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Interaction between exercise training and cold acclimation in rats

J. LeBlanc and P. Diamond

Department of Physiology, School of Medicine, Laval University, Quebec City, Canada, G1K 7P4

Summary. Five groups of 10 rats were used. Group A included sedentary rats kept at 24° C, group B exercised-trained rats and group C rats exposed at −15° C for 2 h every day and kept at 24° C for the remaining time. These 3 groups were kept on this regimen for 10 weeks. In addition group D was acclimated to cold ($2\,h \cdot d^{-1}$ at −15° C) for 6 weeks and subsequently deacclimated at 24° C for 4 weeks. Group E was also acclimated to cold for 6 weeks and during the deacclimation, at 24° C period which lasted 4 weeks, the animals were exercised 2 h per day. Following the 10 week experimental period all animals were sacrified and DNA and protein content of the IBAT as well as its total mass were measured. The results show significant increases in the cold adapted group. Exercise training which had no effect on brown adipose tissue IBAT at room temperature, caused an accelerated reduction in weight, DNA and protein content of the BAT in rats previously acclimated to cold. In spite of this, the thermogenic response to noreadrenaline was significantly enhanced in the group which exercised during the deacclimation period. It is suggested that tissues other than IBAT may explain this enhanced heat production capacity.

Key words: Exercise training — Cold adaptation — Brown adipose tissue

Introduction

A certain amount of work has been carried on the interactions between cold exposure and physical exercise. Chin et al. (1973) and Stromme and Hammel (1967) reported an improved tolerance to cold in exercise trained rats. The question is raised as to wether this action is related to an enhanced brown adipose tissue (IBAT) capacity. The binding of the nucleotide GDP (guanosine diphosphate) to BAT mitochondria has been shown to be an indicator of the thermegenic activity of this tissue. It has been found recently by Arnold et al. (1986), that GDP-binding in the IBAT, which is increased in cold adapted rats, is greatly reduced if the cold exposed rats are simultaneously exercised. In addition in warm-acclimated rats exercise training per se has no action on GDP binding, size and protein content of the IBAT (Arnold et al. 1986). Thus the improved cold tolerance of exercise-trained rats would not seem to be the result of an enhanced capacity of IBAT. In light of the results showing that exercise suppresses IBAT thermogenesis normally associated with cold acclimation, the present investigation was undertaken in order to further elucidate the interaction between cold and exercise training on non-shivering thermogenesis. The approach consisted in comparing the degree of cold acclimation of various groups of rats. The criteria of comparaison between the groups were the body temperature response to norepinephrine as well as the size and protein and DNA content of the IBAT.

Offprint requests to: J. LeBlanc at the above address

Materials and methods

Five groups of 10 male Wistar rats with initial body weight of 170 g were used. Group A included sedentary rats kept at 24° C, group B exercise-trained rats swimming 2 h per day in water maintained at 37° C and group C rats exposed at −15° C for 2 h every day. These last two groups were kept at 24° C when they were not exercised or exposed to cold. These three groups were maintained on this schedule for 10 weeks.

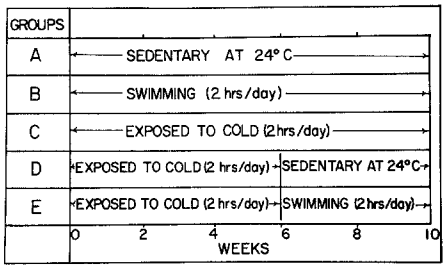

Fig. 1. Diagram showing the procedure used for the various groups of the experiment. The rats were exposed to $-15°C$ and the water temperature was kept at $37°C$

Group D was acclimated to cold ($2\,h \cdot d^{-1}$ at $-15°C$) for 6 weeks and subsequently deacclimatized at $24°C$ for 4 weeks. Group E was also acclimatized to cold for 6 weeks but during the 4 week deacclimation period the rats swam 2 h every day. The diagram shown in Fig. 1, illustrates these various procedures.

After 10 weeks, the body temperature response to subcutaneous noradrenaline was measured 2 h after the injection since preliminary work had shown that the peak response occurred at that time. Three days after the test, the animals were killed and the interscapular brown adipose tissue pads removed to determine the protein by the method of Lowry et al. (1951) and the DNA content by the method of Kapuscinski and Skoczycas (1977). The heart, adrenal glands and epididymal fat were also removed and weighed.

