmospheres. Since the correlation between ΔT and TMR was barely significant ($P = 0.04$ and $P = 0.03$) and the slope was almost zero (Fig. 5), it is likely that ΔT affected TMR only marginally. The RMR predicted from extrapolation of TMR was only about 25–50% of that measured in air (Fig. 5) and the slopes of the significant regression obtained between RMR and ΔT in both atmospheres differed entirely from those for TMR. It is therefore unlikely that the TMR above T_{set} is in effect a downregulated RMR at low ΔT.

Although a reduction in C during torpor was advanced as a possible explanation for low TMR (Snyder and Nestler 1990), the present study does not support

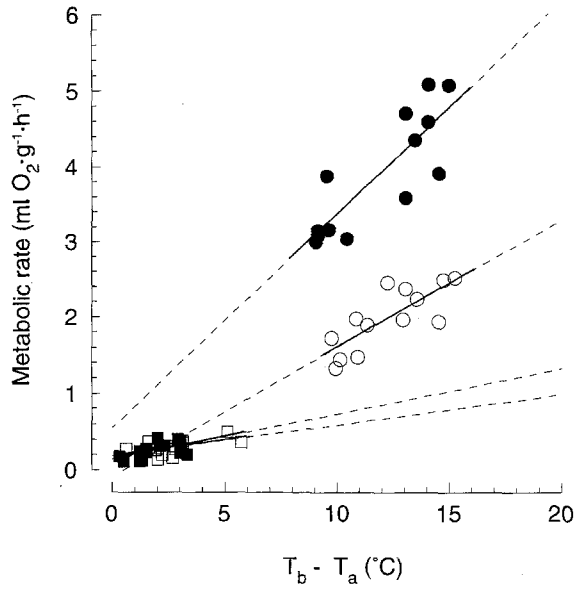

Fig. 5 Metabolic rates of normothermic (*circles*) and torpid (*squares*) *Sminthopsis macroura* in air (*open symbols*) and He-O_2 (*closed symbols*) as a function of the difference between T_b and $T_a(\Delta T)$. Regressions were performed both including and excluding individuals with a slightly elevated TMR in He-O_2 at T_a 18 °C, but no significant difference between the two regressions was detected. Equations for the linear regressions were: Normothermia, air: $MR = -0.09 + 0.17\Delta T$; $r^2 = 0.62$; $P < 0.001$; Normothermia, He-O_2: $MR = 0.55 + 0.28\Delta T$; $r^2 = 0.70$; $P < 0.0001$; Torpor, air: $MR = 0.18 + 0.04\Delta T$; $r^2 = 0.31$; $P = 0.03$; Torpor, HeO_2: $MR = 0.13 + 0.06\Delta T$; $r^2 = 0.31$; $P = 0.04$. The MSe differed significantly between normothermic and torpid individuals in both air and He-O_2 ($P < 0.001$; ANOVA). Differences in slope were observed between normothermic individuals in air and He-O_2

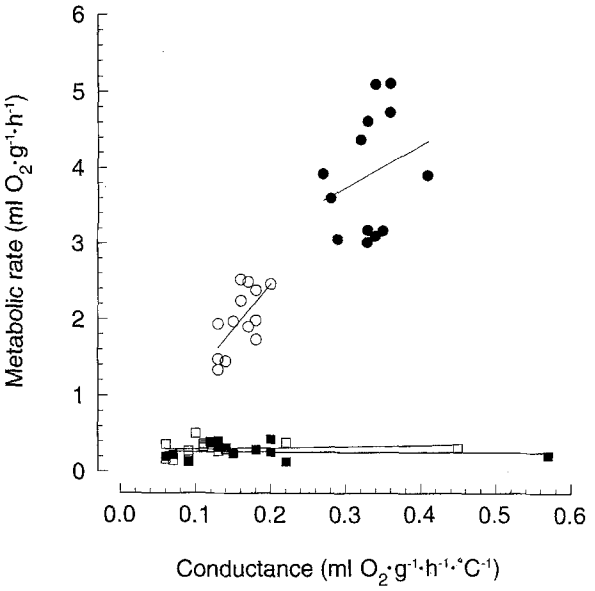

Fig. 6 Metabolic rates of normothermic (*circles*) and torpid (*squares*) *Sminthopsis macroura* in air (*open symbols*) and He-O_2 (*closed symbols*) as a function of the apparent thermal conductance (C). Equations for the linear regressions were: Normothermia, air: $MR = 0.10 + 11.7C$; $r^2 = 0.42$; $P < 0.02$; Normothermia, He-O_2: $MR = 2.05 + 5.54C$; $r^2 = 0.07$; ns; Torpor, air: $MR = 0.28 - 0.09C$; $r^2 = 0.009$; ns; Torpor, HeO_2: $MR = 0.26 - 0.08C$; $r^2 = 0.01$; ns

this hypothesis. If C was involved in the reduction of TMR, the significantly lower TMR in He-O_2 in comparison to air should have resulted in a parallel drop in C, not the opposite as observed here (Fig. 4). Furthermore, as emphasised by linear regression analyses, C was not correlated with TMR in either air ($r^2 = 0.009$; $P > 0.7$) or He-O_2 ($r^2 = 0.01$; $P > 0.7$; Fig. 6). Although a relatively large variation in C was observed in torpid individuals covering the entire range of C measured in normothermic individuals below the TNZ, the TMR in all cases was only a fraction of RMR. This suggests that if a low C is observed during torpor it is a result of, and not the cause of, low TMR. Since TMR is low, circulation can be reduced without ill effects on tissues and the low blood flow should result in a low C.

If C is excluded as an explanation for lower TMR in He-O_2 than in air, and T_b and ΔT provide only a partial explanation for the difference, what could be the likely reason? In normothermic mammals exposure to a lower T_a results in a reduction in peripheral blood flow and it is likely that exposure to He-O_2 which is equivalent to a T_a that is about 20 °C below the measured T_a (Rosenmann and Morrison 1974; present study) causes a similar response. While He-O_2 exposure in torpid individuals above T_{set} will not be equivalent to a 20 °C drop in T_a since the animals are not defending T_b, it is possible that they can sense the greater heat loss which most likely results in an increased peripheral vasoconstriction. This greater vasoconstriction in He-O_2 than in air in torpid animals may, during prolonged exposure, require some form of metabolic inhibition in peripheral tissues to prevent metabolic imbalances in tissues with little blood supply. A reduced TMR will allow a reduction in respiration rate which should reduce both respiratory costs and respiratory heat loss. These factors may in turn be part of the reason why overall TMR in He-O_2 is reduced while C is little affected although the animal is exposed to a highly conductive medium.

Our study shows that exposure to He-O_2 elicits a different physiological response in torpid and normothermic *Sminthopsis macroura*. Exposure to a more conductive atmosphere resulted in an increase in RMR, while TMR decreased well below values measured in air. Our attempt to identify variables responsible for the decrease of TMR in He-O_2 suggests that several factors are involved and that MR reduction during torpor is a most complex phenomenon that requires more detailed physiological investigation.

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