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---------- Datos del solicitante ----------
idusuario: 0534221834
Nombre y Apellidos: Mercedes Galindo Canales
Centro: Facultad de Medicina
Departamento: Por determinar
Telefono: 1362
Direccion Postal: Facultad e Medicina$Ciudad Universitaria s/n
28040 Madrid
Tipo de usuario: Profesor
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Sensitivity of maximal aerobic power to training is genotype-dependent

D. PRUD'HOMME, C. BOUCHARD, C. LEBLANC, F. LANDRY, and E. FONTAINE
Physical Activity Sciences Laboratory, Laval University, Ste-Foy, Quebec, CANADA G1K 7P4

ABSTRACT
PRUD'HOMME, D., C. BOUCHARD, C. LEBLANC, F. LANDRY, and E. FONTAINE. Sensitivity of maximal aerobic power to training is genotype-dependent. Med. Sci. Sports Exerc., Vol. 16, No. 5, pp. 489–493, 1984. Ten pairs of monozygotic twins of both sexes were submitted to a 20-wk endurance-training program, four and five times per week, 40 min per session, at an average of 80% of the maximal heart rate reserve. Testing and training were performed on cycle ergometers. Maximal aerobic power (MAP in ml O_2·min^{-1}·kg^{-1}) and ventilatory aerobic (VAT) and anaerobic (VANT) thresholds (ml O_2·min^{-1}·kg^{-1}) were measured before and after the training program, as well as during the 7th and 14th week to adjust training to changes in maximal heart rate. Considering the 20 individuals as a group, training significantly (P≤0.01) increased MAP (from 44 ± 6 to 50 ± 6), VAT (25 ± 3 to 30 ± 4), and VANT (36 ± 5 to 42 ± 6). Thus, MAP improved by 12% of the pre-test value, while mean changes in VAT and VANT reached 20% and 17%, respectively. There were, however, considerable interindividual differences in training gains as exemplified by a range of about 0% to 41% for MAP. Differences in the MAP response to training were not distributed randomly among the twin pairs. Thus, intraclass correlations computed with the amount of improvement in MAP (ml O_2·min^{-1}·kg^{-1}) reached 0.74 (P<0.01) indicating that members of the same twin pair yielded approximately the same response to training. The same coefficient reached 0.45 and 0.24 for VAT and VANT, respectively (P>0.05). These results suggest that there are considerable individual differences in the adaptive capacity to short-term endurance training. Moreover, sensitivity of maximal aerobic power to such training is largely genotype-dependent.

GENETIC VARIABILITY, GENOTYPE-TRAINING INTERACTION, MONOZYGOTIC TWINS, MAXIMAL AEROBIC POWER, VENTILATORY AEROBIC THRESHOLD, VENTILATORY ANAEROBIC THRESHOLD

Sport scientists are concerned with individual differences observed in the response of maximal aerobic power (MAP) and anaerobic threshold to endurance-training programs. Trainability of MAP reaches generally from 10–30% of the pre-training value, but training effects well about 40% have also been reported (9). Factors such as age, sex, previous training experiences, current phenotype level, and heredity are thought to be related to these individual differences (5).

A question of considerable importance for the understanding of human variability is, therefore, whether the sensitivity of MAP or of the anaerobic threshold to aerobic training is genotype-dependent. In other words, is trainability an inherited trait? Even though doubts have been expressed regarding the presence of a genotype-training interaction effect for MAP (17), there is sufficient circumstantial evidence to support the contention that such an interaction exists for this particular trait (3,6).

One approach to this problem is to submit individuals of the same genotype to an identical training program and study their adaptive responses. This is what we have undertaken with sets of monozygotic (MZ) twins.

The purpose of this study was, therefore, to test for the presence of a genotype-training interaction effect for MAP and ventilatory aerobic (VAT) and anaerobic (VANT) thresholds in 10 pairs of MZ twins submitted to a 20-wk aerobic training program.

METHODS

Subjects. Ten pairs of healthy monozygotic (MZ) twins (6 female and 4 male pairs), with a mean ± SD age of 20 ± 2.9 yr, volunteered to participate in this study. Their zygosity was established by questionnaire and on the basis of morphological similarity as well as genetic similarity in several red blood cell antigenic and enzymatic systems and in the A, B, and C loci of the leukocyte antigens (HLA). All twins were submitted to a 20-wk endurance training program. Fourteen healthy subjects (7 females and 7 males) served as non-trained controls. Mean ± SD age of the control group was 24 ± 4 yr. Written consent was obtained from each subject prior to the study. None of the subjects were highly trained at the time of recruitment, but some were participating in recreational activities.

