TESI DOCTORAL

ESTUDI DELS FACTORS GENÈTICS I AMBIENTALS QUE CONDICIONEN EL RENDIMENT FÍSIC

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DEDICATÒRIA

A l'Esther, la meva amiga, col·laboradora, esposa i mare de la Maria

A la meva família, sempre al meu costat

Als meus companys que sempre m'han ajudat

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- *Estudi de la glucosa i la fructosa amb curses de mig fons*, concedida per la Comissió d'Experts i amb Resolució del Director General de l'Esport (Juliol, 89).
- *Heretabilitat del metabolisme anaeròbic i diferents protocols de mesurar-ho*, concedida per la comissió d'experts i amb Resolució del Director General de l'Esport (Juliol, 90).
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ÍNDEX

I INTRODUCCIÓ I UNITAT TEMÀTICA 7

1. El rendiment físic com a fet multifactorial 7
2. Components genètics del rendiment físic 7
3. Components ambientals del rendiment físic 11
4. Interacció genètica-ambient: com la genètica condiciona la resposta als diversos estímuls ambientals 11
5. Complexitat de la valoració del rendiment físic i possibles errors metodològics 12
6. Bibliografia 12

II OBJECTIUS 14

III RESULTATS 14

1. Component genètic, 14

1.1. Estudis intraracials, 14

1. Could The A2a11 Human leucocyte antigen locus correlate with maximal aerobic power? *Clinical Science 1997; 92: 331-3* 14

2. A2a11 Human Leucocyte antigen locus is not present in athletes with high aerobic power. *Enviat al British Journal of Sports Medicine* 16

3. Heretabilitat de la flexibilitat: un estudi realitzat amb germans bessons. *Apunts. Medicina de l'Esport 1997; 128: 21-7* 18

4. Heritability of running economy. A study made on twin brothers. *European Journal of Applied Physiology 1998; 77: 511-6* 20

5. Heritability of the explosive power and anaerobic capacity. *Acceptat al European Journal of Applied Physiology* 22

1.2. Estudis interracials, 24

6. Cardiorespiratory response to exercise in elite Sherpa climbers transferred to sea level. *Medicine and Science in Sports and Exercise 1997; 7:937-42* 24

7. Normoxic ventilatory response in lowlander and Sherpa elite climbers. *Respiration Physiology* 1998; 113:57-64 26

2. Components ambientals, 28

2.1 L'entrenament físic, 28

8. The effect of prolonged physical exercise on ventricular function. *International Journal of Sports Cardiology 1992; 1: 79-82* 28

9. Efectes de l'entrenament anaeròbic en el múscul esquelètic. *Apunts. Medicina de l'Esport* 1998; 129: 27-36 29

10. A short training programme for the rapid improvement of both aerobic and anaerobic metabolism. *European Journal of Applied Physiology* 2000; 82: 480-6 31

11. The distribution of rest periods affects performance and adaptations of energy metabolism induced by high-intensity training in human muscle. *Acta Physiologica Scandinavica* 2000; 169:157-65 33

2.2 L'entrenament psicològic, 34

12. Assessment methodology on the effectiveness of psychological training. *Actas Proceedings. VIII World Congress of Sport Psychology Lisboa (1993)* 35

2.3 La nutrició, 36

13. The effect of previous ingestion of glucose or fructose on the performance of an exercise of intermediate duration *European Journal of Applied Physiology 1994; 68: 345-9* 37

2.4 L'entorn, 38

14. Comparison of the basal metabolic rate among champions of two nationalities in olympic and world games. *Medicine and Science in Sports and Exercise 1999; 31;5 (Suppl): 909* 39

3. Possibles errors metodològics, 40

15. Differences between lactate concentration of samples from ear lobe and finger tip *Journal of Physiology and Biochemistry* 1999; 55: 333-40 41

IV CONCLUSIONS FINALS 43

V ANNEXES. TREBALLS ORIGINALS PUBLICATS 44

I. INTRODUCCIÓ I UNITAT TEMÀTICA

1. El rendiment físic com un fet multifactorial

El rendiment físic està influenciat per molts factors, que van des de l'aportació energètica –tant per la via aeròbica com per les vies anaeròbiques– fins a l'estat d'ànim i la motivació que poden fer fallar a esportistes d'altra banda ben dotats genèticament.

A l'esquema següent de Keul (1996) podem veure quins són els factors més importants:

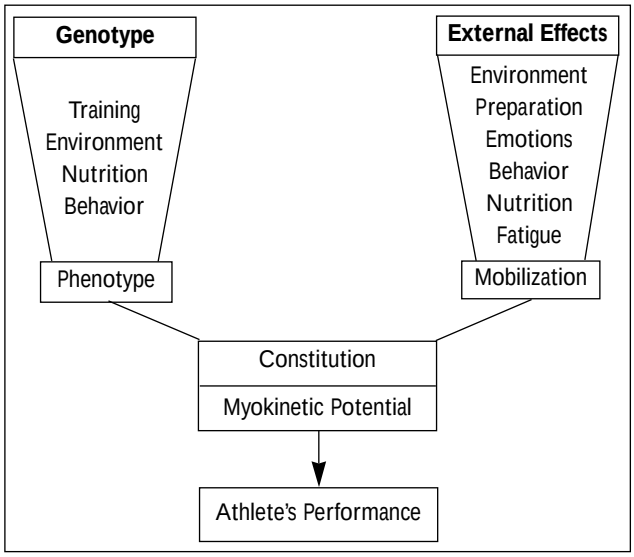

Figura 1. Factors ambientals versus genètics determinants del rendiment esportiu. (Keul i cols., 1996)

2. Components genètics del rendiment físic

Cada vegada coneixem millor el genoma humà, només cal veure en la figura 2 la descodificació del cromosoma 4 del codi genètic humà (cortesia d'US Department of Energy, "Human Genome Project") i com un gran nombre de malalties podran ser diagnosticades i tractades a partir d'aquest mapa genètic.

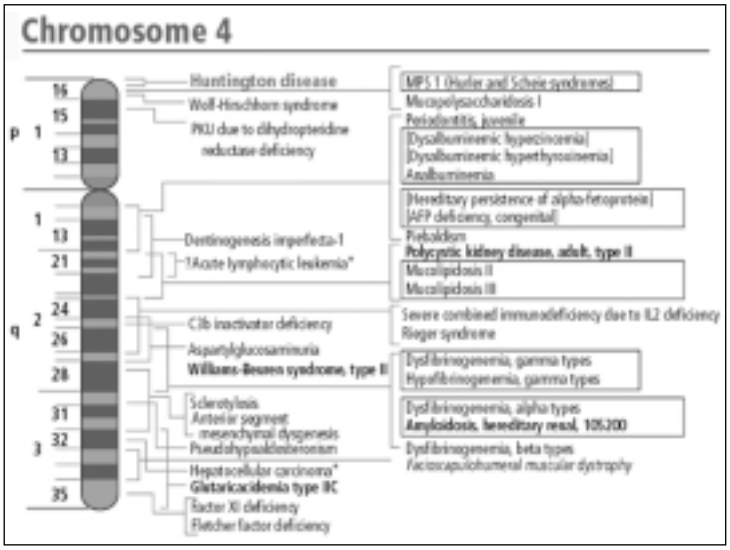

Figura 2. Mapa genètic cromosoma 4

En l'àmbit de la medicina de l'esport destacaríem dues línies importants:

1) la localització de factors genètics associats a determinades patologies relacionades amb l'exercici físic i la patologia associada al sedentarisme.
2) el paper dels gens en la determinació de les qualitats físiques condicionants del rendiment esportiu.

Així, per exemple, es coneix que existeix una relació entre les malalties cardiovasculars i la variació (polimorfismes) en el gen de l'enzim conversor d'angiotensina (ECA). Se sap que aquelles persones que són homozigots per l'al·lel D tindran un major risc de desenvolupar una malaltia coronària i una hipertròfia ventricular i les que tenen l'al·lel I tindran una millor resposta a l'entrenament de resistència.

Un altre camp de gran interès actual és el que fa referència a les causes de l'obesitat, i així se sap que està influenciat per una determinada proporció entre l'estil de vida i els factors genètics. Referent als factors genètics, s'ha postulat que hi ha més de 50 gens implicats en el mapa genètic humà, ja sigui per ells mateixos o per unions entre ells, i per tant, encara cal investigar molt per poder determinar quines associacions són les que determinen en més gran mesura el risc de patir d'obesitat. Avui, però, podem dir, que així com la variabilitat de la massa mineral òssia està en un 85 % aproximadament determinada genèticament, el sobrepès i l'obesitat estan molt aprop del 50 %, i per tant, tenim moltes més possibilitats de canviar aquestes tendències modificant els altres factors implicats en l'etiopatogènia com els diferents estils de vida, la quantitat d´exercici físic, la dieta etc.

La relació entre el que és innat i el que és adquirit s'ha estudiat mitjançant diferents estratègies com ara:

1) L' epidemiologia genètica, on s'estudia la importància dels gens en la contribució de determinats fenotips mitjançant estudis amb germans bessons, monozigots (MZ) i dizigots (DZ) o estudis familiars de diverses generacions.

2) La identificació de marcadors genètics, tant a nivell d'ADN com d'enzims o proteïnes que se sap que hi tenen alguna cosa a veure amb el rendiment físic i que permitran la recerca de sondes per a la identificació de portadors de variacions seqüencials d'ADN que permetran detectar futurs talents de l'esport.

Gràcies als primers estudis sobre la heretabilitat de diferents qualitats físiques coneixem, per exemple, que la capacitat d'un individu per la velocitat està molt més condicionada pels gens que hem rebut del nostres pares que la capacitat aeròbica i, per tant, del seu rendiment en proves de llarga durada.

Així podem veure en la figura 3 diferents índexs d'heretabilitat (IH) que permeten estimar la importància relativa de la influència genètica per un fenotip determinat, és a dir ens dóna una idea de la proporció de la variació atribuïble a la genètica. Un valor de 1,00 voldria dir que tota la variació (100%) és atribuïble al factor genètic i un 0,0 (0%) voldria dir que no hi ha cap component genètic. Aquest valor es pot obtenir mitjançant l'estudi de bessons o de generacions de famílies a les quals se'ls realitzen diferents proves i es miren les variacions que hi ha entre elles. Així, per exemple, quan més petites siguin les diferències entre MZ d'una qualitat determinada i més grans entre els germans DZ, més gran serà la variació atribuïble al factor genètic.

Malaltia/Qualitat	Índex d'Heretabilitat
Luxació congènita de cadera	1,00
Diabetis mellitus	0,75
Flexibilitat	0,75
Força muscular	0,70
Obesitat	0,50
Resistència aeròbica	0,40

Figura 3 . Diferents IH

Avui podem assegurar que la transmissió inherent de pares a fills del $\dot{V}O_2$ màx. està entre un 40 i un 60 % del total de la variació fenotípica. En aquest moment no hi ha una clara evidència que el rendiment físic de la via aeròbica vingui determinat per un sol gen. En aquest àmbit, dos estudis multicèntrics són avui capdavanters en recerca científica en l'àmbit de la genètica i del rendiment físic: l'HERITAGE FAMILY STUDY i el GENATHLETE STUDY.

El primer estudi ha tingut com a objectiu determinar el rol del genotip en la resposta cardiovascular, metabòlica i hormonal en l'exercici físic de característiques aeròbiques, i per això es van utilitzar 90 famílies caucasianes i 40 afroamericanes de més de tres generacions, sedentàries, sanes, entre 17 i 65 anys d'edat, que realitzaven un entrenament de 60 sessions en cicloergòmetre durant 20 setmanes al 50-75% del $\dot{V}O_2$ màx. (Bouchard i cols., 1995). Aquest grup ha publicat recentment sobre quins són els cromosomes humans que tenen major relació amb l'increment del rendiment físic valorat mitjançant el consum màxim d'oxigen previ i posterior a l'entrenament: 1p, 2p, 4q, 6p, 8q, 11p, 14q (on q: braç llarg i p: braç curt) (Bouchard i cols., 2000).

L'altre estudi analitza diferents enzims que, codificats per determinats gens, poden representar un factor genètic que influeix per ell mateix o mitjançant unions de diversos gens a tenir un rendiment físic més alt.

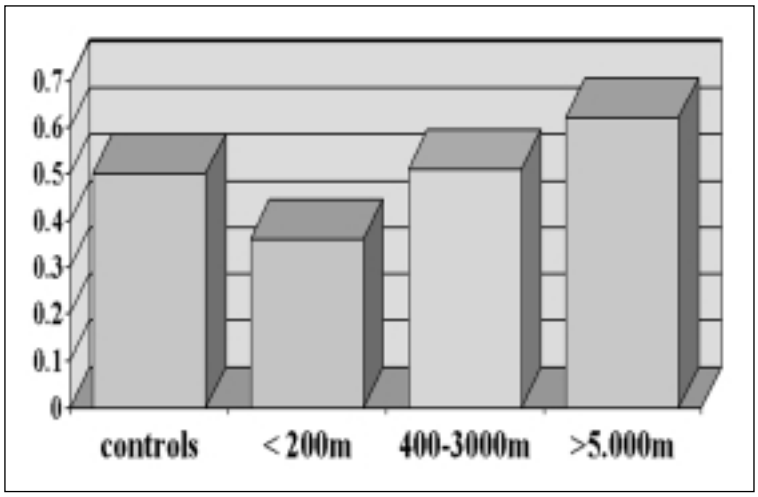

Figura 4. Freqüència relativa de l'al·lel I del gen codificador de l'enzim ECA en atletes anglesos de diferents modalitats

Així per exemple, podem veure en la figura 4 com aquells subjectes que tenen l'al·lel I (genotips I/D i II) de l'enzim conversor de la angiotensina (ACE) situat al cromosoma 17q23 (Saul Myerson i cols., 1999) tenen un millor rendiment esportiu en proves de llarga durada que el grup control i que el grup d'aquells que realitzen proves atlètiques on el factor de resistència aeròbica és menys important.

Però, revisant la bibliografia, aquesta hipòtesi no sembla estar totalment consensuada si bé hi ha indicis evidents. Alguns autors descriuen l'anterior vinculació: Montgomery HE i cols., 1998; Saul Myerson i cols. 1999; Álvarez R i cols., 2000, i en canvi altres no la veuen: Taylor R i cols. 1999; Rankinen T i cols. 2000, per tant, s'ha de ser molt cautelós a l'hora de donar conclusions anticipades.

El cromosoma 22 (figura 5) també ha estat proposat per altres autors com a posseïdor de gens que codifiquen proteïnes que tenen a veure amb el rendiment esportiu. Així se sap que en el braç llarg trobem el gen que controla el transportador de la glucosa, el transcriptor de la miosina o la pròpia mioglobina, però també trobem autors que no corroboren aquesta hipòtesi (J. Gagnon i cols., 1999).

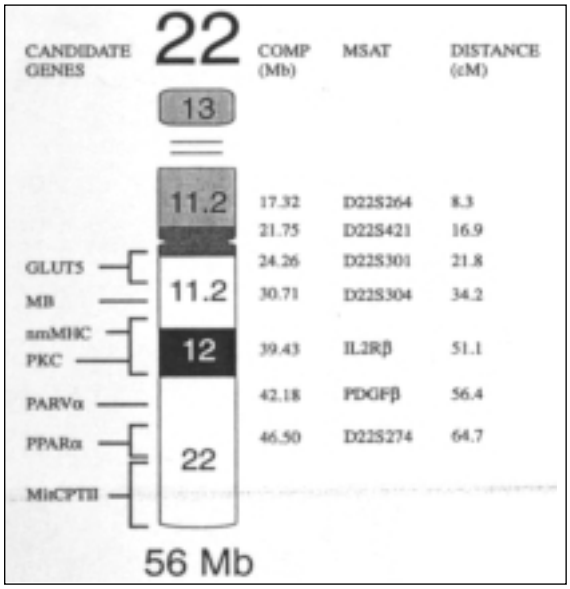

Figura 5 . Mapa genètic del cromosoma 22

L' altre cromosoma molt estudiat és el 6, un dels més petits i que avui sabem que en una zona del seu braç curt es troben els marcadors de la malaltia de l'hemocromatosi. Justament en aquesta porció telomèrica (6p21.3) és on es troben els gens del sistema HLA que, segurament, són marcadors d'altres gens situats als voltants i que, probablement, codifiquen enzims i proteïnes relacionades amb el metabolisme muscular. El nostre grup va trobar que existia una associació entre la presència dels al·lels A2,A11 del locus A situat a la regió p21.3 del Cr 6 humà i la potència aeròbica màxima o $\dot{V}O_2$ màx. (Gil Rodas i cols., 1997).

Per tant, si bé avui s'ha descodificat el genoma humà, encara queda molt per saber mitjançant una gota de sang si els nostres fills tindran predisposició a patir malalties cardíaques, o si arribaran a ser uns esportistes d'elit.

Dintre dels estudis genètics, també hi podem trobar d'aquells que intenten trobar diferències interracials (comparant individus de races diferents) o intraracials (comparant individus d'una mateixa raça). Les diferències genètiques més importants es donarien probablement en els estudis interracials. Cal destacar l'aclaparador èxit esportiu en proves de fons de la població del nord i de l'est de l'Àfrica, mentre que la de l'oest –i les persones amb antecedents familiars d'aquella zona, sobretot de l'època de l'esclavatge, com són part de la població dels Estats Units d'Amèrica, del Canadà i del Carib,...– triomfen en les proves de més curta durada; tot això evidencia el gran component genètic, que diferencia unes races d'altres.

Pel que fa a aquesta qüestió cal incidir en el fet que les persones "de color" no formen una raça –i encara menys una raça homogènia–, sinó que tal com hem comentat des del punt de vista funcional, i també des del punt de vista antropomòrfic, formen ètnies ben diferenciades, probablement força més diferenciades que altres grups de persones, que pel seu menor temps d'existència no s'han pogut diversificar tant –recordem l'origen africà de l'ésser humà–.

Nosaltres hem fet estudis tant intraracials (Rodas i cols. 1997; Rodas i cols., 1998) com interracials (Garrido i cols., 1997; Rodas i cols., 1998) en què vam cercar respostes fisiològiques diferents condicionades genèticament, i els seus marcadors; i també diferències fisiològiques entre races diferents. A més a més, vam analitzar els efectes de l'exercici de predomini aeròbic sobre la contractibilitat miocàrdica i l'aparició de signes de fatiga (Serra-Grima i cols., 1992) i diverses pautes d'entrenament de predomini anaeròbic, l'aparició de fatiga i les diferències en el rendiment (Parra i cols., 2000), al marge de components genètics.

L'eficiència energètica, o capacitat de realitzar més o menys treball amb una energia determinada, ha estat força estudiada per les seves evidents repercussions en el rendiment, però tan sols coneixem un estudi sobre el seu component genètic i és interracial (Saltin i cols., 1995), en el qual comparen l'eficiència de la cursa entre atletes escandinaus i kenyans. No s'ha trobat cap estudi intraracial, per la qual cosa vam realitzar un estudi intraracial mitjançant el mètode de bessons per quantificar el component genètic de l'economia de la cursa (Rodas i cols., 1998).

3. Components ambientals del rendiment físic

Com ja hem vist, la genètica condiciona clarament les diferents variables que determinen el rendiment físic, però això no vol dir que no puguin ser modificades per factors ambientals, que han estat força estudiats al llarg de la història i que per la gran extensió del tema no entrarem a revisar-los, però sí que cal destacar: l'activitat física i l'entrenament (utilitzats des d'èpoques molt llunyanes, i cada vegada més importants pels clars avantatges epidemiològics que comporten); les modificacions dietètiques (cada vegada estudiades més seriosament, sobretot a les darreres dècades, perquè és un camp molt interessant pels fisiòlegs); el suport psicològic (també més emprat); el material esportiu, etc.

Respecte al $\dot{V}O_2$ màx. sembla complir-se a l'Heritage Family Study, les hipòtesis que el principal component genètic és matern –basat en la contribució materna al DNA mitocondrial– i que el component patern seria ambiental (Bouchard i cols., 1998).

Dins d'aquests components ambientals capaços de millorar el rendiment físic, hem fet estudis en el camp de: l'entrenament, les modificacions dietètiques, el suport psicològic i la repercussió de l'entorn, que posteriorment detallarem.

4. Interacció genètica-ambient

La genètica no tan sols condiciona els valors "de sortida" d'una variable concreta sinó que també condiciona la seva modificació per distints estímuls ambientals com l'entrenament, la dieta, ...

En aquest aspecte són molt importants els resultats del ja mencionat Heritage Study, en què es posa clarament de manifest com la magnitud de l'augment de la potència aeròbica màxima, induït per un mateix entrenament, està clarament lligada a la genètica ja que hi ha persones que l'incrementen molt més que altres (Bouchard i cols., 1999).

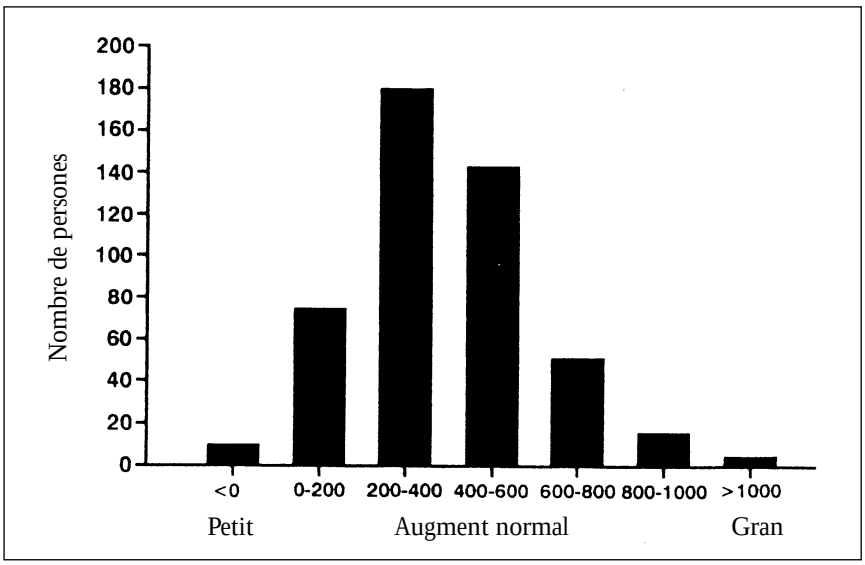

Figura 6. Increment del $\dot{V}O_2$ màx. produït per un mateix entrenament (Bouchard i col, 1999)

Per això, per pronosticar el rendiment esportiu no n'hi ha prou amb saber els valors inicials d'una variable donada, sinó que també s'ha de saber si la persona que es valora respon millor o pitjor als diferents estímuls que intenten fer que millori. O sigui, que unes variables són més susceptibles que altres de ser millorades, però també unes persones són més capaces que altres de millorar una variable concreta.

5. Complexitat de la valoració del rendiment físic i possibles errors metodològics

D'altra banda, davant de la possibilitat de fer una valoració de millora o empitjorament que no sigui real, i com que les determinacions de lactat plasmàtic per micromètode és un dels mètodes més utilitzats, vam fer un estudi comparatiu de les mostres de sang arterialitzada extretes de dos llocs en teoria equivalents: el lòbul de l'orella i la punta del dit, i es va poder apreciar que no eren idèntiques (Feliu i cols., 1999).

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II OBJECTIUS

L'objectiu de la tesi ha estat polièdric, ja que el rendiment físic es multifactorial. S'han intentat enriquir els coneixements sobre això utilitzant diverses metodologies de les més vàlides; i s'ha intentat aportar nous coneixements tant en la vessant genètica com en l'ambiental.

III RESULTATS

RESUMS DELS TREBALLS

1. Could The A2a11 Human Leucocyte Antigen Locus Correlate With Maximal Aerobic Power?
   Gil Rodas, Guadalupe Ercilla, Casimiro Javierre, Eduardo Garrido, Mar Calvo, Ramon Segura i Josep Lluís Ventura
   Clinical Science 1997; 92: 331-3

Abstract

The power of the aerobic metabolic pathway correlates well with successful physical performance in endurance sports events. The ability to alter the pathway through training presents well-known limitations, and consequently a good genetic endowment is essential to participate in elite sporting activities.

In 32 subjects (16 healthy pairs of male twin sportsmen, 8 monozygotic and 8 dizygotic) zygosity was determined by means of the genetic analysis of human leucocyte antigen (HLA) system specificities at class I and II loci and other genetics variants. The subject performed a progressive exercise test on a treadmill to ascertain the maximal oxygen uptake ($\dot{V}O_2$ max), measured by an automatic breath-breath analyzer. We have considered the relation ship between the A,B and C loci of the HLA system and $\dot{V}O_2$ max.

We found a high correlation between the presence of both HLA A2 and A11 and $\dot{V}O_2$ max. In the A2A11 group (n=6) we found a $\dot{V}O_2$ max. (mean±SD) equal to 71± 4 $ml \cdot min^{-1} \cdot kg^{-1}$. The group without this pair of alleles (n=26) showed a much lower aerobic power (58 ± 5 $ml \cdot min^{-1} \cdot kg^{-1}$).

Differences between the two groups were found to be largely significant ($P < 0.001$). It is noteworthy that in two pairs of dizygotic twins, the higher $\dot{V}O_2$ max value correspond to the twin with the A2A11 allele.

The very marked concordance between the presence of the A2A11 locus of the HLA system and the $\dot{V}O_2$ max could be of great interest for the identification of outstanding performers.

Key words: aerobic metabolism, human leucocyte antigen system, physical performance, twins.

Resum

La potència aeròbica màxima correlaciona correctament amb un rendiment físic alt en esdeveniments esportius de resistència. La capacitat de modificar-la per mitjà de l'entrenament presenta limitacions conegudes i, en conseqüència, és essencial un entorn genètic adient per participar en activitats esportives d'elit.
En 32 individus (16 parelles sanes d'esportistes bessons masculins, 8 monozigots i 8 dizigots) es determinava la zigocitat per mitjà d'una anàlisi genètica del sistema antigen leucòcit humà (HLA) especificat en els loci classe I i II i altres variants genètiques. L'individu realitzava un test progressiu sobre una cinta ergomètrica per determinar el consum màxim d'oxigen ($\dot{V}O_2$ màx.), mesurat per un analitzador de gasos automàtic respiració a respiració. Hem considerat la relació entre els loci A, B y C del sistema HLA i el $\dot{V}O_2$ màx.
Trobem una forta correlació entre la presència de l'HLA A2 i l'A11 i el $\dot{V}O_2$ màx. En el grup A2A11 (n=6) trobem un $\dot{V}O_2$ màx. (mitjana ± DE) igual a 71 ± 4 $ml \cdot min^{-1} \cdot kg^{-1}$. El grup sense aquest parell d'al·lels (n=26) mostrava una menor potència aeròbica (58 ± 5 $ml^{\cdot -1} \cdot kg^{-1}$).
Les diferències entre ambdós grups eren clarament significatives ($p<0,001$). En dues parelles de bessons dizigots el valor més alt de $\dot{V}O_2$ màx. correspon als bessons amb els al·lels A2A11.
La molt marcada concordança entre la presència del locus A2A11 del sistema ALH i el $\dot{V}O_2$ màx. podria ser de gran interès per a la identificació dels talents esportius.

Paraules clau: metabolisme aeròbic, sistema antigen leucòcit humà, rendiment físic, bessons.

Comentari

L'estudi mencionat semblava indicar que l'A2A11 del sistema HLA pot ser un marcador prou atractiu de la capacitat d'arribar a aconseguir valors alts de potència aeròbica màxima. A partir d'aquí era important veure si aquest probable marcador era present en esportistes amb grans valors de potència aeròbica màxima, com és el cas dels fondistes d'elit. Això és el que vàrem fer en el següent treball.

2. A2a11 Human Leucocyte Antigen Locus Is Not Present In Athletes With High Aerobic Power.
   Gil Rodas, Guadalupe Ercilla, Casimiro Javierre, Eduardo Garrido, Assumpta Estruch, Ramon Segura, Josep Lluís Ventura
   Enviat al ***British Journal of Sports Medicine***

Abstract

In a previous study we found a high correlation between the presence of A2,A11 antigens in locus A of the human leucocyte antigen (HLA) and high maximal aerobic power ($\dot{V}O_2$ max) in twin brothers.
Nine top-class national endurance athletes who achieved a $\dot{V}O_2$ max near or above 70 ml·min-1·kg-1 in a maximal treadmill stress testing were selected with the aim to determine HLA class I polymorphism by means of the serologic standard microlymphocytoxicity test.
None of athletes studied showed both A2,A11 antigens, while 5 of them (55%) were probably homozygous for HLA A2 locus, higher than expected according to Hardy Weinberg's law.
The absence of the A2,A11 antigens of the HLA system in this sample of high aerobic power athletes suggests that it can not be of use as a marker to detect potential endurance in humans but that perhaps it is related to other HLA haplotypes, mostly HLA A2, which is present in twofold in individuals with high $\dot{V}O_2$ max.

Key Words: maximal oxygen uptake, human leukocyte antigen system, genetic variation, short arm of chromosome 6.

Resum

En un estudi previ realitzat en un grup de germans bessons vam trobar una alta correlació entre la presència dels antígens A2, A11 del locus A del sistema antigen leucocitari humà (HLA) i la potència aeròbica màxima ($\dot{V}O_2$ màx.).
Nou atletes espanyols d'èlit que havien obtingut valors alts de $\dot{V}O_2$ màx. igual o superiors a 70 $ml \cdot min^{-1} \cdot kg^{-1}$ en una prova d'esforç sobre la cinta ergomètrica, van ser seleccionats per determinar els polimorfismes de la classe I del sistema HLA mitjançant tests serològics estàndards de microlinfocitotoxicitat.
Cap dels atletes estudiats va presentar ambdós antígens A2, A11, però cinc d'ells (55 %) eren probablemente homozigots pel locus A2, més del que s'esperava, tenint en compte la llei de Hardy Weinberg.
L'absència dels antígens A2,A11 del sistema HLA en aquesta mostra d'atletes amb alts valors de potència aeròbica màxima, suggereix que no podem utilit-

zar-los com a marcadors genètics per a la detecció d'humans que potencialment tenen una gran resistència aeròbica, però potser estan relacionats amb altres haplòtips del HLA, com l'A2, el qual està present en el doble d'individus amb un alt $\dot{V}O_2$ màx.

Paraules clau: consum màxim d'oxigen, sistema antigen leucocitari, variació genètica, braç curt cromosoma 6.

Comentari

Un cop determinat en el primer article que podria existir una correlació entre l'alt $\dot{V}O_2$ màx. i la presència o absència dels antígens A2A11 del sistema HLA, vam voler trobar aquesta correlació amb esportistes de alt nivell: corredors de fons. No vam trobar aquesta correlació, i en principi aquest fet exclou la possibilitat d'utilitzar aquest paràmetre com a senyal genètic per detectar en els humans característiques genètiques predeterminades per assolir un alt rendiment en proves de resistència, però caldria matiçar i dir que cinc individus (55%) eren probablement homozigots (no es pot assegurar perquè no tenim estudis familiars, i la determinació és qualitativa i no quantitativa) per al locus A2 del HLA, una prevalença major de l'esperada d'acord amb la llei de Hardy Weinberg. Això suggereix que un gen localitzat en el braç curt del cromosoma 6 en una unió desequilibrada amb el locus A podria estar relacionat amb l'alt $\dot{V}O_2$ màx., de manera similar al descobriment recent d'un gen candidat (HLA-H) que provoca hemocromatosi genètica i es localitza en el mateix braç curt del cromosoma 6 proper a la zona de la classe I de la HLA(5). En bessons monozigots, aquest locus es troba en el mateix cromosoma que els gens HLA, però en una població aleatòria, aquest gen podria associar-se amb una altre haplòtip HLA, preferentment A2, present en doble dosi en individus amb un alt $\dot{V}O_2$ màx.

Tanmateix, es necessitaran més treballs per determinar si aquesta hipòtesi es manté més enllà del grup estudiat aquí i per explorar els mecanismes subjacents a aquestes observacions. D'acord amb les nostres troballes, si volem buscar gens localitzats en la proximitat del sistema HLA (probablement codificadors dels enzims relacionats amb el metabolisme oxidatiu del múscul) el que hem de fer es traçar un mapa de la regió telomèrica del locus A situat a la regió p 21,3 del cromosoma 6.

3. Heretabilitat de la flexibilitat: Un estudi realitzat amb germans bessons.
Gil Rodas, Gerard Moras, Assumpta Estruch, Josep Lluís Ventura
Apunts. Medicina de l'Esport 1997; 128: 21-7

Abstract

Flexibility (FL) defined as the scope of movement of one or several joints is a factor of sport performance, which is determined by age, gender, training, anthropomorphic characteristics and genetic factors, of which very little has been studied. Method: To establish the relative importance of genetic and environmental influence on flexibility, we have studied the component, using the Heritability Index (HI at a value of 1.0 indicates that 100% of the variation would be due to genetic variation) in 24 sportsmen split into 12 pairs of twin brothers (6 monozygotes and 6 dizigotes), who did not have any significant perinatal or environmental differences. Zygosity was determined by genetic analysis of the leukocyte human antigen system (LHA), especially the class I and II loci, and other genetic variants . To calculate the FL, the subjects underwent an indirect flexometric test for the trigonometric calculation of the angle of aperture of the escapulohumeral (FL,SHA) and coxo femoral joints (FL,CFA), this later broken down into the horizontal and sagittal fields, for active and forced passive movements and for both legs. RESULTS: We do not find significant differences in the age and anthropomorphic measurements between the two groups (MZ and DZ). The HIs which were statistically significant for $F_{6,6}$ ($DZ \cdot MZ^{-1}$) over 4.2 and $P < 0.05$, are the FL, CFA in the transversal plane for open Iegs and active (0.82) and forced passive (0.77) movement, and in the sagittal plane for the right leg in active movement (0.78). CONCLUSION: We conclude that the genetic influence of flexibility is substantial, especially for the coxofemoral joint (75%) and mainly of the right leg, and our study corroborates the previous results yielded by family studies, at least for the target public of our research.

