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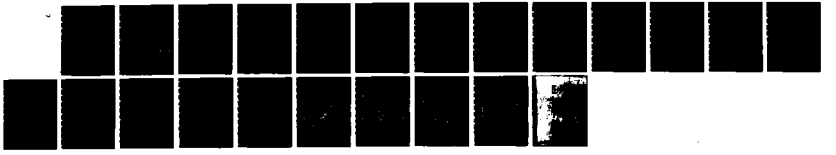

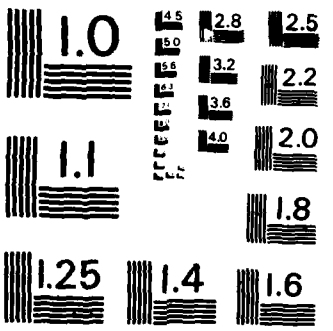

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Thirteen male volunteers performed cycle ergometer maximal oxygen uptake ($\dot{V}O_2$ max) tests in moderate (21°C, 30% rh) and hot (49°C, 20% rh) environments, before and after a nine-day heat acclimation program. This program resulted in significantly decreased ($P<0.01$) final heart rate (24 $bt \cdot min^{-1}$) and rectal temperature (0.4°C) from the first to last day of acclimation. The $\dot{V}O_2$ max was lower ($P<0.01$) in the hot environment relative to the moderate environment both before (8%) and after (7%) acclimation with no significant difference

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(P>0.05) shown for maximal power output (PO max, watts) between environments either before or after acclimation. The $\dot{V}O_2$ max was higher (P 0.01) by 4% after acclimation in both environments. Also, PO max was higher (P<0.05) after acclimation in both the moderate (4%) and hot (2%) environments. The reduction in $\dot{V}O_2$ max in the hot compared to moderate environment was not related to the difference in core temperature at $\dot{V}O_2$ max between moderate and hot trials, nor was it strongly related with aerobic fitness level. These findings indicate that heat stress, per se, reduced the $\dot{V}O_2$ max. Further, the reduction in $\dot{V}O_2$ max due to heat was not affected by state of heat acclimation, the degree of elevation in core temperature, or level of aerobic fitness.

INFLUENCE OF HEAT STRESS AND ACCLIMATION ON MAXIMAL AEROBIC POWER

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Summary. Thirteen male volunteers performed cycle ergometer maximal oxygen uptake ($\dot{V}O_2$ max) tests in moderate (21° C, 30% rh) and hot (49° C, 20% rh) environments, before and after a nine-day heat acclimation program. This program resulted in significantly decreased ($P < 0.01$) final heart rate (24 bt · min^{-1}) and rectal temperature (0.4° C) from the first to last day of acclimation. The $\dot{V}O_2$ max was lower ($P < 0.01$) in the hot environment relative to the moderate environment both before (8%) and after (7%) acclimation with no significant difference ($P > 0.05$) shown for maximal power output (PO max, watts) between environments either before or after acclimation. The $\dot{V}O_2$ max was higher ($P < 0.01$) by 4% after acclimation in both environments. Also, PO max was higher ($P < 0.05$) after acclimation in both the moderate (4%) and hot (2%) environments. The reduction in $\dot{V}O_2$ max in the hot compared to moderate environment was not related to the difference in core temperature at $\dot{V}O_2$ max between moderate and hot trials, nor was it strongly related with aerobic fitness level. These findings indicate that heat stress, per se, reduced the $\dot{V}O_2$ max. Further, the reduction in $\dot{V}O_2$ max due to heat was not affected by state of heat acclimation, the degree of elevation in core temperature, or level of aerobic fitness.

Key Words: Aerobic fitness - Cycle exercise - Maximal exercise - Rectal temperature - Temperature regulation

Introduction

Man's ability to perform maximal effort exercise in the heat has not been well studied. Human subjects acutely exposed to hot conditions have been reported to have lowered (Klausen et al.1967; Rowell et al. 1969; Saltin et al. 1972) or unchanged (Rowell et al. 1965; Williams et al. 1962) maximal aerobic power ($\dot{V}O_2$ max) values. The investigations which demonstrated reduced $\dot{V}O_2$ max in the heat, relative to moderate environmental conditions, all employed subjects who were not heat acclimated. One of the two investigations reporting that $\dot{V}O_2$ max was not altered by acute heat exposure employed heat acclimated subjects (Williams et al. 1962). Therefore, the subject's state of heat acclimation might alter the influence of heat stress on $\dot{V}O_2$ max. The physiological mechanism responsible for heat stress reducing $\dot{V}O_2$ max values, is probably an inability to achieve maximal cardiac output (Rowell 1983; Rowell et al. 1966). Heat acclimated subjects demonstrate improved central circulatory responses during submaximal exercise (Wyndham et al. 1968; Wyndham et al. 1976) and may have a better ability to increase cardiac output during maximal exercise in the heat.