Results

The results are expressed as means ±SE. Data were computed using a one way analysis of variance and a Duncan (1955) multiple-range test for comparison between means of each treatment. The lower final body weight and epidymal fat weight in the B group indicate that the swimming training regimen was sufficiently intensive (Table 1). Even after four weeks group E had significant lower values for those parameters as compared to the A group. The weight of the adrenals giving an indirect indication of the stressing effect of the treatments, was significantly higher in B, C and E groups. The results of Table 1 also show that the heart ratio was larger in both cold exposed and exercise trained groups. After 10 weeks the body temperature response was comparable in groups A, B and D but it was greater in the other two groups, with group C being the most responsive. These results are reported in Fig. 2.

Exercise training (group B) was found to have no effect on the wet weight of IBAT whereas cold adaptation produced a significant increase (Fig. 3). Following deacclimation the size of the IBAT of group D was reduced but still signifi-

Table 1. Body weight and organ weight of rats for each treatments after 10 weeks

	A	B	C	D	E
Final body	379	$336^{**}_{\dagger}$	351^{*}	$357^{*}_{\S}$	$316^{**}_{\S\S}$
weight (g)	±9.7	±8.1	±7.6	±4.2	±4.6
Heart (g)	1.01	1.0	1.06	$0.97^{\dagger}$	1.03
	±0.02	±0.02	±0.03	±0.16	±0.02
Adrenals (mg)	54	62*	60*	$51^{\ddagger\ddagger}_{\dagger}$	$64^{**}_{\S\S}$
	±1.32	±2.1	±0.93	±2.1	±2.2
Epididymal	4.32	$2.66^{**}_{\S\S}$	3.18**	3.67*	$2.4^{*}_{\S\S}$
fat (g)	±0.25	±0.16	±0.21	±0.18	±0.09

Values are means ±SE
Groups: A, sedentary warm; B, trained warm; C, cold adapted; D, cold deadapted; E, cold deadapted + exercise
Significant differences from A, (*), B (+), C (†) and D (§) are indicated by 1 ($P<0.05$) or 2 symbols ($P<0.01$)

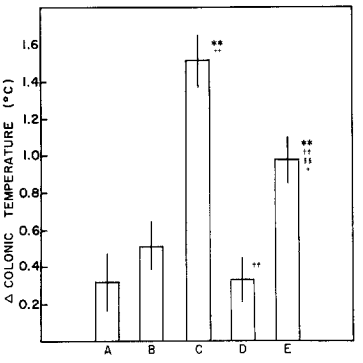

Fig. 2. Body temperature changes as measured by colonic temperatures 2 h after subcutaneous injection of norepinephrine ($300\,\mu g \cdot kg^{-1}$) in A, sedentary warm; B, trained warm; C cold adapted; D, cold deadapted; E, cold deadapted + exercise. Significant differences from A (*), B (+), C (†), and D (§) are indicated by 1 ($P<0.05$) or 2 symbols ($P<0.01$)

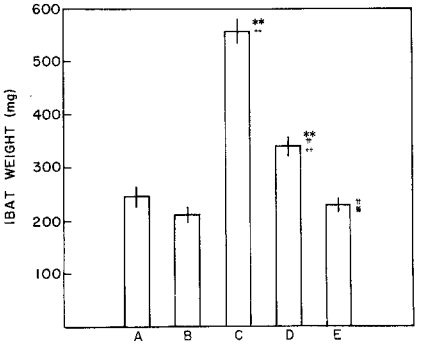

Fig. 3. Interscapular brown adipose tissue (IBAT) weight for A, sedentary warm; B, trained warm; C, cold adapted; D, cold deadapted; E, cold deadapted + exercise were obtained at the end of the 10 w experimental period. Significant differences from A (*), B (+), C (†), and D (§) are indicated by 1 ($P<0.05$) or 2 symbols ($P<0.01$)