Key words: Heritability, Flexibility, Twin Brothers.

Resum

La flexibilitat (FL) definida com el rang d'amplitud de moviment d'una o diverses articulacions, és un factor del rendiment esportiu que ve determinat per l'edat, el sexe, l'entrenament, les catacterístiques antropomètriques i els factors genètics dels quals s'ha estudiat molt poc. MÈTODE: per descobrir la importància relativa de la influència genètica i ambiental en la flexibilitat, hem estudiat el component genètic utilitzant l'Índex d'Heretabilitat (IH= un valor de 1.0 indica

que el 100% de la variació seria deguda a variació genètica) a 24 subjectes barons i esportistes dividits en 12 parelles de germans bessons –6 monozigots i 6 dizigots–, que no presentaven diferències perinatals ni ambientals significatives. La zigositat es va determinar mitjançant l'anàlisi genètica del sistema antigen humà leucocitari (HLA), específicament els locus de la classe I i II, i altres variants genètiques. Per al càlcul de la FL els subjectes van realitzar un test flexomètric indirecte per mitjà del càlcul trigonomètric de l'angle d'obertura de les articulacions escapulohumeral (FL, AEH) i coxofemoral (FL, ACF), aquesta darrera desglossada en el camp horitzontal i sagital, per moviments actius i passius forçats i per ambdues cames. RESULTATS: No trobem diferències significatives pel que fa a l'edat i a les mesures antropomètriques entre els dos grups (MZ i DZ). Els IH que van ser estadísticament significatius per F6,6 (Dz·Mz-1) superior a 4.2 i P<0.005, són el FL, ACF en el pla transversal per cames obertes i moviment actiu (0.82) i passiu forçat (0.77) i en el pla sagital per la cama dreta en moviment actiu (0.78). CONCLUSIONS: Nosaltres concloem que la influència genètica de la flexibilitat és substancial, sobretot de l'articulació coxofemoral (75%) i preferentment de la cama dreta, i que almenys per la població que hem estudiat el nostre estudi corrobora els resultats anteriors fets amb estudis familiars.

Paraules clau: Heretabilitat, Flexibilitat, Germans bessons.

Comentari

Amb aquest treball comprovem la important càrrega genètica en la flexibilitat, similar a l'obtinguda amb estudis amb altres metodologies, i a més, precisem a quines articulacions és més gran el component genètic.

4. Heritability of running economy. A study made on twin brothers.
Gil Rodas, Mar Calvo, Assumpta Estruch, Eduardo Garrido, Guadalupe Ercilla, Antoni Arcas, Ramon Segura i Josep Lluís Ventura.
European Journal of Applied Physiology 1998; 77:511-6

Abstract

Running economy (RE), defined as the steady-state of oxygen uptake ($\dot{V}O_2$) for a given running velocity, is a factor of sports performance the genetic component of which has seldom been reported to date. We studied this component using a heritability index (HI) in a group of 32 male twins, 8 monozygotic (MZ) and 8 dizygotic (DZ) pairs, all sportsmen with similar perinatal and environmental backgrounds. Zygocity was determined by the identity of erythrocyte antigenic, protein and enzymatic polymorphism, and human leukocyte antigen serologic types between co-twins. The subjects exercised twice on a treadmill, once until exhaustion and again at sub maximal intensities. Pulmonary gas exchange was measured continuously using an automatic analyzer system during both tests. Blood samples were obtained during both tests. Blood samples were obtained during the recovery period to determine lactate concentrations. No significant differences were observed between MZ and DZ, in respect of RE at any speed or in maximal $\dot{V}O_2$ relative to body mass. Nevertheless, significant HI ($P < 0.005$) was found in maximal lactate concentrations (HI = 0.75) and in respiratory equivalent for oxigen at two speeds, 7 $Km \cdot h^{-1}$ (HI = 0.71) and 8 $Km \cdot h^{-1}$ (HI = 0.79), differences which probably suggest that there are differences in RE. In conclusion, we did not detect a genetic component in RE or in maximal oxigen uptake, but a genetic component for markers of anaerobic metabolism was present.

Key words: aerobic metabolism, genetic endowment, oxygen uptake, energy cost, twins and exercise.

Resum

L'economia de la cursa (EC) definida com el consum d'oxigen en estat estable ($\dot{V}O_2$) per a una velocitat donada de cursa, és un factor de rendiment esportiu del qual el component genètic no ha estat descrit fins a l'actualitat. Hem estudiat aquest component utilitzant un Índex d'Heretabilitat (IH) en un grup de 32 bessons masculins, 8 monozigots (MZ) i 8 dizigots (DZ), tots ells esportistes amb uns condicionants perinatals i ambientals similars. La zigocitat es determinava per la identitat dels antígens eritrocitaris, per un polimorfisme proteic i enzimà-

tic, i per uns tipus serològics antígens del leucòcit humà entre els cobessons. Els subjectes s'exercitaven dues vegades sobre una cinta contínua, una fins a quedar exhausts i una altra intensitat submàxima. El canvi gasós pulmonar es mesurava de manera contínua utilitzant un sistema d'anàlisi automàtica. En ambdós tests, s'obtenien mostres de sang durant el període de recuperació per determinar les concentracions de lactat. No es van trobar diferències significatives entre MZ i DZ, en relació amb l'EC a qualsevol velocitat o a màximes de $\dot{V}O_2$ relatives a la base corporal. Tanmateix es va trobar un significatiu IH ($p < 0,005$) en concentració màxima de lactat (IH = 0,75) i en equivalències respiratòries per a l'oxigen a dues velocitats, 7 $km \cdot h^{-1}$ (IH = 0,71) i 8 $km \cdot h^{-1}$ (IH = 0,79), distincions que probablement suggereixen diferències en l'EC. Concloent: no detectem un component genètic en EC ni en el consum màxim d'oxigen, però hi ha un component genètic en els registres del metabolisme anaeròbic.

Paraules clau: metabolisme aeròbic, consum d'oxigen, dotació genètica, cost energètic, bessons i exercici.

Comentari

Saltin et al. havien trobat en un estudi interracial (escandinaus i kenyans) que hi havia diferències en l'economia de la cursa entre els dos grups. Nosaltres, en aquest pioner estudi intraracial, no hi trobem diferències significatives. De totes maneres com que l'EC es determina habitualment pel consum d'oxigen en fase estable i s'aprecien augments de la concentració del lactat plasmàtic –amb un component hereditari significatiu–, resulta que calculada i afegida l'aportació energètica per la via làctica podria resultar que sí que hi hagués un component hereditari en l'EC.

5. Heritability of the response of explosive power and anaerobic capacity.
Mar Calvo, Gil Rodas, Miguel Vallejo, Asunción Estruch, Antoni Arcas, Casimiro Javierre, Ginès Viscor, Josep Lluís Ventura.
Acceptat a la revista ***European Journal of Applied Physiology***

Abstract

There is a disparity in the information about the heritability of the response of muscle anaerobic metabolism to exercise and explosive power as well as a lack of information about the genetic determinants of this form of work, measured with the different specific physical tests. We applied a battery of some of the commonly employed procedures (Erojump, Wingate, maximal accumulated oxygen deficit, oxigen debt and delta lactate) to a group of 32 Caucasian male twins, 8 monozygotic and o dizygotic pairs, with similar environmental backgrounds. Results were studied using a heritability index (HI). Zygosity was determined by the identity of erythrocyte antigens, protein and enzymatic polymorphism and human leucocyte antigen serologic types between co-twins. Significant HI values (P>0.005) were found in the following tests: maximal 5 s power (HI = 0.74) and total power 30 s (HI = 0.84) in the Wingate test, maximal lactate (HI = 0.82) and delta lactate (HI = 0.84) in the maximal progressive test, as well as in the second (HI=93) and in the third min (HI=0.92) of recovery, which express the oxygen debt alactacid component. In this intra-racial study, the most relevant findings were : 1) significant HI values for many of the variables studied; 2) the HI values of the parameters used to evaluated explosive power were higher than those of lactic capacity and 3) the HI of certain variables from different test measuring, in theory, similar qualities, were different.

Key words: genetic endowment, anaerobic metabolism, twins, exercise

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Resum

Hi ha una disparitat en la informació sobre la influència de l'herència en la resposta del metabolisme anaeròbic del múscul en l'exercici i la força explosiva, així com una manca d'informació sobre els determinants genètics d'aquesta forma de treball, mesurats en diferents tests físics específics. Hem aplicat una bateria d'alguns dels procediments habitualment utilitzats (Ergojump, Wingate, dèficit màxim d'oxigen acumulat, deute d'oxigen i delta lactat) a un grup de 32 bessons caucasians (homes), 8 monozigots i 8 dizigots, en condicions ambientals similars. Els resultats s'estudiaven utilitzant un Índex d'Heretabilitat (IH). La zigocitat es determinava per la identitat dels antígens eritrocitaris, per un polimorfisme

proteic i enzimàtic, i per uns tipus serològics antígens del leucòcit humà entre els cobessons. Valors d'IH significatius (p<0,005) es van trobar en els següents tests: potència màxima 5 s (IH = 0,74) i potència total 30 s (IH = 0,84) en el test de Wingate; màxim de lactat (IH = 0,82) i delta lactat (IH = 0,84) en el test progressiu màxim, així com en el segon (IH = 0,93) i en el tercer (IH = 0,92) minut de recuperació, que expressa el component alactacit del deute d'oxigen. En aquest estudi intraracial, les troballes més rellevants van ser: 1) valors d'IH significatius per a la majoria de les variables estudiades; 2) els valors d'IH dels paràmetres utilitzats per avaluar el poder explosiu eren més grans que la capacitat làctica; 3) l'IH de certes variables de diversos tests, que mesuraven en teoria qualitats similars, eren diferents.

Paraules clau: dotació genètica, metabolisme anaeròbic, bessons, exercici.

Comentari

Aquest estudi aporta més profunditat al coneixement del component genètic de la potència explosiva i del metabolisme anaeròbic en l'esforç físic, i és un estudi intraracial, ja que les diferències interracials són molt més clares. Cal destacar sobretot el gran pes de l'herència en general, però cal dir que és més important en els tests de curta durada que en els de llarga durada i la diferent heretabilitat de variables de diversos tests que en teoria mesuren el mateix, cosa que no deu ser del tot veritat.

6. Cardiorespiratory response to exercise in elite Sherpa climbers transferred to sea level.
Eduardo Garrrido, Gil Rodas, Casimiro Javierre, Ramon Segura, Assumpció Estruch, Josep Lluís Ventura.
Medicine and Science In Sports and Exercise 1997; 7:937-42

Abstract

Himalayan Sherpa are well know for their outstanding physical performance during ascents to the highest summits. To cast some light on this subject, we evaluated the cardiorespiratory response during exercise at sea level of six of the most acknowledged Sherpa climbers, mean age (±SD) 37 (±7) yr. old. Continuous electrocardiogram and breath-by-breath pulmonary gas exchange until exhaustion were obtained by following the Bruce protocol. We detected a maximal oxygen uptake ($\dot{V}O_2$ max) of 66.7 (±3.7) $mL \cdot min^{-1} \cdot kg^{-1}$, maximal cardiac frequency of 199 (±7) $beats \cdot min^{-1}$, and ventilatory anaerobic threshold at 62 (± 4) % of $\dot{V}O_2$ max. These factors could help to explain the greater performance level shown by several elite climbers of this ethnic group, The high functional reserve demonstrated by this very select group of highlanders could be associated with natural selection and with special physiological adaptations probably induced by long-training in a hostile environment.

Key words: highlanders, oxygen uptake, heart rate, anaerobic threshold, altitude, hypoxia, mountain climbing.

Resum

Els xerpes de l'Himàlaia són ben coneguts per la seva actuació física excepcional durant ascensions als cims de gran altitud. Per saber quelcom sobre aquest tema, hem avaluat la resposta cardiorespiratòria de 6 dels més coneguts xerpes durant d'exercici a nivell del mar, amb una mitjana d'edat (±DE) de 37 anys (±7). S'obtenien el registre electrocardiogràfic i els canvis de gasos pulmonars mitjançant un analitzador automàtic, respiració a respiració, fins a quedar exhausts seguint el protocol de Bruce. Hem detectat un consum màxim d'oxigen ($\dot{V}O_2$ màx.) de 66,7 (±3,7) $mL \cdot min^{-1} \cdot kg^{-1}$, una freqüència cardíaca màxima de 199 (±7) $batecs \cdot min^{-1}$, i un llindar anaeròbic ventilatori de 62% (±4) de $\dot{V}O_2$ màx. Aquests factors podrien ajudar a explicar l'excel·lent nivell d'actuació de diversos escaladors d'elit d'aquest grup ètnic. La gran reserva funcional demostrada per aquest selecte grup d'alpinistes podria associar-se amb la selecció natural i les adaptacions fisiològiques especials induïdes probablement per un llarg entrenament i per un ambient hostil.

Paraules clau: alpinistes, consum d'oxigen, altitud, hipòxia, escalada, ritme cardíac, llindar anaeròbic.

Comentari

En aquest cas hem estudiat el metabolisme aeròbic i la tolerància a càrregues físiques altes en un grup d'homes de l'ètnia xerpa que no seguien cap entrenament especial, però que tenien globalment el més gran historial alpinístic conegut. Les dades foren prou concloents: tant la seva potència com la resistència aeròbica eren molt importants –al voltant d'un 70% més de l'habitual en una població caucasiana estàndard–. Això fa ben palès: a) que aquests grans valors són per un component genètic, ja que el factor ambiental no pot justificar-los de cap manera i b) que el metabolisme aeròbic durant l'esforç, si bé, evidentment, no és l'únic determinant del rendiment en altitud –ja que el rendiment, i mes encara en situacions complexes i variants, és sempre multifactorial–, sí que deu tenir una importància considerable, el que fins ara alguns estudis havien rebutjat, dient que hi havia bons escaladors amb consums màxims d'oxigen poc importants.

7. Normoxic ventilatory response in lowlander and Sherpa elite climbers.
Gil Rodas, Casimiro Javierre, Eduardo Garrido, Ramon Segura, Josep Lluís Ventura
Respiration Physiology 1998; 113:57-64

Abstract

The differences in ventilatory response to exercise of some highland ethnic communities is a controversial issue. We have evaluated the differences in ventilatory response to exercise at sea level between two groups of elite climbers, four Himalayan Sherpas (S) and four Caucasian lowlanders (C), after descent from extreme altitude. All of them performed a progressive-intensity exercise on a treadmill under normoxic conditions. Pulmonary gas exchange was obtained until exhaustion by means of an automatic gas - analyzer system. Significant differences in expired ventilation and carbon dioxide production were found between the two groups, the $\dot{V}E \cdot \dot{V}O_2^{-1}$ being lower in the S at rest (41.9 ± 5) in comparison with C (48.7 ± 9) ($P < 0.05$), higher at medium loads (S = 28.2 ± 4 vs C= 25.7 ± 2; $P< 0.05$) and reaching similar values at higher loads (S= 34.5 ± 2 vs. C= 35.6 ± 4; NS). We conclude that the special ventilatory response observed in these highlanders could explain their adaptation to altitude, allowing higher oxygen blood saturation at medium working loads and reducing the risk of neurological injury caused by a high ventilatory response when exercising at high intensity effort under extreme altitude environment.

Key words: Acclimatization, high altitude, exercise; altitude, exercise, highlanders vs. lowlanders; Exercise, high-altitude acclimatization; gas exchange, pulmonary; mammals, humans.

Resum

Les diferències en la resposta ventilatòria durant l'exercici d'algunes comunitats ètniques de muntanya són un tema controvertit. Hem avaluat les diferències en la resposta ventilatòria durant l'exercici a nivell del mar entre dos grups d'alpinistes d'elit, quatre xerpes (X) de l'Himàlaia i quatre caucasians (C) habitants de terres baixes, després de baixar d'altitud extremes. Tots realitzaven un exercici d'intensitat progressiva sobre un tapís rodant sota condicions nòmades. El canvi de gas pulmonar s'obtenia quan estaven exhausts per mitjà d'un sistema analitzador automàtic de gas. Es van trobar diferències significatives en l'espiració i en la producció de diòxid de carboni en ambdós grups, essent el $\dot{V}E \cdot \dot{V}O_2^{-1}$ menor en els xerpes en descans (41,9 ± 5) en comparació als caucasians (48,7 ± 9) ($p<0,05$), major en les càrregues mitges (X = 28,2 ± 4 *versus* C = 25,7 ± 2; $p<0,05$) i valors

similars en les càrregues altes (X = 34,5 ±2 *versus* C= 35,6 ± 4; NS). Concloent: la resposta especial ventilatòria observada en aquests alpinistes podria explicar la seva adaptació a l'altitud permetent una major saturació d'oxigen a la sang en càrregues de treball mitjanes i reduint el risc de problemes neurològics causats per una resposta ventilatòria alta quan es treballa amb esforços d'intensitat elevada sota ambients d'extrema altitud.

Paraules clau: aclimatació, altitud elevada, exercici; altitud, exercici, homes de muntanya versus homes de terres baixes; exercici, aclimatació a les alçades elevades; canvi de gas, pulmonar; mamífers, homes.

Comentari

L'evidència històrica i diversos estudis mostren que els xerpes tenen, d'una banda, un gran rendiment físic en altitud i, de l'altra, que la repercussió neurològica de la hipòxia en altitud és menor que en els caucasians. Els resultats d'aquest treball –on es van trobar clares diferències en el comportament ventilatori durant l'esforç– poden donar part de la justificació d'aquesta realitat.

8. The effect of prolonged physical exercise on ventricular function.
   Josep Serra Grima, Ignaci Carrió, Manel Estorch, Eugenio Trilla, Luis Berna, Carlos Martínez-Dunker, Gil Rodas.
   International Journal of Sports Cardiology 1992; 1:79-82

Abstract

The aim of this study is to evaluate alterations of the ventricular function in long-distance runners. A radio nuclide ventriculography was recorded at rest and immediately after the athletes ran during 2 h. 30 min. There was a significant difference between basal heart rate and mean heart rate post-exercise. The systolic time was significantly longer after race (P< 0.005). There was not a difference between ejection fraction at rest and post-exercise. The etiology of this phenomenon is unknown.

Key words: long-distance runners, ventricular function, myocardial scintigraphy.

Resum

L'objectiu d'aquest estudi consisteix en avaluar les alteracions de la funció ventricular en corredors de llarga distància. Es va gravar una ventriculografia radionuclear en descans i una altra immediatament després que els atletes haguessin corregut dues hores i mitja. Hi havia una diferència significativa entre el ritme cardíac basal i la mitjana del ritme cardíac postexercici. El temps sistòlic era significativament major després de la cursa (p<0,005). No hi havia una diferència entre la fracció d'expulsió en descans i postexercici. L'etiologia d'aquest fenòmen és desconeguda.

Paraules clau: corredors de llarga distància, funció ventricular, escintigrafia miocardial.

Comentari

En aquest cas es posa de manifest un fet no evidenciat en la recerca fins ara, que podríem definir com a fatiga cardíaca, ja que es troba que després d'un esforç físic important, el rendiment cardíac minva. La causa no està determinada, ja que el metabolisme energètic del múscul cardíac és bastant diferent del metabolisme del múscul esquelètic.

9. Efectes de l'entrenament anaeròbic en el múscul esquelètic

Joan Parra, Gil Rodas

Apunts. Medicina de l'Esport 1998; 129: 27-36

Abstract

The skeletal muscle is a highly adaptable tissue and responds quickly to stressing situations by means of hypertrophy and atrophy mechanisms. Its plasticity is restricted by motor neurons and its synaptic order. Despite all this, the modifications induced to the muscular tissue can be extensive enough and try to be a reflex of what has provoked them.

This way, with a well-programmed training, it would have to be possible to improve the specific muscular characteristics, such as speed. But those criteria cannot be generalised due to the considerable populational differences.

Korni and cols. described that anaerobic performance, as well as the hystochemical and biochemical characteristics of the muscle, exhibited a substantial interindividual variability.

Age, sex, training level and heredity are factors that influence in a great manner on the diversity among individuals, both on anaerobic performance and on the size of muscular fibres or on the enzymatic activities. However, even thought the genetic component is considerably large, the muscle seems to have a disputably wide margin of adaptation to improve with training.

Key words: Phosphocreatine, glycogen, lactate, glucolitis, sporting performance, speed.

Resum

El múscul esquelètic és un teixit altament adaptable i respon ràpidament a situacions d'estrès mitjançant mecanismes d'hipertròfia o d'atròfia. La seva plasticitat ve restringida per l'adaptabilitat de les motoneurones i la seva ordenació sinàptica. Malgrat tot, les modificacions induïdes al teixit muscular poden ser prou extenses i tracten de ser un reflex d'allò que les ha provocat. D'aquesta manera, amb un entrenament ben programat, hauria de ser possible millorar característiques musculars específiques, com la velocitat. Però, aquests criteris no es poden generalitzar a causa de la considerable diferència poblacional. Komi i cols. van descriure que el rendiment anaeròbic, així com les característiques histoquímiques i bioquímiques del múscul, exhibien una gran variabilitat interindividual. Edat, sexe, nivell d'entrenament i herència són factors que influeixen fortament en la diversitat entre els individus, tant en el rendiment anaeròbic com en la mi-

da de les fibres musculars o en les activitats enzimàtiques. Però encara que la component genètica és força gran, el múscul sembla disposar d'un marge prou ampli d'adaptació per poder millorar amb entrenament.

Paraules clau: Fosfocreatina, glicogen, lactat, glucòlisi, rendiment esportiu, velocitat

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Comentari

El rendiment esportiu d'una gran part de les modalitats esportives es veu afectat i es determinant per la capacitat d'adaptació i millora del múscul a l'esforç anaeròbic. A diferència de les adaptacions aeròbiques amb un component extra-muscular molt alt, la sensibilitat a l'entrenament anaeròbic requereix de la comprensió dels mecanismes de que disposa el múscul per suportar aquest ritme de treball mecànic (alta freqüència i intensitat). En aquest article es fa un recull i anàlisi de l'adaptació del múscul a diferents models d'entrenament i els seus efectes a nivell cel·lular; posant de manifest la plasticitat de les respostes del teixit muscular a l'esforç anaeròbic.

10. A short training programme for the rapid improvement of both aerobic and anaerobic metabolism.
Gil Rodas - Josep Lluís Ventura - Joan A. Cadefau - Roser Cussó - Joan Parra
European Journal of Applied Physiology 2000; 82:480-6

Abstract

The aim of this study was to evaluate the changes in aerobic and anaerobic metabolism produced by a newly devised short training programme. Five young male volunteers trained daily for 2 weeks on a cycle ergometer. Sessions consisted of 15-s all-out repetitions with 45-s rest periods, plus 30-s all-out repetitions with 12-min rest periods. The number of repetitions was gradually increased up to a maximum of seven. Biopsy samples of the vastus lateralis muscle were taken before and after training. Performance changes were evaluated by two tests, a 30-s all-out test and a maximal progressive test. Significant increases in phosphocreatine (31%) and glycogen (32%) were found at the end of training. In addition, a significant increase was observed in the muscle activity of creatine kinase (44%), phosphofructokinase (106%), lactate dehydrogenase (45%), 3-hydroxy-acyl-CoA dehydrogenase (60%) and citrate synthase (38%). After training, performance of the 30-s all-out test did not increase significantly, while in the maximal progressive test, the maximum oxygen consumption increased from mean (SD) 57.3 (2.6) ml · min^{-1} · kg^{-1} to 63.8 (3.0) ml · min^{-1} · kg^{-1}, and the maximum load from 300 (11) W to 330 (21) W; all changes were significant. In conclusion, this new protocol, which utilises short duration, high loads and long recovery periods, seems to be an effective programme for improving the enzymatic activities of the energetic pathways in a short period of time.

Resum

L'objectiu d'aquest estudi consisteix en avaluar els canvis en el metabolisme aeròbic i anaeròbic produïts per un nou programa curt d'entrenament. Les sessions consisteixen en repeticions de 15 seg màximes amb 45 seg de període de descans, més repeticions de 30 seg al màxim amb períodes de 12 minuts de descans. El nombre de repeticions anava incrementant-se gradualment fins a un màxim de 7. Es van prendre mostres de biòpsia del múscul *vastus lateralis* abans i després de l'entrenament. Es van avaluar canvis en els resultats mitjançant dos tests, un test màxim de 30 seg i un test màxim progressiu. Es van trobar increments significatius en la fosfocreatina (31%) i en el glicogen (32%) al final de l'entrenament. A més, es va observar un increment significatiu en l'activitat muscular de creatina cinasa (44%), de fosfofructocinasa (106%), d'hidrogenasa (45%), de 3-hidroxiacil-CoA-deshidrogenasa (60%) i de citrat sintasa (38%). Després de l'entrena-

ment, els resultats del test de 30 seg màxim, no van incrementar-se significativament, mentre que en el test màxim progressiu, el consum màxim d'oxigen va incrementar-se des d'una mitjana (DE) de 57,3 (2,6) $ml \cdot min^{-1} \cdot kg^{-1}$ a 63,8 (3,0) $ml \cdot min^{-1} \cdot kg^{-1}$, i la càrrega màxima des de 300 (11) P a 330 (21) P; tots els canvis van ser significatius. En conclusió, aquest nou protocol, de duració curta, càrregues màximes i llargs períodes de recuperació, sembla un programa efectiu per millorar les activitats enzimàtiques de les vies energètiques en breus períodes de temps.

Comentari

En bastants ocasions cal millorar la preparació física en poc temps, i no està ben determinat com aconseguir-ho; més encara quan es volen millorar tant el metabolisme aeròbic com l'anaeròbic. Amb l'entrenament utilitzat sembla prou palès que això és possible, aportant una novetat fisiològica interessant i amb un caire pràctic evident. Cal pensar, a més, que en el procés de la recuperació funcional d'una lesió de l'extremitat inferior, abans de poder córrer es poden millorar les condicions metabòliques musculars fent treball sobre la bicicleta.

11. The distribution of rest periods affects performance and adaptations of energy metabolism induced by high-intensity training in human muscle.
Joan Parra, Joan Aurelio Cadefau, Gil Rodas, Narciso Amigo and Roser Cussó.
Acta Physiologica Sandinavica 2000; 169:157-65

Abstract

The effect of the distribution of rest periods on the efficacy of interval sprint training is analysed. Ten male subjects, divided at random into two groups, performed distinct incremental sprint training protocols, in which the muscle load was the same (14 sessions), but the distribution of rest periods was varied. The 'short programme' group (SP) trained every day for 2 weeks, while the 'long programme' group (LP) trained over a 6-week period with a 2-day rest period following each training session. The volunteers performed a 30-s supramaximal cycling test on a cycle ergometer before and after training. Muscle biopsies were obtained from the *vastus lateralis* before and after each test to examine metabolites and enzyme activities. Both training programmes led to a marked increase (all significant, $P < 0.05$) in enzymatic activities related to glycolysis (phosphofructokinase - SP 107%, LP 68% and aldolase - SP 46%, LP 28%) and aerobic metabolism (citrate synthase - SP 38%, LP 28.4% and 3-hydroxyacyl-CoA dehydrogenase - SP 60%, LP 38.7%). However, the activity of creatine kinase (44%), pyruvate kinase (35%) and lactate dehydrogenase (45%) rose significantly *(P* < 0.05) only in SP. At the end of the training programme, SP had suffered a significant decrease in anaerobic ATP consumption per gram muscle ($P < 0.05$) and glycogen degradation ($P < 0.05$) during the post-training test, and failed to improve performance. In contrast, LP showed a marked improvement in performance ($P < 0.05$) although without a significant increase in anaerobic ATP consumption, glycolysis or glycogenolysis rate. These results indicate that high-intensity cycling training in 14 sessions improves enzyme activities of anaerobic and aerobic metabolism. These changes are affected by the distribution of rest periods, hence shorter rest periods produce larger increase in pyruvate kinase, creatine kinase and lactate dehydrogenase. However, performance did not improve in a short training programme that did not include days for recovery, which suggests that muscle fibres suffer fatigue or injury.

Key words: anaerobic exercise, enzyme activities, glycogen, glycolysis, lactate, recovery, skeletal muscle metabolism, sprint training.

Resum

S'analitza l'efecte de la distribució dels períodes de descans sobre l'eficàcia dels intervals durant l'entrenament de l'esprint. Deu homes, dividits aleatòriament en dos grups van realitzar diferents protocols d'entrenament progressius d'esprint, on la càrrega del múscul era la mateixa (14 sessions), però la distribució dels períodes de descans variava. El grup del "programa curt" (PC) va entrenar cada dia durant dues setmanes, mentre que el grup del "programa llarg" (PL) va entrenar durant un període de sis setmanes, amb un període de dos dies de descans després de cada sessió d'entrenament. Els voluntaris realitzaven un test cíclic supramàxima de 30 seg sobre un cicloergòmetre abans i després de l'entrenament. Es van obtenir biòpsies musculars del *vastus lateralis* abans i després de cada test per valorar els metabolits i les activitats dels enzims. Ambdós programes d'entrenament van mostrar un marcat increment de les activitats enzimàtiques (totes significatives, $p<0,05$) en relació amb la glicòlisi (fosfofructocinasa – PC 107%, PL 68% i aldolasa – PC 46%, PL 28%) i amb el metabolisme aeròbic (citrat sintasa PC 38%, PL 28,4% i 3-hidroxiacil-CoA- deshidrogenasa - PC 60%, PL 38,7%). Tanmateix, l'activitat de la creatina cinasa (44%), del piruvat cinasa (35%) i de la deshidrogenasa lactat (45%) van augmentar significativament ($p<0,05$) només en el PC. Al final del programa d'entrenament, PC havia patit un descens significatiu del consum d'ATP anaeròbic per gram muscular ($p<0,05$) i una degradació de glicogen ($p<0,05$) durant el test postentrenament, sense poder millorar els resultats. Per contra, PL va mostrar uns resultats molt millors ($p<0,05$) encara que sense un augment significatiu en el consum ATP anaeròbic en la taxa de glicòlisi o de glicogenolisi. Aquests resultats indiquen que l'entrenament cíclic d'alta intensitat en 14 sessions millora l'activitat enzimàtica del metabolisme anaeròbic i aeròbic. Aquests canvis estaven afectats per la distribució dels períodes de descans, de manera que períodes curts de descans produïen un major increment en el piruvat cinasa, creatina cinasa i deshidrogenasa lactat. Tanmateix, els resultats van millorar en programes d'entrenament curts que no van incloure dies de recuperació, qüestió que suggereix que les fibres musculars pateixen fatiga o dany.

Paraules clau: exercici anaeròbic, activitat enzimàtica, glucogen, glicòlisi, lactat, recuperació, metabolisme múscul esquelètic, entrenament velocitat.

Comentari

L'estudi de l'entrenament està basat sobretot en les càrregues aplicades, la recuperació entre les càrregues aplicades, i els efectes produïts. En aquest cas, amb metodologia molt completa podem aconseguir una informació força interessant i sobretot de gran aplicabilitat en el camp de la pràctica de l'entrenament. Tan sols confirmar que el càlcul del descans és vital per a una correcta assimilació del treball físic realitzat.

12. Assessment methodology on the effectiveness of psychological training.

Luis Capdevila, Tomás Blasco, Manel Pintanel, Gil Rodas, Luis Valiente, Francisco Villamarín, Jaime Cruz

Proceedings VIII World Congress of Sport Psychology. *International Society of Sport Psychology* (pp. 439-442). Lisboa, June 22-27, 1993

Abstract

Our purpose has been to show how to develop a specific assessment methodology about the usefulness of psychological training programs. Four steps are followed: 1) Pretest levels; 2) Laboratory sessions; c) Application of the psychological training techniques; and d) Assessment on practice and competitive settings. Single-case design is always the best strategy to test the efficacy of psychological training methods, both on laboratory or natural settings, when our purpose is to achieve new goals on the application of sport psychology to individual sports. The single-case designs have some advantages over the traditional group designs, such as: a) fewer number of subjects are required; b) small, but consistent effects, that may be masked in a group design, can be detected, and c) they are more appropriate when working with elite athletes whose performance is unlikely to change clearly from baseline levels.

Resum

El nostre objectiu ha estat el desenvolupament d'una metodologia d'avaluació específica sobre l'efectivitat dels programes d'entrenament psicològic. Se segueixen quatre etapes: 1) Avaluació dels nivells pretest; 2) Sessions de laboratori; c) Aplicació de tècniques d'entrenament psicològic; i d) Avaluació en situacions d'entrenament i de competició. El disseny de cas únic resulta sempre la millor estratègia per a provar l'eficàcia de les tècniques d'entrenament psicològic, tant en situacions naturals com de laboratori, quan es pretenen assolir nous objectius en l'aplicació de la psicologia de l'esport als esports individuals. Els dissenys de cas únic tenen alguns avantatges respecte als dissenys de grup tradicionals: a) es requereix un nombre menor de subjectes; b) permeten detectar efectes petits però consistents, que poden estar emmascarats en un disseny de grup; c) són més adequats quan es treballa amb esportistes d'alt nivell, ja que el seu rendiment pot mostrar petits canvis respecte al seu nivell basal.

Comentari

Des de sempre, el desenvolupament de programes d'entrenament psicològic per a potenciar el rendiment en esportistes d'alt nivell ha estat un objectiu prioritari en l'àmbit aplicat de la Psicologia de l'Esport. S'han proposat un gran nombre de programes i de tècniques d'entrenament psicològic per tal que l'esportista pugui controlar amb èxit problemes com l'ansietat precompetitiva o la manca de concentració, de motivació, o d'autoconfiança. Però, des d'una perspectiva científica, s'han trobat moltes dificultats metodològiques a fi de comprovar l'efectivitat objectiva d'aquestes tècniques en situacions naturals. En molts casos, no estan clars els resultats perquè les tècniques s'apliquen en grups d'esportistes que presenten unes característiques esportives, unes habilitats pròpies i unes necessitats d'entrenament psicològic molt diferents. A més, tampoc no està gens definida la forma amb què es comprova l'eficàcia de les tècniques en la millora del rendiment esportiu.

