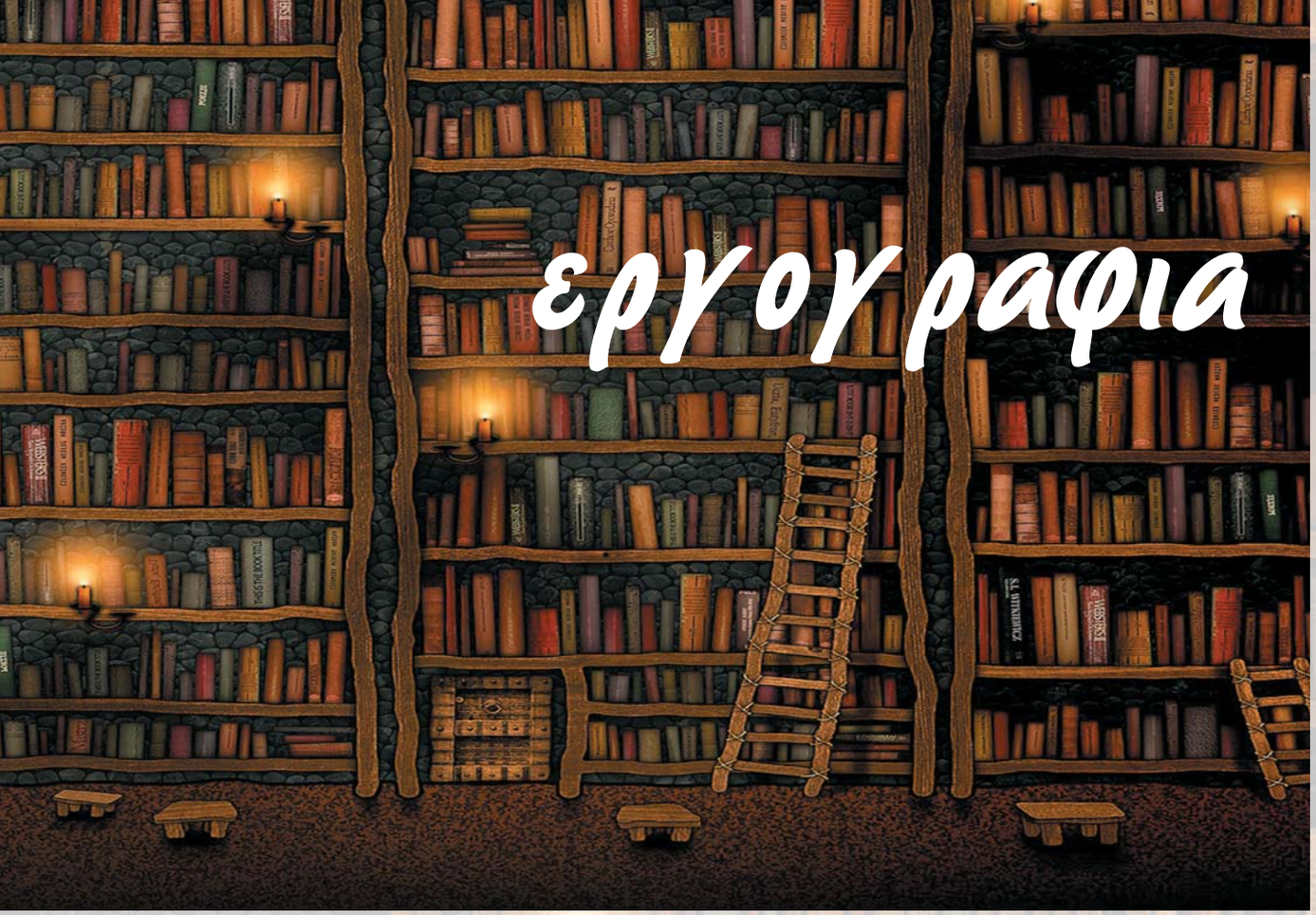
εργογραφια

Στην Ενότητα αυτή σταχυολογείται το συνολικό έργο του τιμωμένου Καθηγητή από το οποίο διαφαίνεται ο πρωταγωνιστικός και καταλυτικός του ρόλος στη θεμελίωση της Εργοφυσιολογίας και κατ' επέκταση της Αθλητικής Επιστήμης στη χώρα μας

Η δημιουργική του δράση ήταν πολυσχιδής. Ανθολογείται όμως και εστιάζεται κυρίως στους παρακάτω τομείς που σχετίζονται με την ανάπτυξη και την εμπέδωση της επιστημονικής σκέψης:



εργογραφια

ΕΝΟΤΗΤΑ 4

Ερευνητική Δράση
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Προοίμιο

Το ερευνητικό έργο του τιμωμένου καθηγητή είναι πλούσιο και πολυσχιδές. Έχει διερευνήσει όλες σχεδόν τις βιολογικές προσαρμογές (αναπνευστικές, καρδιαγγειακές νευρομυϊκές, μεταβολικές, βιοχημικές) κατά τη μυϊκή προσπάθεια, τόσο σε αθλητές όσο και σε ανάσκητα άτομα όλων των ηλικιών. Ακόμα, έχει διερευνήσει κινησιολογικές νομοτέλειες που ρυθμίζουν ή επηρεάζουν τη σωματική απόδοση, ενώ ασχολήθηκε και με την επινόηση εργομετρικών δοκιμασιών και μεθόδων για τη μέτρηση και αξιολόγηση του ανθρωπίνου βιολογικού δυναμικού.

Οι έρευνες αυτές έχουν πραγματοποιηθεί κυρίως στα ιδρύματα στα οποία σταδιοδρόμησε (Πανεπιστήμιο Springfield, Πανεπιστήμιο McGill. Εργομετρικό Κέντρο Αθλητικών Ερευνών, και Εθνικό & Καποδιστριακό Πανεπιστήμιο Αθηνών), στο πλαίσιο μεταπτυχιακών και διδακτορικών διατριβών καθώς και ερευνητικών προγραμμάτων. Έχουν δε δημοσιευτεί σε έγκριτα επιστημονικά περιοδικά και γίνεται συχνή αναφορά σ' αυτές από άλλους ερευνητές στη διεθνή επιστημονική βιβλιογραφία.

Οι έρευνές του όμως που ξεχωρίζουν και αποτελούν ιδιαίτερη συμβολή στην πρόοδο της επιστήμης είναι αυτές που εστιάζονται στη Γενετική. Η δε αρχική του εργασία με τίτλο **"Heritability of Adaptive Variation"** που δημοσιεύτηκε το1971 στο Journal of Applied Physiology, αναγνωρίζεται ως πρωτοποριακή και θεωρείται ορόσημο στο πεδίο αυτό.

Τούτο οφείλεται στο γεγονός ότι εισήγαγε για πρώτη φορά το μοντέλο των διδύμων στη διερεύνηση ενός θεμελιώδους προβλήματος, της σχετικής ισχύος του γονότυπου και του περιβάλλοντος σε φαινοτύπους που προσδιορίζουν τη σωματική απόδοση.

Ο Καθηγητής Rudolf Kovar του Πανεπιστημίου της Πράγας που οι έρευνες του εστιάζονται στη γενετική των σπορ γράφει σχετικά:

"... Σε αυτό το χώρο, η πιο πολύτιμη συμβολή είναι αναμφίβολα το έργο του Εργοφυσιολόγου Καθηγητή Κλεισούρα. Αφετηρία αποτέλεσε η έρευνά του που δημοσιεύτηκε το 1971 στην οποία πραγματεύεται τη γενετική ρύθμιση της

λειτουργικής ικανότητας του οργανισμού. Έρευνα άρτια ως προς τη μεθοδολογία, γεγονός που σε μεγάλο βαθμό συνέβαλε στην πανομοιότυπη επεξεργασία των δεδομένων από άλλους ερευνητές, δίνοντας έτσι πολύτιμα ευρήματα και επιπλέον νέα ερεθίσματα για την παραπέρα διερεύνηση».

Η ερευνητική δε αυτή προσέγγιση έγινε πρόδρομος και δρομοδείχτης μεταγενεστέρων ερευνών ταυτοποίησης γονιδίων, μετά τη χαρτογράφηση του ανθρωπίνου γονιδιώματος. Ακόμα, έγινε γονιμοποιός πολλών εργασιών μέχρι και σήμερα. Εργασίες που υλοποιήθηκαν είτε στα εργαστήρια Εργοφυσιολογίας του τιμωμένου καθηγητή, είτε σε συνεργασία με εξειδικευμένα εργαστήρια διακεκριμένων ερευνητών Πανεπιστημίων άλλων χωρών όπως Φιλανδία, Βέλγιο, Ελβετία, Ιαπωνία, Ιταλία, Γερμανία.

Στην ενότητα αυτή γίνεται επιλεκτική παράθεση ερευνητικών εργασιών του τιμωμένου που θεωρούνται ότι συνιστούν ιδιαίτερη συμβολή στην Αθλητική Επιστήμη γενικά και στην Εργοφυσιολογία ειδικότερα. Στο βασικό του σύγγραμμα «Εργοφυσιολογία» κάνει μνεία πώς άρχισε τις έρευνες στο πεδίο αυτό. Γράφει:

«Οταν πρωτοδίδαξα Εργοφυσιολογία στο Πανεπιστήμιο McGill του Καναδά με εύνοια της τύχης είχα φοιτητή ένα δίδυμο τον Peter Bender, που μου έδωσε το έναυσμα να διερευνήσω συστηματικά τη γενετική βάση των βιολογικών λειτουργιών, προσαρμογών και ικανοτήτων. Μαζί δε με τον μονοζυγωτικό αδελφό του Richard αποτέλεσαν ιστορικά, το πρώτο ζευγάρι διδύμων στο οποίο μετρήθηκε η VO_2 max και οι συνιστώσες του συστήματος μεταφοράς και κατανάλωσης οξυγόνου. Έκτοτε, το θεμελιώδες αυτό πρόβλημα της σχετικής ισχύος του γονότυπου και του περιβάλλοντος στη διαμόρφωση του βιολογικού δυναμικού και της αθλητικής απόδοσης αποτέλεσε αντικείμενο πολλών ερευνών...»

Έρευνες που υλοποιήθηκαν είτε από άλλους ερευνητές είτε από τον ίδιο στα εργαστήρια Εργοφυσιολογίας που ο ίδιος δημιούργησε, αρχικά στο Πανεπιστήμιο McGill και αργότερα στο στο Πανεπιστήμιο Αθηνών. Ακόμα, έρευνες έγιναν με γόνιμες συνεργασίες που αναπτύχθηκαν μεταξύ των εργαστηρίων του τιμωμένου και εξειδικευμένων εργαστηρίων διακεκριμένων ερευνητών σε Πανεπιστήμια άλλων χωρών όπως Φιλανδία, Βέλγιο, Ελβετία, Ιαπωνία, Ιταλία, Γερμανία.

Αξίζει να σημειωθεί ότι η ερευνητική προσέγγιση με τη χρήση του μοντέλου των διδύμων έγινε πρόδρομος και δρομοδείχτης μεταγενεστέρων ερευνών ταυτοποίησης γονιδίων, μετά τη χαρτογράφηση του ανθρωπίνου γονιδιώματος.

Στην ενότητα αυτή γίνεται **επιλεκτική παρουσίαση** ερευνητικών εργασιών που επικεντρώνονται κυρίως στη Γενετική και κατ' επέκταση στην Επιγενετική και θεωρούνται ότι συνιστούν ιδιαίτερη συμβολή στην Αθλητική Επιστήμη γενικά

και στην Εργοφυσιολογία ειδικότερα. Παρουσιάζονται δε στις παρακάτω τρεις υποενότητες:

Γενετικές Έρευνες στο Πανεπιστήμιο McGill

Απαρχή ερευνητικών εργασιών που πρωτοεισήγαγαν το μοντέλο των διδύμων και την έννοια της κληρονομησιμότητας στο πεδίο της Αθλητικής Επιστήμης.

Γενετικές Έρευνες στο Πανεπιστήμιο Αθηνών

Με αδιάπτωτο ενδιαφέρον συνεχίζεται να διερευνάται το θεμελιώδες πρόβλημα της κληρονομησιμότητας στο Εργαστήριο Εργοφυσιολογίας.

Στροφή σε Επιγενετικές Έρευνες

Η ερευνητική δραστηριότητα του τιμώμενου καθηγητή στρέφεται στο αναδυόμενο και πολλά υποσχόμενο πεδίο της Επιγενετικής.

Με την Επιγενετική κλείνει ο κύκλος των ερευνητικών τον αναζητήσεων στο λυκόφως της ακαδημαϊκής τον πορείας, που άρχισαν με τη Γενετική στο λυκαυγές της.

Ερευνητική Δράση

- **Γενετικές Έρευνες στο Πανεπιστήμιο McGill**
- **Γενετικές Έρευνες στο Πανεπιστήμιο Αθηνών**
- **Στροφή σε Επιγενετικές Έρευνες**

Γενετικές Έρευνες στο Πανεπιστήμιο McGill

Οι αρχικές ερευνητικές εργασίες που διεξήχθησαν με το μοντέλο των διδύμων κατά την καθηγεσία του τιμώμενου στο Πανεπιστήμιο McGill, δημοσιεύτηκαν σε έγκριτα περιοδικά και περιλαμβάνονται σ' ένα συλλεκτικό τόμο 234 σελίδων με τίτλο *"Genetic Basis of Ergophysiological variation".*

Εδώ παρατίθενται ενδεικτικά μόνο μερικές από αυτές και εισάγονται μ' ένα Editorial στο Research McGill της εποχής εκείνης:

- EDITORIAL
- HERITABILITY OF ADAPTIVE VARIATION (1971)
- GENETIC LIMIT OF FUNCTIONAL ADAPTABILITY (1972)
- ADAPTATION TO MAXIMAL EFFORT: GENETICS AND AGE (1973)
- GENETIC VARIATION IN NEUROMUSCULAR PERFORMANCE (1973)
- GROWTH & TRAINING WITH REFERENCE TO HEREDITY (1976)

Ακόμα, αξίζει να σημειωθεί ότι στα χρόνια της καθηγεσίας του στο McGill, πραγματοποίησε μια ερευνητική αποστολή στη ζούγκλα της Μαλαισίας, όπου ακόμα επιβιώνει μια πρωτόγονη φυλή **Orang Asli** (ο πρώτος άνθρωπος), για να διερευνήσει την οντολογική επίδραση γονότυπου και modus vivendi στη λειτουργική προσαρμοστικότητα του ανθρώπου.

Στην εικόνα αριστερά ο καθηγητής μετράει την καρδιακή συχνότητα στους Orang Asli πριν ξεκινήσουν για κυνήγι με τα φυσοντούφεκά τους.

Editorial
Research McGill, 39:1-3, 1976

Being Born with the Right Genes

Cecily Lawson-Smith

ABSTRACT: *A McGill University professor has carried out studies on approximately 200 sets of twins in order to determine the relative importance of heredity versus environment in the functional capacity of man. His results demonstrate that heredity is by far the over-riding factor. Physical training can increase Individual functional capacity but only within the limits dictated by the genetic make-up.*

The athletes who took part in the Olympic Games were in peak physical condition. To attain this, they had undergone months and in some cases years of physical training. And many of us not-quite-so-peak-form spectators would like to think that, if we had had the same advantages of youth and training, we could have been out there showing them how to really shave seconds off the records! Could we? Is training really the big variable or is an athlete actually born, not made?

Dr. Vassilis Klissouras, a McGill physiology and education professor, will tell you that the question can't be answered with a clear-cut yes or no, but that studies he has performed in a number of countries over the past eight years indicate the tremendous importance of being born with the right genes.

In 1968, Dr. Klissouras decided that a study of twins was the best way to examine the relative importance of heredity versus environment in the functional capacity of man (which to a large extent dictates athletic ability). Identical twins have the same genetic make-up; therefore, any differences between the two individuals can be ascribed to events which are not hereditary. Fraternal twins, on the other hand, have different genotypes and can be viewed as siblings of the same age.

Since 1968, Dr. Klissouras has carried out studies with approximately 200 sets of twins of all ages. About half were identical, the others non-identical. In order to gauge the functional capacities of the participants, he measured their maximal oxygen uptake after they had exercised to exhaustion. This means that a subject exercises on a stationary bicycle or a treadmill. Air containing a measured amount of oxygen is fed to him to breathe through one apparatus and his exhaled air containing oxygen and carbon dioxide is collected in another apparatus. Computations can thus be made of the amount of oxygen being picked up from his bloodstream by his body tissues. This oxygen uptake reaches its maximum when the subject is close to exhaustion since his tissues then crave oxygen. The test is based on the principle that the higher a person's maximal oxygen uptake per kilogram body wei-

ght, the better his functional capacity and thus athletic ability. In fact maximal oxygen uptake reflects changes in the capacity of the lungs, of the heart, of the oxygen transport system and of the muscle cells. It is the best single indicator of the functional capacity of the organism. The maximal oxygen uptake of an average individual is about 40 millilitre5 per kilogram body weight. Fifty millilitres indicate an above-average individual and most top athletes have maximal uptakes of over 70 and 80 millilitres of oxygen.

Dr. Klissouras has undertaken a number of different types of experiments. In an early study, for example, he worked with 25 pairs of twins, 15 of whom were identical and 10 pairs non-identical. The subjects, who ranged in age from 7 to 13 years, were each asked to perform a series of runs on a treadmill. Measurements were made of maximal oxygen uptake and of maximal blood lactate concentration, which is an indication of anaerobic (non-oxygen) functional capacity. The results showed a much greater difference between the uptake values for non-identical twins than for identical twins. In fact the differences between individual identical twins were so minimal that the researchers concluded that heredity accounts almost entirely for differences in functional capacity.

Because the subjects in this experiment were young, it could be argued that environ-mental influences, which might be more alike for identical than for non-identical twins, had some influence on maximal oxygen up-take. Dr. Klissouras therefore decided to do a follow-up study to determine whether the small differences between identical twins and the marked differences between non-identical twins persist throughout life. Thirty-nine pairs of twins (23 identical and 16 non-identical) of both sexes ranging in age from 9 to 52 years were used as subjects. The results of this study confirmed the conclusion that heredity was the overriding factor.

These two studies illustrate the importance of heredity rather than environment in functional capacity. However, they do not take into account the potential effects of training on athletic ability. To obtain some insight into this question, Dr. Klissouras tested a pair of identical twins over a period of one and a half years. One of them trained as an athlete while the other did not. The untrained twin had a maximal oxygen uptake of 35.9 millilitres per kilogram body weight, whereas his trained brother managed to attain a value of 49.2 millilitres. While this demonstrates the potential effects of training in increasing oxygen uptake, it also points out the limitations which are imposed by heredity. In spite of rigorous training, the trained twin was not able to exceed an uptake of 50 millilitres per kilogram body weight which was the average value for untrained men of his age. This suggests that rigorous athletic training cannot contribute to functional development beyond a limit set by the genetic make-up of the individual. Thus the question "Is an-athlete born or made?" should be rephrased to read "Does everybody have the genetic material which appropriate training can tune to produce a superior athlete?" And the an-

swer is "No". This is not to say that training has no purpose but rather that, even with training, each of us has a ceiling of performance dictated by our genes beyond which we will not pass.

The relative importance of training was the subject of another of Dr. Klissouras' experiments. The participants in this study were all identical twins and the purpose was to determine the effects of physical training at different ages. Twelve sets of twins were studied, four aged 10 years old, four were 13 years old and four were 16 years old. In each case, the twins were split so that one twin went through rigorous athletic training for 10 weeks while his brother did not train. At the end of the training period, the two were compared on a number of variables. As would be expected, the trained 10-year-olds and 16-year-olds increased their functional capacities more than did their untrained brothers. In contrast to this, however, the 13-year-olds' functional capacity changed at the same rate regardless of whether or not they trained. The researchers hypothesized that this was due to the adolescent growth spurt which occurs at this age. It is possible that hormonal activity is optimum at this age and any additional input such as training cannot override its influence. This negates an old hypothesis which held that more might be gained by introducing extra exercise at a time when the rate of growth is greatest.

Another question which Dr. Klissouras has examined in his work is the interaction between genetic material and training. In other words, if two people with different genotypes undergo the same training program, will one increase his functional capacity at a faster rate than the other because he has a stronger genetic make-up? Using data gathered in twin studies, he found that the answer was no. Different genotypes respond to a given training program with a change of the same magnitude.

In this connection, it should be pointed out that not only will two different individuals increase their functional capacities at the same rate, but they will also reach their ceilings of performance at the same time. Each of us can only increase our maximal oxygen uptake by a certain amount; that amount is the same for all of us. The important factor is one's starting level and that is genetically determined.