The purposes of the present investigation were a) to determine if acute heat stress reduces $\dot{V}O_2$ max relative to the levels achieved in a moderate environment and b) to determine if heat acclimation alters the relationship between $\dot{V}O_2$ max values obtained in hot and moderate environments.

Methods

Subjects. Thirteen male soldiers volunteered as subjects. These subjects had a mean ($\pm$SD) age of 22 ± 4 yr, height of 176 ± 10 cm, and weight of 76 ± 12 kg. Previous to all testing, subjects were informed of their involvement in the study, any known risks, and their right to terminate participation at will without penalty. Each signed a statement of informed consent.

Protocol. These experiments were conducted in Natick, MA, during winter (January-March) when subjects were naturally unacclimated to heat. During all test sessions, subjects dressed in shorts, socks and tennis shoes. The subjects' initially completed $\dot{V}O_2$ max tests in a moderate (21°C, 30% rh) and in a hot (49°C, 20% rh) environment both before and immediately after a heat acclimation program. A discontinuous effort but progressive intensity test was employed (Kamon and Pandolf 1972; Young et al. 1982). A pedal rate of 60 rpm was used during all of the cycle exercise tests. The criterion for $\dot{V}O_2$ max was a leveling off or a decrease in oxygen uptake with increasing power output level (Kamon and Pandolf 1972; Young et al. 1982). The subjects rested for 20 minutes in the test environment prior to each maximal effort test.

The heat acclimation program consisted of performing 2-hours of exercise in the heat (49°C, 20% rh) for nine consecutive days. The subjects walked on a treadmill ($1.52\ m \cdot s^{-1}$) at a grade (2-6%) that elicited 40 to 50% of their moderate environment $\dot{V}O_2$ max. Ad libitum water drinking was encouraged for all test sessions.

Physiological Variables. Electrocardiograms were obtained with chest electrodes (CM5 placement) and heart rates were calculated from 20-second recordings. Oxygen uptake, carbon dioxide production ($l \cdot min^{-1}$ STPD) and pulmonary ventilation ($\dot{V}E$, $l \cdot min^{-1}$ BTPS) were determined by open-circuit spirometry. Subjects breathed via a two-way breathing valve (Otis-McKerrow), and expired gases were collected in 150-liter Douglas bags. Expired gases were analyzed for O_2 and CO_2 concentrations by an electrochemical O_2 analyzer (Applied Electrochemistry S-3A) and an infrared CO_2 analyzer (Beckman LB-2), respectively. The volume of expired air was measured by a Tissot gasometer. Rectal temperatures (T_{re}) were measured for subjects with a Yellow Springs Instrument rectal thermistor probe inserted ~10 cm beyond the anal sphincter.

Statistical Analyses. Means, standard deviations, paired t tests, repeated measures analysis of variance, Tukey critical difference tests, and regression analyses were performed on a desktop computer (Hewlett-Packard 9826). Statistical significance was accepted at the $P < 0.05$ level.

Results

Table 1 provides the subjects' final exercise heart rate and rectal temperature values for the first and ninth heat acclimation sessions. The heat acclimation program decreased ($P < 0.01$) mean final heart rate by 24 bt $\cdot$ min^{-1} and mean final rectal temperature by 0.4°C. Physiological acclimation was further demonstrated by nonsignificant ($P > 0.05$) differences between the last three heat acclimation days.

Ther were a total of 52 maximal effort cycle ergometer tests being administered in this investigation. The $\dot{V}O_2$ max criterion of leveling off in oxygen uptake or a decrease in oxygen uptake with increased exercise intensity were achieved in all of these tests. Table 2 presents the $\dot{V}O_2$ max values for the four test sessions. Heat acclimation was found to significantly ($P < 0.01$) increase $\dot{V}O_2$ max by 4% in both environments. In comparison to the moderate environment, significantly lower ($P < 0.01$) $\dot{V}O_2$ max values were elicited in the hot environment: the reduction averaged 8% pre-acclimation and 7% post-acclimation. Figure 1 presents the individual data for $\dot{V}O_2$ max in the moderate and hot environments. In all 26 instances, the subjects had lower values in the hot than moderate environment. Figure 2 illustrates the individual percent decrease in $\dot{V}O_2$ max (from moderate to hot environment) for the pre- and post-acclimation tests. A nonsignificant relationship ($r = 0.37$) was found for the reduction of $\dot{V}O_2$ max from pre- to post-acclimation.