Amb l'objectiu de millorar la metodologia d'avaluació, en aquest treball es proposa el desenvolupament d'una sèrie d'estratègies adreçades a comprovar l'eficàcia de diferents tècniques d'entrenament psicològic en la millora del rendiment esportiu. Així, es proposa un procediment en quatre etapes que està basat en dos aspectes fonamentals:

a) la utilització de dissenys de cas únic, que permeten treballar amb un nombre reduït de subjectes, possibilitant la detecció d'efectes petits però consistents que poden estar emmascarats en un disseny de grup; i
b) l'avaluació contínua o longitudinal de les dades presentades pels casos únics, que permet una comparació més objectiva i realista de l'evolució de l'esportista, tant respecte a les tècniques aplicades com al rendiment esportiu.

Aquesta metodologia ha estat utilitzada en els últims anys pel nostre grup d'investigació, i ha permès aplicar amb èxit en situació natural els resultats obtinguts a partir de l'avaluació rigorosa prèvia de laboratori.

13. The effect of previous ingestion of glucose or fructose on the performance of an exercise of intermediate duration

Josep Lluís Ventura, Assumpció Estruch, Gil Rodas, Ramon Segura

European Journal of Applied Physiology 1994; 68: 345-34

Abstract

The metabolic responses induced by the ingestion of a beverage containing glucose (G), fructose (F) or placebo (W) 30 min before exercise of high intensity and intermediate duration have been investigated: in these conditions the energy processes are mostly dependent on aerobic reactions. A group of 11 male recreation sportsmen ran on a treadmill at an intensity corresponding to 82% of peak oxygen consumption until exhaustion, on three different occasions(after ingestion of beverage containing 75 g G, 75 g of F, or W). Plasma glucose, insulin, and lactic acid concentrations were determined just prior to the ingestion of the beverages, 30 min afterwards and 10 and 30 min after completion of the exercise. The mean endurance time was 644 (SD 261) s after the ingestion of G, 611 (SD 27)s after the ingestion of F and 584 (SD 189) s after the ingestion of the W ($P< 0.05$) between G and W). No differences in the oxygen uptake, respiratory quotient or lactate concentrations between the three trials were observed. Both plasma glucose and insulin concentrations determined in samples obtained immediately before the onset of exercise were higher when G was ingested than when F ($P< 0.005$ and $P<0.005$, respectively) or W ($P< 0.001$ and $P< 0.005$, respectively) were ingested. These findings would suggest that the ingestion of G prior to an effort of intermediate duration may improve physical performance.

Key words: Blood glucose, carbohydrates, endurance.

Resum

S'investiga la resposta metabòlica induïda per la ingesta d'una beguda que conté glucosa (G), fructosa (F) o placebo (W) trenta minuts abans d'un exercici d'alta intensitat i de durada intermitja: en aquestes condicions els processos energètics són majoritàriament dependents de reaccions aeròbiques. Un grup d'11 esportistes masculins afeccionats van córrer sobre un tapís rodant a una intensitat corresponent al 82% de consum màxim d'oxigen fins a quedar exhausts en tres situacions diferents (després de la ingesta de la beguda que contenia 75 g G, 75 g F, o W). Es van determinar les concentracions de glucosa, insulina i àcid làctic al plasma just abans de la ingesta de les begudes, 30 minuts després, i entre 10 i 30 minuts després de completar l'exercici. El temps de resistència mitjà va ser de

644 seg (DE 261) després de la ingesta de G, 611 (DE 27), després de la ingesta de F, i de 584 (DE 189) després de la ingesta de W ($p<0,05$) entre G i W. No es va observar cap diferència en la despesa d'oxigen, quocient respiratori o concentracions de lactat en els tres casos. Tant la glucosa com les concentracions d'insulina en el plasma determinades en les mostres obtingudes immediatament abans de l'inici de l'exercici eren més grans quan G es bevia que quan F ($p<0,005$ i $p<0,005$, respectivament), o W ($p<0,001$ i $p<0,005$, respectivament). Aquests resultats suggereixen que la ingesta de G abans d'un esforç de durada intermitja pot millorar l'actuació física.

Paraules clau: glucosa a la sang, carbohidrats, resistència.

Comentari

L'aportació de glícids més o menys temps abans d'un exercici físic s'ha realitzat bàsicament en exercicis de duració igual o superior a 1 hora i amb la finalitat de posposar el buidament dels dipòsits de glucogen i, conseqüentment, de l'aparició de fatiga. però, hi ha molts exercisis físics de durada inferior a 1 hora, en què la fatiga no depèn del buidament de dipòsits glucídics, i si també són de durada superior als 2 minuts la via aeròbica serà la que aporti més subministrament energètic. La via aeròbica té un rendiment energètic (formació d'ATP per volum d'oxigen consumit) un 12 % més gran quan s'oxida glucosa que quan s'oxiden àcids grassos. La ingestió de glucosa produeix l'alliberament pel budell prim del pèptid inhibit gàstric, que té una gran activitat insulinotròpica i en augmentar la secreció d'insulina pel pàncrees, produeix una reducció de l'activitat lipolítica del teixit adipós; tot això produeix un augment de la utilització de glucosa respecte als àcids grassos en els músculs i exhaureix abans els dipòsits de glicogen, però aixo no succeeix en exercicis de menys d'una hora de durada; i en canvi, sí que era d'esperar que augmentés el rendiment energètic i així va ser en el nostre treball, amb un millora significativa del rendiment físic el dia de la ingestió de glucosa respecte al de la ingestió d'aigua. La ingestió de fructosa produeix un increment més petit de la glucosa i la insulina en el plasma que la ingestió de glucosa i, com que hi havia estudis contradictoris sobre aquest aspecte vam decidir estudiar-lo, però l'increment del rendiment no va ser significatiu.

14. Comparisson of the basal metabolic rate among champions of two nationalities in olympic and world games.

Cintia Biehl, Ramon Segura, Gil Rodas, Josep Lluís Ventura

Medicine And Science In Sports and Exercise 1999; 31;5 (Suppl): 909

Abstract

The recommendation of the FAO/WHO/ONU committee that the basal metabolic rate (BMR) serves as the basis for estimating dietary energy intake has rekindled interest in this fundamental physiological measurement. This study has as objective to compare BMR among champion athletes from Spain (SPA) (n=15) and from Brazil (BRAZ) (n=15), of the masculine sex, of 4 different sports in even numbers of athletes of each sport. The date of evaluation of BMR is characterized in the Sport Calendar as period of "post competition" that happened in the Olympic and World games. An Mijnhardt gases analyzer was used for calculation of $\dot{V}O_2$ and $\dot{V}CO_2$. Routine procedures were strictly followed in the preparation of basal conditions and during its measurements for BMR calculation. The measurement of the oxygen consumption was accomplished alon 35 minutes.

These results suggest that the Brazillian athletes have heavier weight (WT) and had more higher lean body mass (LBM), that could suggest a higher BMR. However, the relative percentage was of less 17.88% of BMR ($LO_2 \cdot min^{-1}$) and 17.10 % of BMR ($ml \cdot kg\ WT^{-1} \cdot min^{-1}$) ($LO_2 \cdot min^{-1}$) and 17.11 % of BMR ($ml \cdot kg\ LBM^{-1} \cdot min^{-1}$)in relation to the Spanish athletes. Conclusion: The champions from Brazil that inhabit a tropical zone presented significant lower BMR ($P< 0.05$) than the Spanish Champions that live in a temperate climate.

Resum

La recomanació del comitè de la FAO/WHO/ONU que el ritme metabòlic basal (RMB) serveix com a base per valorar la quantitat energètica de la dieta, ha revifat l'interès d'aquesta mesura fisiològica fonamental. Aquest estudi té com a objectiu comparar el RMB entre atletes campions d'Espanya (n = 15) i de Brasil (n = 15), de sexe masculí, de quatre esports diferents en un nombre equivalent d'atletes de cada esport. La data d'avaluació de la RMB està descrit en el calendari esportiu com a un període de postcompetició que té lloc després dels Jocs Olímpics i Mundials. Es va utilitzar un analizador de gasos de Mijnhardt per fer el càlcul de $\dot{V}O_2$ i del $\dot{V}CO_2$. Els procediments de rutina es seguien estrictament en la preparació de les condicions basals i durant la seva mesura del càlcul de la RMB. La mesura del consum d'oxigen es realitzava durant uns 35 minuts.

Aquests resultats suggereixen que els atletes brasilers tenen un pes més elevat i tenen més massa corporal muscular que el que podria suggerir un alt RMB. Tanmateix, el percentatge relatiu era de menys del 17,88% del RMB ($LO_2 \cdot min^{-1}$), del 17,10% del RMB ($ml \cdot kgWT^{-1} \cdot min^{-1}$) ($LO_2 \cdot min^{-1}$) i del 17,11% del RMB ($ml \cdot kg\ LBM^{-1} \cdot min^{-1}$) en relació amb els atletes espanyols. Conclusió: els campions de Brasil que habiten en una zona tropical presenten un menor RMB ($p<0,05$) que els campions espanyols que viuen en una zona de temperatura no tropical.

Comentari

El metabolisme basal té moltes i cabdals implicacions i, per això, el seu càlcul és important en la fisiologia en general i en la fisiologia de l'esforç físic. Per la laboriositat de la seva determinació sovint s'utilitzen els valors teòrics. Aquest treball posa en qüestió, entre altres coses, la utilització dels valors teòrics en general, sobretot si es volen determinacions acurades.

15. Differences between lactate concentration of samples from ear lobe and finger tip

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Abstract

Blood lactate concentrations in capillary samples obtained from the ear lobe or from the finger tip are used indistinctly, since they are considered equivalents. The aim of the study reported in this paper was to verify whether that assumption is valid due to the practical implications which any possible differences between these two samplin sites would have in the planning and assessing of an athletic training program. Twenty six healthy male athletes competing in different sports at the national level (9 rowers, 7 cyclists and 10 runners) were studied during the performance of a graded exercise test up to the point of exhaustion, on specific ergometres. In each group, capillary blood samples were obtained simultaneously from both the ear lobe and the finger tip at three different times during the test: 1) in resting conditions 2) when exercising at a submaximal work load and 3) seven minutes after the point of exhaustion. Significant differences were found between the blood lactate concentrations of samples obtained from ear lobe and from finger tip ($P < 0.001$). The method error of repeated measurements for lactate concentrations from paired samples obtained in resting conditions was 27%, when exercising at submaximal work load, 16% and at maximal work load, 3%. Capillary blood samples collected from the finger tip consistenley showed higher values in lactate concentration than those obtained, at the same time, from the ear lobe.

Key word: Blood lactate, Ear lobe sample, finger tip sample.

Resum

Les concentracions de lactat a la sang es poden obtenir a partir de mostres capil·lars del lòbul del pavelló auditiu o de la punta del dit que s'utilitzen indiferentment ja que es consideren equivalents. L'objectiu de l'estudi presentat en aquest article és verificar si aquesta suposició és vàlida per les complicacions pràctiques que qualsevol diferència possible entre aquestes dues zones de mostres tindrien en la planificació i en l'avaluació d'un programa d'entrenament atlètic. Es van estudiar vint-i-sis atletes d'elit (homes) que competeixen en diferents esports a nivell nacional (9 remers, 7 ciclistes i 10 corredors) durant l'actuació en un exercici gradual fins al punt de quedar exhausts sobre ergòmetres específics. En

cada grup, es van obtenir mostres de sang capil·lar de manera simultània del lòbul del pavelló auditiu i de la punta del dit en tres moments diferents del test: 1) en condicions de descans, 2) quan l'exercici estava en el moment màxim de càrrega i 3) set minuts després de quedar exhausts. Es van trobar diferències significatives entre les concentracions de lactat a la sang de les mostres obtingudes del lòbul del pavelló auditiu i les de la punta del dit ($p<0,001$). L'error de mesures repetides per concentracions de lactat de parells de mostres obtingudes en condicions de descans va ser d'un 27%, d'un 16% quan es treballava en càrrega submàximes i d'un 3% en càrregues màximes. Les mostres de sang capil·lar recollides de la punta del dit mostraven de manera consistent valors majors en concentracions de lactat que aquelles obtingudes al mateix temps del lòbul del pavelló auditiu.

Paraules clau: lactat a la sang, mostra del lòbul del pavelló auditiu, mostra de la punta del dit.

Comentari

Sempre s'ha considerat que les mostres sanguínies per a la determinació del lactat donen valors idèntics si s'han obtingut del dit o del pavelló auricular i, per tant, cada grup les obté d'on millor li sembla i compara proves amb determinació del lactat de mostres d'un lloc o de l'altre. Després d'aquest treball, on s'evidencia que el lloc de l'extracció produeix valors diferents, queda prou clar que tan sols es poden comparar proves, valorades mitjançant el lactat, en què les mostres hagin estat obtingudes del mateix lloc.

IV. CONCLUSIONS FINALS

Del contingut de treballs que constitueixen aquesta tesi s'obtenen les següents conclusions:

1. El component genètic de les variables condicionants del rendiment físic pot ser molt diferent d'una variable a un altra, i molt diferent d'un estudi intraracial a un estudi interracial.
2. La determinació d'aquests components genètics ha de permetre la precisió de les expectatives de canvis produïts en una variable determinada per maniobres modificant el component ambiental: exercici, dieta..., ja que en presència d'un IH alt les possibilitats de canvi són menors.
3. Millorar la precisió en les expectatives de canvi mencionades ens ha de permetre saber si un canvi lleuger és degut a la poca qualitat de les mesures dedicades a intentar modificar una variable o, simplement, al gran IH de la variable en qüestió.
4. Tot i que s'ha avançat bastant en la recerca dels marcadors genètics del rendiment físic, la recerca segueix oberta, ja que no hi ha conclusions definitives. A més, s'ha d'anar molt amb compte amb els grups a partir dels quals s'obtenen conclusions, ja que la generalització pot no ser vàlida.
5. Hem millorat en el coneixement del component genètic de la flexibilitat, i a més, hem precisat diferències en el component genètic d'una articulació a altra.
6. Si bé un estudi interracial havia trobat diferències genètiques en l'economia de la cursa, en el nostre estudi intraracial no s'aprecien, suggerint que o bé són molt menors, o que no hi són presents, o que la simplicitat del mètode habitualment emprat per a la seva determinació el fa poc precís.
7. S'aporten força matisos al component genètic de la potència explosiva i del metabolisme anaeròbic durant l'esforç i es confirma el major component d'heretabilitat en les proves de curta durada.
8. Es troben determinades característiques racials, des del punt de vista metabòlic i respiratori, que justifiquen l'excepcional adaptació de l'ètnia xerpa a la gran altitud.
9. S'ha determinat un efecte de fatiga cardíaca, fins ara no conegut.
10. S'han precisat les repercussions de les diferents càrregues de treball i dels períodes de recuperació, amb importants repercussions de caire pràctic.
11. S'han donat aportacions nutricionals que, confirmant conceptes fisiològics, permeten millorar el rendiment.
12. S'aprecien diferències fisiològiques que depenen del clima on es visqui i que de no tenir-se en compte, poden dur a deduccions imprecises.
13. El fet d'haver comprovat les diferents concentracions del lactat en les mostres obtingudes dels llocs més comuns, ens permet evitar errors diagnòstics per comparacions esbiaixades.
14. Com a conclusió final, diríem que és evident la complexitat del rendiment físic, la importància del diferent component genètic dels factors que el condicionen i el gran interés de seguir en el seu estudi, que ens aportarà un interessant aprofundiment científic i resultats de caire pràctic.

V. ANNEXE: TREBALLS ORIGINALS PUBLICATS

Número	Títol	Revista i any	Publicació	Factor d'impacte
1	Could The A2a11 Human leucocyte antigen locus correlate with maximal aerobic power ?	*Clinical Science (1997)*	Internacional	2,271
2	A2a11 Human Leucocyte antigen locus Is not present In athletes with high aerobic power.	*Submitted to the British Journal of Sports Medicine*	Internacional	—
3	Heretabilitat de la flexibilitat: Un estudi realitzat amb germans bessons.	*Apunts. Medicina de l'Esport (1997)*	Nacional	—
4	Heritability of running economy. A study made on twin brothers.	*European Journal of Applied Physiology (1998)*	Internacional	0,983
5	Heritability of the explosive power and anaerobic capacity	*European Journal of Applied Physiology (2001)*	Internacional	0,983
6	Cardiorespiratory response to exercise in elite Sherpa climbers transferred to sea level.	*Medicine and Science In Sports and Exercise (1997)*	Internacional	2,110
7	Normoxic ventilatory response in lowlander and Sherpa elite climbers.	*Respiration Physiology (1998)*	Internacional	1,018
8	The effect of prolonged physical exercise on ventricular function.	*International Journal Sports Cardiology (1992)*	Internacional	—
9	Efectes de l'entrenament anaeròbic en el múscul esquelètic.	*Apunts. Medicina de l'Esport (1998)*	Nacional	—
10	A short training programme for the rapid improvement of both aerobic and anaerobic metabolism.	*European Journal of Applied Physiology (2000)*	Internacional	0,983
11	The distribution of rest periods affects performance and adaptations of energy metabolism induced by high-intensity training in human muscle	*Acta Physiologica Scandinavica (2000)*	Internacional	1,411
12	Assessment methodology on the effectiveness of psychological training	*Actas Proceedings. VIII World Congress of Sport Psychology Lisboa(1993)*	Internacional	—
13	The effect of previous ingestion of glucose or fructose on the performance of an exercise of intermediate duration	*European Journal of Applied Physiology (1994)*	Internacional	0,983
14	Comparison of the basal metabolic rate among champions of two nationalities in olympic and world games.	*Medicine and Science in Sports and Exercise (1999)*	Internacional	2,110
15	Differences between lactate concentration of samples from ear lobe and finger tip	*Journal of Physiology and Biochemistry (1999)*	Internacional	0,385

Clinical Science (1997) 92, 331–333 (Printed in Great Britain)

Rapid Communication

Could the A2A11 human leucocyte antigen locus correlate with maximal aerobic power?

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1. The power of the aerobic metabolic pathway correlates well with successful physical performance in endurance sports events. The ability to alter the pathway through training presents well-known limitations, and consequently a good genetic endowment is essential to participate in elite sporting activities.

2. In 32 subjects (16 healthy pairs of male twin sportsmen, 8 monozygotic and 8 dizygotic) zygosity was determined by means of the genetic analysis of human leucocyte antigen (HLA) system specificities at class I and II loci and other genetic variants. The subjects performed a progressive exercise test on a treadmill to ascertain the maximal oxygen uptake ($\dot{V}O_2$max), measured by an automatic breath-by-breath analyser. We have considered the relationship between the A, B and C loci of the HLA system and $\dot{V}O_2$max.

3. We found a high correlation between the presence of both HLA A2 and A11 and $\dot{V}O_2$max. In the A2A11 group ($n = 6$) we found a $\dot{V}O_2$max (mean $\pm$ SD) equal to 71 ± 4 ml min^{-1} kg^{-1}. The group without this pair of alleles ($n = 26$) showed a much lower aerobic power (58 ± 5 ml min^{-1} kg^{-1}). Differences between the two groups were found to be largely significant ($P < 0.001$). It is noteworthy that in two pairs of dizygotic twins, the higher $\dot{V}O_2$max value corresponded to the twin with the A2A11 allele.

4. The very marked concordance between the presence of the A2A11 locus of the HLA system and the $\dot{V}O_2$max could be of great interest for the identification of outstanding performers.

INTRODUCTION

The power of the aerobic metabolism is usually quantified by means of the maximal oxygen uptake ($\dot{V}O_2$max), which shows a good correlation with performance in endurance sports [1]. The heritability of the $\dot{V}O_2$max has been considered in different studies, although this has always proved to be a source of controversy. The heritability indexes range from 0.84–0.93 [2, 3], to 0.40–0.25 [4, 5]. We have looked for some genetic markers with $\dot{V}O_2$max, in order to identify sportsmen endowed with special capabilities for endurance performance. This communication reports our recent findings.

SUBJECTS AND METHODS

Thirty-two subjects [16 healthy pairs of male twins, 8 monozygotic (MZ) and 8 dizygotic (DZ)], all sportsmen without significant differences in their physical activity, agreed to participate in the study. All subjects regularly practised amateur-competitive sports, mostly team sports such as football and basketball. Zygosity was determined by means of the following genetic analysis: (1) human leucocyte antigen (HLA) class I polymorphism was studied by means of the serologic standard microlymphocytox-

Key words: aerobic metabolism, human leucocyte antigen system, physical performance, twins.

Abbreviations: AcP, acid phosphatase; DZ, dizygotic; HLA, human leucocyte antigen; MZ, monozygotic; PGM1, phosphoglucomutase 1; $\dot{V}O_2$max, maximal oxygen uptake.

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icity test including 60 allelic variants of loci A, B and C [6]; (2) red blood cell antigens (ABO, Rh, MNSs, Duffy, Kidd Lutheran and P systems) were determined by standard methodologies (red cell agglutination or indirect antiglobulin test) and all phenotypes were duplicated by independent workers; (3) the electrophoretic polymorphism was determined in the following plasmatic proteins: transferrin, alfa-1 antitrypsin, group component and haptoglobin [7–9]. Isoelectrofocusing in polyacrylamide gels was employed using different ampholine ranges for each protein. Gels were stained with Coomassie Blue R-250 for alfa-1 antitrypsin, transferrin and haptoglobin. Group component bands were read after simple precipitation with sulphosalicylic acid; (4) enzymic polymorphism: phosphoglucomutase 1 (PGM1) subtypes and acid phosphatase (AcP) phenotypes were determined by isoelectrofocusing in polyacrylamide gels (T5 5%, C 3%) with ampholines pH 5–8 or pH 5–7 for AcP and PGM1 respectively. Isoenzyme visualization was performed according to the method of Sutton and Burgess [10] for PGM1 and Burdett and Whitehead [11] for AcP. Glyoxalate phenotypes were determined by agarose–starch gel electrophoresis with the staining technique described by Parr et al. [12], using the specific-activity technique. Twins were considered MZ when all genetic markers were identical, and DZ when otherwise. DZ twins differed in at least two polymorphic systems.

Each individual performed a progressive exercise test on a treadmill (Jaeger, Germany) until exhaustion. All the stress tests were performed under similar environmental conditions, 3 h after a light breakfast. Pulmonary gas exchange was measured by means of an automatic breath-by-breath analyser (MedGraphics, Sant Paul, MN, U.S.A.). The cardiorespiratory parameters evaluated were continuous heart rate (beats/min) recorded by a modified V5 precordial lead, oxygen uptake ($\dot{V}O_2$, ml/min, standard temperature pressure dry) and $\dot{V}O_2$ related to body mass (ml min^{-1} kg^{-1}, standard temperature pressure dry).

Data were analysed using two-way analysis of variance with repeated measures. The null hypothesis was rejected for $P<0.05$.

The work has been approved by the ethical Committee of the University of Barcelona and consent was obtained from all participants following careful explanation of the aims and processes involved.

RESULTS

The HLA specificities and the $\dot{V}O_2max$ values are shown in Table 1. We found a high correlation between the presence of both antigens, HLA A2 and A11, and $\dot{V}O_2max$. All subjects were classified in two subgroups, one with A2A11 and the other without. In the A2A11 group ($n=6$) we found a $\dot{V}O_2max$ (mean $\pm$ SD) equal to 71 ± 4 ml min^{-1} kg^{-1}. By contrast, the group without this pair of alleles ($n=26$) showed a much lower aerobic power (58 ± 5 ml min^{-1} kg^{-1}). The differences between the two groups were found to be largely significant ($P<0.001$). It is noteworthy that in two pairs of DZ twins, in each of which only one pair-member had the A2A11 phenotype (pair ten: $\dot{V}O_2max = 60$ and 67 ml min^{-1} kg^{-1}; pair eleven: $\dot{V}O_2max = 72$ and 65 ml min^{-1} kg^{-1}), the highest values were shown by subjects having the A2A11 alleles. No other genetic markers correlated in any way with the $\dot{V}O_2max$ performance. No inter-group differences between A2A11 and non-A2A11 groups were

Table 1. HLA system specificities and VO_2max for each subject (individual values are given for each twin in a pair)

Monozygotic			Dizygotic		
Twin	HLA system	$\dot{V}O_2max$ (ml min^{-1} kg^{-1})	Twin	HLA system (ml min^{-1} kg^{-1})	$\dot{V}O_2max$
1	A2,A11	70.4	9	A2,A32	63.1
	A2,A11	69.3		A2,A29	67.5
2	A9,A29	51.7	10	A2,A28	60,0
	A9,A29	57.3		A2,A11	67.0
3	A1,A2	57.0	11	A2,A28	65.0
	A1,A2	57.7		A2,A11	72.0
4	A29,A33	58.0	12	A2,A24	69.0
	A29,A33	54.6		A2,*	49.4
5	A2,A3	59.2	13	A2,A3	47.0
	A2,A3	64.5		A11,*	52.0
6	A2,A11	73.7	14	A3,A28	36.0
	A2,A11	73.2		A2,A3	46.0
7	A29,*	64.5	15	A11,A30	44.0
	A29,*	61.4		A11,A23	50.0
8	A1,A23	50.5	16	A3,A29	61.0
	A1,A23	61.0		A3,A29	59.3

*Antigen not defined.

observed in maximal heart rate during the exercise test, nor were statistical differences found for either weekly training volume or for the period during which the sport had been practised competitively.

DISCUSSION

Given that the training process can improve the $\dot{V}O_2$max up to an average of 20–30% [13], the capacity to predict this high endurance is of major importance in the detection of sports talents capable of achieving high competitive levels. The $\dot{V}O_2$max value is determined by the level of physical activity performed by the subject, although the degree of improvement of this value is strongly conditioned by individual characteristics [4], and thus is only an orientative value. Although several studies have tried to identify a genetic trait of heritability associated with endurance, the results obtained thus far are highly contradictory. In this study a clear association between $\dot{V}O_2$max and the presence of phenotype HLA A2A11 was observed, both in MZ and DZ twins. The HLA complex is located in chromosome 6 (q21) and locus A is its most telomeric marker. This leads us to assume that the antigens A2 and A11 are markers for another gene, in linkage disequilibrium, which has not yet been identified.

Further studies are necessary to confirm this preliminary data. At present we are beginning a study of A2A11 locus presence in a group of top-class national endurance athletes, and a molecular study of the genes located at the extreme of the flanking region of locus A would also be necessary. The apparent disagreement with some recent work [4, 5], in which low heritability for $\dot{V}O_2$max was observed, is probably due to the fact that the proportion of people having the A2A11 locus is rather low. All the subjects studied in this investigation were sportsmen and we cannot therefore exclude that the high $\dot{V}O_2$max observed in the individuals showing the A2A11 locus, in addition to being determined by genetic endowment, is further differentiated by a higher trainability, which is more genetically conditioned than initial fitness level [5]. The present findings suggest that the presence of the A2A11 locus could be a useful marker to predict high endurance in human performance.

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Short Report

A2 A11 human leucocyte antigen locus is not present in athletes with high aerobic power

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Short title: HLA system and $\dot{V}O_2$ max

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ABSTRACT. Aim. To correlate the presence of A2,A11 antigens in locus A of the human leucocyte antigen (HLA) and high maximal aerobic power ($\dot{V}O_2$ max) in athletes.

Method. Nine top-class national endurance athletes who achieved a $\dot{V}O_2$ max near or above 70 $ml \cdot min^{-1} \cdot kg^{-1}$ in a maximal treadmill stress test were selected with the aim to determine HLA class I polymorphism by means of the serologic standard microlymphocytotoxicity test.

Results. None of athletes studied showed both A2, A11 antigens, while 5 of them (55%) were probably homozygous for HLA A2 locus, higher than expected according to Hardy Weinberg's law.

Conclusion. The absence of the A2,A11 antigens of the HLA system in this sample of high aerobic power athletes suggests that it can not be of use as a marker to detect potential endurance in humans but that perhaps it is related to other HLA haplotypes, mostly HLA A2, which is present in double dose in individuals with high $\dot{V}O_2$ max.

KEY WORDS: *maximal oxygen uptake, human leucocyte antigen system, top-class athletes, genetic variation, short arm of chromosome 6*

INTRODUCTION

So far there have been no reports of a specific genetic factor that strongly influences human aerobic capacity, but recently some studies are searching for a possible correlation between genetics and performance[1,2]. The first study found a correlation in the gene encoding angiotensin-converting enzyme with performance at high-altitude and with results in weight-lifting training and the second study found a lack of difference in the NADH dehydrogenase gene of mitochondrial DNA between elite endurance athletes and sedentary controls.

In a previous study performed in twin brothers, we observed a very marked concordance between the presence of A2, A11 antigens in locus A of the human leucocyte antigen (HLA) system and maximal aerobic power ($\dot{V}O_2$ max). The average value was equal to 71 (SD 4) $ml \cdot min^{-1} \cdot kg^{-1}$ in the 6 subjects with the A2,A11 antigens and 58 (SD 5) $ml \cdot min^{-1} \cdot kg^{-1}$. in the 26 subjects without them[3].

According to this data, we have tried to determine whether the heterozygous A2, A11 in locus A could be a good marker for the identification of outstanding performers, by observing the presence or not of this locus in a group of top-class national endurance athletes.

METHODS

$\dot{V}O_2$ max was measured in 9 male top-class national endurance athletes, aged 28 (SD 6) years measuring 175 (SD 5) cm in height, and with 68 (SD 4) kg of body mass.

Pulmonary gas exchange was obtained by means of an automatic breath-by-breath analyser (Medgraphics, Sant Paul, MN, USA) throughout a progressive stress test on a treadmill (Jaeger, Germany) until exhaustion. The presence or absence of the A2 and A11 antigens of the HLA system was determined by means of the serologic standard microlymphocytoxicity test including 60 allelic variants of loci A, B and C.[4]

The study was approved by the ethical Comittee of the University of Barcelona and consent was obtained from the participants following careful explanation of the aims and processes involved.

RESULTS

The average $\dot{V}O_2$ max of the group was 73 (SD 4) $ml \cdot min^{-1} \cdot kg^{-1}$. The A2 antigen was observed in 5 athletes (55%), 3 of which were probably homozygous. One athlete carried the A11 antigen, but none of them were heterozygous for both A2 and A11.

DISCUSSION

The $\dot{V}O_2$ max related to body mass was high, in accordance with the high level of endurance performance of the selected group, but no correlation was observed with the presence or absence of the A2, A11 antigens of HLA. This fact excluded, thereby, the possibility of using this parameter as a genetic marker to detect potential high-endurance in humans.

Five individuals (55%) were probably homozygous for the HLA A2 locus, a prevalence higher than expected according to Hardy Weinberg's law. This suggests that a gene located in the short arm of chromosome 6, in linkage disequilibrium with the locus A, could be related to high VO_2 max., similary to the recent finding of a candidate gene (HLA-H) causing genetic haemochromatosis and located in the same short arm of chromosome 6, close to the HLA class I region[5]. In monozygotic twins this locus is in the same chromosome as the HLA genes, but in random population this gene could be associated with other HLA haplotypes, mostly HLA A2, which is present in double dose in individuals with high $\dot{V}O_2$ max.

Nevertheless, further work will be needed to determine whether this hypothesis holds beyond the limited group studied here and to explore the mechanisms underlying these observations. According to our findings, however, if we want to look for genes located in the proximity of the HLA system (probably codifiers of the enzymes related with the muscle oxidative metabolism) what we should do is map the locus A telomeric region situated in the p 21. 3 region of the 6 chromosome.

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Heretabilitat de la flexibilitat: un estudi fet amb germans bessons

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ABSTRACT. Flexibility (FL) defined as the scope of movement of one or several joints is a factor of sport performance, which is determined by age, gender, training, anthropomorphic characteristics and genetic factors, of which very little has been studied. METHOD: To establish the relative importance of genetic and environmental influence on flexibility, we have studied the genetic component, using the Hereditability Index (HI at a value of 1.0 indicates that 100% of the variation would be due to genetic variation) in 24 sportsmen split into 12 pairs of twin brothers (6 monozygotes and 6 dizygotes), who did not have any significant perinatal or environmental differences. Zygosity was determined by genetic analysis of the leukocyte human antigen system (LHA), especially the Class I and II loci, and other genetic variants. To calculate the FL, the subjects underwent an indirect flexometric test for the trigonometric calculation of the angle of aperture of the scapulohumeral (FL, SHA) and coxofemoral joints (FL, CFA), the l atter broken down into the horizontal and sagittal fields, for active and forced passive movements and for both legs. RESULTS: We do not find significant differences in the age and anthropomorphic measurements between the two groups (MZ and DZ). The HIs which were statistically significant for $F_{6,6}$ (DZ MZ-1) over 4.2 and $P<0.05$, are the FL, CFA in the transversal plane for open l egs and active (0.82) and forced passive (0.77) movement, and in the sagittal plane for the right leg in active movement (0.78). CONCLUSION: We conclude that the genetic influence of flexibility is substantial, especially of the coxofemoral joint (75%) and mainly of the right leg, and our study corroborates the previous results yielded by family studies, at least for the target public of our research.

KEY WORDS: Heriditability, Flexibility, Twin Brothers.