Although maximal oxygen uptake has been Dr. Klissouras' main tool in studying the question of genetics and athletic ability, he has also worked with other methods. One study which was undertaken in collaboration with Swiss colleagues, involved taking muscle biopsies from 16 pairs of twins, 10 of whom were identical while 6 were non-identical. Muscle cells of each individual were analyzed in order to study a number of structural components and to examine the activities of some energy-transforming enzymes. Comparison of their observations showed that there were no significant differences between either identical or non-identical twins. This led the researchers to conclude that variability in muscle cell structure and activity is conditioned primarily by non-genetic influences. All of the subjects in this experi-

ment were also tested for maximal oxygen uptake and it was noted that differences between individuals on this measure could not be explained by differences in their muscle cells.

The various kinds of studies outlined above do more than add to the body of theoretical knowledge on heredity versus environment. They have an obvious practical application in the selection and training of athletes for different sports. But perhaps more important is their potential use in the pedagogical field where such knowledge will enable educators to place their objectives in perspective and to realize the limits of what they can achieve through exercise and practice.

Meanwhile there's still a little solace for the armchair gold medalists among us. Another of Dr. Klissouras's studies demonstrated that the members of a primitive tribe in Malaysia, who are as yet unsullied by industrialization and urbanization, have the same average functional capacity as we do. Their level of physical fitness is also dictated by their genes.

Journal of Applied Physiology 31:338, 1971

Heritability of Adaptive Variation

Vassilis Klissouras

Ergophysiology Laboratory, Department of Physiology and Physical Education, McGill University, Montreal, Canada

The proportional contribution of heredity (Hest) to the interindividual variance of functional adaptability was estimated from an additive model of heredity and environment, based on intrapair differences in maximal aerobic power observed in monozygous (MZ) and dizygous (DZ) twins:

$$H_{est} = \frac{(\sigma^2_{DZ} - \sigma^2_m) - (\sigma^2_{MZ} - \sigma^2_m)}{(\sigma^2_{DZ} - \sigma^2_m)} \times 100$$

The application of the above equation to data obtained from 25 pairs of male twins (15 MZ and 10 DZ), whose ages ranged from 7 to 13 years and who ran on a treadmill to exhaustion, disclosed that the variability in maximal aerobic power is 93.4% genetically determined. On this evidence it was concluded that the variation observed in functional adaptability is almost entirely due to the variety of genotypes which exist in the individuals. Further, the application of the same heritability index to the twins' maximal anaerobic capacity and maximal heart rate disclosed that the interindividual variation observed in these parameters is genetically conditioned to the extent of 81.4% and 85.9%, respectively. The model used assumes that environmental influences between MZ and DZ twins are comparable and that no genotype-environment interaction exists. The tenability of these assumptions is discussed.

genetic variability; genetic endowment; heritability; functional adaptability; maximal aerobic power; maximal anaerobic capacity

MAXIMAL AEROBIC POWER (MAP) represents the upper limit of adaptational response of the organism to physical exertion; and its magnitude has been used as a performance criterion of an individual's functional adaptability (2, 4, 5, 13, 16, 17, 21, 30). There is ample evidence to suggest that MAP is affected on the one hand by extrinsic factors such as altitude, training, and prolonged periods of complete in-activity (10, 12, 21-24), and on the other hand by intrinsic factors, such as sex and age (1-3, 9, 21). However, one is puzzled by the wide interindividual variability of MAP in a homogenous population (2, 12, 21), and wonders to what extent genetic differences may account for existing individual differences.

The genetic contribution to the variance of MAP can be estimated from a simple additive model of heredity plus environment. This model makes use of monozygous (MZ) twins, who presumably have identical heredity, and dizygous (DZ) twins, who do not differ from ordinary siblings. Further, it is based on the assumptions that environmental influences are comparable for MZ and DZ twins and that no genic-environmental interaction is present. Thus, in DZ twins the variance of the differences in MAP between partners is partly dependent on genetic variability, partly due to environmental effects and partly affected by the error of measurement:

$$\sigma^2_{DZ} = \sigma^2_{DZg} + \sigma^2_{DZe} + \sigma^2_{DZm} \quad \textbf{(1)}$$

For MZ twins there is no genetic variability and the intrapair difference is attributed solely to nongenetic influences, namely environment and error of measurement;

$$\sigma^2_{MZ} = \sigma^2_{MZe} + \sigma^2_{MZm} \quad \textbf{(2)}$$

Equations 1 and 2 can be combined and by eliminating the environmental effect, which is assumed to be equal for MZ and DZ twins, we derive the following equation, which denotes the variance in dizygous twins due to genetic difference:

$$\sigma^2_{DZg} = (\sigma^2_{DZ} - \sigma^2_{DZm}) - (\sigma^2_{MZ} - \sigma^2_{MZm}) \quad \textbf{(3)}$$

Further, if we arrange the above equation in a ratio form, and refer to the term as heritability estimate Hest (14, 31) we have:

$$H_{est} = \frac{(\sigma^2_{DZ} - \sigma^2_{DZm}) - (\sigma^2_{MZ} - \sigma^2_{MZm})}{(\sigma^2_{DZ} - \sigma^2_{DZm})} \times 100 \quad \textbf{(4)}$$

This heritability index signifies the proportion of the total variance attributable to genetic variability and will be used in the present study to discover to what extent genetic predisposition accounts for the interindividual variation in maximal aerobic power. It will be further applied to the variation observed in maximal anaerobic capacity and maximal heart rate, in order to elucidate the heritability of these parameters as well.

It is to be emphasized that at no time in the present study were we concerned with the relative worth of physical training, nor were we concerned with the relative potency of heredity and environment in determining functional adaptability. In this latter respect, heredity and environment are presumably inextricably intertwined and their relative potency should be considered axiomatic in the development of functional adaptability, since it is inconceivable for a given organic attribute to develop without a hereditary basis and without an appropriate environment.

METHODS

Twins. Twenty-five pairs of male twins (15 MZ and 10 DZ) raised in the ecological setting of the same large metropolis participated in this study. Their zygosity, anthropometric data, and age distribution are given in Tables 1, 2, and 3, respectively. It will be noted that they ranged in age from 7 to 13 years. The lower age limit was set because younger children were unable to exert themselves maximally and satisfy the criteria set for attainment of maximal oxygen uptake. For this reason data obtained from a pair of 6-year-old MZ twins are not included in this report. The upper limit was set, for as children grow older, the assumption we have made of a shared environment becomes less certain.

Twins were classified as MZ or DZ on the basis of morphological traits and a serological examination. The similarity in physical appearance was used only as a first approximation of genetic identity or nonidentity. An observer can make a rough diagnosis of zygosity from physical similarities and differences, since identical twins have similar hair color, texture and curliness, similar ear lobes, and similar eye color and iris pattern. Mistaken identity by parents and dermatoglyphic analysis, which involves identification of digital patterns and palmar configuration, served as additional subjective criteria of monozygosity (6,18,26).

However, blood and serum examination renders greater precision in zygosity determination than the above criteria. Blood samples were taken from the fingertip to be tested for blood groups ABO, MN, CDE, P, Kell, and Duffy. The antisera employed in the blood typing were anti-A, absorbed anti-A, anti-B; anti-AB; anti-M, anti-N; anti-C, anti-D, anti-E, anti-e, anti-K; anti-Fya, and anti-P. Discordance for a single antisera was regarded as sufficient evidence of dizygosity (29). However, in concordant sets the median probability of monozygosity is more than 95% (8, 15, 19). Thus, the concordance observed in the 15 pairs of twins was considered as compatible with monozygosity.

TABLE 1. Anthropometric data of monozygous twins

Code No.	Twin	Age. yr	Ht, cm	Wt, kg	Lean Body Wt, kg	Vital Capacity,L, BTPS (VC)
50	DL	13	152	46.0	38.5	2.95
51	D1L	13	152	39.2	34.0	2.29
58	BG	8	138	28.9	25.4	2.13
59	B1G	8	136	28.5	25.0	2.18
72	MI	12	151	45.0	38.5	3.34
73	SI	12	150	42.6	36.8	3.05
74	DM	11	136	33.1	28.6	2.33
75	RM	11	139	35.3	30.1	2.63
78	BW	7	119	19.9	17.7	1.81
79	DW	7	119	19.9	17.7	1.44
84	KS	10	132	23.4	20.9	1.79
85	MS	10	134	26.5	23.4	1.82
88	DA	12	146	35.7	30.7	2.55
89	RA	12	144	33.4	28.8	2.62
92	GB	9	128	27.8	24.5	1.94
93	JB	9	130	29.1	25.6	1 .92
94	GJ	8	124	26.5	23.5	1.77
95	JJ	8	127	27.1	23.8	1.71
102	DJ	7	119	21.3	18.9	1.43
103	RJ	7	121	21.4	19.0	1.33
108	CB	10	129	26.8	23.5	2.13
109	KB	10	130	28.1	24.3	2.09
110	DC	13	161	46.1	39.5	3.05
111	SG	13	161	48.1	41.0	3.29
112	PD	13	156	41.8	36.7	3.11
113	LD	13	156	41.8	36.2	3.24
128	PA	9	134	28.3	24.8	2.11
129	P1A	9	134	28.8	25.2	2.13
130	FB	11	144	32.0	28.5	2.76
131	RB	11	142	29.6	26.5	2.50
Mean		10.2	138.1	32.1	27.9	2.31
±σ		±2.1	±12.7	±8.3	±6.9	±0.58
Range		7-13	119-161	19.9-48.1	17.7-41.0	1.33-3.34

TABLE 2. Anthropometric data of dizygous twins

Code No.	Twin	Age. yr	Ht, cm	Wt, kg	Lean Body Wt, kg	Vital Capacity,L, BTPS (VC)
54	RG	10	139	26.2	23.4	1.94
55	PG	10	137	30.7	26.9	1.81
60	SC	10	139	33.1	27.9	2.13
61	BC	10	134	28.1	24.7	1.42
64	BD	12	158	41.6	36.4	2.85
65	GD	12	153	39.4	34.6	2.63
66	HR	13	148	39.1	33.9	2.63
67	KR	13	140	32.7	28.3	2.49
68	TO	11	132	34.4	29.9	2.76
69	MO	11	141	39.8	35.0	2.72
76	MG	7	120	21.9	19.5	1.38
77	BG	7	114	18.7	16.6	1.57
90	EB	7	121	24.1	21.3	1.49
91	JB	7	123	25.1	21.7	1.59
104	BaR	13	158	41.9	36.9	3.00
105	BR	13	181	60.9	53.6	4.20
116	TS	9	128	23.8	21.1	2.08
117	DS	9	128	23.6	21.0	2.25
136	KW	10	132	25.1	22.5	2.00
137	TW	10	134	28.3	25.1	2.10
Mean		10.2	138.0	31.9	28.2	2.25
±o		±2.1	±15.7	±9.8	±8.6	±0.68
Range		7-13	114-181	18.7-60.9	16.6-53.6	1.38-4.20

Experimental plan. There were two testing sessions. The first session was held to a) give the twins a medical examination including a resting ECG, b) diagnose their zygosity, c) assess the type and the amount of physical activity in which they had been participating, d) familiarize them with the experimental procedures and make them used to running on the treadmill, and e) make measurements of their pulmonary function, maximal muscular strength and anthropometric dimensions. In six pairs of twins the cardiologic evidence was not completely normal and they were excluded from further testing.

During the second session the twins performed a series of runs of progressively increasing intensity on a motor-driven treadmill. They all started running on a horizontal level at a speed of 5 mph and proceeded to 6, 7, and 8 mph; thereafter the speed was kept constant at 7 mph while the slope was elevated in increments of 2.5% before each run. Each run lasted 5 min and 15 s (except for the last supramaximal one which was usually of shorter duration), and was followed by a l0-min rest pause.

Twin brothers ran alternately; this created a competitive atmosphere which motivated them to exert themselves to exhaustion at the supramaximal efforts.

Measurements. This report deals primarily with maximal aerobic power and secondarily with maximal anaerobic capacity and maximal heart rate. However, it should be noted that additional measurements were obtained on morphological traits, maximal cardiac output, and cardiorespiratory response to submaximal exercise; findings related to these parameters will be reported elsewhere.

The open-circuit method was used to determine the rate of oxygen consumption during the last minute of exercise (this was from the 4th to 5th min, except for the supramaximal efforts which was of shorter duration; the remaining 15 s of exercise was the time for cardiac output measurements). An asymptote of oxygen consumption was used as the primary criterion of maximal oxygen uptake. Oxygen uptake was calculated by a computerized pro-

TABLE 3. Twin pairs distributed according to age and zygosity

	Age, yr							Total
Zygosity	7	8	9	10	11	12	13	Pairs
MZ	2	2	2	2	2	2	3	15
DZ	2		1	3	1	1	2	10
Total pairs	4	2	3	5	3	3	5	25

gram on the basis of the volume of the inspired air and fractional concentration of oxygen and carbon dioxide in the expired air. The paramagnetic Beckman EO_2 and the infrared Godart Capnograph analyzers, calibrated with known gas concentrations which were determined by the Scholandcr analyzer, were used for gas analysis. A specially designed mixing chamber was used for sampling expired air. The inspired air was measured by a dry gas meter connected in parallel to bellows and continuously recorded on a Visicorder oscillograph. Breathing frequency and tidal volume were also determined from the same spirogram. The resistance of the respiratory circuit was less than 1 cm H_2O at maximal airflows.

A body density equation derived from a population of voting boys (19) was used to estimate the lean body weight (LBW), which was employed as a biomctric unit. The skinfold thickness of two sites (midway between the acromion and the olecranon and at the inferior angle of the scapula) was taken for this purpose by the Lange constant-pressure caliper (10 g per mm^2).

Blood lactate concentration was determined from an arterialized blood sample, taken from a prewarmed finger tip at the 5th min of each recovery period, and analyzed according to the Barker-Summerson method as modified by Strom (28). The maximal blood lactate value was used as an index of the individual's maximal anaerobic capacity.

Cardiac frequency was determined from the subject's electrocardiograph which was recorded from a Sanborn monitor (model 780-7).

The experimental error of the methods was estimated from the variation of the differences observed between duplicate determinations, made on seven subjects. Some of these subjects did not participate in the study, but were well accustomed to the experimental procedure. The values so obtained (Table 4) were used for the derivation of heritability estimates.

Statistical analysis. The single-factor analysis of variance was used to test the significance of the differences, between the mean monozygotic intrapair variance and the mean dizygotic intrapair variance. The. one-sided instead of the two-sided test of Anovar was selected on the grounds, that the mean intrapair variance in DZ twins will always exceed that in MZ twins, since the variability in the former is the result of two compo-

TABLE 4. Experimental error estimated from variation of differences observed between duplicate determinations made on seven subjects

	Oxygen Uptake, L/min, STPD	Oxygen Uptake, ml/Min per kg BW, STPD	Ventil, L/min, STPD	Resp Rate, cycles/ min	Heart Rates, beats min	Blood Lactate, mg/100 ml
No. of duplicate determinations	14	14	14	14	14	14
Mean value	2.26	44.79	63.24	43.3	184.2	77.6
Error $\sqrt{\Sigma d^2/n-1}$	0.04	1.25	1.30	1.8	1.7	3.4
Error in percent of mean value	1.9	2.7	2.1	4.3	0.9	4.4

nents: environmental variability plus genetic variability (the intrapair genetic coefficient of correlation between DZ twins is 0.5, that is on the average they differ in 50% of their genes).

The variance ratio (F) derived from the single-factor Anovar, determined whether or nor further analysis was necessary, if F was not significant at 5% level of confidence, there was no reason for further testing, as any inference thus drawn would have little meaning. But if F was significant at 5% level, we proceeded with the derivation of the heritability quotient, by introducing to equation four, the variance obtained for MZ twins, DZ twins, and error of measurement,

TABLE 5. Metabolic, respiratory and cardiovascular responses of monozygous twins to maximal work

Code No.	Oxygen Uptake, L/min STPD'	Oxygen Uptake, ml/min per kg BW, STPD	Oxygen Uptake, ml/min per kg LBW, STPD	Ventil L/min STPD (Vr)	Vr/VC	Resp Rate, cycles/ min	Tidal Vol, Lit/ cycle	Resp. Exch Ratio (R)	Heart Rate, beats/ min	Blood Lactate, mg mg/100 ml
50	2.65	57.57	68.75	72.53	24.62	60	1.2	1.01	204	75.1
51	2.31	58.82	67.75	60.10	26.23	57	1.1	0.98	204	59.3
58	1.50	51.93	59.08	51.27	24.07	63	0.8	1.05	204	35.0
59	1.46	51.23	58.31	42.29	19.38	52	0.8	1.01	203	29.5
72	2.14	47.46	55.54	67.00	20.04	62	1.1	1.06	194	36.3
73	2.09	49.08	56.77	63.28	20.77	53	1.2	1.08	193	36.6
74	1.58	47.67	55.24	45.30	19.41	48	0.9	1.05	198	44.6
75	1.71	48.48	56.82	50.90	19.35	57	0.9	1.07	197	49.0
78	1.11	55.95	62.89	43.11	23.80	72	0.6	1.09	190	52.8
79	1.08	54.46	61.20	34.10	23.66	64	0.5	0.94	193	40.0
84	1.28	54.77	61.22	51.19	28.08	54	0.7	1.10	197	45.0
85	1.45	54.75	62.02	41.77	22.98	54	0.8	1.00	198	38.9
88	2.03	56.84	66.12	57.28	22.50	60	1.0	1.05	209	79.0
89	1.83	54.66	63.46	49.02	18.69	62	0.8	1.04	201	82.8
92	1.38	49.79	56.47	40.79	21.03	77	0.5	0.96	184	22.1
93	1.48	50.89	57.95	40.39	21.00	58	0.7	0.97	188	21.6
94	1.35	51.05	57.55	37.54	21.23	56	0.7	1.02	210	29.3
95	1.43	52.66	59.97	49.38	28.84	79	0.6	1.04	215	35.4
102	1.04	48.93	55.17	23.81	16.66	75	0.3	0.86	211	
103	1.04	48.56	54.75	22.21	16.70	07	0.3	0.85	208	
108	1.35	50.34	57.53	46.55	21.85	69	0.7	1.05	196	59.6
109	1.32	46.84	54.15	44.37	21.23	64	0.7	1.00	196	50.8
110	1 .07	42.78	49.89	58.09	19.07	46	1.3	1.11	192	53.5
111	2.12	44.10	51.72	58.15	17.67	41	1.4	1.07	190	55.5
112	2.05	48.94	56.48	52.70	16.96	60	0.9	0.95	193	54.5
113	2.12	50.69	57.70	58.38	18.01	56	1.0	0.96	193	59.3
128	1.56	55.06	62.95	47.59	22.61	59	0.8	1.04	201	73.2
129	1.53	53.26	60.77	47.42	22.29	53	0.9	1.14	203	74.7
1:S0	1.73	54.01	60.55	45.70	16.55	49	0.9	0.93	198	
131	1.54	52.05	58.12	51.36	20.51	63	0.8	0.96	202	
Mean	1.64	51.45	58.89	48.43	21.22	59,7	0.83	1.02	199.03	50.09
±σ	±0.41	±3.84	±4.34	±11.12	±3.25	±8.9	±0.26	±0.07	±7.2	±17.49
Range	1.04-2.65	42.78-58.82	49.89-68.75	22.21-72.53	16.55-28.84	41-79	0.3-1.4	0.85-1.14	184-215	21.6-82.8

TABLE 6. Metabolic, respiratory and cardiovascular responses of dizygous twins to maximal work

Code No.	Oxygen Uptake, L/min STPD'	Oxygen Uptake, ml/min per kg BW, STPD	Oxygen Uptake, ml/min per kg LBW, STPD	Ventil L/min STPD (Vr)	Vr/VC	Resp Rate, cycles/ min	Tidal Vol, Lit/ cycle	Resp. Exch Ratio (R)	Heart Rate, beats/ min	Blood Lactate, mg mg/100 ml
54	1.38	52.67	58.95	39.05	20.13	50	0.8	1.00	181	
55	1.35	43.83	50.04	39.50	21.85	59	0.7	1.03	196	
60	1.60	48.27	57.34	51.61	24.26	62	0.8	0.96	206	33.0
61	1.53	54.53	62.04	56.72	40.00	79	0.7	1.10	204	39.2
64	2.33	55.96	63.95	74.35	26.09	67	1.1	1.00	204	65.4
65	2.07	52.51	59.87	52.91	20.12	50	1.1	1.01	198	52.0
66	2.21	56.61	65.23	71.52	27.19	67	1.1	1.17	197	93.8
67	2.04	62.33	72.11	59.47	23.90	65	0.9	1.00	202	75.5
68	1.77	51.43	59.26	56.91	20.66	79	0.7	1.05	209	52.8
69	1.74	43.81	49.89	52.91	19.44	66	0.8	1.07	198	33.9
76	1.21	55.09	61.94	34.85	25.24	66	0.5	0.97	189	30.1
77	0.96	51.18	57.63	27.88	17.79	71	0.4	0.98	188	35.3
90	0.97	40.28	45.60	26.32	17.66	68	0.4	1.02	189	22.7
91	1.24	49.57	57.32	30.20	19.01	69	0.4	0.99	196	42.1
104	2.13	50.78	57.69	55.38	18.48	58	1.0	0.96	213	34.3
105	2.58	42.34	48.68	84.38	20.10	62	1.4	1.04	202	53.8
116	1.38	58.01	65.50	47.17	22.71	81	0.6	0.98	192	27.5
117	1.51	63.81	71.86	35.05	15.56	60	0.6	0.91	202	18.3
136	1.51	60.21	67.25	32.12	21.11	64	0.7	0.94	198	38.1
137	1.53	54.05	60.95	46.00	22.38	71	0.6	0.95	192	35.8
Mean	1.65	52.37	59.63	49.22	22.18	65.7	0.77	1.01	197.8	43.53
±σ	±0.44	±6.45	±7.24	±15.67	±5.16	±8.3	±0.27	±0.06	±7.8	±19.19
Range	0.96-2.58	40.28-63.81	45.60-72.11	26.32-84.38	15.56-40.00	50.0-81.0	0.4-1.4	0.91-1.17	181-213	18.3-93.8

RESULTS

Data obtained are given in Tables 5 and 6 for MZ and DZ twins, respectively. The mean values and their standard deviations for maximal oxygen uptake and maximal blood lactate concentration clearly show the comparability of the two groups.