Figure 3 presents the individual $\dot{V}O_2$ max values obtained in the moderate environment plotted against the percent decrease in $\dot{V}O_2$ max in the hot environment. The correlation coefficients between these variables pre-(r=0.26) and post-(r=0.31) acclimation were not significant. Figure 4 plots aerobic fitness ($\dot{V}O_2$ max in ml $\cdot$ kg^{-1} $\cdot$ min^{-1}) values against the percent decrease in $\dot{V}O_2$ max in the hot environment. The correlation coefficients between these variables was not significant pre-acclimation (r=0.13) but was significant post-acclimation (r=0.55, $P < 0.05$). In addition, insignificant correlation coefficients were found between the elevation in core temperature and the reduction in $\dot{V}O_2$ max in the hot compared to moderate environment pre-(=0.37) and post-(r=0.32)acclimation.

Table 2 presents the power output (PO max) and physiological responses to maximal effort cycle exercise. POmax values were found to be significantly ($P < 0.05$) greater in both the moderate (10 watt) and hot (5 watt) environments during the post-acclimation tests. Environmental condition did not affect ($P > 0.05$) POmax. The $\dot{V}_E \cdot \dot{V}O_2^{-1}$ values were not influenced ($P > 0.05$) by acclimation state nor environmental condition. Maximal heart rate values were significantly ($P < 0.01$) lower post-acclimation in the moderate (7 bt $\cdot$ min^{-1}) and hot (4 bt $\cdot$ min^{-1}) environments. In addition, HR max values were significantly ($P < 0.01$) higher in the hot than moderate environments, both pre- (5 bt $\cdot$ min^{-1}) and post- (8 bt $\cdot$ min^{-1}) acclimation.

Discussion

Research has not been published which attempts to quantify the influence of acute and chronic (heat acclimation) heat exposure on an individual's maximal aerobic power. The present investigation's research design permitted each subject to serve as his own control since they completed both the acute and chronic experiments. Since our subjects had $\dot{V}O_2$ max values ranging from 39-

53 ml • kg^{-1} • min^{-1} which corresponds with fair-to-high fitness levels (Åstrand and Rodahl 1977), we were also able to examine whether high fitness lessened the decrement in $\dot{V}O_2$ max elicited by heat exposure. Our data indicates that high aerobic fitness has a minimal influence on this decrement in $\dot{V}O_2$ max both pre-and post-acclimation (see Figure 4).

Our data clearly demonstrate that heat stress reduces an individual's $\dot{V}O_2$ max relative to the level achieved in a moderate environment. More importantly, the state of heat acclimation did not alter this approximate 0.25 l • min^{-1} decrement in $\dot{V}O_2$ max between environments. The question remains, what physiological mechanisms(s) is/are responsible for these decreased $\dot{V}O_2$ max values? It can be theorized that thermal stress might result in a displacement of blood to the cutaneous vasculature, which could result in a decreased effective central blood volume and thus reduce venous return and cardiac output. Since each liter of blood could deliver ~0.2 l of oxygen (1.34ml O_2/gHb x 15 gHb/100ml of blood), a reduction in maximal cardiac output by 1.2 l • min^{-1} could account for the observed decrement in $\dot{V}O_2$ max with heat exposure. Rowell et al (1966) have reported that during high intensity exercise in the heat the cardiac output was significantly reduced by 1.2 l • min^{-1} below control levels.

The $\dot{V}O_2$ max decrement in the heat was not related to the magnitude of the thermal strain. Although we measured rectal temperature responses for only eight subjects, no clear trend was noted between elevation in rectal temperature at $\dot{V}O_2$ max in the heat and the reduction in $\dot{V}O_2$ max. This finding is in agreement with previous reports that core temperature (Rowell et al. 1969) and ambient humidity (Saltin et al. 1972) are not related to the $\dot{V}O_2$ max decrement. Also, we found that heat acclimation resulted in small but significant improvements of $\dot{V}O_2$ max in both environmental conditions which we attribute

to a "training effect" by the heat acclimation program. This observation was a little surprising as a 50% relative exercise intensity, used to acclimate the subject, is below the level frequently reported to induce a cardiovascular training effect (Pollock 1973). However, examination of previous heat acclimations investigations provides collaborative support for our observation (Nadel et al. 1974; Roberts et al. 1977; Wyndham et al. 1972). Employing a nearly identical protocol, Nadel et al (1974) reported $\dot{V}O_2$ max to increase by approximately 190 ml (T_a = 22°C) after a 10-day heat acclimation program. In addition, Desai and Senay (1984) have reported that heat acclimation will not alter $\dot{V}O_2$ max, if the acclimation program is preceded by an endurance training program.