Testing protocol. An exercise test was administered prior to the training program and at 20th week of training. The test, conducted on an electromagnetically-braked cycle ergometer (Lode Instrument, Holland), was also performed at the 7th and 14th week of training to adjust the training program to changes in individual maximal heart rate. The initial exercise intensity was 50 W and was increased by 25 W in males and 20 W in females.

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every 3 min until exhaustion; rpm was maintained at about 60 throughout the test. Subjects were verbally encouraged to reach their maximal exercise tolerance. Expired air was collected and monitored continuously with an automated open-circuit system (Beckman Metabolic Measurement Cart) to obtain measures of \( \text{VO}_2 \), \( \text{VCO}_2 \), and \( \text{Ve} \). Gas analyzers were calibrated before and after each test with a known mixture of gases. The highest \( \text{VO}_2 \) obtained for 1 min during the incremental exercise test was recorded as the subject’s \( \text{VO}_{2\text{max}} \). The ECG was monitored throughout the test in the CM5-lead position, and heart rate (HR) was measured during the last 10 s of each min.

Ventilatory aerobic threshold (VAT) and VANT were defined as the first and second \( \text{VO}_2 \), respectively, at which the pulmonary ventilation \( (\text{Ve}) \) departs from linearity with respect to the oxygen uptake rate (16). In cases of uncertainty, the ventilatory equivalent for oxygen \( (\text{Ve}/\text{VO}_2) \) on \( \text{VO}_2 \) was used to ascertain VAT and VANT. Procedures for the determination of VAT and VANT and the reproducibility of MAP, VAT, and VANT measurements have been described previously (13).

Measurements of body composition. Body density was obtained by using underwater weighing procedures outlined by Behnke and Wilmore (2). The mean of five valid measurements was retained for the computation of body density; residual volume was assessed by using the method of Wilmore et al. (20); and the sum of six skinfolds was chosen as an indicator of subcutaneous fat (biceps, triceps, subscapular, suprailliac, abdominal, and medial calf)—measurement procedures used were those outlined by Weiner and Lourie (18).

Training characteristics. Both twins of each pair took part in a 20-wk cycle ergometer (Monark) endurance-training program, which involved: 1) an uninterrupted cycling session, 4 times increasing to 5 times a week, 40–45 min per session, starting at 60 and increasing to 85% of the heart rate reserve as computed with the formula of Karvonen et al. (10); 2) intermittent cycling session, twice during weeks 5, 11, and 18, and including series of 3 x 10 min at 80%, separated by 5 min of active recuperation. During each training session, heart rate was monitored every 2 min in order to maintain the pre-determined exercise intensity and to be certain that all subjects were submitted to the same standardized training stimulus. The average intensity of the 20-wk training program was approximately 80% of the maximal heart rate reserve. No effort was made to refrain the twins and the control subjects from participating in their usual recreational activities.

Statistical analysis. Differences between means were tested with the Student t-test. Changes induced by training were investigated for within-pair similarity by intra-class correlation procedures and significance was established as suggested by Haggard (8).

RESULTS

The subject characteristics are presented in Table 1. The control group was significantly heavier and fatter than the twin group at the beginning of the experiment. On the other hand, the twins had higher MAP per kg of body weight, VANT and VAT mean values than the controls. Both groups were comparable in terms of max heart rate, MAP (\( \text{O}_2\text{-min}^{-1} \)) as well as in VAT and VANT (% MAP). Moreover, no significant differences were found between the variances of the twins and the controls for any of the variables.

The effects of the 20-wk aerobic training program are also shown in Table 1. Training significantly increased MAP, VAT, and VANT (all \( P < 0.01 \)). Thus, MAP-kg weight\(^{-1}\) improved by a mean of 12% of the pre-test value, while mean changes in VAT and VANT reached 20% and 17%, respectively. However, there were no changes in VAT and VANT (% MAP) in the sample of twins as a result of 20 wk of aerobic training. On the other hand, no significant differences were found in MAP and VANT for the control group. Significant variations \( (P < 0.01) \) were, however, obtained for VAT in the control subjects. The paired t-tests also revealed that body weight increased significantly in the twins, but remained constant in the controls. The sum of six skinfolds did not change in any of the groups, but body density was slightly reduced in the twins. There was also a significant increase in maximal heart rate in the twins (Table 1).