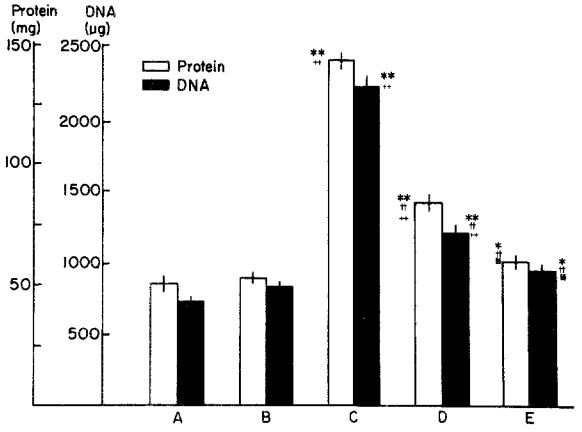

Fig. 4. Variations in protein (mg) and DNA (µg) of interscapular brown adipose tissue over 10-wk period for A, sedentary warm; B, trained warm; C, cold adpated; D, cold deadapted; E, cold deadapted + exercise were obtained at the end of the 10 week experimental period. Significant differences from A (*), B (+), C (†), and D (§) are indicated by 1 ($P<0.05$) or 2 symbols ($P<0.01$)

cantly larger than that of the control group (A). However in group E which was exercised for four weeks after being removed from the cold the size of the IBAT was further reduced and comparable to that of group A. The results on protein and DNA content of the IBAT were comparable to those on the weight of the tissue. Exercise alone had no effect on BAT components whereas marked increases were found with cold exposure. Removal from the cold for four weeks reduced the DNA and protein content of the IBAT and this effect was significantly accentuated when the animals were submitted to an exercise programme. These results are reported in Fig. 4.

Discussion

Trayhurn et al. (1987) have shown in cold acclimated rats removed from the cold for 1 day, a marked reduction of GDP binding without significant change in the uncoupling protein of the IBAT. The results show that the activity of the IBAT declines rapidly upon removal from the cold, whereas its enhanced capacity to produce heat remains unchanged for at least one week and is retained for over three weeks. Similarly the thermogenic response to noradrenaline which is considered as an indicator of IBAT capacity in cold-acclimated animals is retained for many days after cold acclimation. In the present investigation it was found as expected that cold-acclimated rats responded more to noradrenaline and that the weight, protein and DNA content of the IBAT were increased. Four weeks after removal from the cold noradrenaline response was comparable to that of warm-acclimated rats except when the animals were exercised during the deadaptation period. In this case a significant portion of the thermogenic capacity was retained. This occurred at a time when the size of the IBAT and its DNA and protein content were reduced due to exercise training.

This finding is somewhat unexpected. It has been shown by Arnold et al. in 1986 that exercise training suppresses thermoregulatory thermogenesis in warm and cold-acclimated rats. Furthermore the rats which are exercised during the cold deadaptation period have lower total DNA and protein content in the IBAT than those which are kept sedentary. These findings suggest that the enhanced thermogenic response to noradrenaline caused by exercise-training during cold deacclimation may have come from tissues other than the IBAT. Wether it comes from the muscles in which the number of mitochondria is increased during training (Farrar et al. 1981) is not known?

Our results are in agreement with previous studies showing either no significant effect of exercise training on IBAT weight (LeBlanc et al. 1982[a], Harri et al. 1982 and 1984) or a decrease in IBAT weight (Richard et al. 1986) presumably reflecting a reduction in fat and protein content from the whole carcass. It has also been found that the response to noradrenaline of exercise trained rats was comparable to that of sedentary animals. Similar findings have been reported in other studies in which the response to norepinephrine (LeBlanc et al. 1982a, 1982b) or isoproterenol was tested (Harri et al. 1982, 1984). Several studies have reported no difference in GDP-binding, an index of IBAT nonshivering thermogenesis capacity after exercise bout at 24° C (Arnold et al. 1986) or after a long period of exercise training at 24 or 4° C (Bell et al. 1984; Harri et al. 1984 and Richard et al. 1986).

In summary our work provides evidence that exercising rats after cold adaptation causes a faster suppression of IBAT thermogenic capacity than keeping them sedentary. Thus the increased thermogenic response to noradrenaline observed in this condition would indicate the participation of tissues other than the IBAT.

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