Numerous investigators (Davies 1979; Davies et al. 1976; Saltin et al. 1966) have reported that the magnitude of core temperature elevation during muscular exercise is coupled to the relative exercise intensity. This relationship has been reported for relative exercise intensities up to 85% $\dot{V}O_2$ max (Davies 1979) as well as in moderate and hot environmental conditions (Davies et al. 1976). If heat acclimation did enable individuals to achieve higher $\dot{V}O_2$ max during heat stress, then individuals would be at lowered relative intensities at a given submaximal oxygen uptake level. A reduced relative exercise intensity after heat acclimation would account for lowered core temperature responses observed during exercise-heat stress. The present data indicate that the reduced core temperature response during exercise after heat acclimation is not due to a reduction in relative exercise intensity subsquent to improved $\dot{V}O_2$ max. .

In conclusion, this investigation demonstrates that heat stress reduces an individual's $\dot{V}O_2$ max. The magnitude of the reduction in $\dot{V}O_2$ max is not related to an individual's state of heat acclimation, elevation in core temperature, nor level of aerobic fitness.

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Human subjects participated in these studies after giving their free and informed voluntary consent. Investigators adhered to AR 70-25 and USAMRDC Regulation 70-25 on Use of Volunteers in Research.

Table 1. Final exercise heart rate and rectal temperature responses for the first and ninth day of a heat acclimation (49° C, 20% rh) program.

		First Day	Ninth Day
Heart Rate	$\bar{X}$	164	140
(bt • min^{-1})	SD	16	17
	P	<0.01	
Rectal Temperature	$\bar{X}$	39.2	38.8
(°C)	SD	0.4	0.4
	P	<0.01	

Table 2. Power output and physiological responses to maximal effort cycle exercise (n=13)

Environment		Pre-Acclimation Moderate	Pre-Acclimation Hot	Post-Acclimation Moderate	Post-Acclimation Hot
PO max (watt)	$\bar{X}$	266	267	276	272
	SD	42	39	41	43
	P	**	**		
$\dot{V}O_2$ max ($ml \cdot kg^{-1} \cdot min^{-1}$)	$\bar{X}$	3.39	3.13	3.51	3.26
	SD	0.52	0.46	0.56	0.49
	P	††	†, ††		†
$\dot{V}_E \cdot \dot{V}O_2^{-1}$	$\bar{X}$	45	45	44	44
	SD	6	6	5	6
	P				
HR max ($bt \cdot min^{-1}$)	$\bar{X}$	193	198	186	194
	SD	9	7	8	8
	P	††	†, ††		†

PO_{max} is maximal achieved power output; $\dot{V}O_2$ max is maximal aerobic power; $\dot{V}_E \cdot \dot{V}O_2^{-1}$ is ventilatory equivalent of oxygen at $\dot{V}O_2$ max; HR_{max} is maximal achieved heart rate. Hot different from moderate * $P < 0.05$ and† $P < 0.01$; Pre-acclimation different from post-acclimation ** $P < 0.05$ and†† $P < 0.01$