A close examination of the individual values discloses that the intrapair difference tends to be greater between DZ twins than between MZ twins. Such a difference becomes more apparent if the pair values are plotted against each other in a system of Y-X coordinates, as was done in Figs. 1, 2, and 3 for maximal oxygen uptake, maximal blood lactate concentration, and maximal heart rate, respectively. It may be seen that the scores of the monozygous twins tend to cluster around the line of identity and fall within the shaded area which represents the magnitude of the error of measurement, whereas the scores of the dizygous twins arc widely scattered.

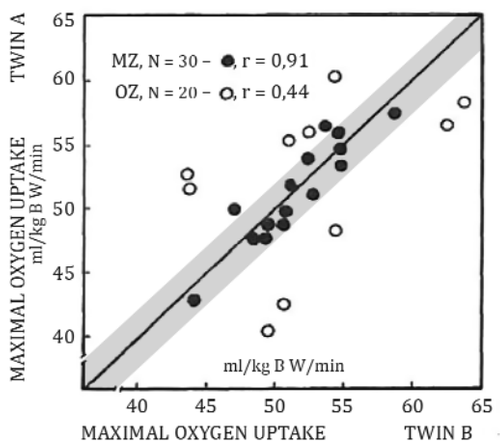

FIG. 1. Intrapair values of maximal oxygen uptake for MZ MZ and DZ twins. Shaded area represents magnitude of the error of measurement.

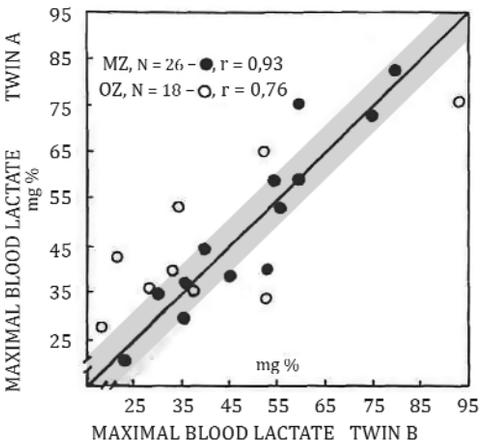

FIG. 2. Intrapair values of maximal blood lactate concentration for MZ and DZ twins. Shaded area represents magnitude of the error of measurement.

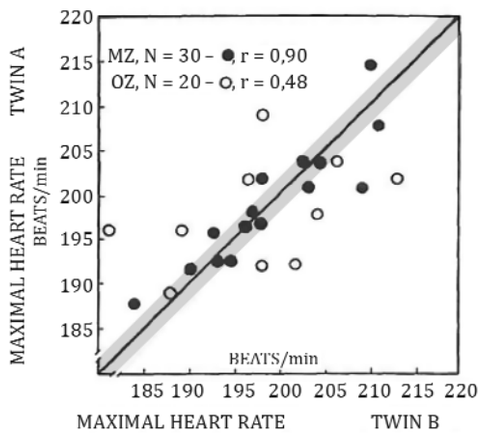

FIG. 3. Intrapair values of maximal heart rate for MZ and DZ twins. Shaded area represents magnitude of the error of measurement.

It was found that the difference of the intrapair variance between MZ and DZ twins was significant well beyond the 1% probability level for maximal oxygen uptake (expressed either in kg BW or kg LBW) and maximal heart rate, and beyond the 5% level of confidence for maximal blond lactate concentration. Thus, we proceeded with the computation of the more elaborate heritability estimates for these variables by applying equation four, and the results so obtained are presented in Table 7. It appears that the variability of maximal oxygen uptake, maximal blood lactate concentration, and maximal heart rate is genetically determined by 93.4%, 81.4%, and 85.9%, respectively.

The intrapair variation of MZ twins was not, however, significantly different from that of DZ twins with regards to respiratory frequency (F = 0.19), tidal volume (F = 0.40), respiratory exchange ratio (F = 0.57), oxygen pulse (F = 4.09), minute ventilation (F = 2.53), and ventilation vital capacity ratio (F = 3.29). Since an F ratio of 4.28 was needed for significant difference at 5 % level (N = 23 η = 1), further analysis of these parameters had no meaning and it was, thus, abandoned.

DISCUSSION

The validity of the heritability estimate of any organic attribute depends on the acceptability of the underlying assumptions. Two basic assumptions were made in the derivation of the present heritability estimates. It was assumed, on the one hand, that environmental influences were comparable for MZ and DZ twins, and, on the other, that no genic-environmental interaction was present.

In regard to the tenability of the first assumption, one has to consider both the prenatal and postnatal environment. It has been argued that differences in intrauterine position and blood supply to the embryo, and accidental differences in the make-up of the cytoplasms may result in structural and biochemical differences between monozygous twins (20, 27). Albeit such differences in the prenatal environment may exist, no phenotypical differences that could be ascribed to their effect were observed in the identical twins of the present study. The mean intrapair difference for maximal aerobic power and maximal anaerobic capacity in MZ twins was well below any significant level (F ratio of 1.04 and 0.50, respectively). This

strongly suggests either the equality of prenatal environment or that existing prenatal differences are not enduring, but are progressively equalized under the influence of a genetic maturational pacemaker (25). This, of course, would only apply to prenatal differences which do not result in injury of a vital organ that in turn may cause some developmental anomaly, or malformations. In any event, prenatal inequalities would only lead to an underestimation of the share of heredity in the discordance of nonidentical twins.

TABLE 7. Estimates of variance within male dizygous twins (σ^2_{DZ}), variance within monozygous male twins (σ^2_{MZ}), variance of error of measurement (σ^2_m), F ratios, and herritability of variation (H_{est}) of selected parameters.

Attribute	σ^2m	σ^2MZ	σ^2DZ	F=σ^2DZ /σ^2MZ	H est %
Max oxygen uptake, ml/min per kg BW	1.30	15.37	214.05	13.9*	93.4
Max blood lactate, mg/100 ml	31.70	156.57	701.88	4.9#	81.4
Max heart rate, beats/min	2.63	40.75	273.81	6.8*	85.9

*Significant well beyond the 1% probability level
#Significant at 5% probability level.

As far as the postnatal environment is concerned it is believed that no differentiating influences were operant, on the following grounds. First, both MZ and DZ twins were reared in the same city and came from families of the same socioeconomic status. This makes it likely that life styles were similar, including upbringing, living standards, and leisure-time activity. Second, comparatively young children (7-13 years) were studied, for as children grow older the assumption of a shared environment becomes less certain. Due to this selection, the twins were to a great extent similar with respect to their participation in physical activity (Fig. 4), presumably the extragenetic agent capable of most strongly affecting functional adaptability. In spite of this, it is conceivable that the intensity of exercise might not have been the same for both identical and fraternal twins. However, there is some evidence to suggest that additional exercise during the prepuberty period does not substantially alter maximal aerobic power (7, 11). These observations speak strongly in favor of the comparability of environ-mental influences.

In regard to the second assumption, it cannot be ascertained from the available data, whether the interaction between heredity and environment is a source of intrapair variation in functional adaptability (FA). It is quite probable that the present simple model of heredity plus environment may not be adequate to explain the observed within pair variance of the dizygous twins, and that it should be modified to include an additional term, signifying the mutual interaction between heredity and environment, notably:

$$\sigma^2_{FA} = \sigma^2_g + \sigma^2_e + \sigma^2_{ge} + \sigma^2_m \qquad \textbf{(5)}$$

However, the inclusion of this multiplicative component requires experimental confirmation. "Split-twin" experiments, in which one twin trains and his genotypically identical partner acts as a control, are currently being conducted in this laboratory.

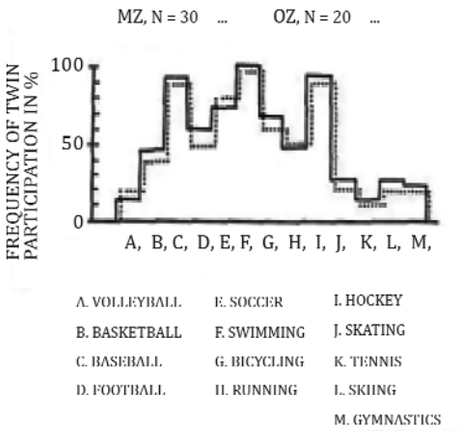

FIG. 4. Histogram showing percentage of twins (MZ and DZ) participating in indicated physical activity.

With data obtained in this manner it will be possible to distinguish three sources of variation: intrapair variation (training effect), interpair variation (genetic disposition), and interaction between the two. In individuals of a vast genetic variability who are subject to a maximal training stimulus, it is plausible that the modus operandi of heredity and environment is that of interaction. In other words, the increment in FA produced by an increment in training may be greater for individuals with a higher FA than for those with a lower one, or mutatis mutandis.

In any event, it is unlikely that such interaction took place to any marked degree in the present subjects. The twins were at an early stage of development and the environmental influences are not as pronounced at this period as they are at later developmental stages. In fact, no twin had participated in organized competitive sports and the training stimulus was certainly far from maximal. Further, MZ and DZ twins had equivalent means and between pair variance on FA (Tables 5 and 6), meaning that even if there were a genotype-environment interaction, it would have been of about the same magnitude for both MZ and DZ twins. Furthermore, DZ twins had a comparable intrapair variability at different levels of functional adaptability (Table 6). Though the assumption that genotype-environment interaction does not exist is not presently susceptible to rigorous proof, it seems justifiable to omit this component from the computations of heritability estimates without risk of serious error.

In conclusion, on the grounds of the evidence obtained it would seem that heredity alone accounts almost entirely lor existing differences in functional adaptability, as measured by maximal aerobic power, in a fairly homogeneous group of individuals. My deep appreciation is extended to the twins and their parents for their enthusiastic cooperation. I am indebted to Messrs. Steven Iscoe, Peter Vaktor, and George Dimakis for their devoted efforts in the collection and computation of data. I am also grateful to Dr. Brian Lowery for the medical examination of the twins and to Dr. Julius Metrakos for his advice in the zygosity determination.

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Genetic Limit of Functional Adaptability

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Summary. A pair of monozygous twins, a trained athlete and his identical untrained counterpart, were tested over a period of 1 % years. Monozygosity was established on the basis of morphological traits and serological criteria. Repeated measurements obtained during basal conditions, submaximal and maximal exercise on a cycle ergometer disclosed that: a) the basal heart rate, respiratory rate, minute ventilation, vital capacity and forced expiratory volume were insensitive to training; b) cardiorespiratory displacement during submaximal work was smaller in the trained than in the untrained twin; and e) the maximum muscular force, mechanical power output, aerobic power and anaerobic capacity were about 10, 50, 37 and 60%, respectively, higher in the trained than in the untrained twin. The increase of the maximal aerobic power was attributed equally to an enhancement of the O2 transport and 02 utilization systems. It was concluded that although exogenous factors are important, a strong hereditary component sets a limit to functional adaptability.

Key words: Genetic Endowment — Monozygous Twins — Functional Adaptability — Training.

Functional adaptability (FA) is an expression of the upper limit of adaptational response of the organism to a biological demand and it is most commonly measured in man by a test of maximal oxygen uptake. In this sense FA is a continuum, and the placement of an individual on this continuum will be dependent upon both genetic and nongenetic influences. If there is no diversity in the amount of environmental influences exerted on a homogeneous population, the genetic factor is the exclusive determinant of individual differences in FA (Klissouras, 1971). However, if the amount of environmental influences is varied by an external agency such as physical training, then any interindividual variation observed in FA should be attributed partly to nongenetic factors; since physical framing is known to affect maximal oxygen uptake strongly (e.g. Robinson et al., 1941; Rowell, 1962; Saltin et at., 1968; Ekblom, 1969 a). The question then arises as to whether an individual's locus on the continuum of FA is primarily conditioned by training or set by the genotype.

The use of cross-sectional and longitudinal studies in envisaging this hypothesis has the obvious limitation that the genetic factor is operant to an unknown degree in different individuals. Using monozygotic twins as subjects, however, obviates this problem since each subject is accompanied by a genotypieally identical control. It was reasoned in. selecting the co-twin analysis that if athletic training, confined to one twin and extended over a period of years, failed to raise his functional adaptability from a low to a superior level, then the upper limit of FA might be assumed to be set by the genotype.

METHODS

Subjects. Measurements were obtained from a set of monozygotic twins, monozygosity being established on the basis of morphological traits, blood factors, serum proteins, concordance of antisera (Dencker et at., 1961; Nickols et al., 1966), and similarity of physical appearance (Table 1).

The twins were 21 years of age at the onset of the study and they were tested periodically over a 17-month period. Both twins had had a very active childhood and from the ages of 8 to 15 years had engaged in athletic training. Thereafter, their lifestyles changed. One twin (RB) became interested in cars and participated only occasionally in the summer in unorganized sports such as swimming and golfing. He became a salesman, driving about 1000 miles a week and generally leading rather a sedentary life. Conversely, his brother (PB) underwent strenuous athletic training for competitive sports at senior high school and at the university where he was a physical education student (and a member of the football and ice hockey teams). His training was year-round and was designed to develop both maximal aerobic and anaerobic power.

In view of these kinetic profiles, the one twin (RB) was designated as untrained (U) and his identical counterpart (PB) as trained (T).

Measurements. Observations on oxygen consumption, oxygen debt, lactate production, cardiac output and heart rate were made during submaximal and maximal work on an Elema-Scholander cycle ergometer.

Oxygen consumption during work was measured by the open circuit method in a manner previously described (Klissouras, 1971). An asymptote of oxygen uptake was used as the primary criterion of the subject's maximal aerobic power.

Both the maximal respiratory oxygen debt and the maximal blood lactate concentration were used as indices of the individual's maximal anaerobic capacity. The post-recovery oxygen level, after maximal exhausting exercise, was taken as a baseline for the calculation of maximal oxygen debt. The recovery period was extended for about 1 hr and it was terminated when minute ventilation became essentially constant. Minute ventilation was monitored by an electronic counter for this

purpose. Blood lactate concentration was determined from an arterialized blood sample taken from a pre-warmed finger tip at the 5th min of recovery and analyzed according to the Barker-Summerson method as modified by Strom (1949).

The cardiac output was estimated by the carbon dioxide rebreathing method (Defares, 1958) using a continuous sampling (Jernerus et al., 1963) and a graphical analysis (Klassen, 1965) as described by Perguson et al. (1968).

Cardiac frequency was determined from the subject's electro-cardiograph, which was recorded from a Sanborn patient monitor (model 780—7).

The isometric force produced by the maximal contraction of the anterior femoral muscles of the dominant leg was measured by means of a specially developed device previously described (Kuroda et at, 1970). The vital capacity was assessed by a Collins 9-1 respirometer.

The fat percentage was predicted from skinfolds taken by the Lange constant pressure caliper (10 g/mm2) and introduced into the equation (Wilmore et αl., 1969):

Percent fat = 5.783 + 0.153 (triceps skinfold + scapula skinfold + abdominal skinfold + suprailiac skinfold).

RESULTS

Table 2 and Fig. 1 present measurements taken under basal conditions and the differences observed in these measurements between the trained and the untrained twin. The basal heart rate, respiratory rate and minute ventilation were not affected by training. The basal oxygen consumption of the trained twin was substantially higher than in the untrained one, but the difference between them disappears when the values are expressed per unit of lean body weight. No differences were observed either in vital capacity or in forced expiratory volume. Protoplasmic tissue was greater in the trained twin (Table 2) which may account for his greater muscular force and the apparent elevation in basal oxygen consumption.

TABLE 1. Diagnosis fo zygosity

A. Morphology

Twin	Hair color	Hair texture	Hair curliness	Eye color	Eye iris pattern	Ear lobe	Digital ridges	Finger prtin patterns	PTC*
Untrained	Blond	Soft	Same direction	Blue	Same	Attached	162	Same	Yes
Trained	Blond	Soft	Same direction	Blue	Same	Attached	170	Same	Yes

B. Serology

	A	B	AL	AB	H	N	C	d	E	c	e	C^W	Fy^a	K	k	P	s
Untrained	+	—	+	+	—	+	+	—	—	+	+	—	—	—	+	+	+
Trained	+	—	+	+	—	+	+	—	—	+	+	—	—	—	+	+	+

*Ability to taste phenylthiocarbamide.

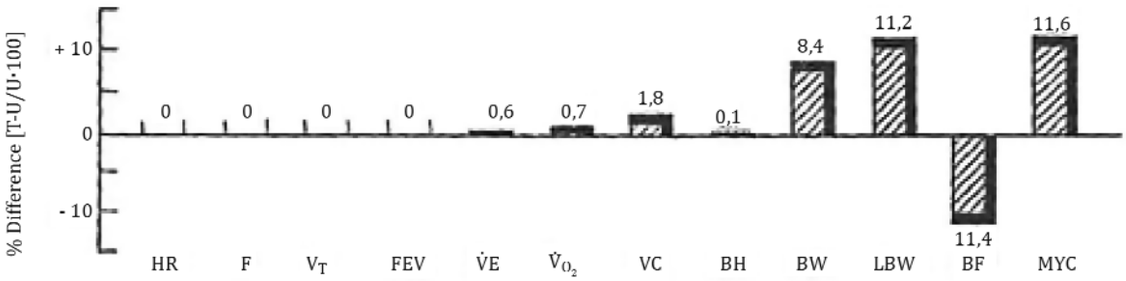


FIG. 1. Deviations in resting parameters of the trained from the untrained twin. Symbols are explained in Table 2

The trained twin exhibited a significantly smaller displacement of cardiorespiratory parameters during submaxirnal work, as shown in Table 3 and Fig. 2. This phenomenon is commonly observed in trained subjects and apparently is not due to inherited static dimensions or superior regulatory capacity. Further, the oxygen uptake and the mechanical efficiency during submaximal work were about the same for both twins. The untrained twin utilized anaerobic energy sources to a greater extent than his brother and hence his work efficiency may have been overestimated, since only the steady state oxygen uptake was considered for its estimation.

A work load which could be sustained and exhaust the subject within 5 min was considered as maximal. The trained twin produced a maximal work output which was 50% higher than that of his untrained counter-part, enhancing his aerobic and anaerobic metabolism by about 40 and 60%, respectively (Table 4 and Fig. 3). It appeared that the higher maximal aerobic power was attained by an equal enhancement of cardiac output and oxygen extraction by the tissues. The greater maximal cardiac output was accomplished by a rise in the stroke volume, since the maximal heart rate was the same for both twins.