RESUMEN: ANTECEDENTS: La flexibilitat (FL) definida com el rang d'amplitud de moviment d'una o vàries articulacions, és un factor del rendiment esportiu que ve determinat per l'edat, el sexe, l'entrenament, per característiques antropomètriques i per factors genètics dels quals, s'ha estudiat molt poc. MÈTODE: Per esbrinar la importància relativa de la influencia genètica i ambiental en la flexibilitat, hem estudiat el component genètic utilitzant l'índex d'heretabilitat (IH = un valor de 1.0 indica que el 100% de la variació seria deguda a variació genètica) a 24 subjectes barons i esportistes dividits en 12 parelles de germans bessons –6 monozygots i 6 dizigots –, que no presentaven diferències perinatals ni ambientals significatives. La zigositat es va determinar mitjançant l'anàlisi genètic del sistema antigènic humà leucocitari (HLA), específicament els locus de la classe I i II, i altres variants genètiques. Pel càlcul de la FL els subjectes vam realitzar un test flexomètric indirecte pel càlcul trigonomètric de l'angle d'obertura de les articulacions escàpulohumeral (FL, AEH) i coxofemoral (FL, ACF), aquesta darrera desglossada en el camp horitzontal i sagital , per moviments actius i passius forçats i per amdues cames. RESULTATS: No trobem diferències significatives en l'edat i les mesures antropomètriques entre els dos grups (MZ i DZ). Els IH que van ser estadísticament significatius per F6, 6 (DZ˙MZ–1) superior a 4, 2 i P<0.05, son el FL, ACF en el plà transversal per cames obertes i moviment actiu (0.82) i passiu forçat (0.77) i en el plà sagital per la cama dreta en moviment actiu (0.78)
CONCLUSIONS: Nosaltres concluïm que la influència genètica de la flexibilitat es substancial, sobretot de l'articulació coxofemoral (75%) i preferentment de la cama dreta, i que al menys per la població que hem estudiat el nostre estudi corrobora els resultats anteriors fet amb estudis familiars.

PARAULES CLAU: Heretabilitat, Flexibilitat, Germans bessons.

INTRODUCCIÓ

L'heretabilitat de les característiques morfofuncionals que contribueixen a un alt rendiment esportiu ha estat àmpliament estudiat. La contribució del efecte genètic relatiu a la variabilitat del fenotip ha estat estudiat mitjançant estudis familiars, estudis amb germans bessons o per dades extenses de descendència o adopcions.[1]

En els estudis amb germans bessons el procediment més utilitzat és el càlcul de l'índex d'heretabilitat (IH) que és un valor numèric que determina la proporció de la variació atribuïble a factor genètic, i així un valor de 1.0 indica que el 100% de la variació seria deguda a variació genètica. Aquest índex s'ha determinat per varis components del rendiment esportiu així com per factors salut-depenents mitjançant diferents procediments matemàtics[2, 3, 4, 5] però els resultats han estat força contradictoris, probablement pel fet de treballar en mostres poc homogènies: sexes diferents, edats diferentes, activitats física diferent, o per que el mètode estadístic per valorar el fenotip no ha estat el més adequat.[6]

La flexibilitat (FL) ha estat definida per el rang d'amplitud de moviment d'una o vàries articulacions,[7] és a dir per moure's fins uns límits naturals. Aquests els constitueixen, la disposició òssia de l'articulació, els lligaments que el reforcen, la càpsula fibrosa, i els músculs i tendons que la creuen.[8] Es un factor del rendiment esportiu que ve determinat per l'edat, el sexe, l'entrenament, per característiques antropomètriques i per factors genètics dels quals, s'ha estudiat molt poc.

Els únics estudis que hem trobat que han estudiat el component genètic de la FL han estat els realitzats per Devor and Crawford (1984),[9] Perusse LC i cols (1988)[10] i Maes H i cols (1996)[11] on determinen en estudis de correlacions familiars –i al darrer també amb germans bessons– uns valors d'heretabilitat de la FL moderadament alts, si bé força dispersos, de 0.35 a 0.85. Son estudis familiars que treballen principalment en mostres molt grans, amb ambdós sexes, en edats infantils, i utilitzant el test de flexió del tronc, el test *sit and reach* de la bateria Eurofit[12] com a valoració de la flexibilitat, i amb diferents anàlisis estadístics.

L'objectiu d'aquest estudi ha estat determinar la diferent contribució dels factors ambientals i genètics de la flexibilitat, seleccionant una mostra suficientment homogènia, un mètode de valoració actualitzat i fiable, i la utilització d'un procediment estadístic adequat. Amb aquest propòsit hem avaluat l'índex d'heretabilitat de la flexibilitat de l'articulació coxofemoral i escàpulohumeral, analitzant una mostra de bessons homes esportistes amb idèntics antecedents familiars i personals i d' edats entre 17 i 30 anys.

MATERIAL I MÈTODE

Un grup de 12 parelles de bessons homes sans, 6 monozygots (MZ) i 6 dizigots (DZ), tots ells esportistes sense diferències significatives en la seva activitat física, han estat d'acord a participar en aquest estudi. Tots els subjectes han practicat regularment exercici físic d'una forma amateur, el bàsquet o el futbol. Les seves principals característiques són (mitjana i DS), els MZ, edat: 22 (DS 2); pes:76 (DS 7); talla: 174 (DS 6) i els DZ, edat: 22 (DS 3), pes: 73 (DS 8) i talla: 174 (DS 8), no havent diferències estadísticament significatives entre els dos grups.

Determinació de la zigossitat

Es va determinar la zigossitat mitjançant l'anàlisi genètica que segueix l'Hospital Clínic de Barcelona per l'estudi de la paternitat i que es el següent:

1) Tipatge serològic del sistema HLA, classe I mitjançant tècnica de microlinfocitotoxicitat, incloent 60 variants al·lèlics dels locus A, B and C.[13]
2) Sistemes antigènics eritrocitaris (ABO, Rh, MNSs, Duffy, Kidd Lutheran i sistema P), que van ser determinats mitjançant tècnica standard d'aglutinació i test d'antiglobulina.[14] Tots els fenotips van ser duplicats i tractats per treballadors independents;
3) Polimorfisme protèic, que va ser determinat perl·les següents proteïnes plasmàtiques: transferrina (Tf), alfa-1 antitripsina (Pi), component de grup (Gc) i haptoglobina (Hp).[15, 16, 17] La Isoelectrofocus en gels que va ser utilitzat seguint diferents rangs de amofilines per cada proteïna. Els gels van ser tenyits amb blau de coomassie R-250 per Pi, Tf and Hp. Les bandes del component de grup van ser llegides després d'una simple precipitació amb àcid sulfosalicíac;
4) El polimorfisme enzymàtic: el fenotip dels subtipus de la fosfoglucomutasa 1 (PGM1) i fosfatasa àcida (AcP) van ser determinats per isoelectrofocusing en gels de poliacrilamide (T5 5%, C 3%) amb ampholines pH 5-8 o pH 5-7 per AcP i PGM1 respectivament. La visualització isoenzymàtica va ser realitzada d'acord a Sutton & Burgess (1978)[18] per la PGM1 i Burdett & Whitehead (1977)[19] per AcP. Els fenotips de la glycoxalasa (GLO) van ser determinats per electroforesi en un gel d' agarosa-starch mitjançant la tècnica descrita per Parr i cols (1977),[20] utilitzant tècnica d'activitat específica. Es va

considerar que els germans bessons eren MZ quan tots els marcadors antigènics eren idèntics, i DZ quan no. Els germans bessons DZ diferien almenys en dos sistemes polimòrfics.

Antropometria

Vam utilitzar una balança seca ajustada fins a 100 gr., un tallimetre i un antropòmetre (Atlantida, Barcelona), un bastó de fusta graduat de 0 a 120 cm i 25 mm de diàmetre i un flexòmetre (flexómetre, Barcelona).

Test flexomètric

Si be existeixen diferentes formes de valorar la flexibilitat,[21, 22] nosaltres vam utilitzar el test flexomètric de Moras i Torres (1989)[23] i Moras G (1992)[24] per ser actualment dels més vàlids i reproduibles, i que es basa en el càlcul trigonomètric de l'angle d'obertura d'un segment corporal.

Per tal de ser molt precisos a l'hora de passar els tests els individus varen realitzar les proves sense escalfament previ específic, descalços, i es va tenint molta cura de que la roba no entorpís el moviment. Totes les mesures les va realitzar sempre la mateixa persona i al costat dret. Per a mesurar la llargada de la cama (Lc) i del braç (Lb) es va seguir la tècnica de Torres i Moras (1990)[25] i per les mesures del diàmetre biacromial (da) i diàmetre bitrocanteri (dt) la tècnica de Ross i cols (1978).[26]

Càlcul de variables

Flexibilitat de l'articulació escapulohumeral (FL, AEH)

Consisteix amb un gir d'espatlles amb bastó (Fig. 1.1). El subjecte agafa amb les dues mans la pica i per assaig i error (procurar no fer més de cinc intents) es determina la mínima distància de separació de braços amb la qual es pot portar la pica des de la part anterior fins a la part posterior del cos, mantenint sempre els braços estirats.

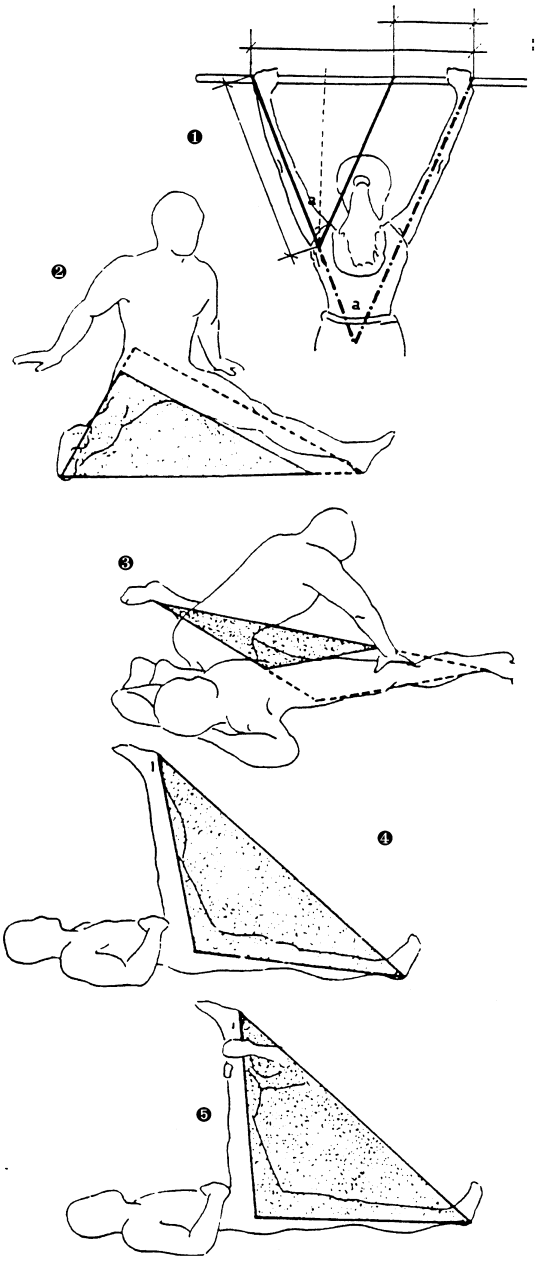
Cal que tots els dits de les mans no perdin contacte amb la pica al fer el moviment. Així mateix, es permet que el subjecte doblegui els braços per tornar a la posició inicial un cop realitzi el moviment. Es determina la distancia de separació entre els dos cantons externs del cinquè metacarpià (ds) i es calcula la flexibilitat de l'articulació escapulohumeral mitjançant la fórmula: FL, AEH = 2arcsin (ds-da)/2Lb.

Flexibilitat de l'articulació coxofemoral (FL, ACF)

Consisteix amb l'abducció de les extremitats inferiors, en el pla transversal i sagital:

Figura 1

❶: esquema de la posició per a la valoració de la flexibilitat de l'articulació escapulohumeral (FL, AEH).
❷: per l'articulació coxofemoral (FL, ACF1) en cames obertes i moviment actiu;
❸: FL, ACF2 en cames obertes passiu forçat;
❹: FL, ACF3, 5 per cama dreta i esquerra actiu;
❺ FL, ACF4, 6 : per cadascuna de les cames passiu i forçat.



I. En el plà transversal es realitza la màxima separació de les cames en :

MOVIMENT ACTIU LLIURE: El subjecte, assegut al terra de manera perpendicular, intenta separar el màxim les cames en un sol moviment i sense rebots (Fig. 1.2). Determinen la distància de separació entre els dos cantons externs del calcani (ds). Així calculem el FL, ACF1 = 2arcsin (ds-da)/2Lc.

MOVIMENT PASSIU FORÇAT: El subjecte, amb l'esquena recolzada al sol i les cames rectes i perpendiculars al mateix, es relaxa al mateix temps que l'ajudant tracciona les dues cames, separant-les fins que l'executor arriba al límit articular no dolorós (Fig 1.3). Es determina la màxima distància de separació entre els dos cantons externs del calcani (ds). Així es calcula la flexibilitat de FL, ACF 2= 2arcsin (ds-da)/2Lc

II. En el pla sagital, es realitza una anteversió de la cama des de decúbit supí.

MOVIMENT ACTIU LLIURE: El subjecte, en decúbit supí, intenta elevar al màxim una cama mantenint el cos i l'altre cama enganxada al sòl (Fig 1.4). Cal evitar moviments ràpids o balístics. Es determina la màxima distància assolida entre ambdós extrems externs dels calcanis (ds) i mitjançant la fórmula = 2arc sin ds/2Lc, calculem per la cama dreta (FL, ACF3) i l'esquerra (FL, ACF5).

MOVIMENT PASSIU FORÇAT: El subjecte en decúbit supí es relaxa al mateix temps que l'ajudant eleva al màxim una cama fins que l'executor arriba al límit articular no dolorós . En tot moment el subjecte ha de mantenir el cos i la cama no mobilitzada en contacte amb el sol (Fig. 1.5). Cal elevar la cama amb lentitud per evitar l'activació del reflex miotàtic. Es determina la màxima distància assolida entre ambdós extrems externs dels calcanis (ds) i mitjançant la fórmula = 2arc sin ds/2Lc, calculem per la cama dreta (FL, ACF4) i l'esquerra (FL, ACF6).

ANÀLISI ESTADÍSTICA

Les dades obtingudes han estat analitzades mitjançant models ANOVA en les quals s'han considerat com a variables observades les mesures de flexibilitat i com a causes de variació, el tipus genètic (MZ i DZ) i el factor parella (sis parelles dins de cada tipus genètic). Ens resulta doncs, un model ANOVA a dos factors on el factor parella està jerarquitzat en el factor tipus genètic. Com a resultat del mateix vàrem decidir si existien o no diferències entre els tipus genètics considerats, i si la variabilitat entre parelles resultava significativa estadísticament. En cas de no obtenir diferències globals entre

Taula 1

Mitjana i desviació estàndard de; Lc, longitud de cama; Lb, longitud de braç; Da, diàmetre biacromial; Dt, diámetro bitrocanteri; FLAEH: Flexibilitat (FL) articulació escàpulo-humeral; FL, ACF1, FL articulació coxo-femoral (ACF) en cames obertes i moviment actiu; FL, ACF2, FL en cames obertes passiu forçat; FL, ACF3, FL cama dreta activa; FL, ACF4 de la cama dreta passiva forçada; FL, ACF5 de la cama esquerra activa; FL, ACF6, cama esquerra passiva i forçada. MZ: Monozigots, DZ: Dizigots; F ratio DZ•MZ-1 : F6, 6 > 4.2 diferències significatives; IH: valor de l'Índex de heretabilitat; * significació estadística per $P < 0.05$.

	DZ Mitjana (DS)	MZ	IH Mitjana (DS)	F ratio DZ-MZ^{-1}
Lc (cm)	90.7 (3)	92.1 (5)		
Lb (cm)	64.8 (3)	66.6 (3)		
Da (cm)	41.5 (2)	41.1 (1)		
Dt (cm)	34.1 (1)	33.8 (1)		
FL, AEH	57.1 (23)	56.2 (21)	0.54	2.21
FL, ACF1	75 (15)	73 (12)	0.82*	5.76
FL, ACF2	92.5 (11)	84.4 (13)	0.77*	4.35
FL, ACF3	82.5 (11)	84.4 (10)	0.78*	4.66
FL, ACF4	100.8 (20)	95 (13)	0.58	2.41
FL, ACF5	82.5 (11)	82.5 (10)	0.49	1.85
FL, ACF6	108 (17)	92 (12)	0.60	1.42

els dos tipus genètics (no significació de l'ANOVA anteriorment descrit), es va dissenyar un model ANOVA per a cada tipus genètic, considerant com a única causa de variació el factor parella. La variança residual de cada un dels ANOVES anteriors serà l'estimació de la variabilitat pel tipus genètic considerat i per tant serà el valor utilitzat en el càlcul del index d'heretabilitat. Aquest es pot entendre com el coeficient de les diferències entre la variància residual de DZ i MZ respecta la variància residual de DZ. Aquest paràmetre pot entendres com el percentatge de la variabilitat intrínseca, depenent únicament del grup genètic considerat. Les diferències interparella entre MZ i DZ van ser considerades estadísticament significatives per $P < 0.05$, i la variabilitat del IH va ser considerat estadísticament significatiu quan $F_{6, 6}$ va ser superior a 4.3 i $P < 0.05$. L'objectiu d'aquesta metodologia es separar els components genètics i ambientals de l'error experimental de la mesura ja que la varianza no controlada ha de ser similar en ambdós grups de bessons.

RESULTATS

En la taula I exposem les dades (mitjans i DS) de les mesures antropomètriques realitzades, els resultats de la FL segons els càlculs trigonomètrics i l'IH amb la seva significació estadística. No van existir diferències significatives ($P < 0.05$) de les mesures antropomètriques ni dels càlculs de la FL entre els dos grups (MZ i DZ).. Respecta els IH, vam consider-los estadísticament significatius quan $F_{6,6} > 4.2$ i $P < 0.05$. A més trobem que:

I. La FL de l'articulació escapulohumeral presenta una menor heretabilitat que l'articulació coxofemoral, en el plà transversal amb un IH de 0.82 pel moviment actiu i 0.78 pel passiu i forçat, ambdós estadísticament significatius.
II. Que en la articulació coxofemoral existeix un component genètic més determinant per la cama dreta (FL, ACF3 = 0.78 i FL, ACF4 = 0.58) que per la esquerra (FL, ACF5 = 0.49; FL, ACF6 = 0, 59), si bé tan sols és estadisticament significatiu el FL, ACF3.
III. Que per l'articulació coxofemoral existeix una major heretabilitat per els moviments actius (FL, ACF 1, 3, 5) que pels passius forçats (FL, ACF 2, 4, 6).

DISCUSSIÓ

Després d'avaluar diferents procediments i tècniques comunment utilitzats per quantificar la contribució del factor genètic i ambiental en la variació d'una qualitat física vam trobar que els estudis de correlació familiar mitjançant models genètics de descendencia d'un fenotip de pares a fills eren molt costosos i menys fiables que els realitzats en bessons[2, 3, 4, 5, 27, 28] que si bé han estat també criticats per alguns autors,[29, 30, 31] segueixen sent un dels més efectius en diferenciar la importància relativa del component genètic respecte l'ambiental, i segueix sent la base d'importants estudis realitzats per importants grups de científics.[32, 33, 34, 35, 36]

D'altra banda existeixen nombroses proves per tal de valorar la flexibilitat, si bé poques han estat correctament validades i acceptades.[21, 22, 24] Nosaltres hem escollit un test que utilitza un flexòmetre, semblant el goniòmetre, que permet medir l'amplitud de qualsevol moviment articular i el càlcul trigonomètric de l'angle d'obertura.[23, 24.]

Els estudis fets fins ara per analitzar el component genètic de la flexibilitat[10, 11, 12] han trobat una heretabilitat de moderada a alta (0.38-0.85) i per tant sembla ser una qualitat molt inherent i per tant no molt modificable per factors ambientals com l'entrenament. Altres qualitats com la potència i la resistència aeròbica, si bé els estudis mostren una alta variabilitat, troben una heritabilitat inferior (< 0.50)[6, 37] i en canvi per la força estàtica i explossiva sembla existir també una forta determinació genètica (0.60 i 0.90).[27, 28.]

El que és comú a tots els treballs que estudien la contribució de factors genetics en la FL,[9, 10, 11] es generalitzar les conclusions per tots els segments corporals mitjançant l'estudi del càlcul de la flexibilitat del tronc amb el test de *sit and reach* de la Bateria Eurofit, que si bé és un test molt reproduible i senzill per realitzar a grans poblacions[38] creiem que presenta importants limitacions en la interpretació dels resultats ja que en la flexió del tronc intervenen més factors com l'elasticitat de la musculatura isquiotibial, dels m. flexors del maluc, dels component càpsulolligamentós de l'articulació coxofemorall i lumbosacra etc.

Els valors de l'heretabilitat de la FL que troba Maes i cols (1996)[9] mitjançant aquest test en un grup de germans bessons, nois i noies de 10 anys és de 0.38 per nois i 0.50 en noies, i mitjançant l'estudi familiar amb pares i fills troba un 0.72 per homes i 0.51 per dones. Aquest valors son semblants (0.48 i 0.66) els que troben en els estudis familiars Devor i Crawford (1984) en ucranians inmigrants a Kansas i Perrusse i cols (1984) amb població canadenca. Aquests valors son lleugerament inferiors els valors que trobem nosaltres per l'articulació femoral, i semblants els que trobem per l'articulació escàpulohumeral. Això pot explicar-se per varis factors:

I. Pel test *sit and reach* que utilitzan ells, i que per tant no valora exactement l'articulació coxofemoral
II. L'edat, en el cas del treball de Maes i cols (1986) son nens de 10 anys. i l'heretabilitat es baixa comparada a nosaltres, potser perquè les diferències de FL és fa més evidents després de la pubertad i per altre banda la flexibilitat es una qualitat involutiva que a partir dels 30 anys si no s'entrena es perd notablement, i per tant la valoració que es faci en els estudis familiars als pares pot també esta molt esbiaixada.
III. Per altre banda sempre s'estudiat l' heretabilitat a partir del càlcul de la flexió del tronc, pero mai de cap altra articulació. Nosaltres estudiem la de l' articulació escàpulohumeral i trobem valors per sota de la coxofemoral. Els valors més baixos que en la coxofemoral podrien ser atribuïble a que existeix un major procés d'entrenament de AEH (són jugadors de bàsquet i futbol), a més que és una articulació amb més graus de llibertat i menys limitacions estructurals. Per tant és lògic trobar valors mes baixos d 'heretabilitat de l'articulació escapulohumeral, ja que el factor ambiental ha de haber influït més que en els nens dels anteriors estudis.

Respecte a les diferències que trobem en les diferents valoracions en moviments passiu i actiu i cama dreta i esquerra, podem dir que:

1. Tots els tests basats en moviments actius-lliures mostren una dispersió més petita perquè són més precisos en determinar el límit del moviment articular, ja que no depenen tant de les sensacions del dolor i dels reflexos corresponents. Par altra banda existeix una major heretabilitat per els moviments actius (FL, ACF 1, 3, 5) que pels passius forçats (FL, ACF 2, 4, 6) ja que en els forçats afegim un altre component al factor ambiental, en part incontrolable, com és el instructor que modifica l'amplitud de moviment.
2. Les diferències més grans trobades en manipular la cama esquerra poden explicar-se si pensem que totes les parelles explorades tenien com a cama dominant la dreta i, per això, podem pensar que l'esquerra disposa d'un pitjor control i regulació del moviment, el qual es reflecteix en tots els factors neuromusculars associats a aquest (reflex miotàtic, receptors del dolor, ...). Per altra banda veiem que existeix un component genètic més determinant per la cama dreta (FL, ACF3 = 0.78 i FL, ACF4 = 0.58) que per la esquerra (FL, ACF5 = 0.49; FL, ACF6 = 0, 59) - si bé tan sols és estadísticament significatiu el FL, ACF3 - pel fet que la cama esquerra és menys treballada generalment i la seva flexibilitat vindrà més determinada per l'entrenament específic que hagi realitzat cada subjecte.

En resum, la flexibilitat és una capacitat o qualitat que presenta un component heretable moderadament -alt, i especialment l'articulació coxofemoral, i per tal de mantenir uns nivells òptims, aquells esportistes que vulguin mantenir un nivell òptim per tal de millorar el seu rendiment o disminuir les incidències lesionals que es poden derivar, haurà de seguir un programa d'entrenament periòdic i correcte. Quan naixem disposem d'uns valors elevats de flexibilitat, que s'aniran perdent progressivament si no es segueixen programes concrets de treball.

CONCLUSIONS

L'efecte del factor genètic pel que fa a la flexibilitat de l'home sembla ser força determinant. Això demostra que per aquelles disciplines esportives on la flexibilitat és fonamental, el genotip hi juga un paper molt important.

Tot i que la flexibilitat és una capacitat física que es pot desenvolupar força -en un sentit relatiu- mitjançant l'entrenament orientat, és clar que els esportistes que parteixin d'uns índexs més alts de la mateixa (potencial genètic), tant a nivell quantitatiu com quantitatiu, podran mantenir més fàcilment una flexibilitat elevada.

El problema de l'entrenament esportiu, pel que fa al factor flexibilitat, probablement no es troba només en el control de les càrregues, en els processos tècnics o tècnicotàctics, sinó en la limitació genètica amb la qual tothom neix, i que en molts casos pot impossibilitar assolir valors òptims per a la competició o provocar una major incidència de lesions esportistes.

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ORIGINAL ARTICLE

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Heritability of running economy: a study made on twin brothers

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Abstract Running economy (RE), defined as the steady-state of oxygen uptake ($\dot{V}O_2$) for a given running velocity, is a factor of sports performance the genetic component of which has seldom been reported to date. We studied this component using a heritability index (HI) in a group of 32 male twins, 8 monozygotic (MZ) and 8 dizygotic (DZ) pairs, all sportsmen with similar perinatal and environmental backgrounds. Zygocity was determined by the identity of erythrocytic antigenic, protein and enzymatic polymorphism, and human leucocyte antigen serologic types between co-twins. The subjects exercised twice on a treadmill, once until exhaustion and again at submaximal intensities. Pulmonary gas exchange was measured continuously using an automatic analyser system during both tests. Blood samples were obtained during the recovery period to determine lactate concentrations. No significant differences were observed between MZ and DZ, in respect of RE at any speed or in maximal $\dot{V}O_2$ relative to body mass. Nevertheless, significant HI ($P < 0.05$) was found in maximal lactate concentrations (HI = 0.75) and in respiratory equivalent for oxygen at two speeds, 7 km · h^{-1} (HI = 0.71) and 8 km · h^{-1} (HI = 0.79), differences which probably suggest that there are differences in RE. In conclusion, we did not detect a genetic component in RE or in maximal oxygen uptake, but a genetic component for markers of anaerobic metabolism was present.

Key words Aerobic metabolism · Genetic endowment · Oxygen uptake · Energy cost · Twins and exercise

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Introduction

It has been suggested that running economy (RE) may be defined by the rate of oxygen uptake ($\dot{V}O_2$) at a steady state at a submaximal running speed (Conley and Krahenbuhl 1980; Daniels 1985), and this would imply that lactate does not accumulate progressively. However, lactate concentrations can increase above rest values contributing extra energy – above the aerobic supply – which is not usually taken into account. This has been shown to be important in sports performance, especially in long distance running, where it plays a significant role (Morgan et al. 1989; Morgan and Daniels 1994). Nevertheless, studies have shown that RE may differ among individuals of the same level of athletic ability (Conley and Krahenbuhl 1980) and may not necessarily show a high correlation with sports performance (Ramsbottom et al. 1987) which is a multifactorial process. Higher values in other parameters may compensate for low values of RE or vice versa (see Daniels 1985).

Heritability indexes (HI, that is numerical values for the proportion of the population variation attributable to genetic variation) for different components of sports performance have been calculated by different equations (Holzinger 1929; Clark 1956; Klissouras 1971; Christian 1979) but contradictory data have been obtained, which have probably been due either to small and not sufficiently homogenous samples of subjects, or to the statistical methods used in the studies (Bouchard 1992). The most studied factor to date has been maximal oxygen uptake ($\dot{V}O_{2\,max}$), although this has always proved

to be a source of controversy. The HI (a value of 1.0 indicates that all of the population variation is attributable to genetic variation) have ranged from 0.84–0.93 (Klissouras 1971; Pirnay and Crielaard 1983), to 0.40–0.25 (Bouchard et al. 1986; Bouchard 1992), and anaerobic power from 0.50 to 0.80 (Weiss 1977; Simoneau et al. 1986).

The only studies relating to heritability of $\dot{V}O_2$ at submaximal exercise intensities have been those of Fagard et al. (1991) who carried out cycle ergometer studies and found an HI of approximately 0.16, and Bouchard et al. (1989) who have found a genetic effect only when performing at a very low exercise intensity on a cycle ergometer, and that of (Danis et al., unpublished work) showing values of 0.44 obtained from a treadmill test performed by children aged 8–13 years.

The main objective of this study was to examine the different contributions of genetic and environmental factors to RE by selecting a sufficiently homogeneous sample and using modern measurement techniques and mathematical procedures. With this aim we have evaluated the HI of RE by analysing a sample of twin sportsmen with similar backgrounds.

Methods

Subjects

A group of 16 pairs of healthy male twins [8 monozygotic, MZ, mean age 21.2 (SD 2.1) years, body mass 72.4 (SD 10) kg and height 175 (SD 5) cm; and 8 dizygotic, DZ, mean age 22.8 (SD 4.4) years, body mass 71.1 (SD 8.6) kg and height 173 (SD 8) cm], all sportsmen, agreed to participate in the study. The 32 subjects participated regularly in amateur competitive sports, mostly team sports such as soccer or basketball. None of them had perinatal or familial differences, and all had similar environmental backgrounds as well as similar patterns of habitual physical training. Zygosity was determined by means of the genetic analysis used in our laboratory for paternity studies:

1. Human leucocyte antigen class I polymorphism was studied by the serologic standard microlymphocytoxicity test including 60 allelic variants of loci A, B and C (see Terasaki and McClelland 1964).
2. Red blood cell antigens (ABO, Rh, MNSs, Duffy, Kidd Lutheran and P systems) were determined using standard methods (AABB 1990): red cell agglutination or indirect antiglobulin test (all phenotypes were duplicated by independent workers).
3. Electrophoretic polymorphism was determined in the following plasmatic proteins: transferrine (Tf), α-1 antitrypsine (Pi), group component (Gc) and haptoglobin (Hp) (see Constans and Viau 1977; Constans et al. 1980; Dykes and Polesky 1981); isoelectrofocusing in polyacrylamide gels was employed using different ampholine ranges for each protein; gels were stained with coomassie blue R-250 for Pi, Tf and Hp; Gc bands were read after simple precipitation with sulphosalicylic acid.
4. Enzymatic polymorphism: phosphoglucomutase 1 (PGM1) subtypes and acid phosphatase (AcP) phenotypes were determined by isoelectrofocusing in polyacrylamide gels (T_5 5%, C 3%) with ampholines pH 5–8 or pH 5–7 for AcP and PGM1, respectively. Isoenzymes were revealed by the method of Sutton and Burgess (1978) for PGM1, and Burdett and Whitehead (1977) for AcP. Glyoxalate phenotypes were determined in agarose-starch gel electrophoresis with the staining technique described by Parr et al. (1977) using the specific activity technique.

Twins were considered MZ when all genetic markers were identical, and DZ when otherwise. The DZ twins differed in at least two polymorphic systems.

The study was approved by the Ethics Committee of the University of Barcelona and consent was obtained from all participants following careful explanation of the aims of the study and the processes involved.

Anthropometry

A balance with a precision of ±100 g was used to determine body mass, and a stadiometer with a precision of ±1 mm was used to measure body height.

Exercise tests

All participants were invited to visit the laboratory prior to the study to familiarize themselves with the procedures and were encouraged to use the ergometer and the face mask. Each individual followed the same protocol in two exercise tests separated by a period of 48 h: first a maximal test and then a submaximal test. All stress tests were carried out in the morning, at the same time of day, in a well-ventilated laboratory, with room temperature ranging from 22 to 24°C, and relative humidity between 55% and 65%. The tests were carried out 3 h after a light breakfast. None of the participants performed any vigorous exercise during the 24 h prior to the stress tests, and all were informed of the importance of having the same amount of rest during the night preceding the tests. We also insisted that they wore the same training shoes and sportswear during the test.

Both exercise tests were performed on a treadmill (Laufergotest LE-6, Jaeger, Germany), recording respiratory data by means of a breath-by-breath automatic gas exchange analyser (MedGraphics, Sant Paul, USA) equipped with a highly sensitive pneumotacograph (Hans Rudolph, Kansas City, USA). Prior to each test, the gas analyser was calibrated with a tank containing a reference gas mixture (MedGraphics Corporation, USA), and reference room air. Volume and flux were also measured using a calibrated syringe (Hans Rudolph). Parameters recorded continuously were: heart rate (f_C, beats · min^{-1}) using modified V5 precordial leads (Simpliscriptor EK-31, Hellige, Germany); pulmonary ventilation ($\dot{V}_E$, litres per minute, body temperature and pressure, saturated), $\dot{V}O_2$ (millilitres per minute, standard temperature and pressure, dry), and expired carbon dioxide production ($\dot{V}CO_2$, millilitres per minute, STPD). Values obtained automatically were: $\dot{V}O_2$ relative to body mass ($\dot{V}O_2$, millilitres per kilogram per minute STPD), respiratory equivalent for oxygen ($\dot{V}_E \cdot \dot{V}O_2^{-1}$) and for carbon dioxide ($\dot{V}_E \cdot \dot{V}CO_2^{-1}$), and oxygen pulse ($\dot{V}O_2 \cdot f_c^{-1}$, millilitres · beat^{-1}).

The RE was calculated as $\dot{V}O_2$ (millilitres per kilogram per minute) at every velocity, being higher when $\dot{V}O_2$ was lower for a given exercise intensity. All cardiorespiratory parameters were evaluated over an average period of 15 s. Plasma lactate concentration ([La$^-$] millimoles per litre) was determined using the Micro Stat PLM4 electroenzymatic technique (Analox, Instruments Ltd., UK) using a standard [La$^-$] solution as a control (Analox reference: GMRD 090/091/092). The capillary tubes contained heparin, sodium fluoride, and sodium nitrate.

