

THERMOREGULATORY RESPONSES IN ATHLETES BREATHING COLD AND WARM AIR DURING EXERCISE IN THE COLD

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INTRODUCTION

During exercise in cold weather the entire body including the respiratory tract are exposed to low temperatures. In a cold environment, the skin blood vessels of the hands constrict, and the inhalation of cold, dry air may produce bronchoconstriction in subjects with asthma. Berk et al. (1) have hypothesized an association between skin and airway reactivity to cold and suggest that a common mechanism may underlie the obstructive response to thermal stimulation of these two organs.

During exercise, the cutaneous circulation is subject to competing thermoregulatory and nonthermoregulatory reflexes (2). Compared to exercise in the heat, when both the skin and muscle demands for blood flow are high, the demand for skin blood flow is relatively low during exercise in a cold environment (3). The initiation of exercise also causes a reduction in skin blood flow because of an elevated vasodilator threshold (4,5), and finger skin temperature typically falls. The heat balance of the hands relies to a great extent upon heat input by warm blood from the body core and, following an exercise-induced rise in body core temperature, finger temperature may also rise (6).

The objective of this study was to investigate the effects of breathing warm versus cold air during exercise at -15°C on thermoregulatory vasoactivity in the fingers.

MATERIALS AND METHODS

Eleven healthy male athletes participated in the study. Their average age, weight, height and maximal oxygen uptake ($\dot{V}_{O_{2\max}}$) were 23.5 ± 5.4 years, 78.6 ± 7.8 kg, 185.7 ± 3.2 cm and 61.3 ± 4.6 ml \cdot kg $^{-1}\cdot$ min $^{-1}$, respectively. The local Ethical Review Committee approved the experimental procedures.

The subjects were tested while running on a treadmill (Challenger) at a 6° uphill gradient in a climatic chamber, the room temperature being maintained at -15°C ($\pm 1.5^{\circ}\text{C}$) and wind velocity at 1.5 m \cdot sec $^{-1}$. They were randomly assigned to conditions of inhalation of moderately cold (2°C) or warm (25°C) air immediately after entering the climatic chamber.

The subjects rested for 20 min at an ambient temperature of 23°C prior to the tests. Five min after entering the climatic chamber, they started on the experimental protocol, which consisted of a warming-up period of 20 min at an exercise intensity of about 50% $\dot{V}_{O_{2\max}}$, followed by periods in which the intensity of exercise was increased by 1 km \cdot h $^{-1}$ every 5 min over 4 different speeds, representing exercise intensities of about 60 to 90% $\dot{V}_{O_{2\max}}$ (7).

The middle fingertip temperature (T_{tip}) and rectal temperature (T_{re}) were measured with a YSI thermistor ($\pm 0.15^\circ\text{C}$) every minute during the tests. Temperature data analyses were carried out for the first 25 min of the tests because small differences in the time schedule occurred after the warming-up period. Mean finger skin temperature (\bar{T}_{finger}) was calculated after the warming-up periods as the mean of the measurements made every minute between 0 and 25 min. The threshold for finger vasodilatation was defined as the point in time at which finger skin temperature rose at least 0.5°C per min (TTR, time to rise). For those individuals who did not show any dilatation during the warm-up period, minute 25 was defined as the threshold. Expired minute ventilation (V_E), oxygen uptake (\dot{V}_{O_2}) and heart rate were measured continuously during the tests by means of a Jaeger Ergo-Oxyscreen and a Polar Sport Tester heart rate recorder respectively. A paired t-test was used to compare differences in response between cold and warm air breathing of the TTR and \bar{T}_{finger} .

RESULTS

During the last 5 min of the 2 resting periods at 23°C , T_{tip} stabilized at $35.0 \pm 0.7^\circ\text{C}$ and $35.2 \pm 0.5^\circ\text{C}$ (Figure 1). After entering the climatic chamber, equal decreases in T_{tip} occurred, independent of whether cold or warm air was being breathed (Figure 1). When the warming-up period started 5 min later, heart rate increased to 129 ± 14 bpm and 128 ± 10 bpm with cold and warm air, respectively, and at the same time, V_E increased to 66.3 ± 7.5 and 65.4 ± 7.0 $\text{l}\cdot\text{min}^{-1}$ under the 2 conditions. T_{re} increased $0.5 \pm 0.2^\circ\text{C}$ (cold air) and $0.6 \pm 0.2^\circ\text{C}$ (warm air) ($n = 6$) during the 20-min warming-up period. However, during the initial phase of the warming-up period T_{tip} continued to fall in both conditions. Finger vasodilatation occurred significantly later when breathing cold than when breathing warm air (Table 1/Figure 1).