TABLE 2. Basal and anthropometric data

Twin	Body height cm (BM)	Body weight kg (BW)	Lean body weigth kg, (LBW)	Body fat % (BF)	Max. isom. force, kg (MVC)	Vital cap. BTPS, lit. (VC)	Forced esp. Vol.% (FEV_1)	Oxygen uptake lit./ min	Oxygen uptake ml/min kg LBW (VO_2)	Heart rate beats/ min (HR)	Ventil. BTPS lit./min (VE/E)	Respir. rate cycles/ min (F)	Tidal volume lit./ min (V_T)
Untrained	177.8	66.0	54.3	17.5	50.53	5.13	87	0,240	4.43	54	8,45	15	66
Trained	177.7	71.6	60.4	15.5	56.43	5.23	87	0,270	4.45	64	8,50	15	56
Percent Diff.	+0.1	+8.4	+11,2	- -11.4	+ 11.6	+1.8	0,0	+ 12,5	+0,7 0.0	+0.6	0.0	0,0	

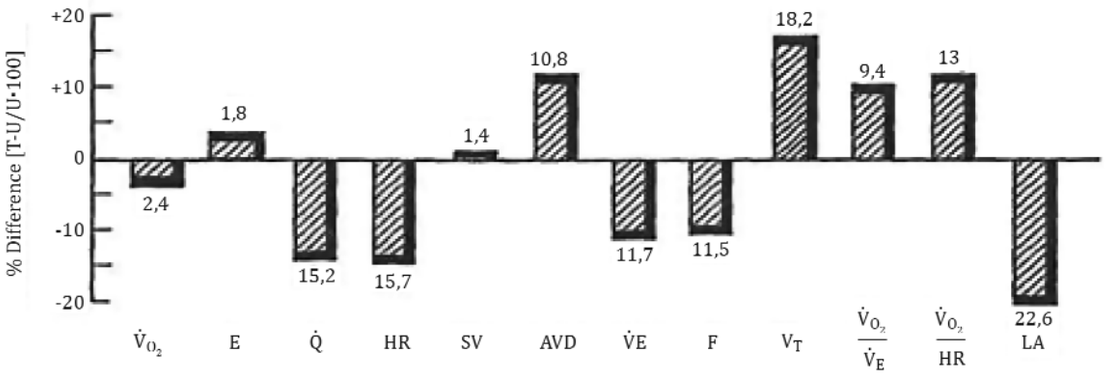
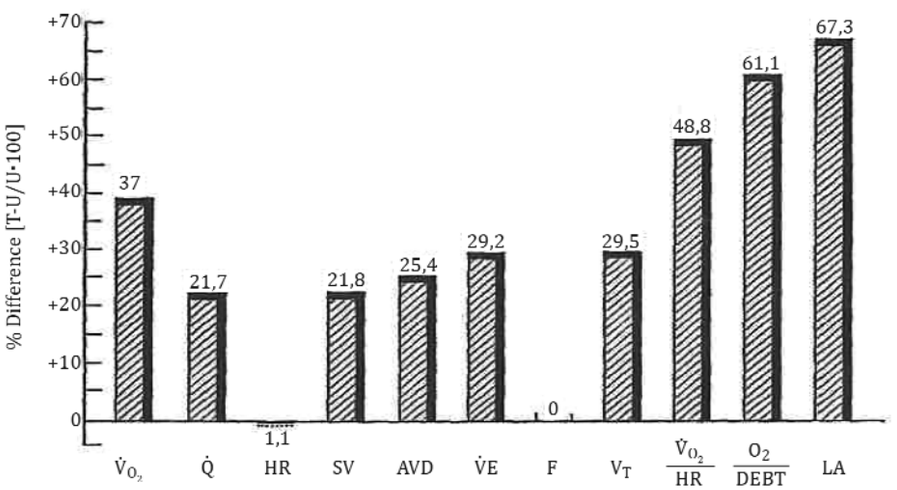


FIG. 2. Deviations in submaximal responses of the trained from the untrained twin. Symbols are explained in Table 3

DISCUSSION

The basal heart rate data support the contention that the bradycardia observed in endurance athletes may not be a result of training, but a genetically determined biological constant. This is further supported by the fact that bradycardia is often observed in non-athletes (Dill et al., 1966). The identicalness of the twins' maximal heart rate may also be indicative of a genetic preponderance.

The kinetics of adaptation and de-adaptation to maximal work as assessed by oxygen uptake and lactate removal showed a similar trend for both twins, when they were expressed in percent of individual maximum values. This strongly suggests, on the one hand, that the time course of the relative displacement of a physiological property might have a genetic basis, and on the other, that the magnitude



PIG. 3. Deviations in maximal responses of the trained from the untrained twin. Symbols are explained in Table 4

TABLE 3. Adaptational response to submaximal work (600 kpm/min)

Twin	Oxygen uptake lit./min (VO_2)	Blood lactate mg% (LA)	Cardiac output lit./min (Q)	Heart rate beats/ min (HR)	Stroke volume ml/beat (SV)	(A-V)O_2 diff. ml/lit. (AVD)	Ventila-tion, BTPs lit./min (VE)	Respir. rate cycle/ min (F)	Tidal volume lit./cycle (VT)	Respir. Equiv.O_2 ml/lit. ($\dot{V}O_2$)/ (VE)	Oxygen pulse ml/beat ($\dot{V}O_2$)/ (HR)	Work effic, % (E)
Untrained	1.25	17,7	11.2	115	97.0	9.68	45.40	26	1.1	33.1	10.0	22.5
Trained	1.22	13.7	9.5	97	98.4	10.72	40.10	23	1.3	36.2	11.3	22.9
Percent Diff.	—2.4	-22.6	—15.2	—15.7	+ 1.4	+ 10.8	- -11.7	—11.5	+18.2	+9.4	+ 13.0	+ 1.8

of displacement constitutes, perhaps, the sole characteristic of adaptation to training. More complete data are needed to illuminate this hypothesis further.

The trained twin's 40% improvement in maximal aerobic power is about twice the expected mean improvement resulting from training. This difference is explainable, since the inter-individual response to training veries greatly (Dill et al., 1966; Saltin et al., 1968) and depends on such factors as intensity, duration and initial level of FA (Saltin et al., 1969).

The most important finding, however, is not the percentage improvement in MAP but the absolute values. The untrained twin had a MAP of 35 ml/min/kg BW, whereas the trained twin had a peak value of only 49 ml/min/kg BW. This latter value is comparable to an average maximum value of about 50 ml/min/kg BW for untrained college men of the same age, well below values reported for top athletes (Saltin et al., 1967). So despite hard and prolonged training, the trained twin was unable to surpass an average level of FA. The reason for this seems to hinge on his low pretraining functional adaptability, as judged from that of his identical counterpart. This observation strongly suggests that rigorous athletic training cannot contribute to functional development beyond a limit set by the genotype.

Several workers have inferred that training can produce results only within the

TABLE 4. Adaptational response to maximal work (Un -1200 kpm/min, Tr =1800 kpm/min)

Twin	Oxygen uptake lit./ min	Oxygen uptake ml/min/ kg BW (VO_2)	Oxygen debt lit. (0_2debt)	Blood lactate mg% (LA)	Cardiac output lit./ min ($\dot{Q}$)	Heart rate beats/ min (HR)	Stroke volume ml/ beat (SV)	(A-V)0_2 diff. ml/ lit. (AVD]	Ventil. BTPS lit. min ($\dot{V}E$)	Respir. rate cycle/ min (F)	Tidal volume lit./ cycle (V_T)	Oxygen pulse ml/beat ($\dot{V}O_2$)/ (HR)
Untrained	2.37	35.9	4.13	73.4	20.3	184	110	11.8	126.20	60	2.10	12.9
Trained	3.49	49.2	6.65	122.8	24.7	182	134	14.8	163.10	6O	2.72	19.2
Percent Diff.	+47.3	+37.0	+61.0	+67.3	+21.7	–1.1	+21.8	+25.4	+29.2	0.0	+29.5	+48.1

TABLE 5. Maximal oxygen uptake in the twins over a 17-month period (values are in millilitre O_2/min/kg B W)

Date of test	Trained	Untrained
December 1968	49.19	35.97
March 1969	47.91	35.64
December 1969	49.66	–
April 1970	48.95	34.39

variability allowed by the genotype (Gedda, 1960; Åstrand, 1967; Robinson, 1968). Åstrand in particular, pointed out that the standard deviation of maximum oxygen uptake in a large homogeneous population with a similar degree of training is 13%. Suppose then that the mean value of maximum oxygen uptake in a given population is 40 ml/min/kg BW; individual values for 95% of the population (2 SD) will range anywhere between 29.6 and 50.4 ml/min/kg BW. With rigorous training the individual with a maximal value of 29.6 will be able to reach a level of about 40 ml/min/kg BW. Such an individual can never expect to achieve any distinction in athletic performance which is dependent upon the maximal aerobic power.

One may wonder, however, whether the training stimulus in the pre-adolescent and/or post-adolescent period was sufficient to elicit the maximum possible adaptation in the trained twin.

We are largely ignorant regarding the question of whether training at an earlier stage of maturation would have produced a higher functional adaptability. However, there is some evidence that training during the pre-puberty period does not substantially alter functional adaptability (Ekblom, 1969 b; Daniels et al., 1970). Values of maximal oxygen uptake, exceeding 65 ml/min/kg BW have been observed in children as young as 8 years of age (Daniels et al., 1970; Skinner et al., 1970; Klissouras, 1971). These values are equivalent to those of high-calibre athletes and are indicative of a strong hereditary component. In this connection it is of interest to note the maximal aerobic power of 70 ml/min/kg BW observed in a pair of identical twin skiers at the age of 25, neither of whom had taken part in organized athletics until age 18 (Douglas, 1967).

The superior development observed in those adolescents who engage in competitive sports (Åstrand et al., 1963) is not in doubt. However, the relative contribution of training to their superior development cannot be clearly separated from that of the genotype. In order to elucidate the relative effect of physical training, on functional adaptability, at different developmental ages, a longitudinal study is currently being conducted in this laboratory using monozygotic twins to hold constant the hereditary factor.

With regard to the training stimulus during the post-adolescent period, there is good reason to believe that it was maximal. Training was carried out to the tolerance limits of the twin and his maximal aerobic power did not change over a 17-month period (Table 5). Indeed, several workers have failed to observe any increase in maximal aerobic power in athletes who have reached their full growth (Grande et al., 1965; Saltin et al., 1967; Ekblom, 1969a).

In conclusion, an individual's functional adaptability, as measured by maximal aerobic power, can be improved substantially by training, but its ceiling is set by the genotype.

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Adaptation to Maximal Effort: Genetics and Age

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SUMMARY. Thirty-nine pairs of twins (23 MZ and 16 DZ) of both sexes, ranging in age from 9 to 52 years, were tested for maximal oxygen uptake, maximal work ventilation, and heart volume. The mean intrapair difference between twin pairs in maximal oxygen uptake was significant for dizygous (DZ) twins, but not for monozygous (MZ). On the basis of this evidence it was concluded that regardless of age, existing individual differences in functional adaptability of man can be attributed to heredity. Further, it was noted that MZ twins demonstrated as much intrapair variability as DZ twins in the other two attributes studied. From this it was concluded that the pulmonary ventilation and the heart volume are nondecisive in the production of intrapair differences in maximal oxygen uptake. The relative importance of environmental forces in modifying the magnitude of intrapair differences is discussed.

twins; age and environmental influences; maximal oxygen uptake; maximal work ventilation; heart volume; vital capacity

AN INDIVIDUAL'S FUNCTIONAL adaptability is the result of the interaction between heredity and environment. However, wide interindividual variability exists in adaptive responses and one wonders to what extent individual differences are attributable to genetic variation and to what extent to environmental conditions. From comparison of intrapair differences between identical and nonidentical twins, it is possible to answer this question since, in effect, phenotypic variability in identical twins is due solely to environmental agents, whereas that in nonidentical twins is due to both genetic fluctuations and extragenetic influences. In a recent study based on the variance of such intrapair twin differences, we found that the contribution of heredity to the interindividual differences in maximal oxygen uptake, was relatively high (4). The latter is used as a performance criterion of the individual adaptability to exercise. Young twins were used as subjects in this study to strengthen the assumption of environmental comparability. However, it can be argued that dizygous pairs would be under more diverse environmental influences

TABLE 1. Twin pairs distributed according to zygosity, sex, and age

Zygosity	Sex	Age, yr 9-13	14-20	21-30	31-40	41-52	Total pairs
MZ	Male	3	1	4	3	3	13
	Female	2	2	1	2	3	10
DZ	Male	1	3	3		2	9
	Female	1	2	2	1	1	7
Total pairs		7	8	10	6	9	39

than monozygous pairs during the developmental period. Thus, the present study was conducted to determine whether the small intrapair differences observed between identical twins and the marked differences between nonidentical twins persist throughout life. In twins exposed to similar environments at different stages in their lives any demonstrable differences between dizygotes as compared with monozygotes must be an expression of the relative strength of the genotype. In those exposed to contrasting environments the resulting differences may provide a measure of this responsiveness to environmental forces.

METHODS

Twins. Thirty-nine pairs of Belgian twins (23 MZ and 16 DZ) of both sexes, ranging in age from 9 to 52 years, were used as subjects. The distribution of the pairs according to age, sex, and zygosity are presented in Table 1. The respective morphological characteristics for males and females are given in Tables 2 and 3.

The value of this study depends to a large extent upon the accurate determination of zygosity and upon a reliable assessment of the relative exposure of the twins to environ mental conditions. Twins were classified either as monozy gous or di-

TABLE 2. Morphological characteristics of monozygous (MZ) and dizygous (DZ) male twins.

ZYGOSITY	TWINS	AGE yrs	WEIGHT Kg			HEIGHT cm			VITAL CAPACITY L, BTPS			HEART VOLUME ml			HEART VOLUME ml/kg		
			A	B	Δ	A	B	Δ	A	B	Δ	A	B	Δ	A	B	Δ
MZ	103/203	22	68	68	0	169	171	2	4.05	4.40	0.35	688	620	68	10.2	9.1	1.1
	106/206	49	64	64	0	164	164	0	2.95	3.23	0.28	640	589	51	10.0	9.3	0.7
	107/207	28	74	85	11	179	181	2	4.88	4.65	0.23	818	961	143	11.1	11.3	0.2
	108/208	42	71	80	9	178	179	1	4.33	4.08	0. 25	606	1,024	418	8.5	12.8	4.3
	109/209	37	63	64	1	175	178	3	4.45	4.86	0.41	973	974	1	15.4	15.2	0.2
	110/210	40	87	77	10	182	182	0	5.16	5.25	0.09	939	970	31	10.8	12.6	1.8
	111/211	28	70	69	1	182	179	3	4.98	5.08	0.10	645	835	190	9.2	12.1	2.9
	112/212	52	85	76	9	172	1 72	0	4.25	4.20	0.05	1,092	861	231	12.8	11.3	1.5
	121/221	23	63	55	8	1 70	167	3	4.30	4.14	0. 16	575	739	164	9.2	13.4	4.2
	124/224	1	47	46	1	158	156	2	3.05	2.85	0. 20	463	403	60	9.9	8.8	1.1
	127/227	20	79	84	5	180	182	2	6.22	6. 70	0.48	903	904	1	11.5	10.8	0.7
	128/228	9	24	24	0	126	125	1	1.78	1.45	0.33	242	236	6	10.3	10.0	0.3
	135/235	10	27	27	0	136	134	2	1.85	1.84	0.01	284	290	6	10.6	10.8	0.2
MEAN		29	63	63	4	167	167	2	4.02	4.06	0.23	682	724	105	10.7	11.3	1.5
± SD		14	20	20	4	18	18	1	1.29	1.43	0.14	259	273	122	1.8	1.9	1.5
DZ	101/201	21	74	57	17	175	168	7	5.45	4.03	1.42	616	675	59	8.3	11.8	3.5
	102/202	22	71	62	9	178	179	1	4.95	4.88	0.07	574	766	192	8.1	12.4	4.3
	114/214	26	85	90	5	176	173	3	4.55	5.18	0.63	795	884	90	9.4	9.9	0.5
	116/216	16	66	50	16	170	162	8	3.45	3.70	0. 25	633	530	103	9.7	10.6	0.9
	120/220	14	56	1.6	10	164	159	5	3.85	3.26	0.59	604	677	73	10.8	14.9	4.1
	122/222	42	72	59	13	161	163	2	3.60	3. 10	0.50	966	668	298	13.4	11.4	2.0
	131/231	13	47	54	7	158	166	8	2.98	3.23	0.25	470	552	82	10.0	10.2	0.2
	134/234	15	73	67	6	175	175	0	4.10	3.94	0.16	769	619	150	10.5	9.2	1.3
	139/239	49	57	72	15	156	164	8	2. 50	3.68	1.18	849	593	256	11.9	10.4	1.5
MEAN		24	67	62	11	168	168	5	3.94	3.89	0.56	697	663	145	10.2	11.2	2.0
± SD		13	12	13	5	8	7	3	0.94	0.72	0.46	157	110	86	1.7	1.7	1.6

TABLE 3. Morphological characteristics of monozygous (MZ) and dizygous (DZ) female twins

		Age	Weight kg			Height cm			Vital capacity L. BTPS			Heart volume ml			Heart volume ml/kg BW		
Zygosity	**Twins**	**yrs**	**A**	**B**	**Δ**	**A**	**B**	**Δ**	**A**	**B**	**Δ**	**A**	**B**	**Δ**	**A**	**B**	**Δ**
	305/405	44	61	62	1	169	161	8	3.62	3.10	0.52	540	525	15	8.9	8.5	0.4
	306/406	25	53	55	2	164	167	3	2.96	3.36	0.39	503	510	7	9.5	9.3	0.2
	309/403	41	78	68	10	177	175	2	4.08	3.54	0.54	931	595	346	11.9	8.8	2.1
	311/411	16	53	51	2	166	167	1	3.33	3.20	0.13	43l	442	11	8.2	8.8	0.6
	317/417	39	53	55	2	158	158	0	3.45	3.40	0.05	647	616	31	12.1	11.2	0.9
MZ	321/421	10	28	27	1	134	135	1	1.80	1.70	0.10	317	288	29	11.5	10.6	0.9
	323/423	11	31	28	3	139	135	4	2.10	1.85	0.25	343	337	6	11.1	12.3	l.2
	324/424	11	42	32	10	150	147	3	1.70	1.75	0.05	462	446	16	11.1	14.l	3.0
	327/427	45	63	61	2	162	162	0	3.28	3.55	0.27	611	673	62	9.8	11.1	1.3
	332/432	35	57	52	5	161	161	0	2.95	2.90	0.05	593	445	148	10.4	8.6	1.8
MEAN		28	52	49	4	158	157	2	2.93	2.84	0.24	538	488	67	10.5	10.3	l.2
± SD		15	15	15	3	13	14	2	0.80	0.76	0.19	176	121	107	1.3	1.9	0.9
	301/401	19	64	62	2	163	168	5	3.05	3.45	0.40	553	549	4	8.6	8.9	0.3
	303/4O3	26	48	43	5	160	155	5	4.00	3.33	0.67	647	715	68	15.6	16.5	0.9
	304/404	32	72	55	17	160	156	4	3.05	3.10	0.05	850	713	137	11.7	12 9	1.2
DZ	308/408	1.6	71	53	18	166	163	3	3.60	3.03	0.57	686	685	1	9.7	13.0	3.3
	314/414	17	45	45	0	154	153	1	2.95	2.55	0.40	669	696	27	14.9	15.5	0.6
	320/420	25	52	51	1	165	163	2	2.83	3.00	0.17	596	547	49	10.1	10.7	0.6
	322/422	12	37	34	3	145	142	3	2.35	2.00	0.35	401	373	28	10.8	11.0	0.2
MEAN		25	56	49	7	159	157	3	3.12	2.92	0.37	629	611	45	11.6	12.6	1.0
±50		11	14	9	8	7	9	1	0.54	0.50	0.21	137	128	47	2.6	2.7	1.1

zygous on the basis of a polysymptomatic com parison of morphological similarity and serological criteria. Discordance in physical characteristics, such as color of the eyes and iris pattern, color texture and distribution of the hair, as well as shape of facial features, were used as a first approximation of dizygosity. However, some dizygous twins are as much alike as monozygous twins and to increase the certainty of the genetic identity or nonidentity of the pairs a number of serological analyses were performed. A venous sample of 10 ml of blood was collected from each, twin and was analyzed for the plasma protein systems: haptoglobin (Hp), group specific substance (Gc), and B-lipoprotein (Agx); for the enzyme systems: acid phosphatase (APh), phosphoglucomutase-locus 1 (PGM1), and adenylate kinase (AK); and for the red cell antigens: A, A2, BO MNSS, Rh (GCwDEce), P1, Lua, Kk, and Fya. In some cases the systems HL-A and adenosine of aminase were also determined (e.g., ref 3). Discordance in a single blood group was taken as proof of dizygosity (13). The probability of misclassifying a twin pair when using morphology and serology as diagnostic criteria is about 5 % (1).

Case histories revealed that none of the twins had been subjected to any extreme environmental deprivation. With the exception of the children and some young adults (see later) who engaged in competitive sports for several years, the popula-

tion under study was largely comprised of rather sedentary individuals. Their consumption of alcohol and tobacco was either modest or nil.

Measurements. Twins were always tested in pairs. First, a medical examination was given and then an interview from which the individual's way of life was assessed. Measurements were made of the anthropometric dimensions, resting electrocardiogram, heart volume, pulmonary function, nerve conduction velocity, maximal muscular force, maximal heart rate, and maximal oxygen uptake. This report deals primarily with responses to maximal effort and secondarily with heart volume measurements.