Prior to the exercise tests the participants rested for 5 min while connected to the electrocardiograph and the gas analyser system to obtain basal data and to familiarize themselves with the instruments. The protocols for the two stress tests were as follows:

1. *Maximal test* – the participants began running on the treadmill for 4 min at a speed of 8 km · h^{-1} with a 2.5% gradient. After that, and at the same gradient the speed was increased by 1 km · h^{-1} each minute until the subject was exhausted. Earlobe blood samples were obtained at rest, immediately following maximal effort and at min 1, 3, 5, 7 and 10 of the recovery period, for [La$^-$] determination.

2. *Submaximal test* – the participants began to walk on the treadmill at 5 km · h^{-1} with a 2.5% gradient. With no change in gradient the speed was increased every 4 min by 1 km · h^{-1} up to a speed of 8 km · h^{-1}, completing four intensities of exercise. The subjects were requested to walk at a speed of 5 and 6 km · h^{-1} and to run when the speed was higher than 7 or 8 km · h^{-1}. Cardiorespiratory data from the last 15 s of each exercise intensity were evaluated. To determine [La^-], blood samples were obtained from the ear lobe at rest and 3 min after completing the final exercise intensity. This second test was performed with the aim of achieving a steady state of $\dot{V}O_2$ in 4 min of each exercise intensity.

Statistical analysis

The data obtained were analysed using ANOVA models where RE and other variables studied were considered as the observed variables, and genetic type (MZ and DZ) and pair factor (8 pairs of each genetic type) as the reason for the variation. This was an ANOVA model with two factors where the twin factor was nested over the genetic type factor, which enabled us to establish whether there were differences between the two genetic types considered, and whether the variability between pairs was statistically significant. If no overall significant differences were obtained between the two genetic types (the previously described ANOVA being non-significant), we designed an ANOVA model for each genetic type and considered the pair factor as the only cause of variance.

The residual variance of each of the previous ANOVA was the estimate of variability for the genetic type considered and, therefore, was the value used to calculate HI. By this method we calculated the coefficient of the difference between the residual variance of DZ and MZ with respect to the residual variance of DZ. This parameter was taken as a percentage of the intrinsic variability, depending solely on the genetic group considered. The interpair differences between MZ and DZ were considered significant when $F_{1,14}$ was more than 4.6, and HI variability when the $F_{7,7}$ was more than 3.8, which would give in both cases $P < 0.05$. The aim of this method was to separate the genetic and the environmental components since the experimental error of the measurement and uncontrolled variance would have been similar in both groups of twins.

Results

Maximal test

Differences between the MZ and the DZ groups ($F_{1,14} < 4.6$) were not significant, although the mean values for the MZ group were higher than those of the DZ group. Only the HI of the maximal [La^-] (HI = 0.75) was statistically significant ($P < 0.05$) as shown in Table 1.

Submaximal test

Figure 1 shows histograms of RE ($\dot{V}O_2$ in millilitres per kilogram body mass per minute divided by the corresponding running speed) at 5, 6, 7 and 8 km · h^{-1} during the submaximal test. The RE did not show any statistical differences at any exercise intensity. No significant differences were found in $\dot{V}_E$, $\dot{V}CO_2$, or $\dot{V}O_2 \cdot f_c^{-1}$.

Figure 2 shows histograms for $\dot{V}_E \cdot \dot{V}O_2^{-1}$ at the corresponding running speed.

At 7 and 8 km · h^{-1} the HI of $\dot{V}_E \cdot \dot{V}O_2^{-1}$ (HI = 0.71 and 0.79) shows statistically significant differences ($P < 0.05$). Table 2 shows the [La^-] values obtained in the submaximal exercise test. There are clear significant differences between MZ and DZ with a better physical level in the MZ brothers, as the $\dot{V}O_{2\,max}$ values indicate.

In some participants, especially from the DZ group, at 3 min following completion of the exercise [La^-] rose clearly above values at rest in the submaximal test,

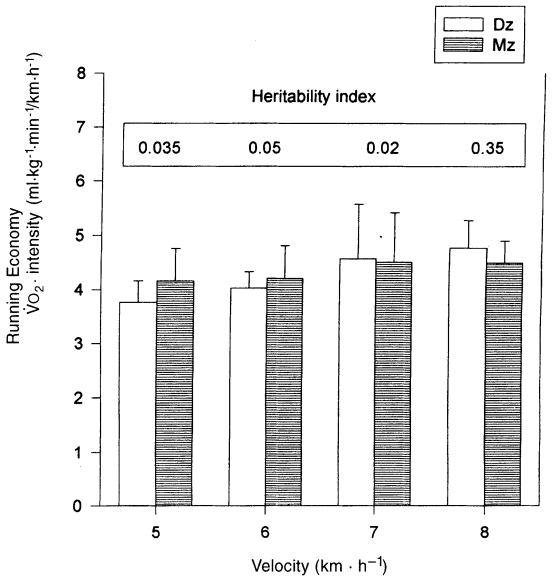

Fig. 1 Running economy at each velocity during the submaximal exercise test. Dizygotes (*DZ*) *unfilled histogram bars*, monocygotes (*MZ*) *hatched histogram bars*; *HI* heritability index value

Table 1 Maximal values during maximal test. $\dot{V}O_{2\,max}$ Maximal oxygen uptake, $[La^-]_{bmax}$ maximal blood lactate concentration. *MZ* monocygotes, *DZ* dizygotes, *F ratio DZ · MZ*$^{-1}$, $F_{1,14} < 4.6$ differences not significant, *HI* heritability index value

	DZ		MZ		*F* ratio DZ · MZ^{-1}	HI
	Mean	SD	Mean	SD		
Speed (km · h^{-1})	14.5	1.5	16.7	2.8	3.54	0.17
$\dot{V}O_{2\,max}$ (ml · min^{-1})	4004	744	4479	929	1.27	0.59
$\dot{V}O_{2\,max}$ (ml · kg^{-1} · min^{-1})	56.8	10.5	61.3	7.4	1.12	0.52*
$[La^-]_{bmax}$ (mmol · l^{-1}	11.0	2.7	11.7	2.4	0.50	0.75

* Statistically significant at $P < 0.05$

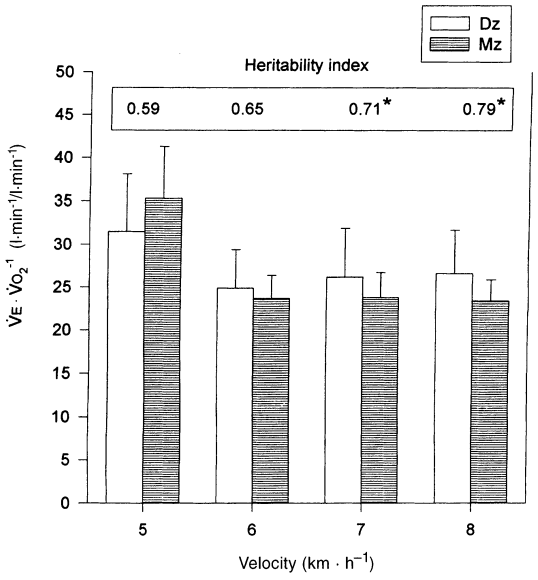

Fig. 2 Ventilation per unit of oxygen uptake ($\dot{V}_E \cdot \dot{V}O_2^{-1}$) at each velocity during submaximal exercise test. *DZ unfilled histogram bars, MZ hatched histogram bars.* Statistically significant at $P < 0.05$. For definitions see Fig. 1

Table 2 Blood lactate concentration $[La^-]_b$ during submaximal exercise test. $[La^-]_b$ Resting lactate, $[La^-]_M$ lactate at final exercise intensity, $[La^-]_R$ lactate at 3rd min of recovery, *MZ* monocygotes, *DZ* dizygotes, *F ratio* $DZ \cdot MZ^{-1}$ $F_{1,14} < 4.6$ not significant differences, *HI* heritability index value

	DZ		MZ		*F* ratio $DZ \cdot MZ^{-1}$	HI
	Mean	SD	Mean	SD		
$[La^-]_b$ (mmol · l^{-1})	1.29	0.38	1.25	0.41	0.037	0.25
$[La^-]_M$ (mmol · l^{-1})	3.59	2.03	2.18	0.85	3.53	0.46
$[La^-]_R$ (mmol · l^{-1})	3.41	1.85	1.81	0.72	5.68*	0.68*

* Statistically significant at $P < 0.05$

interfering with the pure calculation of RE using $\dot{V}O_2$. As the differences among groups were significant, HI values cannot be taken into consideration.

Discussion

After having evaluated the different procedures most commonly used in studies made on twins (Holzinger 1929; Clark 1956; Klissouras 1971; Komi and Karlsson 1979; Pirnay and Crielaard 1983) the purpose of this study was to examine the hereditary component of RE, through studies on twins. Although this procedure has been criticized by several authors (Hrubec and Robinette 1984; Phillips 1993), it has been generally considered to be one of the most effective ways of differentiating the importance of shared genes from that of shared environments, and it has continued to be the basis of many important genetic studies (Bouchard et al. 1990; Stunkard et al. 1990; Heller et al. 1993; Hopper and Seeman 1994; Marenberg et al. 1994).

The HI of $\dot{V}O_{2\,max}$ relative to body mass observed in our study was not significant, but this may have been due to the limited sample size. However, this index was in agreement with that observed by other authors (Bouchard et al. 1986; Fagard et al. 1991) who have reported a lower heritability of $\dot{V}O_{2\,max}$, suggesting that genetic influence is not significant.

The HI of maximal $[La^-]$ was 0.75, which showed statistical significance. The contribution of a genetic factor to this variable may be attributable to differences in percentages of different muscle fibre, as well as to the degree of lipid mobilization and oxidation, implying that genetic conditioning does play a more important role in anaerobic metabolism than it does in aerobic metabolism as has been suggested by Bouchard et al. (1988).

The HI of RE was not significant, with values lower than 1%. The lack of heritability of this component may have been due to various factors:

1. The variability of the analysis of $\dot{V}O_2$ has been estimated by some authors to be approximately 6% (Wright et al. 1978; Katch et al. 1982; Williams et al. 1991) but has been difficult to quantify as its component have been estimated to be 90% biological (Katch et al. 1982) and evidently may conceal the existence of small differences. Nevertheless, a counterbalance between MZ and DZ twins would be expected from such variations in measurement.
2. Intergroup differences in RE are probably small in the majority of cases, as has been deduced from some studies (Dolgener 1982). Dolgener (1982) has not detected RE any differences either in walking or running in untrained, sprint-trained, or endurance-trained women. Bailey and Messier (1991) have not found any differences in RE after a 7-week training programme with novice and trained male runners with variations in stride length, neither have other authors (Brisswalter and Legros 1994) in high level runners after one training season (12–16 km · h^{-1}).
3. The RE may be considerably influenced by varying degrees depending on growth-rate, training, and environmental conditions, as training has been shown to produce changes only in the long term (Schwartz 1977; Daniels 1985; Cavanagh and Kram 1985). It is possible that, more than RE itself, one may inherit the capacity to modify it through training – although this was not the object of our study – as has been found to occur with other physiological variables (Bouchard et al. 1988).
4. The determination of RE, if evaluated from $\dot{V}O_2$ only, cannot be completely accurate if the anaerobic system contributes to the energy supply. In our study the measurement of the energy cost of running at a specified speed from $\dot{V}O_2$ did not necessarily reflect the real total energy cost as there was a small contribution from an anaerobic supply. The results of

blood [La^-] and $\dot{V}_E \cdot \dot{V}O_2^{-1}$ support this possibility. The [La^-] immediately post-effort and at 3 min into the recovery period were above 2 mmol $\cdot$ l^{-1}, and in some cases above 4 mmol $\cdot$ l^{-1}. It is logical to think that this accumulation of [La^-] was partly due to the duration of the stress test, which was 20 min. Although these running speeds of up to 8 km $\cdot$ h^{-1} with a 2.5% gradient were low, they would suggest that the subjects were relatively tired (average f_c equal to 160 beats $\cdot$ min^{-1} at the end of the test).

We were unable to evaluate the HI of [La^-] at 3 min post-exercise as there were statistically significant differences in the variability between MZ and DZ for this variable. The $\dot{V}_E \cdot \dot{V}O_2^{-1}$ increased with each exercise intensity in the submaximal test, coinciding with the incorporation of anaerobic metabolism (see Wasserman et al. 1973), and the HI were 0.59, 0.65, 0.71, and 0.79 at 5, 6, 7, and 8 km $\cdot$ h^{-1}, respectively. In other words, the participation of anaerobic metabolism and its HI increased as the exercise intensity increased, while the HI of aerobic metabolism remained unchanged. It was therefore very likely that, despite our interest in calculating RE at low intensities, such intensities involved an anaerobic component that made imprecise the determination of RE by $\dot{V}O_2$ alone. The [La^-] component must have been present basically at the higher intensity (8 km $\cdot$ h^{-1}) where the difference in $\dot{V}_E \cdot \dot{V}O_2^{-1}$ between MZ and DZ twins became statistically significant. This would imply the participation of anaerobic metabolism, and according to the [increases of [La^-] during exercise, the amount of energy – in oxygen equivalents – can be calculated in both running and swimming (see Margaria et al. 1963; Di Prampero et al. 1978). According to those authors, in running, every increase of 1 mmol in [La^-] represents an amount of energy – in oxygen equivalents – of 3.0–3.3 ml $\cdot$ kg^{-1}. In our study, the increase of [La^-] was: 3.59 mmol −1.29 mmol = 2.30 mmol in the Dz twins and 2.18 mmol −1.25 mmol = 0.93 mmol in the MZ twins (see Table 2). Then, if we multiply the 2.30 mmol of the DZ twins (increase in [La^-] by 3.3 ml $\cdot$ kg^{-1} oxygen equivalent and divide it by 4 min (duration of the exercise) the result is an amount of 1.9 ml O_2 kg^{-1} $\cdot$ min^{-1} which must be added to the measured $\dot{V}O_{2\,max}$ of 38.2 ml $\cdot$ kg^{-1} $\cdot$ min^{-1}, showing an insignificant difference, i.e. 38.2 + 1.9 = 40.1 ml $\cdot$ kg^{-1} $\cdot$ min^{-1}. In the MZ twins it will be: 0.93 mmol (increase in [La^-] times 3.3 ml $\cdot$ kg^{-1} oxygen equivalent) divided by the 4 min equal to 0.77 ml O_2 kg^{-1} $\cdot$ min^{-1} which, added to the measured $\dot{V}O_2$ (36.2 ml $\cdot$ kg^{-1} $\cdot$ min^{-1}) produces a value of 37 ml $\cdot$ kg^{-1} $\cdot$ min^{-1}.

Therefore the total energy expenditure (at these exercise intensities the energy furnished by the phosphocreatine pathway would have been negligible) we measured occurred with no significant difference between DZ 40.1 (SD 10.5) ml $\cdot$ kg^{-1} $\cdot$ min^{-1} and MZ 37.0 (SD 4.6) ml $\cdot$ kg^{-1} $\cdot$ min^{-1} ($P > 0.05$).

Furthermore, it is worth emphasizing the agreement of the results between our study and that of Fagard et al. (1991) using the cycle ergometer. Those authors have not found a significant genetic component in mechanical efficiency or in $\dot{V}O_2$ at a f_c of 150 beats $\cdot$ min^{-1}; however the anaerobic energy generation was significantly conditioned by genetics.

Although our study was carried out in laboratory conditions, where the individuals – as in our study – were correctly instructed on the treadmill, running speeds did not exceed 16 km $\cdot$ h^{-1} and the treadmill gradient was less than 5.7% so the results are applicable to track or field conditions (see Basset et al. 1985).

Finally, regarding the lack of any hereditary factors in RE, although the genotype has been shown to play a significant role in some physiological parameters such as body mass index (Stunkard et al. 1990) and fasting glucose plasma concentrations (Bouchard et al. 1989), other factors such as glucose and insulin responses to a carbohydrate meal appear to have been characterized by lower heritability estimates (Bouchard et al. 1989). Furthermore, different patterns in cerebral surface convolutions in MZ twins have indicated that their brain development is intensely influenced by nongenetic factors, such as environment or chance (Steinmetz et al. 1994).

In summary, we did not detect any genetic component in RE, yet differences were observed in the components of anaerobic metabolism that suggest differences in RE between MZ and DZ twins. However, one must remember that twin studies should be regarded only as a first step in the study of genetic architecture, rather than being definitive.

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Heritability of Explosive Power and Anaerobic Capacity

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Abstract There is a disparity in the information about the heritability of the response of muscle anaerobic metabolism to exercise and explosive power as well as a lack of information about the genetic determinants of this form of work, measured with the different specific physical tests. We applied a battery of some of the commonly employed procedures (Ergojump, Wingate, maximal accumulated oxygen defici, excess post-exercise oxygen consumption, and delta lactate) to a group of 32 Caucasian male twins, 8 monozygotic and 8 dizygotic pairs, with similar environmental backgrounds. Results were studied using a heritability index (HI). Zygosity was determined by the identity of erythrocyte antigens, protein and enzymatic polymorphism and human leucocyte antigen serologic types between co-twins. Significant HI values (P <0.05) were found in the following tests: maximal 5 s power (HI = 0.74) and total power in 30 s (HI = 0.84) in the Wingate test, maximal lactate (HI = 0.82) and delta lactate (HI = 0.84) in the maximal progressive test, as well as in the second (HI = 0.93) and in the third min (HI = 0.92) of recovery after the deficit test. In this study, the most relevant findings were: 1) significant HI values for many of the variables studied; 2) the HI values of the parameters used to evaluate explosive power were higher than those of lactic capacity and 3) the HI of certain variables from different tests measuring, in theory, similar qualities, were different.

Key words: *Genetic endowment, anaerobic metabolism, explosive power, twins, exercise*

INTRODUCTION

The response to exercise of numerous biological variables is determined by the interrelation between genetic and environmental factors. In this respect, the genetic component of several physiological variables - maximal oxygen uptake, running economy, aerobic endurance and anthropometric characteristics - is different (Bouchard et al., 1992, 1999, 2000; Fagard et al., 1991; Rodas et al., 1998; Hamel et al., 1986). Although aerobic metabolism has been extensively studied and the results of these studies seem conclusive, studies on anaerobic metabolism are not definitive (Jones and Klissouras 1985; Komi and Karlsson 1979; Paxinos et al., 1990). Because the methods used to obtain the results of these studies were of lesser validity and reliability and were less standardised than those used in studies of aerobic metabolism, their evaluation is especially difficult.

Paxinos et al. (1990) estimated that the heritability of maximal anaerobic power and that of anaerobic capacity was 86%. Jones and Klissouras (1985) reported a heritability index (HI) of 97% for maximal power and an HI of 83% for maximal isometric force. However, Komi and Karlsson (1979) did not find that the isometric force of the quadriceps was strongly determined by genotype. Concerning the heritability of lactic acid values after exercise, Paxinos et al. (1990) reported an HI of 74% for maximal blood lactate. Komi and Karlsson (1979), however, did not find any significant differences between monozygotic (MZ) and dizygotic (DZ) twins.

Conclusive results on the heritability of explosive power and anaerobic metabolism, of prime importance in many sporting activities, are lacking. Determination of the heritability of the various components which make up the response of anaerobic metabolism to exercise would be valuable in the selection of outstanding performers, and would also be useful in predicting how much improvement could be gained from training, since the greater the genetic component of a given variable, the lesser the possible improvement by training.

The twin method, which compares differences between the variances of MZ and DZ twins, has been widely used to establish the degree of genetic determinism of several physiological qualities (Hrubec and Robinette 1984). It is based on the fact that DZ twins share the same environment but not identical genes, while MZ twins share both environment and genes; consequently, differences in the variances between MZ and DZ twins must be attributable to the genetic component.

The purpose of the present study was to quantify the heritability of the various qualities that determine explosive power and anaerobic performance. To do this, we used some of the most commonly used laboratory tests and the twin method.

SUBJECTS AND METHODS

Subjects

Thirty-two subjects, 16 pairs of healthy male twins, 8 MZ [mean age 21.3 years (SD 2.1), mean body mass 70.4 kg (SD 8.8) and mean height 175 cm (SD 5)] and 8 DZ [mean age 22.8 years (SD 4.4), mean body mass 70.1 kg (SD 8.7) and mean height 173 cm (SD 8)] agreed to participate in the study. None of them had perinatal, pathological or familial differences and all had similar biological age, environmental backgrounds and lifestyle, as well as similar patterns of habitual physical training.

Zygosity was determined by means of the following genetic analyses:

1. Human leucocyte antigen class I polymorphism was studied by the standard_serologic microlymphocytotoxicity test, which includes 60 allelic variants of loci A, B and C (see Terasaki and McClelland 1964).
2. Red blood cell antigens (ABO, Rh, MNSs, Duffy, Kidd Lutheran and P systems) were determined using standard methods (American Association of Blood Banks 1990): red cell agglutination or indirect antiglobulin tests (all phenotypes were duplicated by independent workers).
3. Electrophoretic polymorphism was determined in the following plasmatic proteins: transferrin, α_1 antitrypsin, group component and haptoglobin (see Constans and Viau 1977; Constans et al. 1980), Dykes and Polesky 1981); isoelectrofocusing in polyacrylamide gels was employed using different ampholine ranges for each protein; gels were stained with coomassie blue R-250 for α_1 antitrypsin, transferrin and haptoglobin; group component bands were read after simple precipitation with sulfosalicylic acid.
4. Enzymatic polymorphism, regarding phosphoglucomutase 1 (PGM1) subtypes and acid phosphatase (ACP) phenotypes, were determined by isoelectrofocusing in polyacrylamide gels (T5 5%, C 3%) with ampholine pH 5-8 or pH 5-7 for ACP and PGM1. Isoenzymes were revealed by the method of Sutton and Burgess (1978) for PGM1 and by that of Burdett and Whitehad (1977) for ACP. Glyoxylate phenotypes were determined in agarose-starch gel electrophoresis with the staining technique described by Parr et al. (1977) which uses the specific activity technique.

Twins were considered MZ when all genetic markers were identical and DZ when otherwise. The DZ twins differed in at least two polymorphic systems.

The study was approved by the Ethics Committee of the University of Barcelona and informed consent was obtained from all participants after careful explanation of the aims of the study and the processes involved.

Anthropometry

A balance with a precision of 100 g was used to determine body mass and a stadiometer with a precision of 1 mm was used to measure body height.

Exercise tests

All participants were invited to visit the laboratory prior to the study to familiarise themselves with the procedures. All stress tests were carried out in the morning, at the same time of day, in a well-ventilated laboratory, with room temperature ranging from 22-24 °C and relative humidity between 55% and 65%. The tests were carried out 3 h after a light breakfast. None of the participants performed any vigorous exercise for 24 h prior to the stress tests and all were informed of the importance of having the same amount of rest during the night preceding the tests. We also insisted that they wore the same training shoes and sportswear during the tests.

Physiological variables studied

Table 1 shows the variables analysed, the quality they evaluated, their respective abbreviations and units of measure. Tests used to obtain these variables are also specified.

Table I. Physiological variables indicating anaerobic power and capacity.

Variable	Quality evaluated	Tests used	Abbreviations	Units
Explosive power	Explosive Power	Bosco's squat jump	SJ	cm
Explosive + elastic power	Explosive Power	Bosco's counter movement jump	CMJ	cm
Anaerobic power developed in 15 s	Anaerobic Power	Bosco's 15s jump test	PA15s	W/kg
Maximal power developed in 5 s	Explosive Power	Wingate	Pmax5s	kgm in 5s
Work developed in 30 s	Anaerobic capacity	Wingate	Ptot30s	kgm in 30s
Maximal accumulated oxygen deficit	Anaerobic capacity	Deficit Progressive submaximal Progressive maximal	MAO_2D	ml/kg
Excess Post-exercise Oxygen Consumption	Anaerobic capacity*	Deficit	O_2D	ml/kg
Maximal lactate after maximal progressive test	Anaerobic capacity*	Progressive maximal Blood lactates during recovery	LamaxT	mM/L
Delta lactate after maximal progressive test	Anaerobic capacity*	Progressive maximal Blood lactate: maximal, basal	ΔLaT	mM/L
Maximal lactate after deficit test	Anaerobic capacity*	Deficit Blood lactates during recovery	LamaxD	mM/L
Delta lactate after deficit test	Anaerobic capacity*	Deficit Blood lactates: maximal, basal	ΔLaD	mM/L

* and complex factors

Protocols

1. Variables determined by Bosco's tests

The following variables were determined by a battery of Bosco tests, carried out on an Ergo-Jump Bosco System platform (Digitest OY, Muurame, Finland) using the method described by Bosco et al, 1983 -explosive power was measured with the squat jump test; explosive plus elastic power with the counter movement jump and power developed in 15 s with the 15-second jump test. Moreover, we calculated the elastic quotient obtained by subtracting explosive power from explosive plus elastic power.

2. Variables determined by the Wingate test

The variables determined by the Wingate test, carried out on a cycle ergometer (Monark 818, Sweden) following the protocol described by Tharp et al., 1984,

were: maximal power developed in 5 s ($P_{max}5s$), work developed in 30 s ($P_{tot}30s$) and fatigue index ($P_{max}5s$ – minimum power developed in 5s)/ $P_{max}5s$.

3. Maximal accumulated oxygen deficit (MAO_2D).

The calculation method proposed by Medbø et al. (1988) was used in a simplified form, MAO_2D being the difference between the oxygen demand and the oxygen consumption measured during the deficit test. Maximal oxygen deficit equals the sum of the differences between demand ($ml.kg^{-1}.min^{-1}$) and oxygen uptake (VO_2) ($ml.kg^{-1}.min^{-1}$) measured during the deficit test for each min that the subject is able to maintain maximum velocity. Maximum velocity was previously determined for each subject. Demand was calculated by extrapolating least squares. Oxygen uptake values, on which the extrapolation was based, were those which corresponded to the mean of the values measured in the last 2 min of each of the four steps that constituted the submaximal progressive test. The tests were carried out on a treadmill (Laufergotest LE-6, Jaeger, Germany) with a "breath by breath" gas ergoanalizer (CPX II, MedGraphics, USA) and with a mask (Hans Rudolf, USA). To determine the MAO_2D, three exercise tests were needed: (i) the maximal progressive test was performed with a constant gradient of 2.5%, with a 4-min warm-up at a velocity of 8 km h^{-1}, followed by increments of 1 km h^{-1} per min until exhaustion. The parameters measured during the last 15 s before the end of the test were considered maximal. (ii) The submaximal progressive test was performed at a constant gradient of 2.5%, an initial velocity of 5 $km.h^{-1}$, with four, 4-min steps with increments of 1 $km.h^{-1}$ every 4 min. Subjects were asked to walk when v = 5 and 6 km h^{-1} and to run when v = 7 and 8 km $h.^{-1}$ Ventilation parameters were obtained every 15 s and mean values of the last 2 min of each step were obtained. With this protocol a steady state was reached in each of the steps and the exercise performed was clearly predominantly aerobic. (iii) The deficit test was performed at a constant gradient of 2.5%, at an initial velocity (warm-up) of 8 $km.h^{-1}$ for 4 min followed by the maximum velocity reached in the maximal triangular test. During the deficit test, subjects had to run for as long as possible (a minimum of 2 min).

4. Maximal blood lactate ($La_{max}T$, $La_{max}D$) and delta lactates (Δ LaT, Δ LaD).

Maximal blood lactate was the highest value obtained during recovery, analysed at 3, 5, 7 and 10 mins after both the maximal progressive test ($La_{max}T$) and the deficit test ($La_{max}D$). Delta (Δ) lactate is the difference between the highest value of blood lactate and the basal value (at rest) determined before beginning the tests and referring to both tests (Δ LaT and Δ LaD). Capillary blood samples were obtained from the ear lobe. Quantification of blood lactate was performed with the electroenzymatic technique (Micro Stat P-LM4, Analox Instruments Ltd., UK).

5. Excess Post-Exercise Oxygen consumption (O_2D).

Calculation of the O_2D was based on the data collected during each min of the first 10 min of recovery after the deficit test (oxygen uptake at each minut of recovery after the deficit test: VO_2R_x PD, $_x$ number in subscript indicate the minute of recovery). Theoretical basal oxygen uptake was considered as 3.5 ml/kg/min.

General chronological method

The battery of tests was performed on 3 different days (non consecutive). On the first day of the study a complete medical history was taken and the maximal tests were performed. On the second day, the submaximal tests were performed so that oxygen demand could be calculated. One and a half hours later, the deficit tests were performed. On the third day, tests from the battery of Bosco's tests were performed followed, half an hour later, by Wingate's test.

Statistical analysis

The data obtained were analysed using ANOVA models where the variables studied were considered as the observed variables and genetic type (MZ and DZ) and pair factor (8 pairs of each genetic type) as the reason for the variation. In consequence, an ANOVA model with two factors was applied, where the twin factor was nested over the genetic type factor, which enabled us to establish whether there were differences between the two genetic types considered and whether the variability between pairs was statistically significant. If no overall significant differences were obtained between the two genetic types (the previously described ANOVA being non-significant) we designed an ANOVA model for each genetic type and considered the pair factor as the only cause of variance.

The residual variance of each of the previous ANOVA was the estimate of variability for the genetic type considered and, therefore, was the value used to calculate the HI (Rodas G. et al., 1998). Using this method we calculated the coefficient of the difference between the residual variance of DZ and MZ with respect to the residual variance of DZ. This parameter was taken as a percentage of the intrinsic variability, depending solely on the genetic group considered. The inter-pair differences between MZ and DZ were considered significant when $F_{1\ 14}$ was more than 4.6 and HI variability was considered significant when $F_{7,7}$ was more than 3.8, which in both cases would give $P < 0.05$. The aim of this method was to separate the genetic and the environmental components since the experimental error of the measurement

and uncontrolled variance would have been similar in both groups of twins.

Before calculating the corresponding HI values, in accordance with the mathematical base necessary to be able to calculate them, the independence between twin-type and the variables studied was confirmed by a hierarchical analysis of variance for each of the variables analysed.

RESULTS

The statistics for the variables analysed are shown in Table II.

It is remarkable that the adjustments used to estimate the residual mean squares of the two groups of twin-types and later the HI were high, then the reliability of the method was high.

Table III shows the heritabilities estimated for the pairs of twins (the HI values), the F value and the statistical significance of the HI.

The heritabilities estimate were striking high, even though the genetic component reached significance ($P < 0.05$) in only 6 variables or biological indicators of power and anaerobic capacity: $P_{max}5s$, $P_{tot}30s$, $La_{max}T$, Δ LaT, VO_2R_2PD and VO_2R_3PD.

Table II. Statistical data for the variables analized

Variables	Mean DZ	Std DZ	Mean MZ	Std MZ
Explosive power	31.1	4.1	30.0	4.2
Explosive + Elastic power	35.2	4.7	33.7	4.5
Elastic quotient	4.8	2.5	3.7	1.9
Anaerobic power developed in 15s	20.7	3.5	20.5	3.1
Maximal power developed in 5s/ body weigh	5.1	0.5	5.0	0.5
Maximal power developed in 5s	363.0	60.2	361.2	71.3
Fatigue index	42.3	5.1	39.9	7.7
Work ideveloped in 30s	1769.0	293.5	1772.8	295.9
Maximal lactate progressive test	10.9	2.7	10.2	2.7
Δ lactate progressive test	9.3	2.7	9.0	2.9
Maximal lactate deficit test	12.4	2.0	10.9	2.2
Δ lactate deficit test	10.3	1.6	9.2	2.5
Maximal accumulated oxygen deficit	100.9	72.1	51.0	49.2
Excess post-exercise oxigen consumption	101.8	30.6	101.3	11.8

Table III. Heritability of the variables indicating anaerobic power and capacity

Variables	Heritability index	F
Explosive power	0.6694	3.02
Explosive+elastic power	0.4488	1.81
Elastic quotient	0.6951	3.28
Anaerobic power developed in 15s	0.6262	2.67
Maximal power developed in 5s	0.7442	3.90*
Fatigue index	0.4346	1.76
Work developed in 30s	0.8361	6.10*
Maximal lactate progressive test	0.8161	5.43*
Δ lactate progressive test	0.8393	6.22*
Maximal lactate deficit test	0.5920	2.45
Δ lactate deficit test	0.7022	3.35
VO_2R_1PD **	0.6958	3.28
VO_2R_2PD	0.9297	14.22*
VO_2R_3PD	0.9246	13.26*
VO_2R_4PD	0.6602	2.94
VO_2R_8PD	0.7185	3.55
VO_2R_9PD	0.5931	2.45
$VO_2R_{10}PD$	0.4260	1.74
Maximal accumulated oxygen deficit	0.2221	1.28
Excess pos-exercise oxigen consumption	0.5613	2.27

* P<0.05

** Oxygen uptake at the first minut of recovery after the deficit test. Numbers in subscript indicate the minute of recovery

DISCUSSION

Homogeneity of the sample analysed

Given the difficulty of finding twins with identical antecedents, who were willing to take part in the numerous tests required, we chose to use a sample that was relatively small but numerically representative. To minimise non-genetic variance as much as possible, the environmental background of the sample was highly homogeneous. Thus, the general approach adopted in previous studies of genetic influence in sports performance in which more numerous samples were used was followed (Hamel et al. 1986; Klissouras, 1971; Komi and Karlsson 1979; Pirnay and Crielaard 1983). Moreover, the exhaustive immunogenetic analyses used in our study enabled us to establish the reliability of the sample analysed since it guaranteed the correct classification of the twins studied into monozygotic and dizygotic pairs.