### Table 1. Characteristics of the twin subjects before and after training and of the control subjects tested under similar conditions.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Twin Subjects (N = 20)</th>
<th>Control Subjects (N = 14)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre-training</td>
<td>Post-training</td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>56.8 ± 10.5</td>
<td>59.2 ± 10.8</td>
</tr>
<tr>
<td>Sum 6 skinfolds (mm)</td>
<td>57.1 ± 16.0</td>
<td>57.0 ± 15.4</td>
</tr>
<tr>
<td>Body density (g/cm²)</td>
<td>1.071 ± 0.016</td>
<td>1.068 ± 0.014</td>
</tr>
<tr>
<td>Maximal heart rate (beats-min(^{-1}))</td>
<td>197 ± 10</td>
<td>200 ± 8</td>
</tr>
<tr>
<td>( \text{VO}_{2\text{max}} ) (1-min(^{-1}))</td>
<td>2.6 ± 0.7</td>
<td>2.9 ± 0.8</td>
</tr>
<tr>
<td>( \text{VO}_{2\text{max}} ) (ml ( \text{O}_2\cdot \text{min}^{-1} \cdot \text{kg}^{-1} ))</td>
<td>44.2 ± 6.0</td>
<td>49.7 ± 6.0</td>
</tr>
<tr>
<td>VAT (ml ( \text{O}_2\cdot \text{min}^{-1} \cdot \text{kg}^{-1} ))</td>
<td>25.0 ± 3.3</td>
<td>29.8 ± 4.1</td>
</tr>
<tr>
<td>VANT (ml ( \text{O}_2\cdot \text{min}^{-1} \cdot \text{kg}^{-1} ))</td>
<td>36.0 ± 4.9</td>
<td>41.5 ± 5.5</td>
</tr>
</tbody>
</table>

Values are mean ± SD.  
$\text{§}$ Significant difference between the twins and the controls before the experiment \( (P \leq 0.05) \).  
*$P \leq 0.05$; **$P \leq 0.01$  
VAT = ventilatory aerobic threshold, VANT = ventilatory anaerobic threshold.
Figure 1 illustrates the considerable interindividual differences found in training gains for MAP, VAT, and VANT. The magnitude of changes expressed in relative value ranges from approximately 0 to 41% for MAP, from 0 to 50% for VAT, and from 2 to 32% for VANT. These differences in the training responses were not distributed randomly among the twins, as revealed by the intraclass correlations computed with the changes brought about by training. These coefficients reflect the importance of the within-pair similarity in the response to training in comparison to the between-pair variation.

The intraclass correlation computed with the magnitude of improvement in MAP reached 0.74 (P<0.01) (Figure 2) and 0.82 (P<0.01) when scores are expressed in ml O₂·min⁻¹·kg⁻¹ and in percentage changes, respectively. The same coefficients were 0.43 (Figure 3) and 0.54 (P<0.05) for VAT and 0.24 (Figure 4) and 0.33 for VANT. As a rule, coefficients are superior when computed with the percentage of improvement over the pretraining value rather than with gains in ml O₂·min⁻¹·kg⁻¹.

As expected, a significant correlation (r = −0.52; P<0.05) between the pre-training MAP per kg phenotype and the magnitude of changes in MAP caused by endurance training was observed. No significant correlations were found between the initial level of VAT (−0.41) and of VANT (−0.34) with their respective changes induced by training.
DISCUSSION

When considering the 20 individual twins as a group, the magnitude of changes in MAP are in agreement with the data reported in a study in which training was on bicycle and testing was performed on a cycle ergometer (19). Other investigators (7,14) have, however, reported larger mean training gains over shorter training periods. On the other hand, when threshold measurements are considered, training effects of 44% and 70% were reported by Davis et al. (7) and Ready and Quinney (14), respectively, for the anaerobic threshold. These results are two and three times higher, respectively, than the training gain observed for VANT in the present experiment. The largest improvements in MAP and VANT found in these studies may be associated with a difference in training intensity, lower pre-training scores of their subjects, differences in sensitivity to training on the part of their subjects, or methodological variations. On the other hand, the significant improvement observed for VAT (ml £O,-min-1·kg-1), is similar to the findings of McLellan and Skinner (11).