The open-circuit spirometry was used to determine the maximal oxygen uptake. Atmospheric air was inspired from a Tissot spirometer and the volume displacement was recorded on a kymograph. Two such spirometers were employed alternately at 1-min intervals, so that when the one was in use the other was being filled with air. The change was made automatically by means of a timer and magnetic valves. The expired air was sampled from a mixing chamber into small meteorological balloons and analyzed for oxygen and carbon dioxide concentrations. The paramagnetic Servomex and the infrared Goddart capno-graph analyzers, calibrated with known gas concentrations, were used for gas analysis. The signals from these analyzers were amplified and recorded on a Honeywell recorder (electronic 194),

TABLE 4. Metabolic, respiratory, and cardiovascular responses of monozygous (MZ) and dizygous (DZ) male twins to maximal effort

ZYGOSITY TWINS		OXYGEN UPTAKE L/min STPD			OXYGEN UPTAKE ml/min/kg BW STPD			RESPIRATORY EXCHANGE RATIO (R)			HEART RATE Beats/min			VENTILATION,L/m OXYGEN UPTAKE L/min		
		A	B	Δ	A	B	Δ	A	B	Δ	A	B	Δ	A	B	Δ
M	103/203	2.84	2.68	0.16	42.1	39.4	2.7				196	193	3			
	106/206	2.00	2.02	0.02	31.3	31.0	0.7	1.11	1.14	0.17	180	176	4	50.6	35.6	15.0
	107/207	4.10	4.36	0.26	55.8	51.2	4.6	1.04	1.08	0.0k	199	195	4	29.4	26.0	3.4
	108/208	1.84	2.36	0.52	25.9	29.5	3.6	0.93	1.08	0.15	142	147	5	21.9	33.5	11.6
	109/209	2.60	2.79	0.19	41.3	43.6	2.3	1.13	1.02	0.11	180	174	6	31.8	25.6	6.2
	110/210	3.29	3.21	0.08	37.8	41.7	3.9	1.16	1.14	0.02	160	152	8	33.6	30.6	3.0
	111/211	3.93	3.66	0.27	56.1	53.1	3.0	1.11	1.01	0.10	194	182	12	31.9	25.2	6.7
	112/212	2.83	2.60	0.23	33.2	34.2	1.0	0.99	0.96	0.03	165	170	5	30.4	26.3	4.1
	121/221	3.48	3.00	0.48	55.7	54.6	1.1	1.15	1.18	0.03	211	200	11	28.5	32.8	4.3
	124/224	1.98	2.03	0.05	42.4	44.4	2.0	1.20	1.32	0.12	194	205	11	37.1	41.7	4.6
	127/227	3.47	3.70	0.23	44.0	44.2	0.2	1.14	1.14	0.00	201	192	9	32.0	29.2	2.8
	128/228	0.94	0.94	0.00	40.1	40.0	0.1	0.96	0.93	0.03	189	190	1	44.3	36.1	8.2
	135/235	1.27	1.18	0.09	47.3	44.1	3.2	0.98	0.98	0.00	202	192	10	43.0	45.3	2.3
KEAN +- SD		2.66 1.00	2.66 0.98	0.20 0.16	42.5 9.5	42.5 7.7	2.3 1.4	1.09 0.11	1.08 0.05	0.06 0.05	186 20	182 18	7 4	34.5 7.9	32.3 6.5	6.0 3
DZ	101/201	3.91	3.19	0.72	52.8	56.0	3.2				203	189	14			
	102/202	3.15	3.25	0.10	44.3	52.4	8.1				185	195	10			
	114/214	4.61	5.36	0.75	54.2	59.9	5.7	1.06	0.97	0.09	198	215	17	29.0	22.9	6.1
	116/216	2.57	2.83	0.26	39.2	56.5	17.3	0.93	0.93	0.00	194	178	14	25.7	26.2	0.5
	120/220	2.38	2.75	0.37	42.5	60.3	17.8	1.05	0.98	0.07	182	182	0	32.2	31.2	1.0
	122/222	2.44	2.33	0.11	33.9	39.7	5.8	1.28	1.22	0.06	162	168	6	39.4	31.8	7.6
	131/231	2.67	2.44	0.17	48.2	45.1	3.1	1.19	1.26	0.07	199	192	7	37.4	33.2	4.2
	134/234	3.27	3.76	0.49	44.5	56.2	11.7	1.04	1.05	0.01	196	204	8	25.3	27.6	2.3
	139/233	1.82	3.23	1.41	31.9	45.0	13.1	1.08	0.99	0.09	173	190	17	36.5	28.8	7.7
MEAN +- SD		2.94 0.89	3.24 0.91	0.49 0.42	43.5 7.7	52.3 7.4	9.5 5.7	1.09 0.11	1.06 0.13	0.06 0.04	188 14	190 14	1 0	32.2 5.7	28.8 36	4.2 3.0

TABLE 5. Metabolic, respiratory, and cardiovascular responses of monozygous {MZ) and dizygous (DZ) female twins to maximal effort

zygosity	TWINS	oxygen uptake L/min STPD			OXYGEN UPTAKE ml/min/kg BW STPD			RESPIRATOTY EXCHANGE RATIO (R)			HEART RATE Beats/min			VENTILATION, l/min OXYGEN UPTAKE L/min		
		A	B	Δ	A	B	Δ	A	B	Δ	A	B	Δ	A	B	Δ
MZ	305/405	1.93	1.78	0.15	11.9	290	2.9	1.06	0.95	0.11	179	174	5	33.4	35.0	1.6
	306/406	1.87	1.50	0.37	31.6	27.3	4.3	1.12	1.09	0.03	180	198	18	36.2	33.0	3.2
	309/409	2.06	1.89	0.17	26.4	27.8	1.4	0.96	1.09	0.13	165	187	22	28.4	37.5	9.1
	111/411	1.86	1.88	0.02	35.5	36.9	1.4	0.96	0.92	0.04	199	198	12	31.4	16.2	4.8
	317/417	1.77	1.76	0.01	33.2	31.9	1.3	1.33	1.34	0.01	181	179	25	37.0	34.8	2.2
	321/421	0.92	1.01	0.09	33.4	37.2	38	1.10	0.99	0.11	198	200	3	46.7	40.3	6.4
	323/423	1.20	1.04	0.16	38.7	37.7	1.0	1.03	1.07	0.04	184	179	17	44.9	46.7	1.8
	324/424	1.46	1.23	0.23	34.6	38.9	4.3	1.30	1.10	0.20	208	205	12	42.6	39.8	2.8
	327/427	1.37	1.48	0.11	21.9	24.5	2.6	0.98	0.91	0.07	164	147		30.0	28.1	1 9
		196	1.57	0.39	34.4	30.3	4.1	0.90	1.01	0.11	190	178				3.4
MEAN ± SD		1.64	1.51	0.17	32.2	32.2	2.7	1.07	1.05	0.09	185	185	9	36.1	36.2	2.7
		0.38	0.33	0.13	4.8	5.2	1.4	0.14	0.13	0.06	14	17	8	6.6	5.3	
DZ	301/401	1.60	2.31	0.71	25.0	37.2	12.2	1.18	1.27	0.90	205	195	10	44.6	39.9	4.7
	303/403	1.33	2.11	0.78	27.9	48.9	21.0	0.98	1.06	0.08	177	195	18	40.7	34.2	6.5
	304/404	1.90	2.23	0.33	26.3	40.4	13.9	1.21	0.97	0.24	190	180	10	35.6	29.6	6.0
	308/408	2.44	1.37	1.07	34.4	26.0	8.4	1.11	0.97	0.14	172	156	16	33.9	31.7	2.2
	314/414	1.67	1.91	0.24	37.1	42.5	5.4	0.97	1.07	0.10	188	196	8	33.4	33.1	0.3
	320/420	2.06	2.09	0.03	34.9	41.0	6.1	1.07	1.09	0.02	177	175	2	28.3	34.1	5.8
	322/422	1.66	1.30	0.36	44.7	38.3	6 4	1.16	1.25	0.09	190	196	6	37.9	38.0	0.1
MEAN ± SD		1.81	1.90	0.50	32.9	39.1	10.5	1.09	1.09	0.22	186	185	10	36.3	34.4	
		0.36	0.41	0.36	7.0	6.9	5.6	0.09	0.12	0.31	11	15	5	5.3	3.5	

in order to increase the accuracy of reading. The testing procedure used in this study has been previously described (8).

Briefly, it involves continuous performance on a treadmill at progressively increasing intensities. The treadmill was set at an inclination of 10%. The subject starts by walking at 4 km/hr for 4 min and thereafter the speed is increased every 2 min by an increment of 2 km/hr, until the subject is unable to continue. Measurements are taken at the last minute of each work load. An asymptote of oxygen intake or, when this was not observed, the respiratory exchange ratio and the heart rate were used as objective criteria of maximal oxygen intake. Heart rate was determined from a thoracic lead of the electrocardiogram, recorded on a Mingograf 34 (Elema-Schonander). Heart volume was estimated roentgenographically with the

TABLE 6. Analysis of differences between twin pairs (o+p) for selected parameters

Attribute	Intraclass Correlation Coefficient MZ	Intraclass Correlation Coefficient DZ	Mean Intrapair Differences MZ	Mean Intrapair Differences DZ	Intrapair Variance, σ^2 MZ	Intrapair Variance, σ^2 DZ	F Ratio, σ^2 DZ/σ^2 MZ
Max oxygen intake, ml/min per kg BW	0.95	0.36	2.54	10.06	3.87	63.66	16.45 P<0.001
Max heart rate, beats/min	0.84	0.62	7.77	10.16	44.96	65.72	1.46
Max oxygen pulse, ml/beat	0.97	0.81	1.07	2.12	0.90	3.69	4.00
Max work vent, l/ min/vital cap, 1/min	0.60	0.16	11.26	16.12	12.81	11.70	0.91
Max resp equivalent, l/ml	0.60	0.61	4.7	3.9	17.71	11.33	0.64
Heart vol, ml/kg BW	0.45	0.56	1.37	1.59	1.64	2.21	1.34
Vital capacity, 1 (BTPS)/min	0.98	0.72	0.23	0.56	3.87	18.15	4.70 P<0.05

subject in the standing position (6). Lateral and posterior-anterior projection were used successively, and values were introduced to the equation derived by Reindell and others (9). Although heart volume may decrease as a result of orthostatic changes (7), in this study we were concerned mainly with intrapair differences and the standing position of testing that we employed does not affect the results. Further, to reduce the variability of measurements due to systolic variations in heart volume, the trigger was activated by the QRS signal. Test-retest of this procedure gave an average error of less than 4 %.

RESULTS

Descriptive statistics for metabolic, respiratory, and cardiovascular responses to maximal effort are presented in Tables 4 and 5 for male and female twins, respectively; corresponding morphological data are shown in Tables 2 and 3. In the same tables, in addition to absolute values, the means and their standard deviations for intrapair differences are presented. These statistics give an approximate measure of the comparability of the groups and of their intrapair variability, with respect to the variables measured.

A statistical comparison of the intrapair differences for selected parameters is summarized in Table 6. The significance of the intrapair differences between MZ and DZ twins was computed by using the F ratio, $F = \sigma 2DZ/\sigma 2MZ$, which is based on the intrapair variances. The intraclass correlation was also derived from the total and the intrapair variances.

The data obtained from both sexes were grouped and scrutinized for zygosity only since an examination of the mean intrapair differences of male and female twins did not indicate any sex influence to be operant for the parameters studied.

It is apparent from Table 6 that dizygous twins demonstrated as much diversity as the monozygous twins for all the variables, except for the maximal oxygen uptake. The intrapair variance between MZ and DZ twins being significant well beyond the 1 % level of confidence. The intrapair differences in maximal oxygen uptake for MZ and DZ twins is plotted as a function of age in Fig. 1. No trend altering the magnitude of these differences with age can be detected.

In Fig. 2, the intrapair differences in maximal oxygen uptake are plotted against intrapair differences observed in heart volume for the same twin pairs. It will be noted that the values are markedly scattered and there is no suggestion of dependability of intrapair differences observed in maximal oxygen intake on heart volume differences.

The relation between the intrapair differences in maximal oxygen uptake and the corresponding intrapair differences in vital capacity is shown in Fig. 3. The absence of interdependence between the two variables is also evident. Similar trends were observed when the intrapair differences in maxima] oxygen uptake were related to the maximal work ventilation.

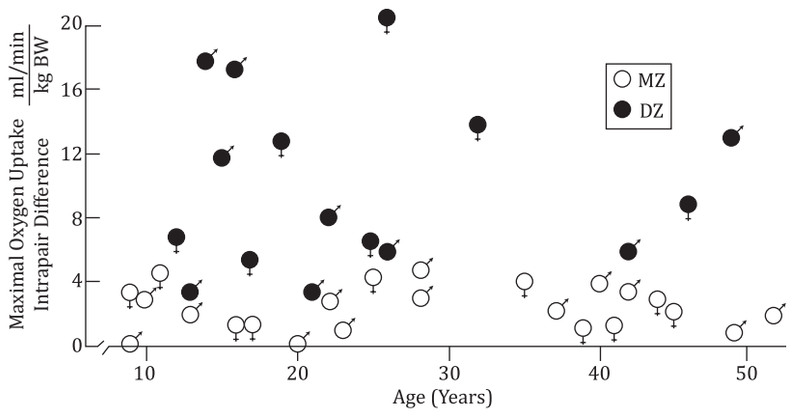

FIG. 1. Intrapair differences in maximal oxygen intake, ml/min per kg BW as a function of age. Filled symbols denote dizygous twins and unfilled symbols monozygous twins

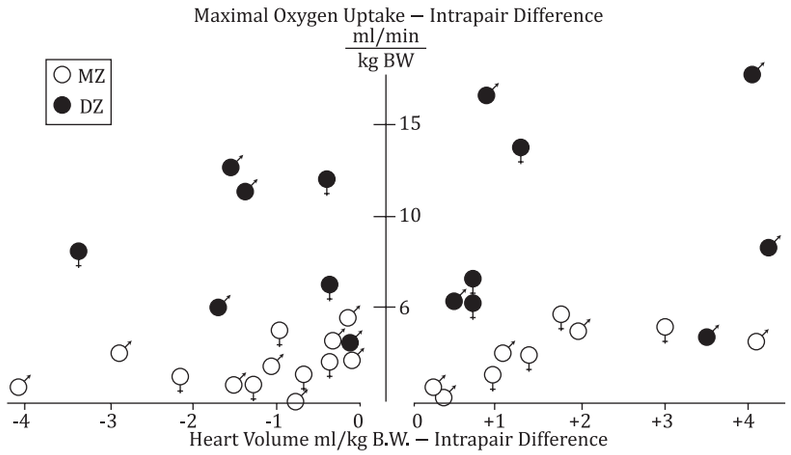

FIG. 2. Intrapair differences in maximal oxygen intake, ml/min per kg BW, against intrapair differences in heart volume, ml/kg BW. Filled symbols denote dizygous twins and unfilled symbols monozygous twins.

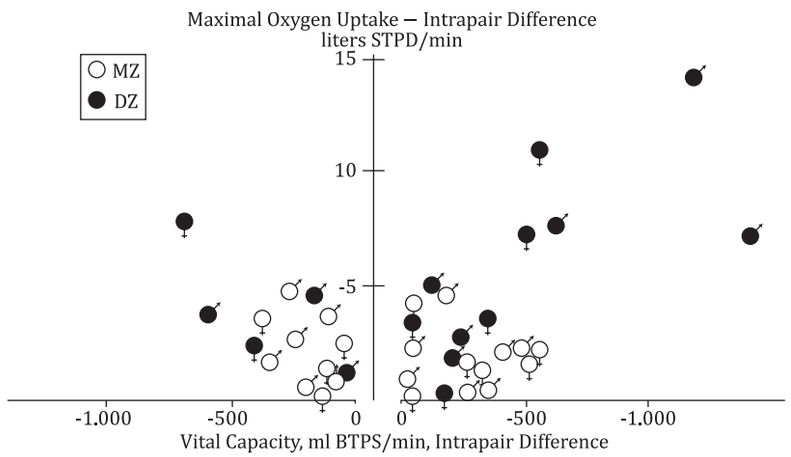

FIG. 3. Intrapair differences in maxima] oxygen intake, I (stpd)/ min per kg BW, against intrapair differences in vita] capacity, ml (BTPs)/min. Filled symbols denote dizygous twins and unfilled symbols monozygous twins.

DISCUSSION

There appears to be a significant resemblance in functional adaptability as measured by maximal oxygen intake between identical twins, whereas there is a divergence between nonidentical twins., who have not been affected by extreme envi-

ronmental forces. This observation is in agreement with previously published findings on preadolescent twins (4). Thus, in view of the age range of the twins used in the present study, our earlier conclusion that heredity alone accounts almost entirely for existing differences in functional adaptability seems to be valid for man regardless of his age. It will be also recalled that fraternal twins are genotypically the same in 50% of their genes, and therefore one would expect divergences in adaptability among unrelated persons to be even more pronounced.

One may wonder, however, whether intrapair differences could possibly be related to differences in mode of life. In this connection, reference may be made to some case studies from our files. In one case (code 101/201) of non-identical twins, aged 21 years, who lived apart since 16, one twin had trained strenuously for competitive middle-distance running, whereas his brother had never participated in sports of any kind. It was therefore surprising to find that the untrained twin had a maximal oxygen uptake of 56.0 ml/min per kg BW as compared with a value of 52.8 for his trained counterpart. One cannot escape the inference that if it were not for the physical training the intrapair difference between this twin pair would have been greater. The increase in maximal oxygen uptake as a result of training is well documented (e.g., 2, 10, 11) and thus there is little doubt that intensive environmental stimulation elicits proportionate changes. However, the magnitude of change must remain within the individual limits of genetic potentialities (5).

Further, the implicit postulate oi this observation is that some individuals with a weak genotype have to use a greater amount of physical activity to attain an average adaptive value, whereas those within a native endowment may not need more than a threshold exposure to maintain their already high adaptive value. The adaptive value of an individual is decreased with hypokinesis (12) suggesting, that collaboration of environmental factors with the genes is essential for the genotype to reach its full expression.

Another two cases are also intriguing. Identical twins (code 110/210), 40 years of age, had been separated at age 12 and had had different lifestyles. More important, one twin had engaged in vigorous training for competitive basketball (12-30 years, 18-30 on the national level), whereas his brother was only moderately active during the same period. For the last 10 years neither of them had been involved in regular physical exertion. When tested, their maximal oxygen uptake was closely similar—the absolute values being 37.8 and 41.7 ml/min per kg BW for the trained and untrained twin, respectively.

In another case, nonidentical twins (code 139/239) had a maximal oxygen uptake of 31.9 and 45.0 ml/min per kg BW at age 49. They had lived together all their lives, had the same profession, and both played competitive soccer from early childhood until they were 22 years of age. These observations support the notion of reversibility of function, that is to say, environmental stimulation of the developing

organism may not have lasting effects. It seems that if there is not a continuous stimulation of sufficient magnitude, the adaptive value of an individual will be set by his genotype. As Galton put it "the one element that varies in different individuals, but is constant in each of them, is the natural tendency which inevitably asserts itself."

Finally, an important question remains unanswered. How is the intrapair variability to be explained? What are those biological attributes which are responsible for the production of the large differences between DZ twins and small differences between MZ twins, with respect to maxima] oxygen intake? We need to obtain experimental evidence in MZ and DZ twins in the partitioning of transport and oxygen utilization systems to factor out variables that play a determining role in setting the degree of intrapair differences. In view of the evidence obtained in the present study we can exclude pulmonary ventilation and the heart volume as being nondecisive factors.

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Genetic Variation in Neuromuscular Performance

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ABSTRACT. Using a simple cumulative model of heredity plus environment, based on intrapair differences observed in monozygous (MZ) and dizygous (DZ) twins, the relative contribution of heredity to the interindividual variance in several neuromuscular parameters was determined with 16 pairs of male (8 MZ and 7 DZ) and 14 pairs of female (7 MZ and 7 DZ) twins ranging in age from 10 to 14 years. The data disclosed that in boys the variabilty in maximal muscular power was 99.2% genetically determined under the environmental conditions of the study. The corresponding heritability estimate values for the patellar reflex time and reaction time were 97.5% and 85.7%, respectively. In girls the heritability estimates could not be computed, because the MZ twins seemed to show almost as much diversity as the DZ twins in all of the variables studied. On the basis of the obtained data it is suggested that the variation observed in maximal mechanical power, patellar reflex time' and reaction time may be more susceptible to environmental modifications in girls than in boys.

Key words: Genetic Variability — Neuromuscular Function — Mechanical Power — Reflex Time — Reaction Time.

In evolutionary terms, the adaptive value of an individual can be characterized primarily by-the speed, magnitude and duration of biological responses with which he confronts an adaptive task. However, the individual effectiveness varies markedly in this respect and the question then arises as to what extent genetio differences account for this variability in biological response patterns.