The intra-ethnic group nature of the study (among Caucasians) should also be highlighted since it is well-known that the heritability of the same variables varies according to different ethnic groups. Thus, the Caucasians have a higher percentage of type-1 muscle fibres and a lower percentage of type-IIA muscle fibres than do native people from the west of Africa. Moreover, the activity of CK, HK, PFK and LDH is lower in Caucasians (Ama et al. 1986).These data suggest that originary people from the west of Africa are better predisposed to perform well in sports events of short duration and are in agreement with the results obtained in short-duration competitive events where people of colour usually obtain the best world results, while they very seldom perform outstandingly well in events in which endurance is required. Because of their west-African origin, the same is true for African Americans from the United States, Canada and the Caribbean, while people of colour from the north and east of Africa are especially suited to events requiring endurance. For these reasons, in the present study heritability was studied in the same ethnic group, although substantial differences might have been found had it been studied in other groups..

Heritability of explosive power.

1. Heritability of explosive power measured with Bosco's tests

The HI values obtained for explosive power measured with the battery of Bosco's tests indicated that, although genetically determined, environmental factors may modify this quality, because the heritability of explosive power (0,67) and power developed in 15 s (0,62) was very similar. The HI value of the elastic quotient was the highest of the group (0.70), which suggests that an additive effect is produced since elastic quotient depends as much on explosive as on elastic force. Even though the significance of the power developed in 15s is complex, it is related, to some extent, with anaerobic metabolism, because a strong correlation has been found between it and 60m dash (Bosco et al., 1983).

The values obtained in the present study were lower than those obtained by other authors (Pirnay and Crielaard 1983; Weiss 1977) and were possibly determined by differences in methodology and in the samples used. In the present study highly reliable methods and a strict protocol were used and, as previously mentioned, the sample was highly homogeneous. Importantly, in some studies the mean age of the sample was low (Margaria et al., 1966; Simoneau et al. 1986a) and consequently force and power were still not developed as this takes place after puberty. Therefore, the genetic influence in a particular quality may not be equally manifested in different ages.

The results of studies evaluating the effect of training on such qualities have shown that they are clearly improved by training (Bosco et al., 1983; Bosco and Komi 1979; Bosco et al. 1984, 1986). These data suppport the HI values obtained in the present study

2. Heritability of explosive power measured with Wingate's test.

The heritability of explosive power ($P_{max}5s$), measured with Wingate's test reached statistical significance with an HI value of 0.74. Likewise, power relative to body weight also showed a high and significant HI value (0.86. These results are the same as those of Paxinos et al. (1990) who used the same test. Moreover, using different, but mainly similar tests, other authors have obtained similar HI values (Komi and Karlsson 1979; Pirnay and Crielaard 1983; Jones and Klissouras, 1985).

3. Overall heritability of explosive power

As previously mentioned, different estimates have been made of the heritability of explosive power, according to whether Bosco's or Wingate's tests was used.

Although, *a priori*, these data could be surprising, it should be noted that the work performed in these tests is different: it is simultaneous in Bosco's tests and alternate in Wingate's; in addition, duration is greater in Wingate's test. These factors lead to differences in the reuse of elastic energy.

Therefore, the higher HI values for energy utilization obtained with Wingate's test ($P_{max}5s$) probably mean that the heritability of the metabolic component is higher than the heritability of the biomechanical component (composed of muscular viscoelastic properties and of neuromuscular functioning), which is better represented by the values obtained with Bosco's tests.

Heritability of anaerobic capacity

1. Heritability of anaerobic capacity measured with the Wingate test

In agreement with other authors (Gastin et al. 1995), we did not find that Wingate's test, because of its brevity, was able to estimate anaerobic lactacid capacity. Therefore, it is more likely that the power developed in the 30 s of this test measures mainly anaerobic alactacid capacity, plus some degree of lactic metabolism.

The HI value of 0.83 found for $P_{tot}30s$ was very high. This result was similar to that of Paxinos et al. (1990). Moreover, the results of studies evaluating the possible improvement produced by training agree with our own, given that increments (between 3% and 5%) were minimal (Bar-Or, 1981). The heritability of the fatigue index is included in this section as this variable is derived from Wingate's test, although it does not reflect either capacity or alactacid anaerobic power. The HI value determined was not significant (0.43). Comparison of this value with those of of $P_{max}5s$ and $P_{tot}30s$, can indicate that, although the explosive power and lactacid-alactacid capacity developed can be modified only tp a small extent, the resistance of this pathway can indeed modified and consequently, can improve anaerobic output.

2. Heritability of anaerobic capacity measured by analysis of blood lactate

Maximal blood lactate after the exercise it is the common indicator of several mechanisms although it is only and indirect, and not very accurate, method to evaluate maximal anaerobic capacity. We have used this variable because it is relatively often utilised, has a correlation with running time for 400m, and it increases (17%) after anaerobic training (Vandewalle et al., 1987). Maximal blood lactate and delta lactate, obtained with the maximal progressive triangular test, showed high HI values (0.82 and 0.84, respectively). However, the HI values corresponding to the same variables obtained with the deficit test were lower and not differ significantly (0.59 and 0.70, respectively). Such differences could be due to the different intensity and duration of these tests, which involve different proportions of energy requirements covered by the aerobic and anaerobic pathways and consequently reflecting differences in lactate production and possibly different intensities in the activity of lactate carriers (Wilson et al., 1998).

In the present study, blood lactate concentrations obtained by the deficit test were considered indicators of anaerobic capacity. Blood lactate obtained after the maximal progressive triangular test provided different information concerning the interaction between aerobic and anaerobic metabolism being the heritability of aerobic metabolism a determining factor in their elevated HI values (Bouchard et al. 1986; Fagard et al. 1991; Klissouras 1971; Komi and Karlsson 1979; Pirnay and Crielaard 1983).

The HI values corresponding to maximal lactate and delta lactate obtained after the deficit test suggest that non-genetic factors significantly influence anaerobic capacity (measured by maximal blood lactate) although the heritability of these variables is not negligible. These results are in agreement with those of other studies in which increases in maximal lactate after training have been observed (Vandewalle et al., 1987; Jacobs 1986). Moreover, Komi and Karlsson (1979) did not consider the heritability of maximal lactate after intense physical activity to be significant. We believe the high HI values for maximal blood lactate found by other authors (Paxinos et al. 1990; Klissouras 1971) can also be attributed to the different tests used.

3. Heritability of anaerobic capacity measured by accumulated maximal oxygen deficit

The HI value of MAO_2D (0.22) was low, the estimated heritability being the lowest of all the variables analysed. This result is in agreement with the finding of significant changes in this parameter after anaerobic training (Medbø and Burgers, 1990).

4. Heritability of anaerobic capacity measured by the maximal Excess post-exercise oxigen consumption

auslity of the excess post-exercise consumption is complex, reflecting a general disturbance following exercise and not just the lactacid "O2 debt" (Gaesser and Brooks, 1984) and in consequence not being a very accurate tool to measure anaerobic capacity, it is no only a classical physiological concept, but a useful indicator, since their highest values have been found in elite-speed athletes -with values about four times more than in untrained healthy people- and in some cases has been used for validation of anaerobic tests (Vandewalle et al., 1987).

Heritability of O_2D was low, the HI value obtained being 0.22. However, analysing oxygen consumption values during each min of recovery after the deficit test revealed that the heritability of O_2D is very high in the second and third min. These findings suggests a high geneic component in the PCr resynthesis and repletion of the O2 stores of myoglobin and venous blood, which mainly occurs during the first 3 min of recovery.

Although few studies have analysed the influence of training on O_2D, it is believed to be susceptible to substantial improvement (Fox E.L. et al, 1989).

5. Overall heritability of anaerobic capacity

The above-mentioned results suggest that the overall heritability of anaerobic capacity is low although that of explosive power is high.

The variable with the highest HI value (0.70) was Delta lactate, evaluated after the deficit test while the HI value for MAO_2D was the lowest (0.22). In view of these results, as well as the fact that the results of blood lactate were notably determined by anaerobic lactic capacity, the heritability of anaerobic capacity can be estimated to be between 22% and 70%.

In addition to agreeing with the results of the studies cited in each of the above paragraphs, our results also agree with those of Simoneau et al. (1986b) who suggested that the heritability of anaerobic lactic capacity was probably lower than that determined by other authors, since they report that training produced significant improvements, with a mean increase of 33%. However, the same authors observed wide inter-individual variation and, more interestingly, found that response to training was associated with genotype as 65% of the variances were due to genetic factors.

Although, as previously mentioned, little is currently known about the heritability of miotypology and/or the various muscular enzymes, analysis of the results of these studies suggests that the heritability of general anaerobic capacity is lower than that of other qualities (Bouchard et al., 1992; Simoneau et al. 1986b).

In summary, the HI estimates for the various indicators of anaerobic metabolism were high. Heritability seems to be much greater in explosive power and anaerobic alactacid capacity than in general and anaerobic lactic capacity. HI estimates should be analysed according to the ethnic group of the subjects studied. Similar studies in different groups should be performed since anaerobic metabolism and its heritability may widely vary .

The heritabilities determined with different tests, designed to evaluate the same quality, were different. These results confirm the belief that such tests do not measure exactly the same qualities. Further studies are needed to clarify this question and improve the validity of such tests. To do so, such aspects as duration, intensity, which muscle groups are used, whether the activity is simultaneous or alternate and whether the force measured is instantaneous or average, should be considered.

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physical fitness and performance

Cardiorespiratory response to exercise in elite Sherpa climbers transferred to sea level

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ABSTRACT

GARRIDO, E., G. RODAS, C. JAVIERRE, R. SEGURA, A. ESTRUCH, and J. L. VENTURA. Cardiorespiratory response to exercise in elite Sherpa climbers transferred to sea level. *Med. Sci. Sports Exerc.*, Vol. 29, No. 7, pp. 937–942, 1997. Himalayan Sherpas are well known for their extraordinary adaptation to high altitude and some of them for their outstanding physical performance during ascents to the highest summits. To cast some light on this subject, we evaluated the cardiorespiratory response during exercise at sea level of six of the most acknowledged Sherpa climbers, mean age (±SD) 37 (±7) yr old. Continuous electrocardiogram and breath-by-breath pulmonary gas exchange until exhaustion were obtained by following the Bruce protocol. We detected a maximal oxygen uptake ($\dot{V}O_{2max}$) of 66.7 (±3.7) $mL \cdot min^{-1} \cdot kg^{-1}$, maximal cardiac frequency of 199 (±7) $beats \cdot min^{-1}$, and ventilatory anaerobic threshold at 62 (±4) % of $\dot{V}O_{2max}$. These factors could help to explain the greater performance level shown by several elite climbers of this ethnic group. The high functional reserve demonstrated by this very select group of highlanders could be associated with natural selection and with special physiological adaptations probably induced by long-training in a hostile environment.

HIGHLANDERS, OXYGEN UPTAKE, HEART RATE, ANAEROBIC THRESHOLD, ALTITUDE, HYPOXIA, MOUNTAIN CLIMBING.

The extraordinary adaptation and physical resistance that some highland ethnic groups present at high altitudes are widely acknowledged; and, without any doubt, the most prestigious example is that of the Sherpas, famous for their performance during ascents to the highest peaks of the world. This small ethnic group directly descended from Tibetans who settled in northeast Nepal some 500 yr ago within the abruptly orographical area around Mount Everest and *Cho-Oyu* in the south Himalayas at an altitude of between 3,000 and 4,900 m above sea level. Although the majority of Sherpas are farmers, it is the only Himalayan community to boast a notable number of individuals who participate in extreme-altitude expeditions and have earned worldwide recognition for their outstanding performance during ascents to the earth's highest summits.

Some studies have demonstrated the existence of physiological characteristics that would explain the excellent performance of native highlanders. It is well known that highland dwellers make greater use of glycidic substrates (13) and show a particular enzymatic adaptation that attenuates pyruvate to lactate flux (14). Likewise, they show better ventilatory efficiency (18), lower pulmonary resistance (10), and differences in the acid-base balance (20). However, other studies have not found any physiological or biochemical bases that could account for their superior performance. Apparently, their muscle capillary or mitochondrial volume density is not greater than those of lowland dwellers acclimatized to high altitude, nor indeed than those of sedentary individuals (17), showing low enzymatic activity in both the oxidative as well as glycolytic anaerobic pathways (12). Nevertheless, Moore et al. (23) suggested that the effectiveness of the oxygen transport system and uptake improves with successive generations exposed to high altitude hypoxia, and another report showed that Tibetan newborns had higher arterial oxygen saturation at birth and during the first months of life than Han Chinese newborns (24).

However, in the majority of the previous studies to evaluate functional capacity in Himalayan highlanders or Andean Amerindians, average values of $\dot{V}O_{2max}$ between 37 and 52 $mL \cdot min^{-1} \cdot kg^{-1}$ have been observed (5,13,17,21,27,34,38). It is noteworthy that the majority of studies performed on Himalayan natives have been carried out at high altitude, or using low-specific exercise tests, such as the bicycle ergometer for Sherpa natives (21,27), with the exception of the use of this ergometer for the north-Himalaya population groups, accustomed to cycling along the Tibetan plateau (34,38), or walking uphill test (31), or the step test (5,17). Another report showed results based on an indirect estimation of oxygen uptake (17). Likewise, it should also be pointed out that only two reports include natives with great experience in ascents at extremely high altitude (5,31), one of which was carried out with the collaboration of Tensing Norgay

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(31), the Sherpa famous for the first human conquest of Everest in 1953.

With the aim of defining the aerobic profile and functional reserve of some select individuals of this ethnic group, we proposed: 1) the study of an elite group of Sherpa climbers and evaluate their performance at sea level; 2) the application of an exercise test as specific as possible, and as close as possible to their usual physical activity in the mountains; and 3) the determination by means of direct methodology of certain cardiorespiratory parameters, including the noninvasive ventilatory threshold, hitherto unstudied in Himalayan highlanders.

METHODS

Subjects. Six males selected from the 25 best Sherpa climbers on the basis of best climbing records and greatest number of ascents performed at extreme altitude. The subjects were 37 (±7) yr old. All of them were born and had spent practically all of their lives within the *Solu Khumbu* region of northeastern Nepal at an average altitude of 3,150 (±650) m above sea level. Only one of them (subject 2) had recently spent periods of time at low altitude (1,300 m) in Kathmandu (Nepal) between alpine expeditions. The whole group had successfully completed, without the use of supplementary oxygen, a total of 67 ascents to above 7,000 m, 45 of which were above 8,000 m. They had reached 25 summits above that altitude, including the summit of Mt. Everest (8,848 m) on 12 occasions. Four subjects had recently climbed beyond 8,000 m (48 d before the study), and two of these had reached the summit of Mt. Everest. None of them had followed a physical training program, other than their usual participation in high-altitude expeditions once or twice a year. One of them (subject 3) also worked as a guide for a trekking agency when not participating in expeditions at extreme altitude. The present study was carried out coinciding with a company-sponsored visit of the Sherpa climbers to our country. The subjects were informed of the purpose and the experimental noninvasive procedures to which they all granted their consent in accordance with the Institutional Review Board. Previously, a medical history was taken (with the assessment of a native expert translator from the Nepal Consulate), and a routine physical examination was performed.

Anthropometry. Weight was determined using a scale with a precision of ± 0.1 kg (Seca, Soenhle, Germany), and height by means of a tape with a precision of ± 1 mm (Holtain Ltd., Crymych, UK). Skinfold thickness (triceps, subscapular, suprailiac, and quadriceps) was measured using a caliper with a precision of ±0.2 mm (Holtain Ltd.). Skeletal diameters (wrist and knee) were measured by means of a pachymeter with a precision of ± 0.01 mm (Mitutoyo, Tokyo, Japan). Limb circumferences (arm and calf) were measured using a metal spring-loaded tape (Holtain, Ltd.). The unilateral measurements were always taken on the right side, following the procedure proposed by Ross et al. (30). Body fat (% fat) was calculated according Durnin and Womersley (7).

Stress test. The study was carried out in a laboratory located at 125 m above sea level at a time when all the natives had been at low altitude (1,300 m or below) for only 2 wk. The room temperature ranged between 22 and 24°C, and the relative humidity between 55 and 65%. The subjects were asked to abstain from strenuous exercise during the 3 d before the test. The evaluation took place in the morning, 3 h after a light breakfast. A standard 12-lead electrocardiogram (Schwarzer Cardioscript CD-6000, Picker, Germany) was obtained with the subjects at rest.

Afterward, all subjects completed an exercise test on a treadmill (Laufergotest LE-6, Jaeger, Germany) under normoxic conditions until exhaustion; the Bruce protocol was followed (4). Pulmonary gas exchange was measured using a breath-by-breath automated gas analysis system (CPXII, MedGraphics, St. Paul, MN). The instrument was calibrated before each test, both in relation to volume and flow by means of a 3-L capacity syringe (Hans Rudolph, Kansas City, MO), and gases obtained from a tank (Medical Graphics Corp., Plumsteadville, PA) containing a reference mixture (5% CO2, 12% O2, balanced N2), and with atmospheric air (well-ventilated laboratory). The following parameters were recorded: pulmonary ventilation ($\dot{V}E$, $L \cdot min^{-1}$ BTPS), tidal volume ($\dot{V}T$, $mL \cdot min^{-1}$ BTPS), respiratory frequency (fR, min^{-1}), end-tidal PCO_2 (PET_{CO2}, mm Hg), end-tidal PO_2 (PET_{O2}, mm Hg), oxygen uptake ($\dot{V}O_2$, $mL \cdot min^{-1}$ STPD), and expired CO_2 ($\dot{V}CO_2$, $mL \cdot min^{-1}$ STPD). The following data were automatically calculated: respiratory quotient [$R = (\dot{V}CO_2 \cdot \dot{V}O_2^{-1})$], oxygen uptake relative to body mass ($\dot{V}O_2$, $ml \cdot min^{-1} \cdot kg^{-1}$ STPD), respiratory equivalent for oxygen ($\dot{V}E \cdot \dot{V}O_2^{-1}$), respiratory equivalent for carbon dioxide ($\dot{V}E \cdot \dot{V}CO_2^{-1}$), and energy expenditure expressed in metabolic units (MET). The different respiratory values were obtained from the average of the last 30 s of each stage and the same was done during exhaustion, rejecting the maximum peak values obtained. Continuous electrocardiogram (Simpliscriptor EK-31, Hellige, Germany) with instantaneous determination of cardiac frequency (fC) was obtained during the test by means of the CM5 precordial lead, and we chose the average fC of the last 15 s of each stage and during exhaustion. The maximum peak values were also rejected. Oxygen pulse was calculated as $\dot{V}O_2 \cdot fC^{-1}$ ($mL \cdot beat^{-1}$). We estimated the anaerobic threshold (AT) by means of the noninvasive "V-Slope" ventilatory method proposed by Beaver et al. (3).

As the subjects had never experienced a medical exercise test before, before the test they were invited to walk on the treadmill for a few minutes wearing the mask, for them to become familiarized with the experi-

TABLE 1. Values (mean ± SD) of the respiratory parameters obtained during maximal effort.

Parameters	Maximal
$\dot{V}E$ ($L \cdot min^{-1}$)	134 ± 22.8
VT ($mL \cdot min^{-1}$)	2994.7 ± 449.5
fR (min^{-1})	45.2 ± 3.8
$\dot{V}CO_2$ ($mL \cdot min^{-1}$)	4741.6 ± 523.5
$VO_2 \cdot fC^{-1}$ ($mL \cdot beat^{-1}$)	20.2 ± 2.6
$PETO_2$ (mm Hg)	124 ± 3
$PETCO_2$ (mm Hg)	41 ± 5
$\dot{V}E \cdot \dot{V}CO_2^{-1}$	28.1 ± 3.3
$\dot{V}E \cdot \dot{V}O_2^{-1}$	33.6 ± 3.6
R	1.19 ± 0.02
METs	19 ± 1

mental procedure. Before starting the exercise protocol, the subjects remained at rest for 3 min connected to the gas analyzer with the aim of obtaining the optimal basal data and achieving equilibrium in the gas-exchange tubes.

RESULTS

Anthropometry. Body mass was 59.4 (± 4) kg, height was 163.5 (± 5.2) cm, with mean body surface area equal to 1.6 (± 0.1) m^2, and mean body fat proportion equal to 11.9 (± 1.3) %. Wrist and knee skeletal diameters were equal to 5.5 (± 0.9) and 9.4 (± 0.2) cm, respectively. Arm and calf limb circumferences were equal to 26.3 (± 1.2) and 33.9 (± 1.1) cm, respectively.

Functional assessment. Three subjects reached exhaustion point during the 2nd or 3rd min of the fifth stage, whereas the other three subjects completed this stage. We observed that the natives all showed an apparently deficient mechanical efficiency on the treadmill with a peculiar walking and running style. The average (30 s) data of the different respiratory variables obtained during maximal effort are shown in Table 1. Figure 1 shows the evolution of the $\dot{V}E$, $\dot{V}O_2$, and fC throughout the exercise test. Table 2 shows individual data for $\dot{V}O_{2max}$, maximal fC, percentage of $\dot{V}O_{2max}$ at ventilatory AT, and fC at ventilatory AT. Five subjects showed the ventilatory AT during the third stage of the protocol at an average $\dot{V}O_2$ of 2.56 (±0.38) $L \cdot min^{-1}$ (43.4 ± 3.3 $mL \cdot min^{-1} \cdot kg^{-1}$). One of them (subject 5) showed an unclear ventilatory AT, which was difficult to determine with precision.

DISCUSSION

In relation to the anthropometric testing, it is worth pointing out that the subjects constituted a homogeneous group, with a medium stature, body build, and low body fat in accordance with the results previously reported by Sloan and Masali (33). With the exception of the higher limb circumferences and total skinfold thickness average values obtained by us, all the other anthropometric measurements were almost identical to those previously reported for the same age group in a wide sample of the

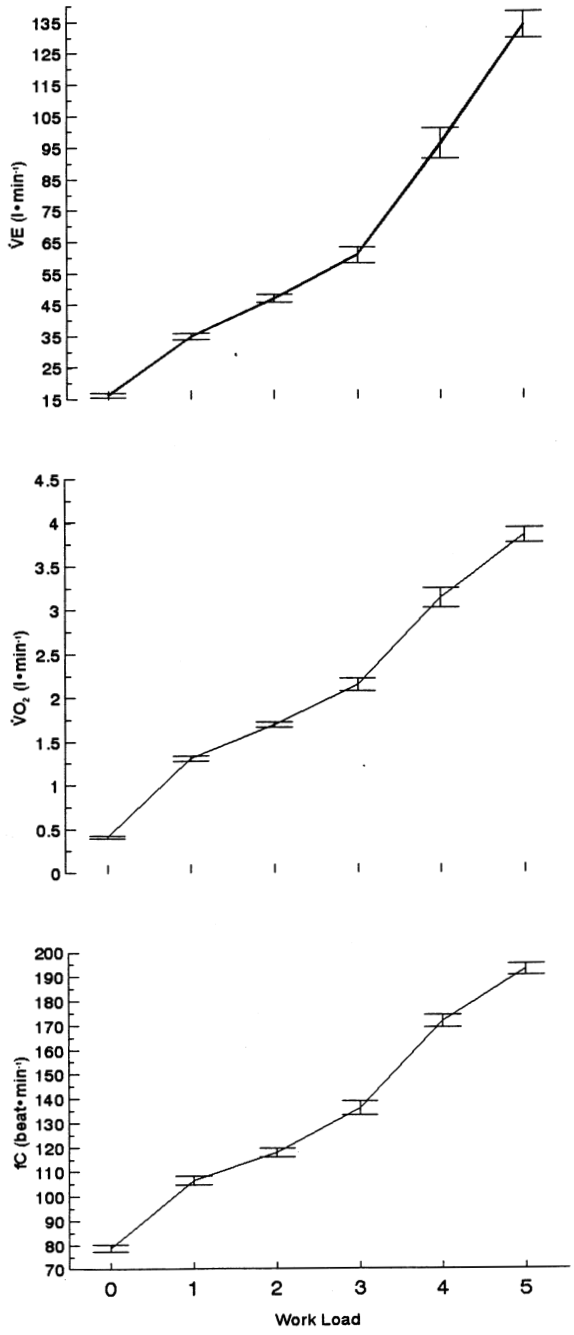

Figure 1—$\dot{V}E$, $\dot{V}O_2$, and fC (mean ± SE) throughout the exercise test.

male Sherpa population (33). This fact may be explained by the higher physical activity and, generally, better nutrition status of climbers in relation to farmers of the Sherpa highland community.

Subjects with higher $\dot{V}O_{2max}$ at sea level had higher uptakes at the simulated high altitudes (6). Although this fact does not seem to ensure tolerance of high altitude

TABLE 2. Some individual data at maximal effort and at anaerobic threshold

Subject	Age (yr)	$\dot{V}O_2max$		fCmax (beat·min^{-1})	Ventilatory AT	
		ml·min^{-1}	ml·min^{-1}·kg^{-1}		%$\dot{V}_2$max	fC (beat·min^{-1})
1	53	4373	66.7	199	70	164
2	36	3966	69.6	189	62	138
3	34	3754	66.5	210	58	143
4	33	3673	66.2	196	59	149
5	33	3405	59.7	203	*	*
6	32	4637	71.8	194	62	142
Mean ± SD	36.8 ± 7.3	3968 ± 420	66.7 ± 3.7	198.5 ± 6.7	62 ± 4	147 ± 9

* Indeterminate value. $\dot{V}O_{2max}$: maximal oxygen uptake; fC: heart rate; AT: anaerobic threshold.

(6,25), it is, however, absolutely necessary to meet minimal oxygen requirements in certain conditions, such as those presented in extreme altitude where levels almost incompatible with life are reached (37). Given that the $\dot{V}O_{2max}$ gradually decreases with altitude-hypoxia (5,6,35), elevated levels of $\dot{V}O_{2max}$ at sea level should allow a better aerobic metabolism and performance at extremely high altitudes (6,29). It is possible that a minimum sea-level $\dot{V}O_{2max}$ between 49 and 61 mL·min^{-1}·kg^{-1} is necessary to reach the summit of Mt Everest without supplementary oxygen (29).

With regard to the studies carried out to date that assess the functional capacity of Himalayan highlanders, we would like to emphasize that: 1) with the exception of the step testing (5,17), and walking uphill with load performed on Sherpas (31), or cycle-ergometer in Tibetans (34,38), the other studies were performed with nonspecific ergometric devices such as the cycle-ergometer in Sherpa natives (21,27); 2) apparently contradictory values of $\dot{V}O_{2max}$ have been obtained at low altitude (17,18,31) or even at sea level (5,21), when compared with several of those obtained at high altitude (21,27,34) where lower values should be expected. Pugh et al. (27) obtained a $\dot{V}O_{2max}$ of 43 mL·min^{-1}·kg^{-1} in one Sherpa tested at 5,800 m using a cycle-ergometer; Lahiri et al. (21) reported values of 49 and 46 mL·min^{-1}·kg^{-1} in two Sherpas at 4,880 m by means of the same ergometer; and Sun et al. (34) reported values between 50 and 58 mL·min^{-1}·kg^{-1} in nine Tibetans tested in the same manner at 3,658 m. Likewise, high $\dot{V}O_{2max}$ values have also been found in Andean Amerindians tested at high altitude: Vogel et al. (36) reported values of 53 and 57 mL·min^{-1}·kg^{-1} in two Peruvian Indians tested by means of cyclo-ergometer at 4,350 m; Hurtado (15) informed of functional capacity equivalent to an average of ~55 mL·min^{-1}·kg^{-1} by using a treadmill in 12 subjects of the same Indian group tested at 4,550 m. If we take into account that altitude causes a well-documented exponential reduction of $\dot{V}O_{2max}$ (6,35,37), and that this relationship is approximately the same in highlanders and lowlanders (5), the above-mentioned values obtained between 3,600 and 5,800 m should correspond to a $\dot{V}O_{2max}$-increase of between ~18 and 40% at sea level. Consequently, this fact would suggest that all the aforementioned Himalayan and Andean highlanders could have high values of oxygen uptake during maximal exercise at sea level. However, some of these isolated cases reported in the literature may be applicable to very select individuals, as has been previously stated (5).

On the other hand, in all studies carried out previously, specifically on Sherpa natives at low altitude—two subjects tested at 2,100 m (31), five subjects at 1,300 m (17), three subjects (5), and one subject (21) at sea level—$\dot{V}O_{2max}$ values similar to those observed in climbers of lowland dwellers have been reported. Although there are several exceptions that have shown high $\dot{V}O_{2max}$, the most frequent value in Western high-altitude climbers of similar age ranged between an average of 50 and 60 mL·min^{-1}·kg^{-1} (8,25,29). However, it is noteworthy that in three of these studies performed on Sherpas, the natives did not reach their theoretical maximum functional values of ventilation or heart-rate response (17,21,31). The higher maximal aerobic power found by us could very well be one of the physiological variables that contribute to the high performance of this select group of elite native climbers tested. This fact could be attributable, partially, to their exceptional athletic endurance (this group boasts the best climbing background of all the present Sherpa population) and also perhaps to the treadmill test never previously performed on Sherpas, who are rural highlanders not accustomed to cycling because of the nature of their local terrain. In addition to this, $\dot{V}O_{2max}$ has been reported as 20% higher for treadmill testing than the cycle ergometer in mountain climbers both before and after one high-altitude expedition (8). However, the marked *genu-valgum-recurvatum* and flat foot condition presented by all the subjects tested by us, which produces a peculiar walking and running style on a smooth surface (personal observation), probably contributed to a certain efficiency limitation on the ergometer especially during the final phase of the exercise test. To support this observation, a greater mechanical efficiency of the Himalayan Sherpas and Andean Quechuas in their natural environment has been suggested (15,31). This fact could explain the high oxygen-uptake levels shown for each stage of the test (Fig. 1).

In addition to the aerobic power and cardiac chronotropic reserve shown by this select group at sea level, their high performance on the mountain could be due to: 1) the greater utilization of glucose rather than fatty acids

as energetic substrate, as demonstrated by Hochachka et al. (12,13), which allows a larger production of ATP per volume of oxygen consumed; 2) the higher ventilatory efficiency particularly at high altitude (5,21,27); 3) the existence of a higher anaerobic threshold—which in our study is elevated in comparison with those individuals who do not follow regular and intensive training and show a threshold of between 40 and 60% of the $\dot{V}O_{2max}$ (32)—that can reach levels almost as high as the $\dot{V}O_{2max}$ detected in the average results shown by the other studies performed on Sherpas previously at low altitude (5,17,21,31). This high anaerobic threshold can be of special value given that, although the Sherpa natives generally exercise at relatively low work intensity, they do so in a hypoxic environment.

As regards the implications of the metabolic threshold, it is worth pointing out that: 1) we calculated the ventilatory anaerobic threshold, which should correspond to a concentration of serum lactate of around 2 $mmol \cdot L^{-1}$ (9); 2) given the enzymatic modifications of the highlanders that attenuate the production of lactate (14), it is presumable that they could sustain, in a stable manner, levels of physical exercise substantially higher than those corresponding to the ventilatory anaerobic threshold. It is well-known that exercise intensities producing plasma lactate levels below 4 $mmol \cdot L^{-1}$ can be sustained for long periods of time (22).

The aerobic qualities, maximal oxygen uptake, and anaerobic threshold found in our study are even more striking if the following factors are taken into account: the age of the subjects, their absence of training program, and, as reported, the relatively low intensity of their routine physical activity at high altitude since childhood which reduces the speed of task-execution, compensating for the environmental hypoxic conditions (26). However, we cannot reject the possibility of the existence of a special genetic endowment in Himalayan (19,23) and Andean ethnic groups (13). This assumption is borne out by the existence of higher blood-oxygen saturation at birth and during the first months of life reported in Tibetans compared with Chinese Han descendants born at the same altitude (24). Furthermore, the $\dot{V}O_{2max}$ values reported for the adult population of both ethnic groups living in Lhasa (Tibet, 3,658 m) were shown to be higher in Tibetans (34,38).

Also noteworthy are the very high maximal cardiac frequencies reached, taking into account the age of the members studied by us. It is well known that a few days of exposure to hypobaric hypoxia induces a decrease in the maximal chronotropic cardiac response to exercise, as is well documented for lowland dwellers (1,11), as well as in Sherpa natives exposed to an altitude higher than that of their habitual residence (5). This appears to be due to a reduction in the density of myocardial β-adrenoceptors (2), adenosine receptors (1), and to an increased density of muscarinic receptors (16). Such cardiac chronotropic "downregulation" is rapidly reversible upon return to lower altitudes, at least, in Caucasian lowland dwellers exposed to chronic hypoxia (28). However, a long-term exposure to high altitudes tends to bring the maximal chronotropic cardiac response back to normal (1,21). The elevated maximal heart rate observed in Himalayan highlanders has been well documented by several studies (18,27,38), which have even detected maximal cardiac frequencies of 210 bpm in two 24-yr-old (mean) Tibetans tested at an altitude of 3,658 m (34), and 195 bpm in one 42-yr-old Sherpa at 4,880 m (21). Although more studies are needed to understand what really happens at the level of myocardial receptors in native highlanders transferred to sea level, our results support the idea that the corresponding maximal heart rates detected during the exercise test could be similar to those reached by them during maximal effort levels when climbing at high altitude. Nevertheless, we cannot rule out the possibility that a certain "rebound effect" could take place after the rapid withdrawal from chronic exposure to hypoxia.

In conclusion, our results appear to be in accordance with some other studies performed on highland ethnic groups tested at high altitude. However, the present work is novel in that it reports for the first time on six elite Sherpa climbers transferred to sea level who showed both high aerobic capacity and very high maximal heart rate. Although these findings have been obtained in a very select sample of the Himalayan population, determining respiratory gas exchange by means of state-of-the-art technology and applying a relatively specific stress test protocol, we believe that they may provide valuable information as to the metabolic reserve shown by some professional climbers of the Sherpa community. This report supports the idea that natural selection and long-training in a hostile environment enable some humans to reach the world's highest summits repeatedly without the use of supplementary oxygen.