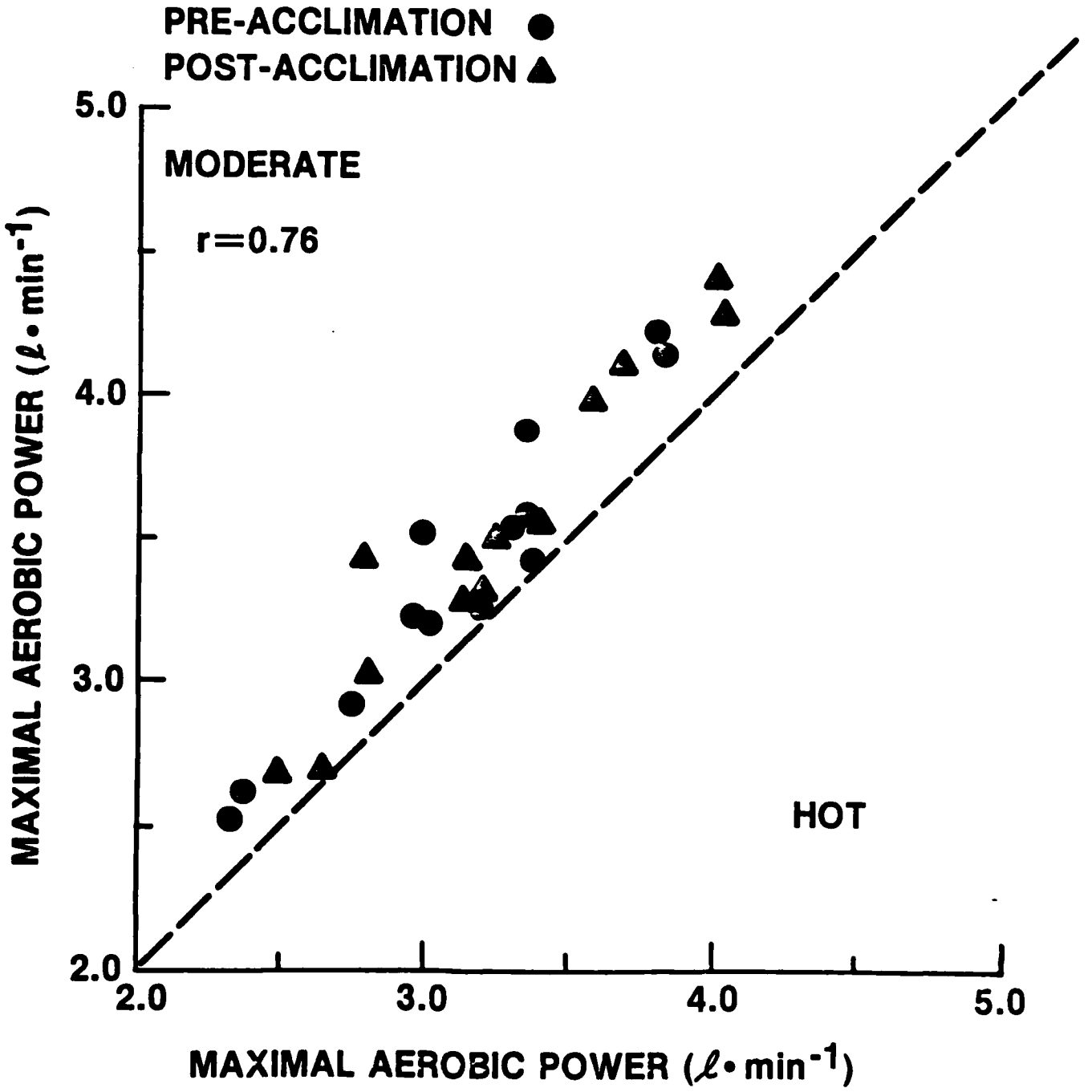
Figure Legends

Fig. 1. Maximal aerobic power values for the pre-and post-acclimation tests in both environmentals.

Fig. 2. The percent decrease of maximal aerobic power in a hot environment relative to a moderate environment during the pre-and post-acclimation tests.

Fig. 3. The percent dicrease in maximal aerobic power as a function of each individuals' absolute maximal aerobic power.

Fig. 4. The percent decrease in maximal aerobic power as a function of each individuals' aerobic fitness.