Using a simple cumulative model of heredity plus environment, based on intrapair differences observed in identical and non-identical twins, we have recently been able to ascertain the relative contribution of heredity to the inter-individual

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variance in maximal oxygen uptake, which is used as the performance criterion of an individual's adaptive value (Klissouras, 1971). To obtain further insight into the nature of the process of adaptation, we need to know the relative importance of the genetic predisposition to the functional variation of the neuromuscular apparatus, which is the mediator and executor of the adaptive responses. It cannot be overemphasized that the degree, to which we can influence the expression of the genotype, depends to a considerable extent upon our knowledge of the relative strength of the genotype. Thus, the present study was undertaken to determine first, the heritability of the maximal mechanical (anaerobic) power, force output, reflex and reaction time, and nerve conduction velocity and secondly the influence of sex upon their variability.

METHODOLOGY

The relative contribution of the hereditary component to the variation observed in the parameters under study, was defined by determination of the degree to which monozygous (MZ) and dizygous (DZ) twins resemble each other in these parameters. Since the phenotype variability in MZ twins is due solely to environmental agents, whereas that in DZ twins is due to both genetic fluctuations and extra-genetic influences, the heritability of a given parameter will be derived by the equation (Holzinger, 1929; Partanen et al., 1966; Klissouras, 1971):

$$H_{\text{est}} = \frac{s^2\text{DZ} - s^2\text{MZ}}{s^2\text{DZ} - s^2\text{e}} \qquad \textbf{(1)}$$

where s2DZ and «SMZ denote intrapair variability of an attribute in MZ and DZ twins, respectively, and 8*e signifies the variance due to experimental error. It should be emphasized that the variability quotient obtained in this manner does not mean that the genetio factor has an etiologic role in the observed variability, but the quotient is of some intrinsic value for it provides an insight into relative strength of the genotype.

TWINS

The population Register of Finland provided us with the names and addresses of those families that had had two or more children born on the same day in the oity or the surrounding communities of Jyvaskyla between the years of 1950 and 1960. Through this we were able to locate the twin pairs ranging in age from 10 to 20 years. To obtain a homogenous age distribution the final sample was limited to those aged from 10 to 14, and it was composed of 15 male (8 MZ and 7 DZ) and 14 female (7 MZ and 7 DZ) twin pairs. Care was taken to ensure that environmental influences for the MZ and DZ pairs were comparable. The distribution of the pairs according to age, sex and zygosity is given in Table 1. Tables 2 and 3 show the ph-

TABLE 1. Twin pairs distributed according to age and zygosity

Zygosity	Boys						Girls					
	Age (years)					Total pairs	Age (years)					Total pairs
	10	11	12	13	14		10	11	12	13	14	
MZ	1	1	3	2	1	8	1	4	1	-	1	7
DZ	2	2	1	—	2	7	1	3	1	2	—	7
Total pairs 3	3	4	2	3	15	2	7	2	2	1	14	

ysical and performance characteristics of the entire sample. Initially, the twins were classified as MZ and DZ pairs on the basis of similarities in physical appearance. To increase the certainty of the genetio identity or non-identity of the twin pairs a number of serological analyses were performed. For these analysis venous samples of 7 to 8 ml of clotted blood were collected from each subject at the local Red Cross Blood Transfusion Service and sent for examination to the Central Laboratory of the same organization. Antisera for the determination of the following red cell antigens were used: A1A2BO, MNSs, Rh(CODEce), P1 Lub Kt, Fyb and Fyb, and Jkb. The serum was separated and frozen for later investigation. The following proteins and enzymes were determined: Haptoglobin (Hp), Group specific substance (Gc), Acid phosphatase (APh), Adenylate kinase (AK) and Phosphogluco-mutase (PGM). For references see Race and Sanger (1969) and Giblett (1970).

In the 14 cases of observed dizygosity discordance was observed in average of more than five antigens or serum proteins. In none of the pairs dizygosity was based on less than three discordant markers.

MEASUREMENTS

Several parameters were selected to represent the functional and performance characteristics of the neuromuscular system. These included neuromuscular (anaerobic) power, muscle forces (concentric, eccentric and isometric), patellar reflex time, ulnar nerve conduction velocity, visual reaction time and the integrated electromyographic activity (IEMG) of the forearm flexors. In addition, an estimation of the physical working capacity was obtained to characterize the sample populations.

Maximal Muscular Power. The maximal muscular power was measured by a method which is essentially the same as that introduced by Margaria et al. (1966). It is based on the observation that energy liberation from oxidative phosphorylation and lactic acid production via anaerobic glycolysis is a delayed process and that the energy required for a maximal effort of a few seconds duration is derived from the splitting of high energy phosphate compounds. The measurement of the maximal running velocity on a staircase meets this criterion. Instead of an electronic

clock, as employed by Margaria et al. (1966), a tachogenerator was used to record this Telocity. One end of a thread was wound around the rotating wheel of the tachogenerator and the other end was attached to the back of the subject's belt. When the subject started to run pulling the thread, the tachogenerator developed a voltage proportional to the rotating velocity of the wheel. Thus, the faster the subject ran the greater was the voltage output of the tachogenerator. The running velocity was calculated from the following formula:

$$(m/sec) = c \times V,$$

where V - voltage generated by the tachogenerator (obtained from the graph recorder)

c - constant = 0.5 m/vs.

The ejected length of the thread during one revolution was 210 cm, thus

$$c = \frac{210 - 10^{-3}}{0{,}41 \text{ vs}} = 0{,}5 \frac{\text{m}}{\text{vs}}$$

The velocity value was then converted to the vertical component of the rumning velocity {Vυ) and mechanical power (kpm/sec) was computed on the basis of the subject's Vv and his body weight. The error of this method was less than 2%.

TABLE 2. Physical and performance characteristics of the male monozygous (MZ) ond dizygous (DZ) twins

	Twins	Age years	Height, cm			Weight, kg			PWC205 (kpm/min)		
	A B		A	B	Δ	A	B	Δ	A	B	Δ
MZ	100/101	13	151.3	150.0	1.3	40.1	38.0	2.1			
	104/105	12	143.0	142.0	1.0	31.5	31.9	0.4	830	845	16
	108/109	13	167.2	168.0	0.8	48.0	48.2	0.2	690	605	15
	110/111	12	159.8	157.5	2.3	46.9	46.1	0.8	420	480	60
	112/113	14	153.5	157.0	3.5	43.0	45.8	2.8	912	944	32
	114/115	10	151.3	149.3	2.0	37.8	36.4	1.4	530	630	100
	116/117	12	151.8	154.0	2.2	36.7	38.0	1.3	790	815	25
	118/119	11	132.2	133.5	1.3	26.6	27.4	0.8	1320	1080	240
	Mean	12.1	151.3	151.4	1.8	38.8	39.0	1.2	770.3	771.3	69.6
	±S.D.	1.2	10.4	10.5	0.9	7.3	7.3	0.9	299.8	210.1	81.1
DZ	200/201	14	177.5	171.5	6.0	76.2	56.4	19.8	1430	900	530
	202/203	11	136.8	134.0	2.6	35.8	30.2	5.6	495	340	155
	208/209	14	149.2	159.6	10.3	36.2	45.8	9.6	1005	765	250
	210/211	10	154.5	148.2	6.3	51.4	37.6	13.8	730	680	50
	212/213	10	135.5	129.8	5.7	34.6	28.4	6.2	735	585	150
	214/215	12	150.2	142.5	7.7	34.0	29.0	5.0	735	655	80
	218/219	11	162.2	153.8	8.4	54.0	42.5	11.5	935	560	375
	Moan	11.7	152.3	148.5	6.7	46.0	38.6	10.2	866.4	639.3	227.1
	±S. D.	1.7	14.6	14.6	2.4	15.7	10.4	5.3	297.9	174.2	172.4

TABLE 3. Physical and performance characteristics of the female monozygous (MZ) and dizygous (DZ) twins

	Twins A B	Age years	Height, cm A	Height, cm B	Height, cm Δ	Weight, kg A	Weight, kg B	Weight, kg Δ	PWC205, A	PWC205, B	PWC205, Δ
MZ	264/265	10	147.2	146.5	0.7	33.4	37.1	3.7	785	720	65
	150/151	11	139.0	139.5	0.5	28.5	29.2	0.7	460	580	120
	154/155	12	142.0	144.0	2.0	30.0	32.6	2.6	520	560	40
	156/157	14	155.5	154.0	1.5	44.6	43.6	1.0	1005	1000	5
	158/159	11	147.8	146.5	1.3	37.6	39.2	1.6	440	465	25
	160/161	11	144.0	148.0	4.0	33.3	35.4	2.1	415	365	60
	260/261	11	142.2	139.2	3.0	36.4	37.2	0.8	615	690	75
	Mean	11.4	145.4	145.4	1.9	34.8	36.3	1.8	605.7	625.7	54.3
	±S.D.	1.3	5.4	5.1	1.3	5.4	4.6	1.1	217.6	205.5	37.4
DZ	250/251	13	161.5	150.8	10.7	41.8	43.0	1.2	980	910	70
	252/253	13	158.0	146.8	11.2	41.0	33.8	8.2	505	705	200
	256/257	11	155.0	157.5	2.5	49.4	47.8	1.6	810	965	155
	258/259	11	139.0	139.2	0.2	28.8	28.8	0.0	755	740	15
	262/263	12	159.2	158.0	1.2	50.6	43.6	7.0	615	690	75
	264/265	10	147.0	148.8	1.8	45.2	36.8	8.4	875	580	295
	266/267	11	154.0	144.0	10.0	34.4	30.6	3.8	520	590	70
	Mean	11.6	153.4	149.3	5.4	41.7	37.8	4.3	722.9	740.0	125.7
	± S.D.	1.1	7.9	6.9	5.0	7.9	7.2	3.6	181.7	148.0	96.7

Leg Force, The maximum voluntary isometric contraction of the quadriceps muscle group of the right leg was recorded with a strain gauge system installed on a steel wire connected to a cuff fastened around the subject's ankle. The subject sat on the edge of the table with his knee at an angle of 160 degrees and the back support at a 60 degree angle with the horizontal. The steel wire was positioned so that the pull of the cuff was always at a right angle to the line joining the ankle and knee joints. To ensure that each subject assumed the same position for each trial a belt was fastened around the subject's waist during the effort.

Forearm Flexor Forces. A special electrical dynamometer was used to record the isotonic (concentric and eccentric) and isometric forces of the forearm flexors. The dynamometer and its performance characteristics have been reported elsewhere (Komi, 1971). Briefly, it was an electro-mechanical system that allowed rapid selection of the direction, and velocity of movement. The geometry of the dynamometer lever arm system was so arranged that the rate of contraction of the muscle (e.g. m. Biceps Brachii) could be kept constant over the movement range of 120 degrees. This corresponds to a 7 cm change in the length of the biceps muscle of an adult man. The velocity selected for the isotonic (eccentric and concentric) contraction was 2.4 cm/sec [refers to the measurements taken for the adult man of an average size (Komi, 1971)]. In isometric contraction the dynamometer lever arm was positioned so that the elbow angle was at 100 degrees. The dynamometer also

had a special wrist cuff that allowed accurate fixation of the wrist at any point between full supination and full pronation. The supinated position was selected for all types of contraction.

Integrated Electromyographic Activity (IEMG). The maximal isometric EMG activity was picked up with a pair of surface electrodes (E & M Intruments Co.) placed on the Biceps Brachii muscle according to the method described elsewhere (Komi and Buskirk, 1970). Because the subjects studied had relatively small biceps muscles, the inter-electrode distance was reduced to 3.5 cm. The EMG signal was amplified with a Tektronix RM122 amplifier with a gain of 1000 and then fed into an electronic integrator. Both the recorded EMG and its integral were displayed on a Honeywell 1206 visicorder.

Patellar Reflex Time. The quadriceps reflex time was recorded with an instrument which was similar in principle to the one described by Tipton arid Karpovich, (1966). The unconscious response to the stimulus of a hammer strike to the patellar tendon was, however, measured directly from the rectus femoris muscle with a pair of surface electrodes placed for each subject at the same position and distance relative to the line joining the patella and tubercle of the crest. Consequently the reflex time values were substantially lower than those reported in the literature (Kroll, 1968; Tipton and Karpovich, 1966). In our opinion, the use of an electromyographic method gives a "truer" value for the reflex time. The reflex time was then measured from the screen of a storage oscilloscope on which both the stimulus and the EMG signal were displayed.

Reaction Time. Visual reaction time was recorded with a standard reaction timer. The subject responded to a green light stimulus by pressing the tapper with the forefinger.

Ulnar Nerve Conduction Velocity. A method similar to Hodes el al. (1948) was used to measure the conduction velocity along the ulnar nerve. The principle of this technique involved the stimulation of the ulnar nerve successively at two marked points along its length: proximal to the elbow and at the wrist. The time delay in the response of the hypothenar muscles was determined by measuring both the moment of stimulation and the moment of the action potential from the oscilloscope screen. Since we knew the time delay difference for the action potential and the distance between the stimulus points, the velocity of conduction could be computed. A Disa ministim type 14 E 01 was used for stimulation with a square-wave pulse of 0.1 msec duration and with, a strength of 10 to 15 volts.

Working Capacity. To obtain an estimate of the subject's physical working capacity (EWC205) a 12 min bicycle Ergometer test comprised of four three-minute workloads (150, 350 and 600 kpm/min) was used (Rusko and Karvinen, 1971). Through the standard extrapolation method the work capacity value (kpm/min) was read to correspond to the heart rate of 205, which was taken as an average maximal heart rate for the age group of the twin population.

EXPERIMENTAL PROCEDURE

The twins were first contacted so that a diagnosis could be made of their zygosity on the basis of physical similarities, and so that information could be obtained as to the extent of their physical activities. Three separate laboratory testing sessions were arranged at weekly intervals 1. to give a medical examination, take anthropometric measurements, draw blood samples for a more accurate zygosity determination and to familiarize the subjects with all testing procedures; 2. to measure and record the muscular forces, IEMG and muscular power; 3. to assess the reaction time, patellar reflex time and the nerve conduction velocity. Accuracy of measurement is of utmost importance because the magnitude of the intrapair difference is small. Both members of each twin pair were measured at the same time to eliminate diurnal variation. The experimental error of the measurements was obtained from duplicate determinations, made on all attributes.

Statistical analysis. The heritability quotient was derived from the Eq. (1). The Wilcoxon matched-pairs signed ranks test (e.g. Siegel, 1956) was used to ensure that there were no statistically significant intrapair differences in the MZ twin pair groups. The intercorrelations were computed between each measured variable to factor out variables that could be considered for derivation of heritability estimation. Then intrapair differences between MZ and DZ twins were computed by using both the Mason-Whitney U test (Siegel, 1956) and the F ratio,

$$F = \frac{s^2\mathrm{DZ}}{s^2\mathrm{MZ}}$$

The Sat was calculated only if both of these tests showed a significant intrapair variance difference at a level of $P < 0.05$.

RESULTS

Descriptive statistics for neuromuscular data obtained from male MZ and DZ twins are shown in Tables 4, 5 and 6; data obtained from the female MZ and DZ twins are summarized in Table 7. It is apparent from these tables that the monozygous twins constituted comparable groups with regard to their neuromuscular performance.

TABLE 4. Muscle force of monozygous (MZ) and dizygous (DZ) male twins

Zygosity	Twins	Quadriceps Isometric force (kg)			Forearm flexor force Isometric			Eccentric			Concentric		
	A B	A	B	Δ	A	B	Δ	A	B	Δ	A	B	Δ
MZ	100/101	30.5	30.8	7.7	11.2	11.2	0.0	14.2	13.8	0.4	9.2	10.3	1.1
	104/105	28.5	29.4	0.9	8.6	9.2	0.6	12.1	12.7	0.6	6.2	5.9	0.3
	108/109	51.7	46.2	5.5	22.4	20.0	2.4	22.1	23.1	1.0	10.6	19.3	8.7
	110/111	38.5	43.2	4.7	9.1	9.0	0.1	11.4	14.8	3.4	9.4	12.3	2.9
	112/113				11.0	7.4	3.6	9.1	8.3	0.8	8.3	7.2	1.1
	114/116	20.7	23.3	2.6	10.8	10.8	0.0	9.1	9.8	0.7	8.0	7.1	0.9
	116/117	27.9	30.9	3.0	7.3	7.8	0.5	5.8	8.9	3.1	4.8	6.3	0.6
	118/119	17.6	22.5	5.0	8.5	8.1	0.4	8.4	7.4	1.0	7.1	5.8	1.3
	Mean	31.9	32.3	4.2	11.1	10.4	1.0	11.5	12.4	1.4	8.0	9.2	2.1
	S.D.	11.8	9.1	2.2	4.8	4.1	1.3	5.0	5.1	1.2	1.9	4.8	2.8
DZ	200/201	41.2	53.9	12.7	6.8	4.5	2.3	12.1	10.3	1.8	14.8	8.8	6.0
	202/203	34.2	31.9	2.3	11.4	10.9	0.5	11.2	13.1	1.9	8.9	8.6	0.3
	208/209	30.9	33.3	2.4	16.2	23.8	7.6	20.4	23.9	3.5	16.0	21.4	6.4
	210/211	25.9	30.2	4.3	13.0	10.2	2.8	10.5	14.1	3.6	7.5	12.5	5.0
	212/213	24.0	21.2	2.8									
	214/215	27.0	19.5	7.5	10.0	8.5	1.5	12.4	9.9	2.5	7.2	6.9	0.3
	218/219	39.4	37.2	2.2	8.6	10.3	1.8	12.4	9.9	2.5	7.6	7.1	0.4
	Mean	31.8	32.5	4.9	11.0	11.4	2.8	13.2	13.5	2.6	10.2	10.9	3.1
	S. D.	6.7	11.4	3.9	3.3	6.5	2.5	3.6	5.4	0.8	3.7	5.5	3.0

Average intrapair differences for male and female MZ and DZ twins are presented schematically in Fig. 1. A statistical comparison of the intrapair variance revealed that the female dizygous twins demonstrated as much diversity as the mono-

TABLE 5. Neural data of monozygous (MZ) and dizygous (DZ) male twins

Zygosity	Twins	Patellar reflex (m/sec)			Reaction time (msec)			Ulnar nerve conduction velocity (m/sec)			Maximal isometric IEMG forearm flexors		
	A B	A			A			A			A		
MZ	100/101	17.3	17.3	0.0	200	193	7	54.3	55.8	1.5	622	581	41
	104/105	15.8	16.7	0.9	215	225	10	43.7	44.2	0.5	286	366	70
	108/109	18.5	19.5	1.0	190	195	5	52.5	53.7	1.2	566	816	250
	110/111	19.0	17.5	1.5	203	168	35	51.2	54.6	3.4	342	444	103
	112/113	17.3	17.7	0.4	225	255	30	56.7	53.9	2.8	557	297	260
	114/115	18.7	18.3	0.4	255	265	10	42.9	35.5	7.4	418	327	91
	116/117	16.8	16.6	0.2	210	225	15	56.8	59.4	2.6	503	719	216
	118/119	14.8	15.2	0.4	245	230	15	39.5	41.4	1.9	1028	575	453
	Mean	17.3	17.4	0.6	217.9	219.5	15.9	49.7	49.8	2.7	540.3	514.4	185.4
	S.D.	1.5	1.3	0.5	22.5	32.6	10.9	6.7	8.4	2.1	228.6	190.2	127.5
DZ	200/201	23.5	20.1	3.2	205	160	45	58.6	53.1	5.5	550	608	58
	202/203	14.7	15.2	0.5	208	200	8	57.5	58.8	1.3	290	697	407
	208/209	16.5	18.8	2.3	190	230	40	49.5	49.4	0.1	472	561	89
	210/211	16.3	19.0	2.7	250	328	78	55.1	54.0	1.1	271	612	341
	212/213	16.8	14.0	1.8	223	203	20	35.4	49.0	13.6	407	442	35
	214/215	17.7	13.0	4.7	200	178	23	52.8	51.9	0.9	882	689	193
	218/219	_	_	_	205	238	33	46.0	35.6	10.4	604	730	127
	Mean	17.4	16.7	2.5	211.6	219.6	35.3	50.7	50.3	4.7	496.4	619.9	178.6
	S.D.	3.1	3.0	1.4	19.6	55.0	22.6	8.1	7.2	6.4	210.1	98.5	144.1

TABLE 6. Maximal mechanical power output and running velocity for monozygous (MZ) and dizygous (DZ) male twins.