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RESPIRATION PHYSIOLOGY

Normoxic ventilatory response in lowlander and Sherpa elite climbers

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Abstract

The differences in ventilatory response to exercise of some highland ethnic communities is a controversial issue. We have evaluated the differences in ventilatory response to exercise at sea level between two groups of elite climbers, four Himalayan Sherpas (S) and four Caucasian lowlanders (C), after descent from extreme altitude. All of them performed a progressive-intensity exercise test on a treadmill under normoxic conditions. Pulmonary gas exchange was obtained until exhaustion by means of an automatic gas-analyzer system. Significant differences in expired ventilation and carbon dioxide production were found between the two groups, the $\dot{V}E \cdot \dot{V}_{O_2}^{-1}$ being lower in the S at rest (41.9 ± 5) in comparison with C (48.7 ± 9) ($P < 0.05$), higher at medium loads of the test ($S = 28.2 \pm 4$ vs. $C = 25.7 \pm 2$; $P < 0.05$) and reaching similar values at higher loads ($S = 34.5 \pm 2$ vs. $C = 35.6 \pm 4$; NS). We conclude that the special ventilatory response observed in these highlanders could explain their adaptation to altitude, allowing higher oxygen blood saturation at medium working loads and reducing the risk of neurological injury caused by a high ventilatory response when exercising at high intensity effort under extreme altitude environment.

Keywords: Acclimatization, high altitude, exercise; Altitude, exercise, highlanders vs. lowlanders; Exercise, high-altitude acclimatization; Gas exchange, pulmonary; Mammals, humans

1. Introduction

Increase in pulmonary ventilation is the primary response to high altitude and constitutes one of the initial phenomena determining acclimatization to a hypoxic environment. A higher hyper-

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ventilatory response has been related to a lower incidence of acute mountain sickness episodes (Hackett et al., 1982), and appears to be associated to greater success when climbing at extreme altitude (Schoene, 1982). The degree of this ventilatory response to hypoxia shows a profile that varies among both individuals and ethnic groups (Hackett et al., 1980), demonstrating a clear genetic basis through studies performed in twins (Kawakami et al., 1982). Nevertheless, while ethnic groups well-adapted to high altitude present a blunted hypoxic ventilatory response (Lahiri, 1984), the study performed with the largest sample of elite climbers from the Sherpa ethnic community shows a brisk acclimatization response, hyperventilating at both low and high altitudes (Hackett et al., 1980). However, these findings contradict data obtained in other studies performed specifically in Sherpa highlanders (Lahiri et al., 1967; Santolaya et al., 1989). It must be noted that in lowland dwellers, acclimatization changes persist for at least 35–40 days after completing a Himalayan expedition (Masuyama et al., 1986), and it seems that years of exposure to sea level conditions are necessary to restore normal ventilatory response to hypoxia in native highlanders (Lahiri, 1984).

The main purpose of the present study is to determine whether there are differences in ventilatory response to exercise under normoxic conditions at sea level between two different groups of elite climbers, Sherpa highlanders and Caucasian sea-level residents, shortly after a ascent to > 8000 m in the Himalayas.

2. Materials and methods

2.1. Subjects

Eight male climbers, four Himalayan Sherpa (S) and four Caucasian lowlander (C), were chosen based on their recent exposure to extreme altitude, their outstanding climbing background, and their similar age (mean ± SD) S: 38.7 ± 8.3 years old; C: 32.7 ± 5.5 years old).

The group of S climbers (height 163.6 ± 6.5 cm; body mass 60.9 ± 3.9 kg) were born at an altitude of ≈ 3800 m (three natives) and at ≈ 2500 m (one native), and had spent most of their life at altitudes ranging from 2500 to 4800 m in the Solu-Khumbu Everest area of north-eastern Nepal. None of them had any known low-altitude progenitors. Climbs to > 6000 m were quite common for each of them and, as a whole, they had participated in 56 expeditions to > 7000 m, 39 of which were to > 8000 m. On 24 occasions they had reached the top of the highest peaks, including 12 ascents to the summit of Mount Everest (8848 m). A total of 49 days before the study, three of them had reached the summit of Mt. Everest and the other native had climbed to 8100 m. They had never used supplementary oxygen during their climbs. None of them had followed a physical training program, other than their usual participation in alpine expeditions once or twice a year. They were all non-smokers.

The group of C climbers (height 175.7 ± 7.2 cm; body mass 65.2 ± 8.3 kg) were born and usually lived at sea level. As a whole, they had participated in 16 expeditions to > 7000 m, 11 of which were to > 8000 m. On seven occasions they had reached the top of the highest summits, including three ascents to Mt. Everest. Also 49 days before the study (the same period applied to the S group) two of them had reached the summit of Mt. Everest, one had climbed to 8758 m, and the other had reached an altitude of 8300 m. Only one of them had climbed the final summital meters of Mt. Everest using supplementary oxygen. All of them followed a regular physical training program (climbing, cross-country skiing, cycling and/or jogging). They were all non-smokers.

Both groups (S and C) withdrew from the Mt. Everest base camp (5350 m) 30 days before the study and spent 6 days hiking to the village of Lukla (2900 m), where they took a plane to Kathmandu (1300 m). Upon arrival at sea level in Europe they were submitted to a routine physical examination and a complete medical history was taken with the help (for the S group) of a native expert translator from the Nepali Consulate in order to rule out eventual pathologies. The subjects were informed of the purpose of the study and the experimental non-invasive procedures to be performed, to which they all granted their

consent (in accordance with the guides of the Human Research Committee of the University of Barcelona, Spain).

2.2. Functional assessment

The study was carried out (5 days after their arrival from Nepal) in a laboratory located at 125 m above sea level (PB $\approx$ 750 Torr), with room temperature ranging between 22 and 24°C, and relative humidity ranging from 55 to 65%. The subjects had not performed any strenuous exercise during the 72 h prior to the test. The evaluation took place in the morning, 3 h after a light breakfast. A standard electrocardiogram (model Schwarzer Cardioscript CD-6000, Picker, Germany) was obtained with the subjects at rest. Afterwards, all subjects completed a progressive-intensity exercise test on a treadmill (model Laufergotest LE-6, Jaeger, Germany) under normoxic conditions until physical exhaustion, following the Bruce protocol (load 1, 2.7 km h^{-1} running speed and 10% slope; load 2, 4 km h^{-1} and 12%; load 3, 6.5 km h^{-1} and 14%; load 4, 7.6 km h^{-1} and 16%; load 5, 8.5 km h^{-1} and 18%). Pulmonary gas exchange was measured using a breath by breath automatic analyzer system (model CPX II, MedGraphics, Sant Paul, MN) equipped with a pneumotacograph (model Hans Rudolph, Kansas City, MO). Before each test, the instrument was calibrated in relation to both volume and flow by means of a 3 L capacity syringe (model Hans Rudolph), and by means of gases obtained from a tank (Medical Graphics, Plumsteadville, PA) containing a reference mixture (5% CO_2, 12% O_2, balanced N_2), and atmospheric air (well-ventilated room). The respiratory parameters recorded were: expired ventilation ($\dot{V}E$, L min^{-1} BTPS), tidal volume (VT, ml), respiratory frequency (fR, min^{-1}), partial pressure of oxygen in end-tidal air (PET_{O_2}, Torr), partial pressure of carbone dioxide in end-tidal air (PET_{CO_2}, Torr), oxygen uptake ($\dot{V}_{O_2}$, ml min^{-1} STPD) and expired CO_2 ($\dot{V}_{CO_2}$, ml min^{-1} STPD). The following data were calculated automatically: oxygen uptake relative to body mass ($\dot{V}_{O_2}$, ml kg^{-1} min^{-1} STPD), respiratory exchange ratio ($RER = \dot{V}_{CO_2} \cdot \dot{V}_{O_2}^{-1}$), and ventilatory equivalent for both oxygen ($\dot{V}E \cdot \dot{V}_{O_2}^{-1}$) and carbon dioxide ($\dot{V}E \cdot \dot{V}_{CO_2}^{-1}$). A continuous electrocardiogram (model Simpliscriptor EK-31, Hellige, Germany) with instantaneous determination of cardiac frequency (fH) was obtained during the exercise test using an equivalent V5 precordial lead. Oxygen pulse was calculated as $\dot{V}_{O_2} fH^{-1}$ (ml $beat^{-1}$). Cardiorespiratory data average was obtained every 30 sec and the same was done during exhaustion, rejecting the maximum peak values obtained. As the S had never been subjected to a physical exercise test, they were invited to walk on the treadmill for a few minutes wearing the mask prior the test, in order to become familiarized with the experimental procedure. After a 30 min resting period in the laboratory, and before the beginning of the exercise test, the subjects were connected to the gas analyzer for 3 min with the aim of obtaining stable cardiorespiratory basal data and achieving the appropriate equilibrium in the gas-exchange tubes.

2.3. Statistics

In order to evaluate the influence of race and different work loads (independent variables) upon cardiorespiratory adaptation to the different work loads (dependent variable) all data were analyzed by means of analysis of variance (ANOVA), with a confidence level of 95%. Statistical significance was considered when $P < 0.05$.

3. Results

Upon carrying out the intergroup comparison of several variables we have observed a different response pattern to the increasing work loads. Table 1 shows a greater $\dot{V}E$ during the first stages of the stress testing in the S group, which decreased, in comparison to the C group, during both the 4th and 5th stage, with statistically significant differences ($F = 6.7$, $P < 0.001$). $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ showed a pattern similar to that described for $\dot{V}E$, with statistically significant differences for both variables between the two groups ($\dot{V}_{O_2}$: $F = 8.2$, $P < 0.001$; $\dot{V}_{CO_2}$: $F = 4.0$, $P = 0.001$). If we relate the $\dot{V}E$ and $\dot{V}_{CO_2}$ to the body mass in both groups we appreciate the lack of significant differ-

Table 1
Data (mean ± SE) of expired ventilation ($\dot{V}_E$, L min^{-1}; BTPS), oxygen uptake ($\dot{V}_{O_2}$, ml·min^{-1} STPD), expired carbon dioxide ($\dot{V}_{CO_2}$, ml·min^{-1} STPD) and ventilatory equivalent for oxigen ($\dot{V}_E \cdot \dot{V}_{O_2}^{-1}$)[a]

Stage	0	1	2	3	4	5
$\dot{V}_E$						
Sherpas	18.4 ± 2	36.5 ± 5	47.8 ± 8	59.3 ± 15	94.6 ± 26	135.5 ± 24
Caucasians	14.5 ± 3	29.4 ± 7	38.8 ± 9	60.7 ± 12	110.7 ± 19	155.8 ± 26
Significance	S	S	S	NS	S	S
$\dot{V}_{CO_2}$						
Sherpas	435 ± 8	1154 ± 204	1630 ± 184	2132 ± 556	3452 ± 884	3928 ± 754
Caucasians	258 ± 9	766 ± 156	1228 ± 308	2127 ± 414	3747 ± 501	4795 ± 697
Significance	S	S	S	NS	S	S
$\dot{V}_{O_2}$						
Sherpas	448 ± 87	1340 ± 157	1715 ± 184	2095 ± 431	3048 ± 566	3905 ± 539
Caucasians	313 ± 112	1020 ± 221	1535 ± 315	2360 ± 463	3508 ± 493	4394 ± 686
Significance	NS	S	S	NS	S	S
$\dot{V}_E \cdot \dot{V}_{O_2}^{-1}$						
Sherpas	41.9 ± 5	27.2 ± 2	27.7 ± 3	28.2 ± 4	30.5 ± 3	34.5 ± 2
Caucasians	48.7 ± 9	29.2 ± 5	25 ± 2	25.7 ± 2	31.7 ± 4	35.6 ± 4
Significance	S	NS	S	S	NS	NS

[a] Detected in the two groups of climbers at rest and throught the stages of Bruce stress testing. S, $P<0.05$; NS, $P>0.05$).

ences between S and C regarding the maximal $\dot{V}_E$ (L kg^{-1}) ($F=7$; $P<0.001$) but the persistence of significant differences ($F=4.9$; $P<0.001$) regarding the maximal $\dot{V}_{CO_2}$ (ml kg^{-1}). $\dot{V}_{O_2}$max were 3905 ± 539 ml min^{-1} (66.5 ± 5.1 ml kg^{-1} min^{-1}), and 4394 ± 686 ml min^{-1} (68.6 ± 2.8 ml kg^{-1} min^{-1}) for the S and C, respectively (differences statistically non-significant).

There were also differences in fH, with higher frequencies in the S group during the first two stages, but leveling off in the last three work loads (Fig. 1). The fH response was statistically different between both groups ($F=7.8$, $P<0.001$). The fR also showed a statistically significant difference in both groups, being always higher in the S group ($F=7.8$, $P<0.001$).

The $\dot{V}_E \cdot \dot{V}_{O_2}^{-1}$ ratio, likewise, showed different statistically significant evolutions ($F=8.2$, $P<0.001$). This was also the case for $\dot{V}_E \cdot \dot{V}_{CO_2}^{-1}$ ($F=17.2$, $P<0.001$). The S group showed significantly lower $\dot{V}_E \cdot \dot{V}_{O_2}^{-1}$ values at rest respect to the C group, higher at 2nd and 3rd stages and achieving similar values during 4th and 5th stages. In both cases the curves for S and C are similar, but the values at the points corresponding to the same work loads are different, making the differences statistically significant (Fig. 2).

4. Discussion

The major finding of this study was the different ventilatory response to exercise under normoxic conditions shown by two elite-climber groups from different ethnia and residing at different altitudes who were transferred to sea level after exposure to extreme altitude. To our knowledge, to date there are few studies dealing with pulmonary function in Sherpa highlanders studied specifically at low altitude (Hackett et al., 1980; Kayser et al., 1991), and even fewer involving isolated cases of natives concerning to this ethnic community transferred to sea level (Lahiri et al., 1967; Cerretelli, 1980). We are not aware of any comparative studies of functional capacity between elite climbers of this Himalayan ethnia and Caucasian lowlanders tested at sea level with modern technology.

In our study the initial relatively higher ventilation was observed in S even after a short trial to

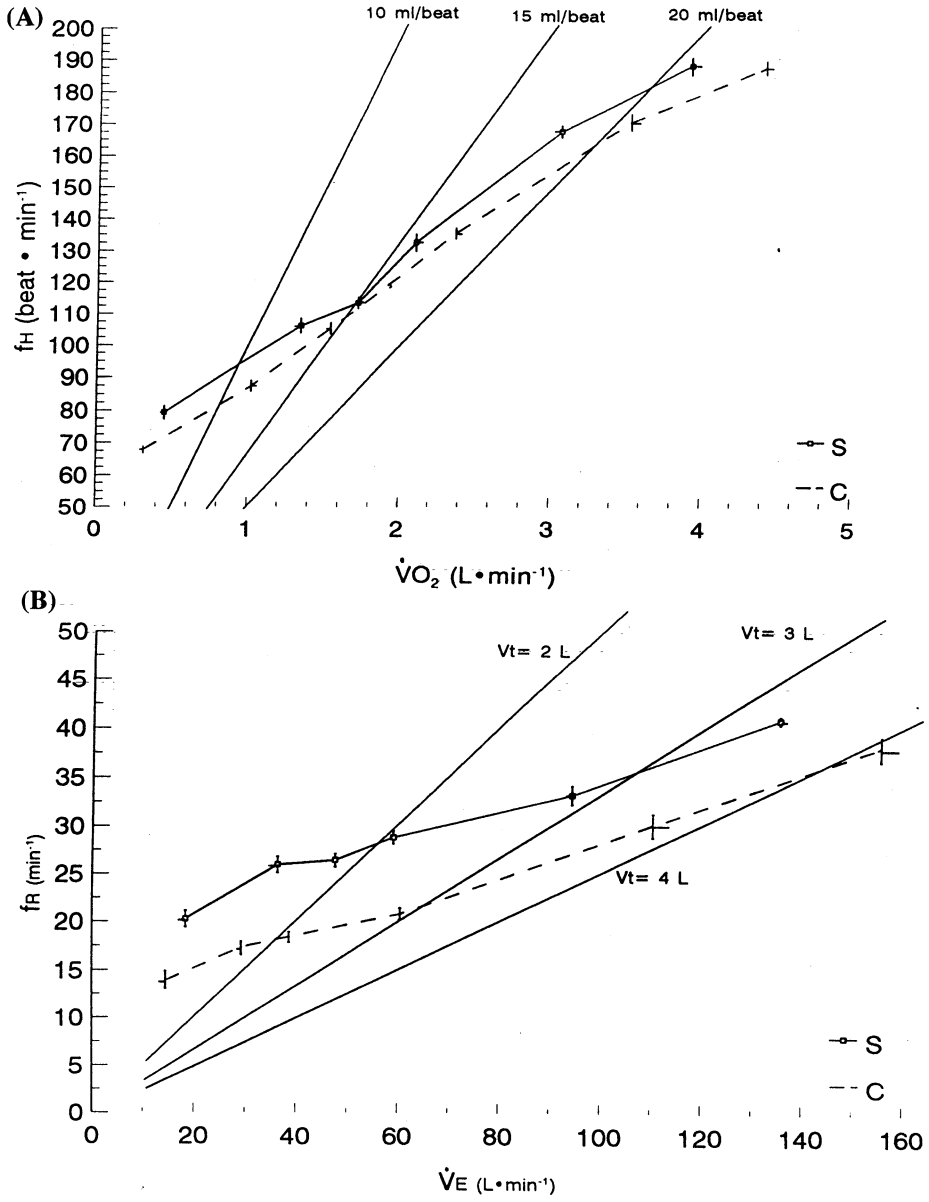

Fig. 1. (A) Evolution of cardiac frequency (f_H) versus oxygen uptake ($\dot{V}_{O_2}$, ml min^{-1}) and (B) evolution of respiratory frequency (f_R) versus expired ventilation ($\dot{V}_E$, L min^{-1}) throughout the exercise test. Values for S (continuous line) and C (discontinue line) expressed in mean and SE. Isopleths of O_2 pulse ($\dot{V}_{O_2} \cdot f_H^{-1}$, ml $beat^{-1}$) and tidal volume (V_T, ml) in the upper and lower panel, respectively.

familiarize them with the experimental procedure and after a 30 min resting period in a friendly atmosphere, talking to them by means of the always present translator. No emotional stress or psychological tension was detected. Also the fact that S showed lower resting $\dot{V}_E \cdot \dot{V}_{O_2}^{-1}$ than C precludes the possibility of emotional hyperventilation, for it is well known that this factor can increase this parameter. The different ventilatory response to exercise shown by the S with respect

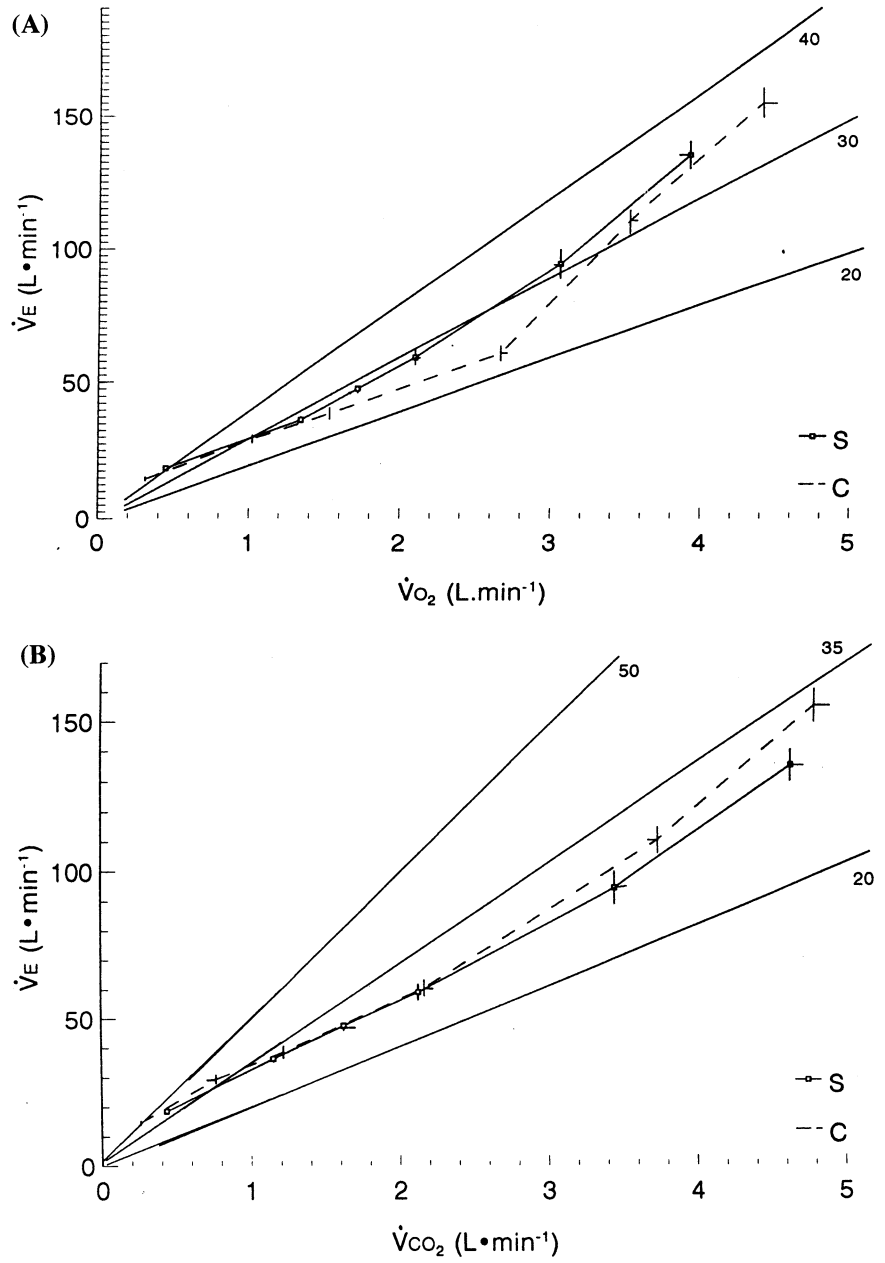

Fig. 2. (A) Evolution of expired ventilation ($\dot{V}_E$, L min^{-1}) versus oxygen uptake (V_{O_2}, ml min^{-1}) and (B) evolution of ($\dot{V}_E$, L min^{-1}) versus expired carbon dioxide ($\dot{V}_{CO_2}$, ml min^{-1}) throughout stress testing. Data for S (continuous line) and C (discontinue line) expressed in mean and SE. Isopleths of $\dot{V}_E \cdot \dot{V}_{O_2}^{-1}$ and $\dot{V}_E \cdot \dot{V}_{CO_2}^{-1}$ in the upper panel and lower panel, respectively.

to C in this study takes place essentially during walking at medium work loads when the differences in $\dot{V}_E \cdot \dot{V}_{O_2}$ become more evident, showing the S a higher ventilation—giving place to a higher haemoglobin oxygen saturation at the more common physical efforts during climbing.

We have detected a high $\dot{V}_{O_2}$ max value in both ethnic groups. This is indeed surprising in

Himalayan highlanders for previous studies which have shown $\dot{V}_{O_2}$max not to be excessively high at low altitudes (Lahiri et al., 1967; Cerretelli, 1980; Kayser et al., 1991). Paradoxically, high $\dot{V}_{O_2}$max have also been found in Himalayan dwellers at high altitude (Lahiri et al., 1967; Sun et al., 1990) where lower values should be expected due to the well-documented exponential reduction of $\dot{V}_{O_2}$max with altitude (Cerretelli, 1980). It has also recently been reported high $\dot{V}_{O_2}$max values in S transferred to sea-level (Garrido et al., 1997). Our findings can be related to the specific stress test performed in the present study and the group of elite Sherpa climbers selected for their extraordinary climbing background. The mean $\dot{V}_{O_2}$max value observed in the lowlanders of this study was slightly higher than the average values reported for Europeans climbers who participate in Himalayan expeditions (Oelz et al., 1986; Richalet et al., 1988), a fact that could be related to the habitual aerobic training of the lowlanders included in our study.

In relation to other metabolic variables measured in this study, we can add that the lower $\dot{V}_E \cdot \dot{V}_{CO_2}^{-1}$ showed by the S is probably due to a lower degree of alveolar ventilation/gas-flow unevenness, which can be determined non-invasively by the different $\dot{V}_E \cdot \dot{V}_{CO_2}^{-1}$ (Wasserman et al., 1987). This is not due to a lower functional dead space, since the Sherpas show a greater increase in respiratory rate to increasing work loads. Nor is it due to a greater arterial carbon dioxide pressure—which would determine a greater alveolar CO_2 tension—since the S show higher ventilation at low working loads.

A brisk ventilatory response to high altitude confers some advantages: lower incidence of acute mountain sickness or other altitude-related complications (Hackett et al., 1982) and better performance at extreme altitude (Schoene, 1982), which are basically attributable to a greater haemoglobin oxygen saturation, as a result of hyperventilation. Nevertheless, this brisk response could have adverse effects such as hypocapnea-induced brain ischemia, whose symptoms can be reversed through inhalation of CO_2-rich gases (Harvey et al., 1988). Moreover, brisk ventilatory response to hypoxia—in our study the C showed significantly higher $\dot{V}_E \cdot \dot{V}_{O_2}^{-1}$ at rest than the S—is necessary to produce sleep periodic breathing (Lahiri et al., 1983), which results in profound hypoxemia levels during periods of nocturnal apnea, repeated short episodes of severe hypoxia probably being more detrimental than low average oxygen blood pressure (Milledge, 1986). It seems to be well demonstrated that a greater hyperventilatory response to hypoxia correlates with more residual impairment of the central nervous system (Hornbein et al., 1989), which is probably related to structural damage to certain brain territories observed in high-altitude climbers (Garrido et al., 1993, 1995).

Finally, we would suggest that the ventilatory response of the type shown in this study by the S could have definite advantages since a greater ventilation at a moderate exercise-intensity would allow maintenance of higher oxygen blood saturation. Nevertheless, this was not the case during maximal exercise testing. This fact, in addition to other physiological changes in oxygen transport observed in Himalayan natives such as minor polycitemic altitude response (Samaja et al., 1979), absence or minimal nocturnal Cheyne–Stokes breathing pattern (Lahiri et al., 1983) and higher arterial oxygen saturation since birth (Niermeyer et al., 1995), could contribute to explanation that clinical and structural cerebral repercussions on S are minimal, despite their repeated ascents to extremely high altitudes without supplementary oxygen, as we previously reported (Garrido et al., 1996).

In conclusion, after an extreme altitude exposure and during the deaclimatization period, Himalayan Sherpas and lowlander elite climbers showed differentiated ventilatory response at rest and during medium intensity exercise under normoxic conditions at sea level. According to $\dot{V}_E \cdot \dot{V}_{O_2}^{-1}$, the lowlanders group showed higher resting pulmonary ventilation, the Sherpa natives showed higher ventilation at medium work loads and similar ventilatory adaptation during high exercise-intensities. This profile of response could favor performance at moderate physical effort and might prevent brain injury during vigorous efforts performed at extreme altitudes, specially in those himalayan highlanders who repeatedly conquered the worlds highest summits.

Acknowledgements

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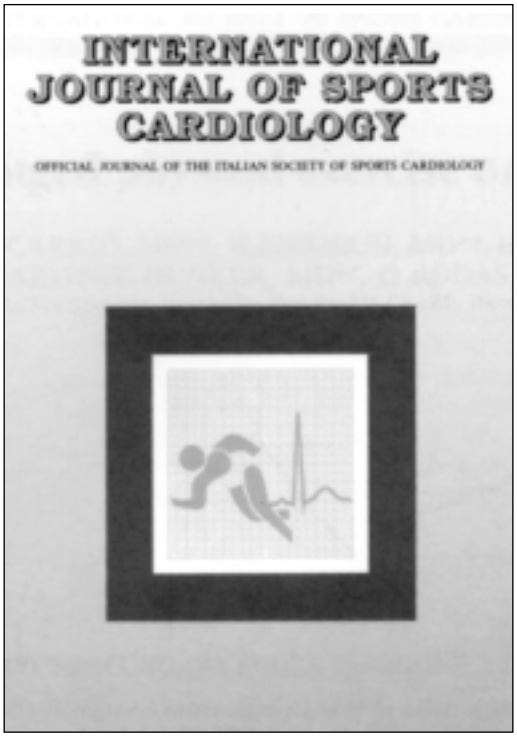

The effect of prolonged physical exercise on ventricular function

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EFFETTO DELL'ESERCIZIO FISICO PROLUNGATO SULLA FUNZIONE VENTRICOLARE

Il significato di alcune alterazioni della funzione ventricolare rilevate in atleti sottoposti ad uno sforzo prolungato è ancora incerto. In questo studio su 10 maratoneti di età media 39.6 anni, gli Autori hanno voluto valutare le modificazioni di alcuni parametri cardiaci attraverso l'esecuzione di una ventricolografia radioisotopica a riposo e subito dopo una competizione ufficiale. Tutti gli atleti erano ben allenati (70-150 km a settimana) ed esenti da cardiopatie. In ognuno furono presi in considerazione, prima e dopo sforzo, la pressione arteriosa, la frequenza cardiaca, il peso, la frazione di eiezione del ventricolo sinistro, il max Dv/Dt sistolico e diastolico, il Dv/Dt sistolico e diastolico e il time sistolico. Nessuna correlazione significativa fu rilevata tra i parametri esaminati eccetto per un interessante e significativo ($p < 0.005$) allungamento del time sistolico (%) dopo esercizio, rispetto ai valori basali, e per un lieve incremento della frequenza cardiaca ($p < 0.005$). Il rilievo di tale alterazione a carico della funzione sistolica del ventricolo sinistro è di dubbia interpretazione e lascia ancora aperti molti interrogativi sull'eziopatogenesi del fenomeno.

Int J Sports Cardiol 1992; I: 79.

Parole chiave: **Maratoneti, Funzione ventricolare sinistra, Scintigrafia miocardica.**

The long-distance runner follows a program consisting of a great volume of training over long periods of time. This is essential in order to be able to stand up to the effort involved in running non-stop for two hours, in the most varied atmospheric conditions (cold, humidity, wind, etc).

The type of work performed is mainly dynamic during most of the race, and intensity ranges between 70% and 90% of VO_2 max. The type of work performed during this activity can not therefore be considered as strenuous, except for the last minutes, when the physical output reaches a maximum.

In the well-trained long-distance runner the gradual loss of physical capacity, especially in the last 10 km, is a consequence of several circumstances, including psychological, energetic-metabolic, and hypohydration factors.

It has been demonstrated that there is a significant rise of enzymes due to injury and to muscular wear, thus leading us to question whether other structures, such as the myocardium, may be injured.

The aim of this study was to evaluate left ventricular function in well-trained marathon runners with considerable experience in this type of competition.

Supported by a grant from the Secretaria Gral de l'Esport de la Generalitat de Catalunya.

METHODS

We studied a group of 10 marathon runners aged between 32 and 47 years (mean 39.6 years). All athletes had participated in at least one official marathon race (42.195 km) and ran between 70 km to 150 km (112 miles-240) per week, with periods of greater activity immediately prior to competitions (Tab I).

Multigated equilibrium blood pool imaging was performed after in vivo labelling of red blood cells with 20

Table I

Age (years)	Weight loss (g)	Km/week (miles)	Racing time (min)
38	1650	150 (83)	150
32	2100	120 (74)	150
38	1900	90-110 (55-62)	150
47	2900	100 (62)	150
41	1000	70-80 (43-55)	150
45	2400	100 (62)	150
35	1900	90 (55)	150
47	1950	100 (62)	150
34	1700	110 (68)	150
39	1000	80-90 (49-55)	150

mCi of technetium -99 m sodium pertechnêtate according to a standard technique[1]. Resting equilibrium gated blood pool scintigrams were performed supine.

The immediately post-effort ventriculography was recorded following a 90 to 150 min race performed between 3.00 and 6.00 in the afternoon. The climatic conditions included temperature of 21°C and humidity of 70%. The rhythm of the race varied according to each athlete's technical preparation and training. The number of kilometers covered varied between 25 and 35 km over land with great many gradients, making it difficult to keep the rhythm of the race regular.

No athlete drank water or other liquid during the race although they were free to do so. Blood pressure, heart rate and body weight were recorded before and after the race.

The parameters evaluated in a basal situation and post-effort were left ventricle ejection fraction, max systolic Dv/Dt, max diastolic Dv/Dt, time of systolic and diastolic Dv/Dt and systolic time.

RESULTS

Data concerning the different parameters evaluated both at rest and post-exercise are shown in table II. There was no significant difference between ejection fraction at rest and immediately after exercise.

There was a significant difference between the basal heart rate and mean heart rate 10 min after exercise. The most significant finding in this study was the lengthening of the maximum systolic time immediately post-exercise ($p < 0.005$) (%) (Fig 1).

The decrease in body weight varied between 1 and 2.9 kg. Such loss is due to perspiration and pulmonary ventilation. None of the athletes showed signs of extreme exhaustion after the race.

DISCUSSION

The intensity of work performed by long-distance runners during training or in competition, oscillates between 70 and 90% of VO_2 max. Work levels of greater intensity can not be maintained for long periods, since exceeding the aerobic threshold leads to progressive accumulation of lactic acid and consequent exhaustion. In order to study this phenomenon is not necessary to determine the level of lactic acid as it is sufficient to know the threshold heart rate previously determined by effort test[2]. By strenuous we mean the type of work demanding maximal physiologic tolerance to exercise, usually of short duration.

Unless there are other complications, physical capacity in marathon runners is most frequently limited by muscle exhaustion. ECG monitoring during this type of competition shows that heart rate rarely exceeds the anaerobic threshold[3]. If this does occur, it lasts only a brief period and metabolic balance is immediately restored without performance being affected in any way.