PRE-ACCLIMATION
POST-ACCLIMATION
MODERATE
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HOT
MAXIMAL AEROBIC POWER (ℓ • min-1)
MAXIMAL AEROBIC POWER (ℓ • min-1)
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4.0
3.0
2.0
2.0
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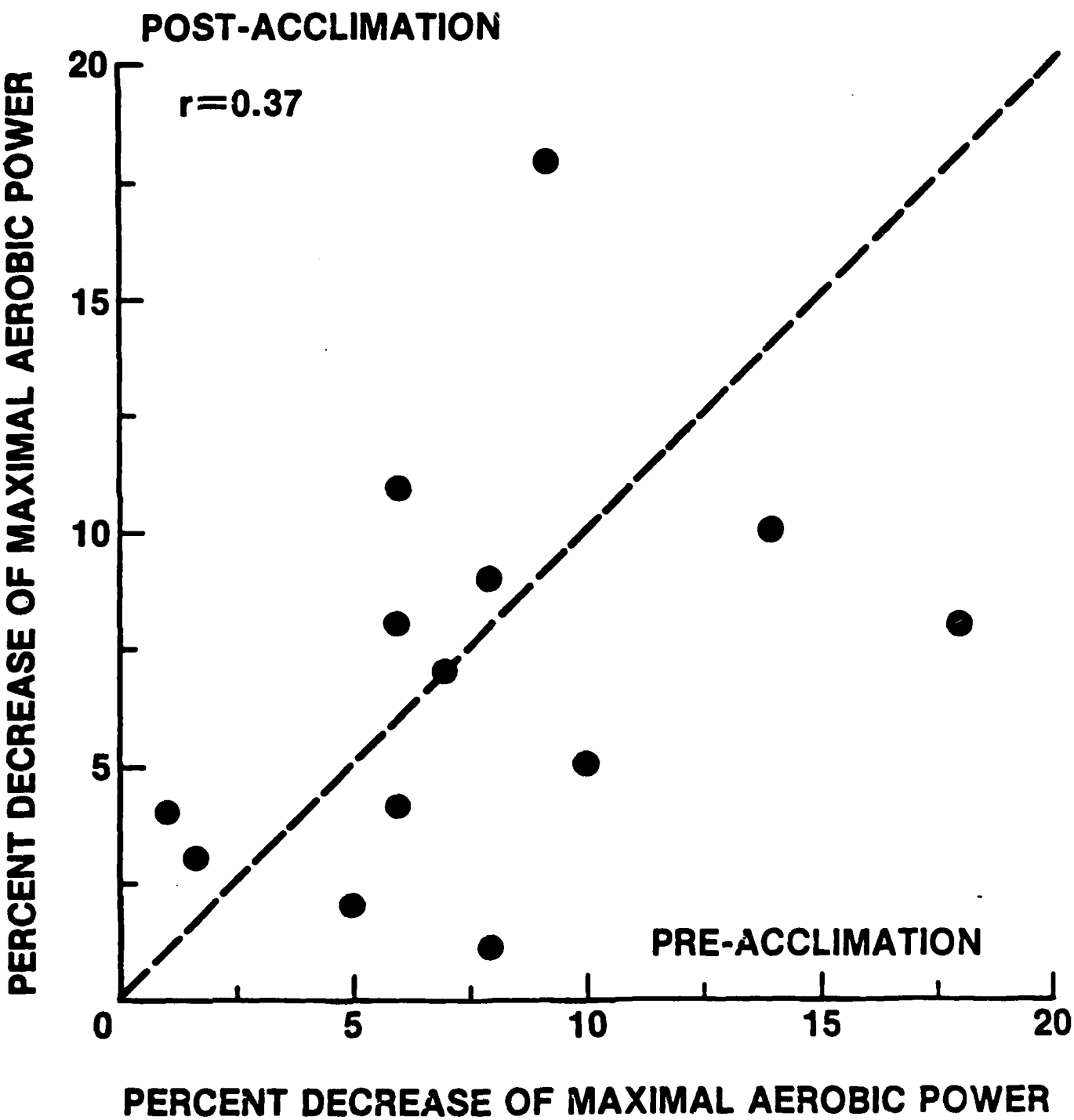
4.0
5.0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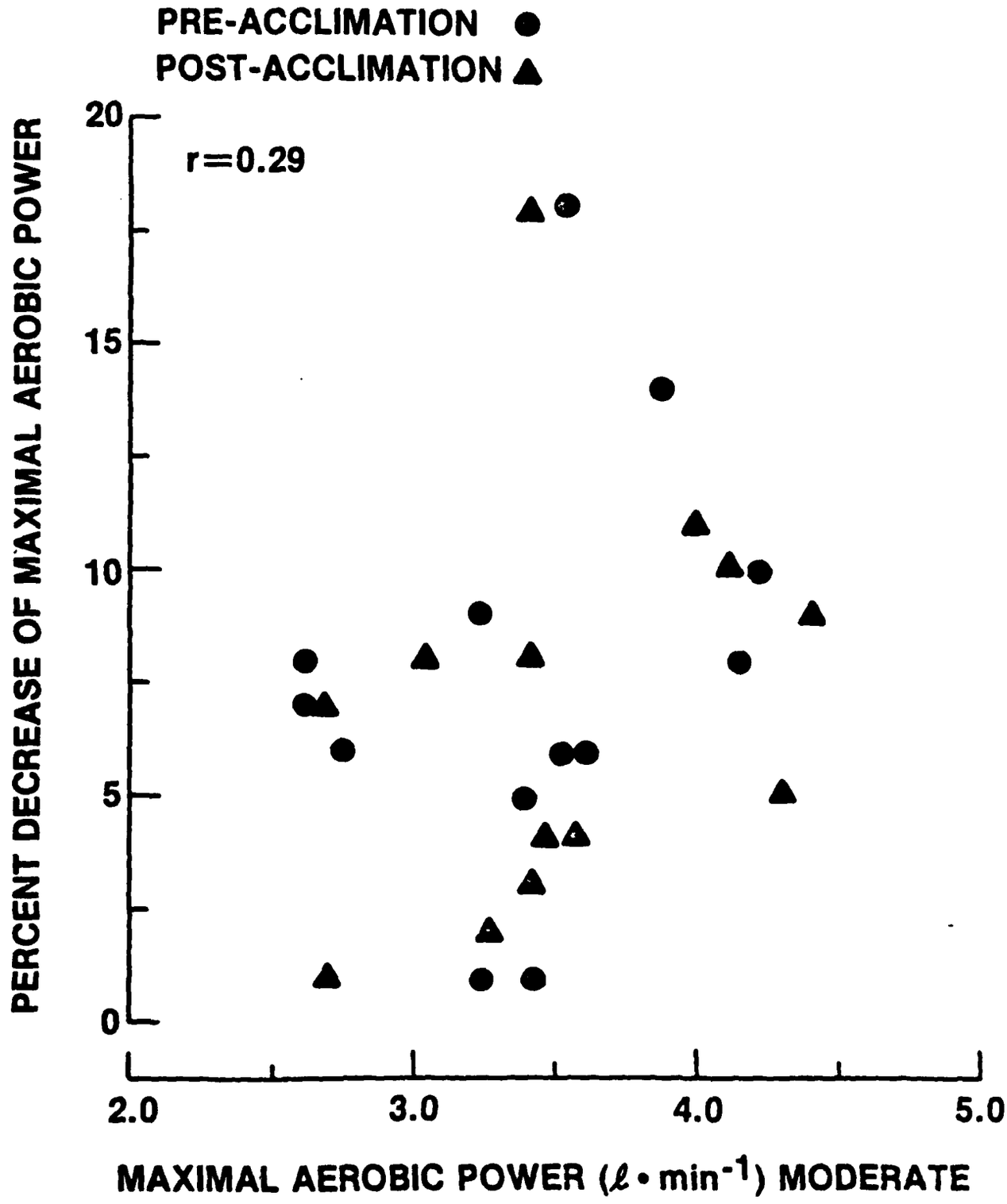
PRE-ACCLIMATION
PERCENT DECREASE OF MAXIMAL AEROBIC POWER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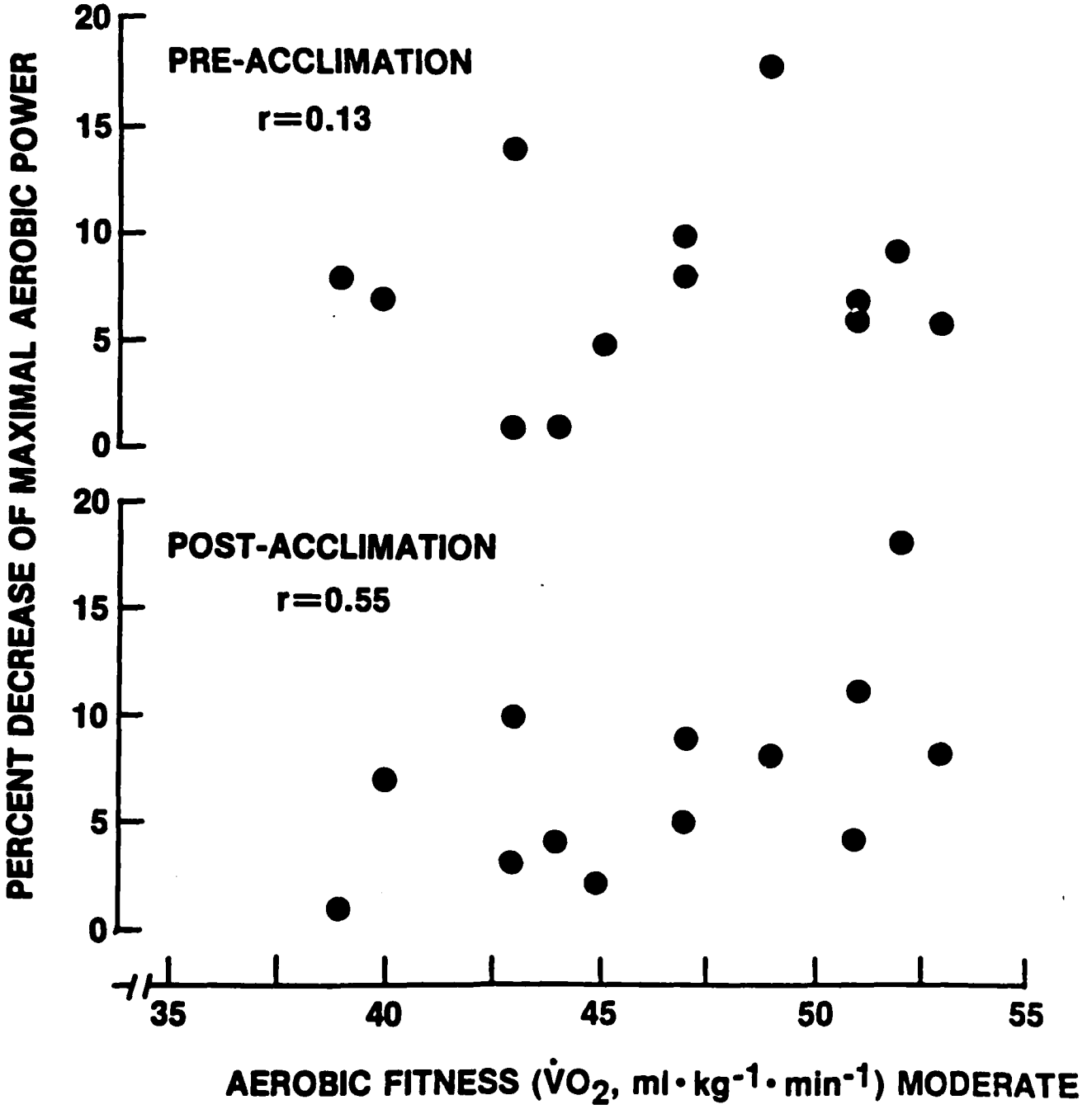