Table 1. Finger skin temperature Time to Rise (TTR) and mean finger skin temperature (\bar{T}_{finger}) during exercise at -15°C ¹

	<u>Breathing cold air (2°C)</u>	<u>Breathing warm air (25°C)</u>
TTR	20.3 ± 4.8 min*	17.6 ± 5.1 min
\bar{T}_{finger} (0-25 min)	$28.7 \pm 2.4^\circ\text{C}$ *	$30.1 \pm 2.2^\circ\text{C}$

¹Values shown are mean \pm SD. $n = 11$.

*Significant difference between breathing air temperature conditions, $P < 0.05$.

DISCUSSION

At the onset of exercise 5 min after entering the climatic chamber, T_{finger} continued to fall under both experimental conditions. This vasoconstriction may be explained by an increase in heat loss due to increased cold and wind exposure associated with arm movement during exercise. Reduced skin blood flow caused by the onset of exercise itself has also been demonstrated under normothermic conditions (2,8).

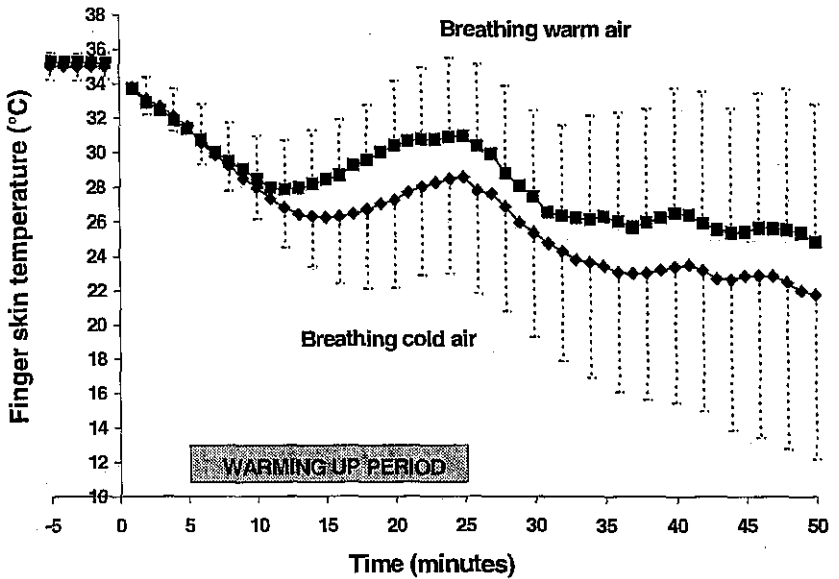


Figure 1. Finger skin temperature response before and during exercise at -15°C when breathing cold and warm air (mean \pm SD, $n = 11$)

In the present study, T_{tip} suddenly increased during the warming-up period under both experimental conditions, but a significantly longer time to rise for T_{tip} was measured when cold air was inspired. During exercise in a cold environment, the vasodilatory temperature threshold is high (3) and increased finger blood flow is entirely due to release of vasoconstrictor tone (8,9). Since finger temperature depends largely on the fingers' supply of warm arterial blood, T_{tip} may rise following an exercise-induced rise in body core temperature (6). However, because the exercise intensity in this study was the same under both experimental conditions, the different result is explained by the additional heat supply provided by warm air. These experiments demonstrate that breathing a certain amount of warm air causes a thermal input to the thermoregulatory center, which results in an earlier vasodilator response in the finger than during cold air inhalation. The earlier onset of the rise in finger skin temperature when breathing warm air also explains the significantly higher \bar{T}_{finger} during the first 25 min of the test.

CONCLUSIONS

The observed differences in threshold values for peripheral vasodilatation between cold- and warm-air breathing conditions may be explained by differences in thermal input to the thermoregulatory center from the respiratory tract. A correlation between skin and airway thermal sensitivity has previously been demonstrated (1). The increased airway reactivity to cold in asthmatics may be partly explained by an enhanced thermal sensitivity upon cold stimulation. We

might, therefore, expect that breathing cold air during exercise at low temperatures would lead to a more pronounced delay in vasodilatation in asthmatics than in healthy athletes.

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