Zygosity	Twin	Maximal muscular power output, kpm/sec			Maximal uphill running velocity, m/sec		
	A B	A	B	Δ	A	B	Δ
MZ	100/101	43.8	41.5	2.3	1.09	1.09	0.00
	104/105	38.6	40.6	2.0	1.22	1.27	0.05
	108/109	59.3	57.2	2.1	1.23	1.19	0.04
	110/111	50.2	48.4	1.8	1.07	1.05	0.02
	112/113	48.5	46.0	2.5	1.13	1.07	0.06
	114/115	38.3	37.8	0.5	1.01	1.04	0.03
	116/117	40.1	40.7	0.6	1.09	1.07	0.02
	118/119	28.6	29.8	1.2	1.08	1.09	0.01
	Mean	43.4	42.8	1.6	1.16	1.11	0.03
	S. D.	9.3	8.1	0.8	0.08	0.08	0.02
DZ	200/201	88.4	72.6	15.8	1.16	1.29	0.13
	202/203	40.6	35.8	4.8	1.13	1.19	O.O6
	208/209	40.9	51.5	10.6	1.13	1.12	0.01
	210/211	46.3	41.4	4.9	0.90	1.10	0.20
	212/213	31.5	25.7	5.8	0.91	0.91	0.00
	214/215	34.2	30.3	3.9	1.01	1.04	0.03
	218/219	63.0	47.7	15.3	1.17	1.12	0.05
	Mean S.	49.3	43.6	8.7	1.06	1.11	0.07
	S.D.	20.1	15.7	5.1	0.12	0.12	0.07

zygous twins in all of the variables studied: whenever there was a difference it was not statistically significant at the required confidence level of $P < 0.05$. For this reason further analysis of the measurements obtained from the female twins was abandoned and these measurements were used only to test the hypothesis of the sex influence on the genetic and nongenetic variability.

For the male twins the mean and the standard deviations of the difference distribution in muscle forces were greater within the DZ twins than between MZ pairs (Table 4), but the statistical test applied did not differentiate the intrapair variance at the required significance level. The intrapair variances between DZ and MZ male twins were significantly different for patellar reflex time ($P< 0.01$) and reaction time ($P< 0.05$), as shown in Table 5. In view of these significant differences we proceeded with the calculation of heritability estimates for these variables. The respective Hest Values for the reflex and reaction time were 97.5% and 85.7% as shown in Table 8. The close correspondence of MZ twins in reflex time is presented also graphically in Kg. 2. With regard to maximal muscular power, a clear intrapair difference ($P < 0.01$) was observed between the MZ and DZ twins for boys (Table 6). Kg. 3 clarifies this intrapair difference in MZ and DZ twins showing that in the X-Y coordinate system almost all the points for the MZ pairs are within the shaded area, which represents the experimental error, while the DZ pair values are markedly scattered around the line of identitity. The computed Hest as can seen in Table 8, was close to unity (99.2%).

DISCUSSION

Interpretation of twin data requires that both the validity and the true meaning of

the heritability estimate (Hest) be considered. The Hest is based on the thesis of comparability of environmental influences. This premise is open to serious question when psychological traits are the subject of investigation, as has been pointed out by many workers, but when somatic correlates are measured, as in the present study, then its acceptability can be substantiated (Klissouras, 1971). To strengthen this presupposition further caution was observed in obtaining comparable MZ and DZ groups with regard to their socioeconomic and health status, age and physical characteristics. With regard to the true value of the Hest one has to avoid making broad generalizations and inference about the relative powers of nature and nurture. The Hest is only an estimate of the extent to which heredity affects the variation of a given organic attribute, in a given population exposed to common environmental influences at a given time.

TABLE 7. Mean (M) and standard deviation (SD) values of selected variables for monozygous and dizygous female twein pairs (A, B) and their difference distribution (Δ)

Attribute		Monozygous (8 pairs) A			Dizygous (7 pairs)		
		A	B	Δ	A	B	Δ
Maximal muscular power	M	37.3	39.2	2.3	41.9	39.1	3.2
(kpm/sec)	SD	6.0	3.9	2.0	8.8	8.0	2.7
Vertical component of	M	1.07	1.08	0.03	1.01	1.03	0.05
maximal velocity (m/sec)	SD	0.04	0.06	0.03	0.09	0.06	0.02
Patellar reflex time	M	16.5	16.9	0.8	17.6	18.7	1.7
(msec)	SD	4.3	4.2	1.0	2.1	3.9	2.2
Reaction time (msec)	M	227.0	239.7	37.6	242.3	255.3	35.0
	SD	47.8	34.7	21.0	19.5	38.8	23.9
Nerve conduction velocity	M	52.4	55.8	3.9	51.9	51.1	3.1
(m/sec)	SD	5.6	3.1	3.8	4.3	5.1	2.2
Maximal isometric	M	422.1	594.7	217.1	421.3	542.4	192.9
IEMG μv χ sec/sec)	SD	123.1	241.4	199.4	104.3	217.5	125.6
Maximal quadriceps	M	27.9	27.8	2.3	32.6	31.6	3.9
isometric force (kg)	SD	5.3	3.5	2.1	6.3	6.0	1.9
Maximal forearm flexor	M	10.8	10.8	1.3	9.2	8.2	1.0
isometric force (kg)	SD	1.6	1.9	1.2	1.8	2.0	0.7
Maximal forearm flexor	M	12.1	12.7	3.0	8.7	8.5	1.7
eccentric force (kg)	SD	3.4	2.9	7.1	7.6	3.3	1.3
Maximal forearm flexor	M	8.5	8.8	1.2	6.5	5.5	1.1
concentric force (kg)	SD	2.0	1.6	0.8	1.6	1.4	0.9

TABLE 8. Estimates of variance within dizygous twins (s^2DZ) variance within monozygous twins (s^2MZ), variance of error of measurement (s^2), and heritability of variation (H_{est}) of selected physiological variables for male twins.

Attribute	s^2e	s^2MZ	s^2DZ	H_{est}
Patellar reflex time (msec)	0.20	0.29	4.03	97.6
Reaction time (msec)	43.80	178.06	842.21	85.7
Muscular power (kpm/sec)	1.20	1.68	49.43	99.2

Knowledge acquired from Hest should be complemented with information obtained from co-twin analysis on the potency of various modes of conditioning at different developmental ages and only then can the nature-nurture problem be

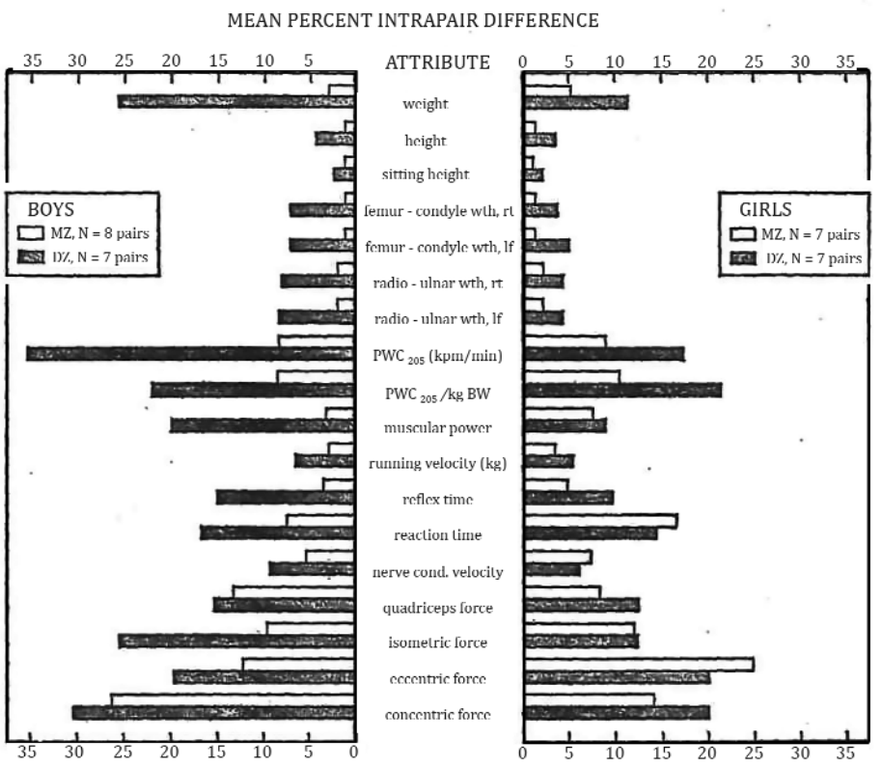

FIG. 1. Mean percent intrapair difference in MZ and DZ twins for antrhopometric data and all variables sudied in boys (left) and girls (right).

placed in perspective and the environmental forces upon hereditary predisposition be evaluated.

The marked heritability estimate obtained for the maximal muscular power in the male twins indicates that constitutional factors predominate in the production of energy through the anaerobic process. However, a statistically significant genetic variation was not observed either in mechanical power output expressed in kpm/kgBW/sec or in maximal running velocity. This implies that the variance in maximal muscular power may not be due to an individual difference in the splitting rate of high energy phosphate compounds, but to quantum, that is, to a greater amount of adenosinetriphosphate and phosphocreatine which are made available as a result of a greater mass of protoplasmic tissue. The data in Kg. 4 gives further support to the notion that maximal power output is dependent upon the total amount of energy drawn from anaerobic alactic sources.

The high heritability values for reflex and reaction time and the wide intrapair variability in nerve conduction velocity for both MZ and DZ twins point to the preponderance of the synaptic transmission rate of neural impulses in determining the speed with which a motor movement is executed. It is possible that dimensional factors such as height have a causal effect on the reflex time values obtained, as suggested by the fairly high correlation coefficients shown in Table 9, but the close relationship between two variables should not necessarily be interpreted to mean interdependence. Whether training can modify these variables cannot be ascertained from the present data. It is plausible however that the faster reaction and reflex

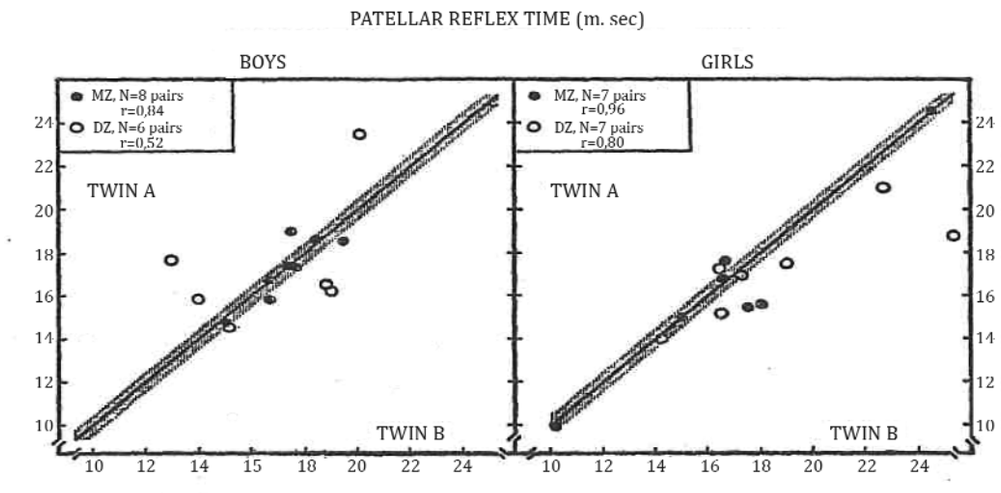

FIG. 2. Intrapair values of patellar reflex time for MZ and DZ twins in boys (left) and girls (right). The shaded area represents magnitude of the error of measurement.

time observed in athletes may be largely genetically determined. Further, we have some evidence on the relative effect of environmental forces on neural function. For example, we do know that semistarvation and vitamin deficiency have a profound effect on intellective tests, such as space perception and perceptual speed and psychomotor tasks such as manual speed and tapping rate (Simonson, 1971). In this connection it is important to mention that we have excluded measurements from a pair of MZ female twins aged 14 from our data presentation. One twin had been on a strict diet to reduce body weight and hence the assumption of a common environment, on which the heritability model is based, could not be sustained. We observed in a comparison of these twins that a 20% reduction in body weight was

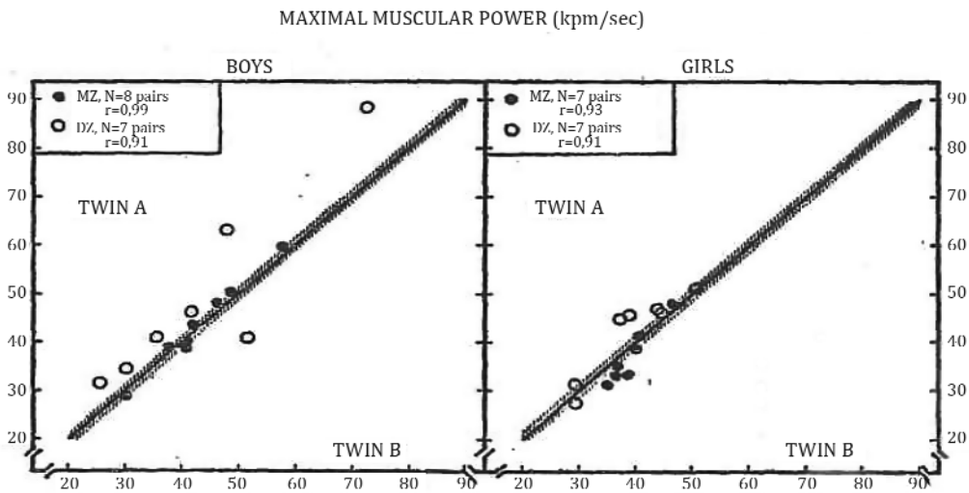

FIG. 3. Intrapair values of maximal muscular power for MZ and DZ twins in boys (left) and girls (right). The shaded area represents magnitude of the error of measurement.

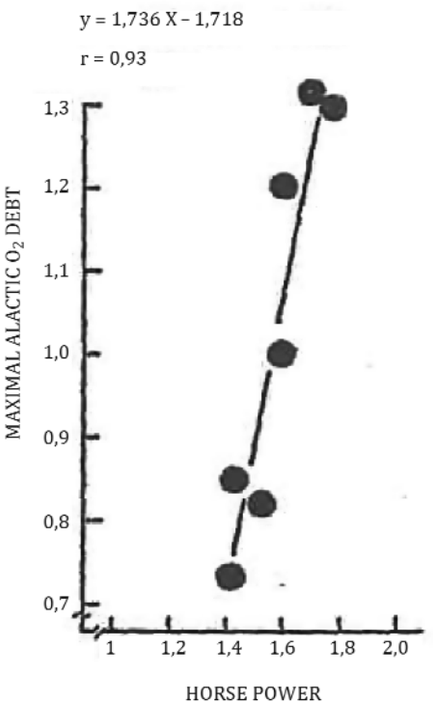

FIG. 4. The relationship between maximal alactio O_2 debt and maximal muscular power. The alactic O_2 debt is a net O_2 measured during the first 2 min of recovery from an all-out effort on a staircase (unpublished data from the Ergophysiology Laboratory, McGill University, obtained in collaboration with A. E. Wall)

accompanied by a 10% decrease in reflex and reaction time and a 20% decrease in nerve conduction velocity.

On the basis of the obtained heritability estimates, it would appear that maximal muscular power and reflex and reaction time are more susceptible to environmental modification in females than in males. No satisfactory explanation can be given for the basis of the mechanism in which these differences between sexes may originate. We do know however, that hormones are responsible for differences in morphological, functional and tissue composition, and the observed variability may be attributable to the sex-differential hormone secretions. The sex influence upon the variability of the various measurements may be appraised by separate comparison of the mean variance of the MZ male and female twins and of the DZ male and female twins. The former comparison will yield some information regarding the sex influence upon extragenetic effects, while the latter will give some idea of the effect of sex upon genetic differences. The variance in maximal muscular power between male and female monozygous twins was not significant, while that in dizygous twins was significant beyond the 0.05 level of confidence, suggesting a sex effect upon the genetic differences in

TABLE 9. Correlation matrix of selected variables for the twins (male - left; female - right)

	Wt.	Ht.	Muscular power	Reflex time	Wt	Ht.	Muscular power	Reflex time
Height	0.90				0.81			
Muscular power	0.96	0.90			0.93	0.75		
Reflex time	0.82	0.83	0.83		-0.00	0.06	-0.08	
Quadriceps force	0.69	0.77	0.80	0.61	0.90	0.74	0.84	-.06

maximal muscular power. This observation is puzzling and calls for further experimentation.

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Growth and Training with reference to Heredity

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Identical male twins (four sets aged 10 yr, four sets aged 13 yr, and four sets aged 16 yr) were divided so that one twin underwent strenuous endurance training for 10 wk, while his brother served as a control without training. Intrapair comparisons of the training-period changes in aerobic, anaerobic, and cardiorespiratory responses to maximum work on a bicycle ergometer disclosed that 1) 10-yr-old trained twins improved more than untrained twins did in VO_2 max, 2) 16-yr-old trained twins increased their $V0_2$ max, O, pulse, blood lactate, ventilation, and respiratory rate and decreased their maximal heart rates more than their untrained counterparts did, and 3) the trained and the untrained 13-yr-old twins changed commensurately. Further, interage comparisons of the intrapair differences showed that both the 10- and 16-yr olds improved their $V0_2$ max more than did the 13-yr olds. In view of this evidence, the old hypothesis that more may be gained by introducing extra exercise at the time when the rate of growth is greatest is not tenable.

Adolescent twins; genotype-training interaction; maximal aerobic power; maximal aerobic capacity; cardiorespiratory responses

ALTHOUGH WE KNOW THAT PHYSIOLOGICAL responses to training vary with age and that older men cannot improve their adaptive capacity as much as younger men can (16), we are still largely ignorant of whether increased physical exertion is more effective in altering functional adaptability at an earlier stage of maturation than at a later one. Possibly hormonal activity during some stage of the growing period overcomes any significant effect of additional stimuli on functional adaptability during this period. There is evidence that growth hormone in normal animals causes proportionately similar increases in skeletal muscles undergoing work-induced hypertrophy and in their controls. This is not to deny the pronounced development observed in adolescents who engage in competitive sports (1,2) but simply to point out that we have not been able, up to the present, to separate distinctly the relative importance of genetic disposition, growth, and amount of training at different developmental ages.

The use of cross-sectional and longitudinal studies in elucidating this problem has the obvious limitation that the degree to which the genetic factor is operant in different individuals is not known. The split-twin method obviates this problem, because each trained twin is accompanied by a genotypically identical control (12). Thus we used identical twins of three developmental ages (10, 13, and 16 yr) to study the effects of physical training on variables related to the oxygen transport system.

METHODS

Twins. Twelve pairs of identical twin boys (four sets aged 10 yr, four sets aged 13 yr, and four sets aged 16 yr) were used as subjects. Their zygosity was determined on the basis of morphological traits and a serological examination, as previously described (11). The physical characteristics of the three age groups are given in Table 1.

TABLE 1. Group means of physical characteristics before and after training, and mean values of individual % changes.

	Body Wt. kg	Standing Ht, cm	Lean Body Wt, kg	Vital Capacity, liters BTPS
10-Yr-oId twins				
Trained				
Before	28.6± 4.7	134.8 ± 12.3	25.1 ± 4.00	2.2± 0.4
After	29.7±5.3	137.0 ± 12.7	26.0±4.5	2.3± 0.4
% Diff	3.8 ± 1.8	1.7 ± 0.7	3.4 ± 2.3	10.8 ± 7.6
Untrained				
Before	28.6 ± 4.2	134.8 ± 11.7	25.1±3.6	2.2± 0.4
After	30.1 ± 4.9	136.8 ± 11.7	26.4 ± 4.2	2.3± 0.4
%, Diff	5.0 ± 2.1	1.5 ± 0.1	4.6 ± 2.7	7.5 ± 4.3
13-Yr-old twins				
Trained				
Before	49.9 ± 5.69	159.3 ± 6.1	39.03 ± 3.50	3.34 ± 0.36
After	51.20 ± 5.49	162.3 ± 5.9	40.4 ± 4.35	3.43 ± 0.42
% Diff	2.73± 2.47	1.90 ± 0.54	3.50 ± 3.8	2.8 ± 2.10
Untrained				
Before	48.90 ± 3.78	160.0 ± 5.9	38.53 ± 2.03	3.33 ± 0.20
After %	51.25 ± 4.58	163.0 ± 6.1	40.33 ± 3.42	3.45 ±0.20
Diff	4.78 ± 3.3	1.88 ± 0.49	4.65 ± 6.55	3.7 ± 3.8
16-Yr-old twins				
Trained				
Before	61.35 ± 3.19	171.8 ± 0.96	54.10 ± 3.26	4.57 ± 0.45
After	62.98 ± 5.21	172.8 ± 0.96	53.28 ± 4.31	4.66 ± 0.36
% Diff	2.58 ± 2.66	0.60 ± 0.49	- 1.63 ± 1.97	2.4 ± 6.57
Untrained				
Before	61.03 ± 4.57	171.5 ± 1.7	53.83 ± 5.25	4.77 ± 0.35
After	61.63 ± 5.44	173.3 ± 1.7	51.18 ± 4.43	4.73 ± 0.43
% Diff	0.90 ± 1.61	1.05 ± 0.30	-4.85 ± 1.96	-0.93 ± 3.82

Training plan. The twins were split so that one trained while his identical brother acted as a control. The twin brothers decided between themselves as to who would train. The 10-wk training was of the endurance type, designed primarily to improve maximal aerobic power. Essentially, it involved 1) three times a week, running 1 mile each time, to maximal effort; 2) three times a week, continuous step work lasting for an average of 8.5 min and eliciting an average heart rate of 162 beats/min; 3) once weekly cycling intermittently on a Monark bicycle ergometer at a pedaling frequency of 60 rpm. The exercise lasted for 3 min and elicited maximal heart rates; during recovery, the subject continued performing at a reduced work rate until his heart rate, continuously recorded, dropped to about 150 beats/min; this work/recovery pattern was carried on until exhaustion. In addition, the subjects trained on the average once weekly for competitive hockey or rugby. No effort was made to keep the control twin from his usual kinetic repertoire, but it was limited to normal daily routine and participation in regularly scheduled physical education classes.