MAXIMAL AEROBIC POWER (ℓ • min-1) MODERATE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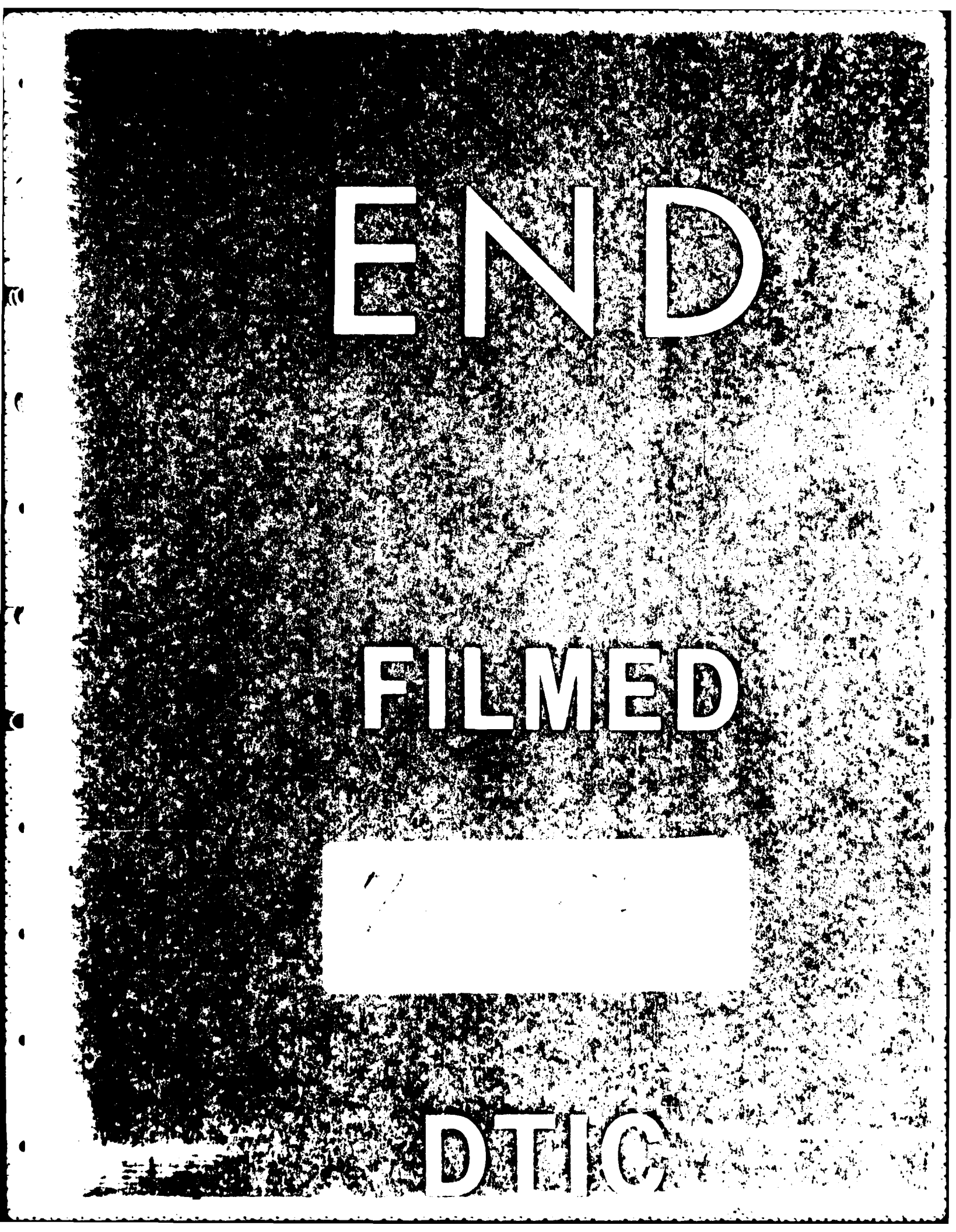
AEROBIC FITNESS ($\dot{V}O_2$, ml • kg^{-1} • min^{-1}) MODERATE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