While the aforesaid changes in the twin group are in the expected direction, the significant improvement observed for VAT in the control group is not typical. This could be the result of a larger error variance associated with the determination of VAT than of VANT and MAP. Another explanation could be that the control subjects, after being sensitized by the testing results to their low fitness level, modified their activity patterns slightly with ensuing effects on submaximal measurements like VAT. Indeed, some control subjects could have experienced changes in their usual recreational activities over the 5-month period of the study without perceiving them as major shifts and thus not reporting them. Finally, it should also be emphasized that submaximal exercise data are more readily influenced by extraneous factors such as the level of habituation with the testing situation and emotional status prior to the exercise test and, for these reasons, they might have yielded small but significant variations in VAT.

The response to training is highly variable, as shown by the interindividual differences observed in the training gains for MAP, VAT, and VANT. Such variations in the response to aerobic training have been reported previously (4). In the present study, these differences in the training response were not distributed randomly. Indeed, as noted above, the sensitivity to training was generally similar in members of the same MZ pair for MAP and more moderately so for VAT and VANT. Even though there were significant variations in body weight with training, the pattern of changes in MAP per kg was not affected by them. Thus, MAP (l £O,-min-1) was characterized by the same trend of a similar training response in members of a given MZ pair. The intraclass coefficient for changes in MAP (l £O,-min-1) brought about by training reached 0.77 (results not shown).

It has been suggested that the following factors could be associated with the differential sensitivity to training (5): age and sex of the individual, past training experiences, current phenotype level, and relevant genotypic characteristics. In the present study, subject age was fairly homogeneous and was not related to training-induced changes. The sample of twins comprised both male and female pairs; however, there were no sex differences in the response to training, with males improving MAP on the average by 11% and females improving by 13%. Both sexes obtained identical training gains in VAT (20%) and VANT (17%). As to previous training experience, none of the twins were athletes or had training experiences on a bicycle. Thus, current phenotype level and genetic variation were the major variables that could be related to the changes brought about by aerobic training.

In the present experiment, the intraclass coefficient represents the extent of the within-pair resemblance in the response to training in contrast to the total variation induced by training in this sample. From these coefficients, it can be estimated that only approximately 20–25% of the training-induced variance in MAP could be associated with within-pair differences in the response, i.e., 75–80% of the variance in response to training are associated with genotype differences. The latter estimate reaches approximately 50% of the training response in VAT and approximately 30% in VANT. These observations strongly suggest that the sensitivity of the organism to training is, to a degree, genotype-dependent, which varies with the biological property under consideration, with the genotype dependency being highest when the organism is under a maximal stress condition. Moreover, this differential sensitivity to endurance training may account for the observation that some individuals did not improve (~ 0% gain) or gained very little with training.

Within the genotype-training interaction described above, a certain fraction seems to be associated with the initial phenotype level. This is certainly implied in the interclass correlation found in this study, as well as in others (4,12,15), between the initial level of MAP and training gains in MAP. Thus, a correlation of −0.52, or a common variance of about 27%, was found for the 20 individual twins of the study. One could therefore suggest that approximately one-fourth of the genotype-training interaction is associated with the initial value of MAP.

In conclusion, these findings support the notion that sensitivity of maximal aerobic power to training is largely genotype-dependent. This observation is in contrast with previous reports (1,17). On the other hand, the genotype-training interaction effect found for the aerobic and anaerobic ventilatory thresholds is quantitatively more moderate, but could nevertheless be biologically meaningful. Furthermore, a portion of the genotype dependency of the response to training is associated with the initial level of MAP, VAT, and VANT, which is also in-
fluenced by the genotype in sedentary individuals. From a quantitative point of view, approximately 75% of the variance found in the response of maximal aerobic power to endurance training appear to be genotype-dependent.

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Reprints may be obtained from Claude Bouchard.

Claude Bouchard and Fernand Landry are Fellows of the American College of Sports Medicine.

REFERENCES