Measurements. Twins performed a series of work loads at progressively increasing intensity on an Elema Schonander bicycle ergometer before and after training. Each work rate lasted 5 min and 15 s (except for the last supramaximal one, which was usually of shorter duration) and was followed by a 10-min rest pause. Twin brothers exercised alternately; this created a competitive atmosphere which motivated them to exert themselves to exhaustion during the supramaximal efforts.

Measurements of oxygen uptake, blood lactate, heart rate, and cardiac output were obtained for each work load.

Oxygen uptake was determined by the open circuit method during the last minute of exercise (this was from the 4th to the 5th min, except for the supramaximal efforts, which were of shorter duration). The Beckman paramagnetic and infrared gas analyzers, calibrated with known gas concentrations which were determined by the Scholander analyzer, were used for O_2 and CO_2 determinations of the expired air. A specially designed mixing chamber was used for sampling expired air, which was collected into small rubber bags and directed at a later time to the O_2 and CO_2 analyzers through a system of solenoid valves, which made it possible to avoid contamination errors. The inspired air was measured by a calibrated dry-gas meter connected in parallel with a set of bellows, in order to provide a reserve and increase the accuracy of the ventilatory measurements during maximal flows. In addition, a flow-sensitive potentiometer was connected at the mouth of the bellows in order to monitor breathing frequency. Readings of the gas analyzers, gas meter, and potentiometer were displayed on digital meters to facilitate recording. Finally, an electric clock was coupled in the assembly to take the precise time of measurements.

Blood lactate concentration was determined from an arterialized sample taken from a prewarmed fingertip at the 5th min of each recovery period and analyzed according to the Barker-Summerson method as modified by Strom (19). Cardiac frequency was determined from the subject's electrocardiogram, which was recorded with a Sanborn patient monitor. Cardiac output was estimated by the CO_2 rebreathing method using a continuous sampling and a graphical analysis, as described by Ferguson et al. (7). Rebreathing was done during the last 15 s of each work rate immediately following the measurement of oxygen uptake. Vital capacity was assessed by a Collins 9-liter respirometer. Lean body weight was predicted from skinfolds taken by the Lange constant-pressure caliper (10 g/mm^2) and introduced into the equation developed in children by Parizkova (14).

RESULTS

The metabolic and respiratory responses of the twins to maximal work and their cardiac responses to near maximal work, before and after training, are presented in Tables 2 and 3. A comparison of the percentage changes during the 10-wk period

TABLE 2. Group means of metabolic and respiratory responses to maximal work before and after training and mean values of individual % changes

	Oxygen Uptake, liters/min	Heart Rate, beats/min	Respiratory Quotient	Blood Lactate, mg 100 mL	Ventilation, liters/min BTPS	Respiratory Rate/ min
10-Yr-old twins						
Trained						
Before	1.59 ± 0.23	189.8 ± 2.5	1.13 ± 0.11	58.6 ± 26.1	68.35 ± 7.75	65.0 ± 13.5
After	1.96 ± 0.28	188.3 ± 8.0	1.03 ± 0.03	65.9 ± 28.4	78.84 ± 8.20	63.5 ± 9.6
% Diff	23.48 ± 6.12	-0.82 ± 3.08	7.96 ± 7.10	21.6 ± 56.2	16.44 ± 17.91	0.55 ± 25.1
Untrained						
Before	1.58 ± 0.13	189.8 ± 6.4	1.10 ± 0.08	55.0 ± 27.5	63.84 ± 4.68	56.5± 6.6
After	1.77 ± 0.18	189.3 ± 3.3	1.10 ± 0.08	54.2 ± 18.2	72.58 ± 8.58	65.6±4.4
% Diff	11.82 ± 4.99	-0.22 ± 1.82	0.78 ± 4.38	9.4 ± 48.5	14.08 ± 15.53	16.71 ± 11.0
13-Yr-old twins						
Trained						
Before	2.19 ± 0.20	195.5 ± 4.4	1.17 ± 0.08	75.4± 7.11	89.05 ± 11.29	50.0±6.3
After	2.49 ± 0.17	194.5 ± 4.2	1.06 ± 0.04	79.5 ± 14.45	95.61± 6.07	52.8 ± 5.5
% Diff	14.17 ± 7.28	-0.5 ± 0.6	-9.05 ± 3.7	6.5 ± 5.1	8.05 ± 6.09	6.7 ± 12.2
Untrained						
Before	2.18 ± 0.23	195.5 ± 4.4	1.12 ± 0.08	79.1 ± 6.5	85.62±13.92	49.3 ± 8.5
After	2.51 ± 0.10	194.8 ± 4.6	1.05 ± 0.08	73.5 ± 7.8*	92.74 ± 6.85	51.0 ± 5.8
% Diff	15.95 ± 8.95	-0.38 ± 0.5	-5.9 ± 9.3	-7.0 ± 0.0	9.7 ± 12.2	4.4 ± 7.2
16-Yr-old twins						
Trained						
Before	2.95 ± 0.25	183.5 ± 2.4	1.13 ± 0.05	67.8 ± 18.6	119.07 ± 7.06	56.5 ± 7.7
After	3.55 ± 0.30	189.8 ± 3.8	1.09 ± 0.04	85.3 ± 18.8	146.01 ± 21.7	65.0 ± 7.8
% Diff	20.53 ± 0.56	3.38 ± 0.81	-2.7 ± 6.8	27.3 ± 26.8	22.3 ± 12.7	15.4 ± 7.8
Untrained						
Before	2.95 ± 0.40	190.0 ± 5.6	1.24 ± 0.05	77.6± 21.0	133.28 ± 25.94	62.4 ± 14.7
After	3.03 ± 0.22	187.3 ± 8.1	1.09 ± 0.03	71.7± 18.2	122.40 ± 12.28	52.0 ± 10.6
% Diff	3.20 ± 4.71	-1.68 ± 4.41	-12.6 ± 1.9	-7.5 ± 4.9	-7.0 ± 8.2	-16.2 ± 5.9

* For technical reasons, this value is for two twins only.

TABLE 3. Group means of cardiac responses to near maximal work and mean values of individual % changes

	Cardiac Output, liters/min	Heart Rate, beats min	$(a\text{-}v)O^2$, Diff, vol%
10-Yr-old twins			
Trained			
Before	10.30 ± 1.48	181.8 ± 9.6	12.72 ± 2.40
After	11.33 ± 1.88	186.5 ± 10.7	15.12 ± 2.28
% Diff	12.06 ± 28.81	4.43 ± 5.46	21.50 ± 25.72
Untrained			
Before	10.83 ± 2.18	181.8 ± 13.8	11.79 ± 1.28
After	10.43 ± 1.08	178.5 ± 14.1	12.99 ± 1.14
% Diff	-2.43 ± 8.46	-1.76 ± 3.22	11.79 ± 21.36
13-Yr-old twins	12.15 ± 1.76	181.5 ± 17.23	16.18 ± 0.76
Trained	13.98 ± 3.59	178.5 ± 19.21	16.54 ± 1.61
Before	14.2 ± 17.84	-1.75 ± 2.06	2.5 ± 12.08
After			
% Diff	12.88 ± 1.75	183.0 ± 11.49	15.36 ± 1.03
Untrained	14.05 ± 3.51	174.0 ± 14.7	16.11 ± 1.9
Before After	7.95 ± 12.51	-5.0 ± 6.73	5.43 ± 16.35
% Diff			
16- Yr-old twins			
Trained	16.1 ± 0.63	168.0 ± 12.0	16.0 ± 1.49
Before	17.95 ± 5.01	169.5 ± 14.18	16.45 ± 1.72
After	10.93 ± 28.43	0.75 ± 2.87	3.43 ±14.22
Diff			
Untrained			
Before	16.15 ± 2.67	169.5 ± 21.56	14.98 ± 1.55
After	16.35 ± 3.73	165.0 ± 12.49	15.88 ± 1.19
% Diff	0.53 ± 7.91	-2.0 ± 9.56	6.75 ± 13.14

of observation indicated a trend toward a change of practically all the variables for both trained and untrained twins. To find out whether or not the intraindividual and the intrapair differences produced as a result of the training period were statistically significant, the Wilcoxon matched-pairs signed ranks test was used (17).

No significant difference was found between trained and untrained 13-yr olds for any of the variables measured. The same holds true for the 10-yr olds with the exception of their maximal oxygen uptake, which improved more in the trained than in the untrained twins at a significant level of $P < 0.02$. The 16-yr-old trained twins increased significantly their maximal oxygen uptake ($P < 0.01$), maximal oxygen pulse ($P < 0.01$), maximal blood lactate concentration ($P < 0.05$), maximal work ventilation ($P < 0.02$), maximal respiratory frequency ($P < 0.01$), and maximal work ventilation to vital capacity ratio ($P < 0.05$), while in the remaining parameters there was no difference. The mean differences before and after training for all the variables under study are summarized in Table 4.

The difference of differences between age groups was tested for significance with the Mason-Whitney U test (17). No significant differences were observed for any of the measurements between 10- and 16-yr olds. There was a difference between 10- and 13-yr olds only in maximal oxygen uptake, at a level of significance of $P < 0.05$. This interage difference appears because the intrapair difference was not changed in the 13-yr olds, but it was altered significantly in the 10-yr olds, as noted in the preceding paragraph and shown in Fig. 1. A statistical comparison of the difference of differences between 13- and 16-yr olds revealed that in the latter, training had produced greater changes in maximal oxygen uptake ($P < 0.02$), maximal

TABLE 4. Mean differences before and after period of observation, difference of differences between age groups, and their significances for selected parameters.

Variable	10-Yr-Old Twins		13-Yr-Old Twins		16-Yr-Old Twins		Probability of Significant Difference of Differences Between Age Groups	
	T	U	TU		T U		10-13 yr	13-16 yr
Body weight, kg	1.03	1.48	1.30	2.35	1.63	0.60	>0.05	>0.05
Standing height, cm	2.25	2.00	3.00	3.00	1.00	1.75	>0.05	>0.05
Lean body weight, kg	0.95	1.33	1.38	1.80	-0.83	-2.65	>0.05	>0.05
Maximal oxygen uptake, ml . kg^{-1}.min^{-1}	10.61*	4.04	4.16	4.67	8.56*	0.73	<0.05	<0.02
Maximal oxygen pulse, ml/beat	2.09	0.66	1.86	2.13	2.84*	0.67	>0.05	<0.02
Maximal heart rate, beats/min	-1.50	-0.50	-1.00	-0.75	6.25	-2.75	>0.05	<0.05
Maximal blood lactate, mg/100 ml	7.25	-0.85	4.13	-14.63	15.00*	-5.95	>0.05	>0.05
Maximal ventilation, liters/min	10.49	8.74	6.56	7.12	26.94*	-10.86	>0.05	<0.05
Maximal respiratory rate/min	-10.50	1.00	2.75	1.75	8.50*	-10.50	>0.05	<0.02
Maximal tidal volume, ml	0.18	-0.01	-1.00	0.08	0.15	0.20	>0.05	>0.05
Maximal V_I/VC	2.21	1.68	1.26	1.20	5.06*	-1.84	>0.05	<0.05
Maximal cardiac output, liters/min	1.03	-0.40	1.83	1.18	1.85	0.20	>0.05	>0.05
Maximal stroke volume, ml	3.90	-0.83	9.18	10.58	8.65	4.05	>0.05	>0.05
Maximal (a-v)0_2 difference, vol%	2.40	1.19	0.36	0.75	0.45	0.90	>0.05	>0.05
Maximal cardiac index, 1.min. m^{-1}	0.79	-0.62	0.94	0.45	0.92	-0.01	>0.05	>0.05

* Confidence level of 0.05 or more.

oxygen pulse ($P < 0.02$), maximal heart rate ($P < 0.05$), maximal work ventilation ($P < 0.05$), maximal respiratory frequency ($P < 0.02$), and maximal work ventilation to vital capacity ratio ($P < 0.05$). All intra-age and interage differences are given in Table 4. The intraindividual and intrapair differences observed in maximal oxygen uptake before and after training are presented in Fig. 1.

DISCUSSION

The commensurate increase in VO_2 max in trained and untrained 13-yr-old twins

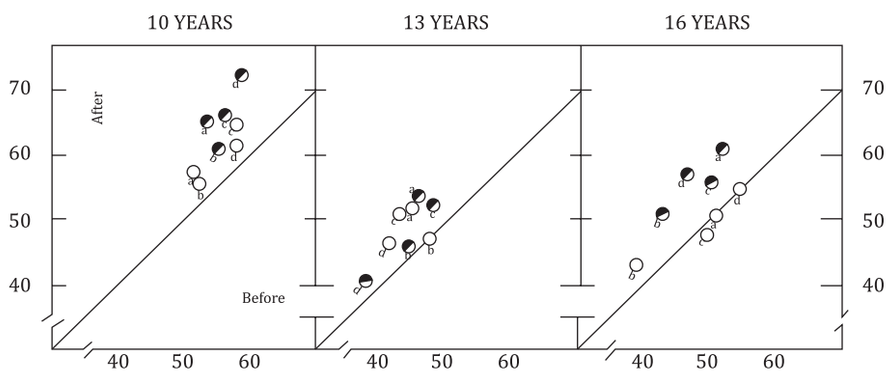

FIG. 1. Intraindividual values of maximal oxygen uptake (ml x kg^{-1} x min^{-1}) before and after period of observation training for all age groups. Partly filled and unfilled circles denote trained and untrained twins, respectively. Each set of letters represents a twin pair.

is striking. The most likely explanation hinges on the adolescent growth spurt, which occurs at this age as evidenced by the increase in height (20). The annual increase in body height of the 13-yr olds was 8 cm (Fig. 2). A rate of growth of such magnitude is expected to occur only during the adolescent spurt. Whether or not this growth spurt is directly related to growth hormone or to other hormonal factors is still speculative. It is possible that hormonal activity is maximal during this age and any additional stimuli such as training cannot override its influence. In this connection one thinks of the anabolic activity of the growth hormone, which stimulates the transport of amino acids across cell membranes and the synthesis of protein. However, some other factors must play an essential role, because the blood growth hormone levels in children and adolescents are not different from those observed in adults during rest (9, 10) and in response to muscular work (6).

From studies in rats, it has been reported that injections of growth hormones have caused elevation of plasma alkaline phosphatase levels (13), and such elevated levels have also been observed in boys of 13 yr. Some findings from animal studies confirm the hypothesis advanced here that during the growing period, hormones probably play a more dominant role in the development of functional adaptability than does physical activity. Indeed, Goldberg and Goodman (8) found that when rat muscle (soleus and plantaris) is injected with growth hormone, the muscle appears to increase in size by the same numeric factor whether there is atrophy from denervation or hypertrophy from increased work. These authors suggest that the hormone determines the absolute changes in muscle size, which result from changes in muscular work.

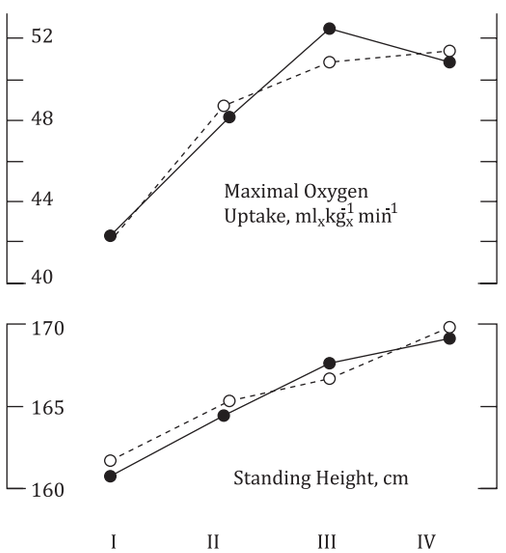

FIG. 2. Data obtained from 3 sets of 13-yg-old trained (solid line) and untrained (dotted line) twins. Time intervals: II = 2 1/2 mo, III = 8 mo and IV = 12 mo.

Several workers have observed no improvement in VO_2 max in boys who trained during the pubertal growth period. Sprynarova (18) noted no differences over a period of 3 yr in two active and one inactive group of 114 11-yr-old boys who had participated in sporting activities of various intensities. Parizkova and Sprynarova (14) observed a significant difference at age 13 between their most and least active groups, but when VO_2 max was expressed in biometric units of body weight, the difference disappeared. This was confirmed by Ekblom (4) and by Daniels and Oldridge (3). Ekblom found an absolute increase of 55% for 5 11-yr-old boys who had trained for 32 mo as compared to 37% for the reference group, but the relative increase was the same (7%) in the two groups. Daniels and Oldridge also observed in a study of 14 boys aged 11-15 yr, who trained for 22 mo, that the increase in VO_2 max was not at a gre-

ater rate than that at which weight increased. At variance with these findings is the recent report by Eriksson (5), who noted 16% increase to 48.5 ml•kg^{-1}•min^{-1} of body weight in 12 boys (11-13 yr) who trained for 4 mo.

It may be argued that our 10-wk observation period was too short to relate to the growth spurt the changes noted in the 13-yr olds. For this reason, the testing of three pairs of the 13-yr-old twins was extended over 1 yr. It was found that both trained and untrained twins continued to improve their VO_2 max at the same rate until the third testing period (Fig. 2). Nevertheless, the improvement over this observation period was about 22%, which is substantially more than the improvements noted in the training studies on adolescent boys that have already been cited.

It cannot be decided from the studies mentioned so far whether or not training at prepubescent age can have a greater influence on VO_2 max than that at a later age. Most probably the ontological time factor is decisive in the development of functionally important structures. However, it remains uncertain to which developmental period the growth-promoting stimuli that act upon the tissues should be applied. The old hypothesis that more might be gained by introducing extra exercise at the time when the growth impulse is the strongest is no longer tenable in view of the evidence obtained in the present study.

A question of considerable theoretical and practical importance is whether different genotypes respond to a given training stimulus with changes of different magnitude. Our split-twin experiments make it possible to separate the observed intrapair variance into its three components: that due to heredity, that due to training, and that due to the interaction between heredity and training. To avoid the growth spurt effect, only data obtained from the 10- and 16-yr-olds were used. Mean VO_2 max for all experimental and control twins was 51.9 ml•kg^{-1}•min^{-1}, with nonsignificant intrapair differences. Interpair variability ranged from 41.1 to 58.6 ml•kg^{-1}•min^{-1}, so that the interaction hypothesis could be tested. Mean VO_2 max after training was 59.4 ml•kg^{-1}•min^{-1}, with adjustments for changes observed in the nontrained twins, and the range was 45.2 to 69.3 ml•kg^{-1}•min^{-1}. Treatment of the results with analysis of variance revealed that the interaction between genotype and training does not contribute significantly to the total variance (Table 5).

These findings do not support the notion that the magnitude of improvement in VO_2 max depends on the relative strength of the genotype. Thus, the inverse relationship occasionally observed between initial level of VO_2 max and relative improvement (16) should be attributed to the amount and intensity of physical activity, which presumably modifies the initial level of VO_2 max. Further, it is surprising to find that in spite of strenuous training, the main cause of the total variance in VO_2 max is still the genetic predisposition. In this context, it should be pointed out that the partitioning of VO_2 max does not refer to individual values but to the variation in a population. In view of the available evidence, it seems that variability in VO_2 max

TABLE 5. Analysis of variance in $\dot{V}O_{2max}$ in 8 twin pairs.

Vo$_2$ max in 8 twin pairs

Sources of Variation	Mean Squares	Variance in Percent of Total Variance
Training	221.72	42
Heredity	69.04	51
Interaction	4.39	7

Estimates of variances, in actual figures, are computed in the following way (n = number of twin pairs): heredity = mean sq. heredity - mean sq. interaction/2;training = mean sq. training -mean sq. interaction/n.

observed in a population that has been exposed to common environmental forces may be almost entirely determined by heredity (11), but its relative contribution to the total variance may be reduced to about 50% with the operation of extreme environmental conditions such as training.

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