It is of great interest to determine whether during prolonged effort is it possible to detect any cardiovascular functions of effort claudication. Seals et al[4] analyzed ventricular function by echocardiography in a group of healthy sedentary individuals who performed exercise of an intensity reaching 70% maximum VO_2. Under these conditions a decrease in the fraction of the circumferential fibre shortening is produced.

Niemelä et al[5] show that ventricular function in ultramarathon runners is depressed after having completed the distance between 114 and 227 km. These Authors recommend a strict selection of athletes who wish to participate in this type of competition in order to avoid cardiac fatigue. The decrease in cardiovascular function during prolonged exercise may occasionally produce pulmonary edema, in absence of organic cardiopathy[6].

By means of radionuclide ventriculography during exercise, Foster et al[7] observed a decrease in the ejection fraction in healthy men undergoing stress testing with progressively greater loads until they reached exhaustion.

ECG alterations were observed in some cases, possibly as a consequence of subendocardial ischemia. The myocardium is most vulnerable to the restrictions of normal coronary flow. Tachycardia and pressure overload influence this phenomenon as they are directly linked to the myocardial O_2 intake.

An increase in the systolic period from 39.9% to 56.5% could seem excessive given an increment of heart rate from 55 to 86. However, the increase in relative systolic time observed after the race can also be related to a decrease in peripheral resistances after long-distance running, and subsequent lengthening of the passive phase of the systolic period.

Prior studies have interpreted changes in left ventricular function (decrease in stroke dimension with increase[5] or no change[4] in end-systolic dimension) observed after prolonged exercise as due, at least in part, to a depressed inotropic state. This phenomenon was seen in the 10 athletes who presented diverse liquid loss (Tab II), suggesting a lack of correlation between cardiac preload and the ventricular function.

Maintaining of ventricular function during exercise allows cardiac output to adjust to the energetic demands of the active muscle without systemic and intracardiac pressure being altered[8]. However, on the other hand, a significant decrease may suggest:

Table II. Different parameters evaluated both at rest and post-exercise

	Basal		Post-exercise	
	Mean	SD	Mean	SD
Heart rate	55	6	86	12*
Ejection fraction	63	3	62	5
Dv/Dt systolic	2.9	0.6	3.8	0.8
Dv/Dt diastolic	3.6	0.6	3.8	0.8
Time a Dv/Dt S	136	74	117	38
Time a Dv/Dt D	146	26	169	64
Systolic (%)	39.9	3	56.6	6*

D=diastolic; S=systolic. *$p < 0.005$.

1) a variant from normality as observed in sedentary women with no heart disease[9];
2) the myocardium uses up a great deal of energetic substrates to produce phosphates of high energetic value, especially aerobically. Normally, 60-70% comes from free fatty acids. In intense and prolonged work, glucolisis is transformed into the most important source of high energy phosphate production by means of the aerobic glucolisis[10]. The deficit in the glucose deposits could produce changes in the contractile function of the myocardium;

particular conditions could provoke cardiac fatigue which is immediately seen in the systolic function curve.

In conclusion, it appears that alterations in left ventricular function develop during prolonged effort. Apparently, the change in morphology of this curve has no relation with the cardiac preload.

Although the etiology of this phenomenon is unknown, we can not discard energetic-type factors and/or an increase in the energetic demands from the myocardium which provoke cardiac fatigue and subsequent alterations in the systolic function.

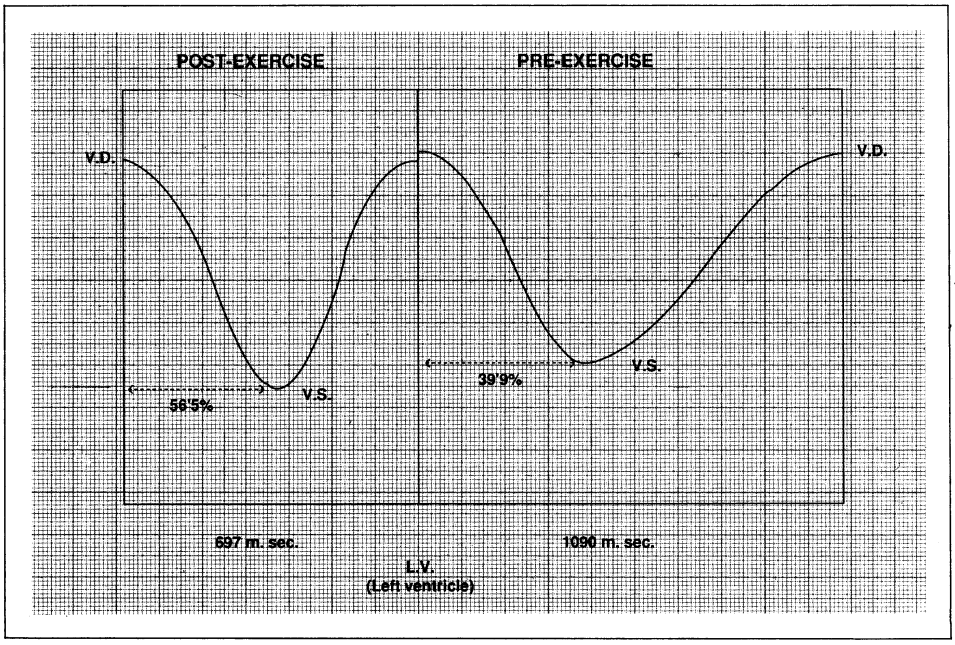

FIGURE 1
Comparison of volume curves pre-exercise (average R-R interval of 1090 msec) and post-exercise (average R-R interval 697 msec). Percentage of systolic time post-exercise was 56.5% and 39.9% pre-exercise ($p < 0.005$). VD= end-diastolic volume; VS= end-systolic volume.

3) the increase in MVO_2 max as a consequence of tachycardia and a greater left ventricular systolic pressure, would provoke a physiological ischemia in zones of the subendocardium with more difficulties for the coronary flow.

One of the limitations in prolonged effort is created by exhaustion of the energetic deposits and metabolic alterations. It can not therefore be ruled out that transient alterations in cardiac function could be related to this phenomenon. None of the athletes had consumed an excess of carbohydrates before the race even though this is frequently the case in order to guarantee sufficient supplies of hepatic and muscle glucogen. These

SUMMARY

The aim of this study is to evaluate alterations of the ventricular function in long-distance runners. A radionuclide ventriculography was recorded at rest and immediately after the athletes ran during 2 hr 30 min. There was a significant difference between basal heart rate and mean heart rate post-exercise. The systolic time was significantly longer after race ($p < 0.005$). There was not a difference between ejection fraction at rest and post-exercise. The etiology of this phenomenon is unknown.

Key words: **Long-distance runners, Ventricular function, Myocardial scintigraphy.**

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Efectes de l'entrenament anaeròbic en el múscul esquelètic

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El múscul esquelètic és un teixit altament adaptable i respon ràpidament a situacions estresants mitjançant mecanismes d'hipertròfia o d'atròfia. La seva plasticitat ve restringida per l'adaptabilitat de les motoneurones i la seva ordenació sinàptica[1,2]. Malgrat tot, les modificacions induïdes al teixit muscular poden ser prou extenses i tracten de ser un reflexe d'allò que les ha provocat. D'aquesta manera, amb un entrenament ben programat, hauria de ser possible millorar característiques musculars específiques, com la velocitat[3]. Però, aquests criteris no es poden generalitzar degut a la considerable diferència poblacional. Komi i cols[4] van descriure que el rendiment anaeròbic, així com les característiques histoquímiques i bioquímiques del múscul exhibien una gran variabilitat interindividual. Edat, sexe, nivell d'entrenament i herència són factors que influeixen fortament en la diversitat entre els individus, tant en el rendiment anaeròbic[5] com en el tamany de les fibres musculars[6] o en les activitats enzimàtiques[7]. Però encara que la component genètica és força gran, el múscul sembla disposar d'un marge discutiblement ampli d'adaptació per poder millorar amb entrenament[8].

ENTRENAMENT ANAERÒBIC

Estructuració de l'entrenament anaeròbic

Contràriament als criteris utilitzats per obtenir millores del sistema aeròbic, on el paràmetre més important de l'entrenament sembla ser el volum de les càrregues, pels anaeròbics la distribució dels períodes d'activitat esdevé decisiva. L'estructura de l'entrenament és clau i petites modificacions en la programació són responsables de diferències significatives en el resultat. Com a components importants del disseny d'un entrenament, la distribució dels períodes de treball i de descans, així com la intensitat a desenvolupar, van ser els primers punts d'estudi[9]. Aquest descans pot modificar l'estratègia muscular d'adaptació, de manera que períodes de descans llargs afavoriran la millora de la glucòlisi anaeròbica, mentre que períodes curts afavoriran la connexió entre la via anaeròbica i el potencial oxidatiu. La resposta adaptativa del múscul dependrà directament de la intensitat, duració i patró temporal de l'activitat física on el descans pren una gran importància[1].

Els criteris concrets a aplicar en cada cas variaran segons la disciplina esportiva per la que es prepari anaeròbicament al múscul. Carrera i ciclisme (normalment en cicloergometre) han estat els models d'exercici més estudiats per avaluar l'efecte d'un entrenament anaeròbic en la fisiologia muscular i la millora del rendiment. Aquestes disciplines esportives són fàcilment reproduibles en condicions de laboratori, on es pot evitar la variabilitat mediambiental.

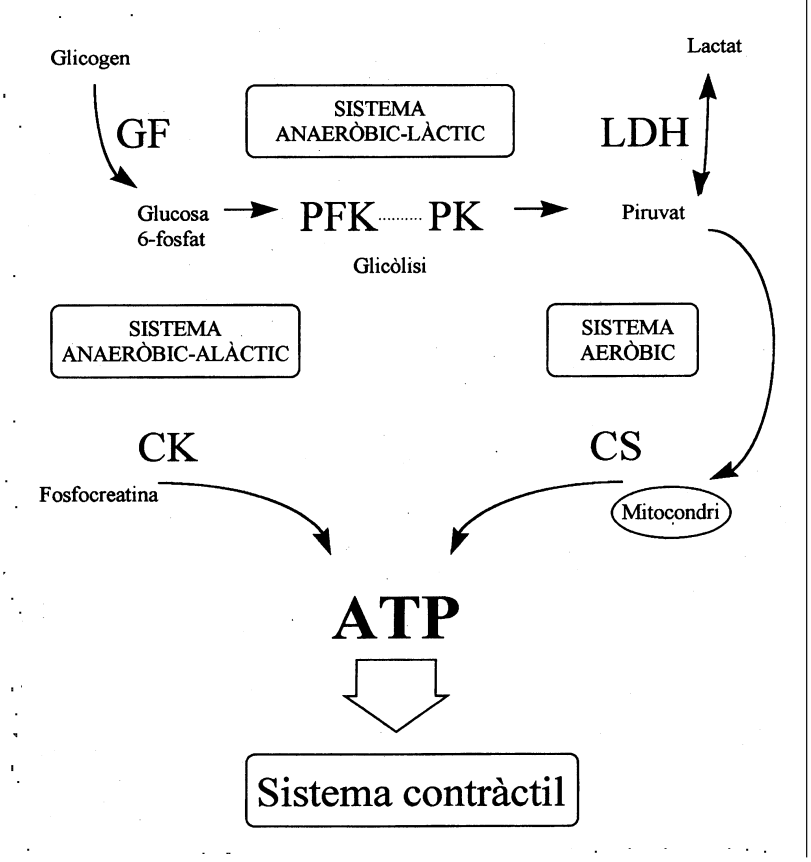
Tipus de descans

El temps necessari per restaurar les condicions basals del múscul és un dels factors més difícil de controlar en tot entrenament anaeròbic i és determinant per poder desenvolupar esforços anaeròbics màxims en exercicis encadenats. La concentració de lactat pot arribar a mantenir-se per sobre del nivell basal més de 20 minuts després de realitzar un exercici intens de curta durada, mostrant la dificultat del múscul per poder retornar ràpidament als seus valors basals[10]. Linossier i cols[11] en cicloergòmetre, així com Balsom i cols[12] en carrera, van trobar variacions en la producció de lactat i en el consum d'oxigen en esforços de molt curta dura-

da, quan s'aplicaven temps de descans variables. Diferències entre 30 o 120 segons de descans representaven una diferència d'un 60% en la producció de lactat i un 20% en l'augment de consum d'oxigen. Aquesta elevada dependència respecte al temps de descans diversifica la resposta muscular a l'entrenament i dificulta de manera extrema l'estudi comparatiu dels protocols d'entrenament i dels seus resultats publicats a la bibliografia (taula 1).

Però, no solament el temps de descans afectarà el rendiment, si no que també ho farà el tipus de descans. La recuperació activa sembla ser la manera més eficaç d'eliminar el lactat acumulat encara que desgasta lleugerament la reserva de glicogen[13,14]. Per altre banda, el descans passiu aconsegueix restaurar el nivell de glicogen muscular encara que no és tan eficaç en la neteja del lactat produït[15]. Conseqüentment, cada tipus de descans afavorirà una adapatció diferents.

Figura 1 Esquema dels mecanismes d'obtenció d'energia. Les abreviatures corresponen a: CK, creatina quinasa; GF, glicogen fosforilasa; PFK, fosfofructoquinasa; PK, piruvat quinasa; LDH, lactat deshidrogenasa; CS, citrat sintasa; ATP, adenosin 5'-trifosfat.



ADAPTACIONS MUSCULARS INDUÏDES PER L'ENTRENAMENT ANAERÒBIC

L'entrenament anaeròbic indueix alteracions que permeten al múscul millorar el seu rendiment. Aquestes adaptacions es produeixen tant a nivell del metabolisme energètic com de les proteïnes constitutives del sistema contràctil.

Efecte sobre el metabolisme

El consum d'ATP en contracció muscular a la màxima intensitat és d'uns 9-10 mmol/Kg dt/seg[16]. Aquest fluxe d'ATP és prou elevat per que solament la producció per via anaeròbica d'ATP hi pugui participar durant els primers instants. Aquesta producció, que com a màxim arribarà a 370 mmol/kg (teixit sec), es veu repartida de la següent manera: 80% a la glucòlisi anaeròbica, 16% a la reserva de fosfocreatina (PCr) i 4% al descens de les pròpies reserves d'ATP. Per poder millorar el metabolisme muscular i d'aquesta manera el rendiment[17] (Hirvonen 87), la fibra muscular ha de patir modificacions a diferents nivells (esquema i abreviatures a la figura 1):

1. Adaptació energètica: Augment en la concentració de substracte energètic tant sigui de consum immediat (fosfocreatina), com a substracte per la glucòlisi (glucosa).
2. Adaptació enzimàtica: Augment en les activitats enzimàtiques implicades en els mecanismes de producció d'energia tant dels enzims de la via alàctica (CK) com de la làctica (PFK, GF, PK, LDH entre d'altres).
3. Adaptació a la acumulació de lactat: Augment en la capacitat tamponadora del múscul que permet

suportar una major producció de lactat i un canvi més fort en el pH intracel.lular.

4. Adaptació aeròbica: Augment de la capacitat aeròbica i del fluxe de producció d'ATP.

Adaptació energètica

La disponibilitat de substracte energètic és un dels principals factors limitants del rendiment. Encara que l'ATP és la molècula energètica del múscul, són la reserva de fosfocreatina en primera instància i la de glucosa-glicogen en segona les que s'encarreguen de subministrar l'energia. La primera de manera directa a través de la reacció de la creatina quinasa i les segones a través de la glucòlisi. Degut a la ràpida conversió de fosfocreatina a ATP, la quantitat de fosfocreatina en reserva sembla involucrada en el rendiment anaeròbic. Encara que l'entrenament de velocitat sembla millorar la utilització de fosfocreatina especialment en les fibres lentes[18], no estar clar que la seva concentració augmenti com a conseqüència d'un entrenament. Per altre banda, s'ha observat que es pot incrementar la seva concentració muscular per ingestió de creatina i que aquest augment de la reserva millora el rendiment anaeròbic i disminueix la producció de lactat[19,20]. Encara que possiblement aquestes millores solament poden ser constatades en exercicis on el mecanisme de la fosfocreatina sigui determinant per l'obtenció l'energia[21].

En treballs anaeròbics d'alta intensitat i curta durada la utilització de glucosa circulant és molt baixa, i és el glicogen muscular el responsable d'aportar la glucosa necessària. Katz i cols[22] van observar que la glucosa externa que s'incorpora al múscul durant un exercici curt i intens és pràcticament despreciable. Sembla ser que el glicogen comença a degradar-se des del primer segon de l'exercici de manera que ja es pot trobar un descens significatiu del 15% després de 6 segons d'esprint[23,24] que pot arribar fins un 20%-30% en exercicis intensos de 30 segons de córrer[24] o de pedalar en bicicleta[26,27]. La concentació muscular de glicogen disminueix sensiblement a gairebé tots els tipus d'entrenament, però la seva restauració és depenent del tipus d'exercici. El desgast produït en una sessió d'entrenament de resistència es recupera normalment en 24 hores, mentre que es necessita unes 48 hores per recuperar el glicogen consumit en un entrenament intervàlic[28,29]. Però, en tots els casos, es troba un increment o "sobrecompensació" de la reserva de glicogen en la fase de repòs[30,31]. L'abast d'aquesta adaptació és depenent del tipus d'entrenament, i també es produeix en entrenaments lleugers de velocitat[23,32]. Curiosament, no s'ha observat un efecte clar de millora del rendiment anaeròbic com a conseqüència de tenir el glicogen sobrecompensat. Vandenberghe i cols[33] van observar que sobrecompensant el glicogen d'un grup de voluntaris amb una dieta rica en carbohidrats i comparant el seu rendiment amb el d'un grup control mitjançant tests anaeròbics no hi va trobar cap diferència en els resultats. Tot sembla indicar que la quantitat total de glicogen no seria un factor energètic limitant degut al fet que es consumeix en un petit percentatge alhora de realitzar un test anaeròbic. Possiblement seran els enzims que el processen els que determinaran la velocitat d'obtenció de glucosa.

Tot i això, no totes les fibres consumeixen glicogen al mateix ritme. Si l'exercici s'inicia amb alta freqüència, molt per sobre del VO_2max, les primeres fibres en perdre glicogen són les ràpides[34] i s'ha vist que hi ha una depleció selectiva, de manera que en primer lloc es consumeix el glicogen en les fibres més ràpides i al final en les més lentes[35,36]. Aquestes fibres ràpides que són les primeres en consumir glicogen també són les primeres en recuperar-lo durant els 90 minuts posteriors a l'exercici intens[37].

Malgrat la millora dels mecanismes de restauració de l'ATP, un entenament anaeròbic intens pot disminuir la quantitat total de nucleotids d'adenina (ATP+ADP+AMP) en múscul, comprometen seriosament el rendiment esportiu[38,39].

Adaptació enzimàtica

Com a resposta a l'estres provocat per l'entrenament, es produeix una adaptació a nivell de proteïna que en part es veu reflectida per un augment en la concentració dels enzims implicats en els mecanismes d'obtenció d'energia. Simbolitzat per la creatina quinasa, la sensibilitat del mecanisme anaeròbic alàctic a un entrenament de velocitat no està gaire clara. Alguns autors[40,41] troben increments significatius en l'activitat de la CK mentre que en altres casos resta invariable a l'entrenament[42,43]. Sembla que l'adaptació de la CK és sensible a algun paràmetre de la programació de l'entrenament que encara no està determinat. De tota manera la seva concentració muscular és molt elevada i fa de la concentració de fosfocreatina el punt més limitant.

La glicogen fosforilasa és l'enzim que subministra glucosa a partir de la reserva de glicogen. Costill i cols[44,45] no van trobar diferències significatives en l'activitat de la GF entre individus sedentaris, atletes de mig fons i atletes de fons, però si van trobar valors més alts de GF i lactat deshidrogenasa en atletes sprinters. La GF sembla ser poc sensible a entrenaments de velocitat de curta durada, i és possible que necessiti períodes llargs d'entrenament per mo-

Taula I Representació esquemàtica d'alguns entrenaments anaeròbics i els seus efectes sobre el múscul (↑ indica augment, ↓indica disminució, = indica falta de canvis significatius). Es poden trobar més tipus d'entrenament a Spriet[104].

Entrenament: Tots els voluntaris són moderadament actius excepte especificació expresa (W: prova de Wingate 30" en cicloergòmetre) a excepció d'aquells que presenten una C (entrenament de córrer), d'una sesió diària i a la màxima intensitat excepte en el cas en que es digui una altra cosa. Abreviatures; set: setmanes d'entrenament, d/set: dies d'entrenament per setmana, rep.: repeticions, rec.: tempsde recuperació, ses/d: sessions por dia.
Metabolits: TAN: quantitat total de nucleotids d'adenina, IMP: Inosina monofosfat, Producció: es refereix durant una prova d'esforç comparativa entre abans i després de l'entrenament.
Enzims: MDH malat deshidrogenasa, HADH 3-hidroxiacil-CoA deshidrogenasa, OGDH oxoglutarat deshidrogenasa, ADK adenilat quinasa, AMPasa AMP desaminasa, HK hexoquinasa, ASAT aspartato aminotransferasa, ALAT alanina aminotransferasa, SDH succinat deshidrogenasa, la resta es troben a la figura 1.

Referència	Entrenamento	Metabolits	Enzims	Tipus de fibres	Consum d'oxígeno	Rendiment
Stathis et al	5 set 3 d/set 3-10 W 4' rec. 2 set 3 d/set 10 W 3' rec.	TAN, ↓Producció IMP			↑	↑
Allemeier et al.[95]	3 set 2d/set 3 W 20' rec. 3 set 3d/set 3 W 20' rec.			=	↑	=
Nevill et al.[103]	8 set 3-4d/set 2 d/set 2x30" 10' rec. 1 d/set 6-10x6" 54" rec. 1 d/set 2-5x2' C 5' rec.	Producció de lactat ↑			↑	↑
Simoneau et al.	15 set 4-5d/set 25ses 30' C contínua 19ses 10-15 rep. 15-30" 16ses 4-5 rep. 60-90"		MDH ↑, HADH, ↑ OGDH ↑, CK=, PFK=, LDH =	I↑ IIb↓ IIa =		↑
Esbjörnsson et al.[93]	6 set 3d/set 15x10" 50" rec. 1 set 2ses/d 3d/set 15x10" 50" rec.			I↓ IIb↓ IIc = IIa ↑		
Hellsten et al.[101]	6 set 3d/set 15x10" rec:50"	TAN ↓	ADK=, AMPasa, ↓ PFK ↑		=	=
Hellsten-Westing et al.[38]	6 set 3d/set 15x10" rec: 50" 1 set 2ses/d 7d/set 15x10" rec: 50"					↑=
Hellsten-Westing et al.[38]	1 set 2ses/d 7d/set 15x10" rec: 50"	TAN ↓				↑=
Linossier et al.[11]	7 set 4d/set 2x8x5" rec:15' i 5" Intensitat variable.	Producció de lactat ↑	PFK ↑, LDH ↑, CS=, HADH=, HK	I↑ II↓	=	↑
Roberts et al.[78]	5 set 3-4d/set 2x4x200m C rec: 10' i 2' Intensitat del 90%		GF ↑,PFK ↑, LDH ↑, MD ↑, SDH=			↑
Cadefau et al.[43]	Atletes velocistes entrenant durant una temporada		GF ↑, GS ↑, LDH ↑, PK↑, PFK ↑, ASAT↑, ALAT ↑,	I↑		=

dificar-se, especialment a partir del moment que l'activitat fosfofructoquinasa ja s'ha incrementat.

La PFK és l'enzim clau i el més regulat de la glucòlisi. També és el més sensible a un entrenament anaeròbic, millorant significativament sota una gran varietat de protocols[32,42,43,44]. Els entrenaments de resistència o clarament aeròbics provoquen una disminució de la seva activitat, fins i tot abans de que es produeixin altres mo-

dificacions muscular més clarament aeròbiques com variacions en el contingut d'enzims al mitocondri[47].

Denis i cols[26] van trobar la mateixa activitat de PFK i LDH en atletes velocistes de 100 metres i en atletes mig fondistes de 800 metres altament entrenats, però els primers tenien menys activitat en marcadors del metabolisme aeròbic com la citrat sintasa (CS, cicle de krebs) i la hidroxiacil-CoA deshidrogenasa (HAD, degradació dels àcids grassos).

Respecte a la LDH, enzim productor del lactat, alguns autors troben augments en la seva activitat després d'entrenaments de curta durada i alta intensitat[11], mentre que d'altre no en troben[41]. Sembla ser que la seva elevada activitat no esdevé limitant i per tant no seria necessària incrementar-la per millorar el rendiment anaeròbic. Aquest fet deixeria aleatoria la seva adaptació. Fins hi tot, alguns autors[48] no han trobat diferències entre els valors corresponents a individus sedentaris i a atletes, encara que en entrenaments de resistència s'ha trobar un decrement de la seva activitat total[49,50].

Adaptació a la acumulació de lactat

Quan el múscul treballa per sota del 60-70% del consum d'oxigen màxim (VO_2max), la producció de lactat és petita i el mecanisme aeròbic és qui s'encarrega en major part del subministrament d'energia. Per sobre d'aquest umbral anaeròbic, el lactat s'acumula proporcionalment a la intensitat de l'activitat física. Aquest increment en la concentració de lactat és conseqüència d'una incapacitat per part del mitocondri de metabolitzar-lo al ritme que és produït[51].

En front d'un mateix esforç, individus entrenats produeixen menys lactat que els sedentaris degut a que tenen el VO_2max més alt i per tant entren més tard en anaerobiosi[52]. També s'ha observat que després d'un entrenament i al repetir el mateix test control, els voluntaris produeixen menor quantitat de lactat, demostrant que el mateix exercici després de l'entrenament no representava igual grau d'esforç[53]. Però, com a resposta a un test màxim de capacitat anaeròbica, el lactat generat és més alt en atletes velocistes que en sedentaris tant si el test és en cicloergòmetre[10,26] com en carrera[54]. Així com també, s'ha observat que en entrenaments de velocitat avaluats per un test realitzat al màxim de la capacitat voluntària, el nivell màxim de lactat en sang presenta un increment entre el valor abans i després de l'entrenament que ve acompanyat d'una millora en el rendiment[55,56,57,58]. Però, no sempre hi ha una bona correlació entre el lactat generat i la marca en competició entre atletes entrenats per velocitat i mig fons[48,59]. Correlació que si van trobar Cheetham i cols[25] entre els descensos de pH en múscul i en sang amb el rendiment d'esportistes durant una carrera de 30 segons a la màxima velocitat, o Granier i cols[10] entre el resultat en un test de Wingate i el lactat generat en atletes velocistes.

Linossier i cols[11] van proposar que aquest augment de producció de lactat amb l'entrenament és conseqüència d'un augment en l'activitat de PFK i LDH. Malgrat tot, no tots el exercicis són purament anaeròbics provocant que algunes correlacions entre lactat i rendiment siguin baixes[60].

Per evitar una acumulació excesiva, el lactat és eliminat per via sanguínia o metabolitzat a l'interior de la cèl.lula. No està gaire clar quin és el percentatge de lactat que s'allibera a sang però sembla ser entre un 10%[61] i un 35%[62]. Una altra part és resintetitzat a glicogen sobre tot en les fibres ràpides més que en les lentes[63]. La resta de lactat, pràcticament la majoria, és oxidat completa i aeròbicament sense tornar a glucosa, provocant un excés en el consum d'oxigen en la fase de repòs (deute d'oxigen), procés afavorit per activitat lleugera[64].

L'entrenament fa augmentar la quantitat de lactat produït, però també afavoreix canvis per suportar aquest increment. Característiques musculars com el transport de lactat a sang o la capacitat tampó són susceptibles de millora amb l'entrenament. La capacitat tampó o "buffer" (ß) permet al múscul esmorteir l'increment de concentració d'ions hidrogenions en exercicis anaeròbics. Una millora en aquest mecanisme permet allargar la utilització de la glucòlisi anaeròbica fins que el pH limitant és assolit[65], però el seu mecanisme encara està en discussió. Parkhouse i cols[66] van trobar positiva i alta correlació entre ß, la concentració de carnosina (un dels components del tampó muscular), el percentatge de fibres ràpides i el rendiment en carrera d'alta intensitat en atletes entrenats per velocitat. Encara en controvèrsia, altres autors no han trobat correlació entre ß i la distribució del tipus de fibra[67] o la concentració de lactat després de l'exercici[68]. Adicionalment, la capacitat tampó o "buffer" (ß) sembla ser susceptible de millora amb l'entrenament, trobant-se valors més alts en atletes que en sedentaris[57,68]. S'ha suggerit a partir de resultats en animals que la capacitat tampó està altament correlacionada amb la capacitat glucolítica i que ambdues co-adapten amb l'entrenament[69,70]. Conjuntament amb els components del tampó muscular, s'ha descrit un paper alcalinitzant de la fosfocretaina[71]

El transport de lactat a sang també és més gran en persones entrenades que en sedentàries i la capacitat de

transport de lactat a través del sarcolema sembla estar relacionada amb la distribució dels tipus de fibra[72].

Adaptació aeròbica

Una manera de poder assegurar la participació del metabolisme aeròbic en treballs de curta durada i alta intensitat és mitjançant el consum d'oxigen[73]. Encara que present en qualsevol exercici anaeròbic, no s'han arribat a trobar bones correlacions entre el consum d'oxigen i el resultat en tests anaeròbics[74]. Però, s'ha observat que corredors entrenats per mig fons consumien més oxigen que velocistes durant el test de Wingate[10].

En entrenaments de velocitat, les modificacions del metabolisme aeròbic es solen presentar de forma secundària. La millora en paràmetres relacionats amb el metabolisme aeròbic pot indicar situacions de sobreesforç muscular i millora del rendiment de les fibres lentes en detriment de les ràpides. D'altra banda, una lleu millora dels mecanismes aeròbics, sense ser determinant en el rendiment de proves supramaximals, esdevé favorable en les fases de recuperació. Amb una millor component aeròbica es resintetiza més ràpidament la fosfocreatina i s'obtenen millors resultats en exercici supramaximals encadenats[24].

Per l'estudi de l'adaptació aeròbica, la citrat sintasa és utilitzada com a marcador i s'han descrit augments de la seva activitat en entrenaments de velocitat[42]. Normalment, els entrenaments de velocitat que fan augmentar el VO_2max acostumen a produir un augment de la concentració d'enzims oxidatius[75], encara que el més normal és no trobar canvis[11,76,77,78].

La millora aeròbica ve induïda per la durada del treball muscular. Es proposa que si l'exercici és prou curt no es generarà deute d'oxigen i per tant no s'induiran millores en la resposta aeròbica a l'exercici[41]. S'ha proposat que el deute d'oxigen post-exercici està relacionat amb la metabolització del lactat i afavoreix millores aeròbiques[79].

Durant el repòs posterior a tota activitat física es produeix un augment del consum d'oxigen respecte al valor basal corresponent (deute d'oxigen) que serveix per pagar la mancança d'oxigen (dèficit d'oxigen) produïda a l'iniciar l'esforç[80]. Del deute d'oxigen s'obtindrà l'energia necessària per tornar l'organisme a l'estat de repòs i és generalment més elevada que el dèficit d'oxigen. Per restaurar les condicions basals, el múscul empra energia en la refosforilació de la creatina, el retorn de la mioglobina a oximioglobina, el retorn de la sang al seu estat d'oxigenació habitual i l'eliminació de l'excés de lactat present per oxidació al mitocondri[9,81]. La diferència entre deute i dèficit podria venir explicada pel cost extra d'oxigen necessari per dur a terme la gluconeogènesi[55], però hi ha controvèrsia en la distribució del lactat produït i la quantitat d'aquest dedicat a la gluconeogènesi[82]. Paràmetres relacionats amb la restauració de la homeostasi, l'augment de temperatura corporal o l'increment d'activitat hormonal poden també estar implicats en la diferència entre dèficit i deute d'oxigen[83].

El deute d'oxigen és un paràmetre millorable amb l'entrenament i presenta valors més grans (de 3 a 4 vegades) en esportistes velocistes que en persones sedentàries. S'ha trobat una bona correlació d'aquest paràmetre amb la marca en proves de velocitat[55]. Similar comportament s'ha observat amb el deficit que correlaciona sobretot en proves atlètiques més curtes de 400 metres[59]. Amb un entrenament mixte per velocitat i resistència lleugera obtenim un augment en el deute d'oxigen, juntament amb una major producció de lactat que estan en relació amb una millora del rendiment[84].

Efecte sobre la distribució fibrilar

El múscul està format per fibres que presenten unitats motores amb diversitat d'umbrals d'activació i cada fibra és reclutada segons les característiques de l'exercici. Sempre que l'esforç és inferior al VO_2max, s'utilitzen les fibres lentes independentment de la freqüència de contracció[85]. Segons Gollnick i cols[85], hi ha dues formes d'activar les fibres ràpides: exercicis per sobre del VO_2max o continuar l'exercici fins que les fibres lentes esgoten la reserva de glicogen. Friden i cols[86] proposen que en un esprint es recluten tant les fibres ràpides (II) com les lentes (I), però s'ha vist que en estimuls màxims d'alta intensitat, les primeres fibres en ser reclutades són les fibres ràpides[87] i de manera pràcticament exclusiva[88]. Aquest reclutament diferencial no sembla ser condicionat per la motoneurona, sinó que encara que arribi el senyal a les fibres lentes no hi ha contracció[89].

La transformació de fibra lenta a ràpida no està del tot clara. Un cas seria el desús o el desentrenament[90], però no és realment una canvi de fibres de tipus I a II, sinó una pèrdua selectiva de fibres de tipus I. A diferència de les fibres de tipus II, sembla que les de tipus I requereixen de l'activitat contràctil continuada per mantenir-se. S'ha vist que en persones entrenades el percentatge de fibres IIC és més alt que en sedentaris, passant de valors pràcticament inapreciables fins el 12-15%. Aquests tipus de fibres són intermediàries i estan involucrades en processos de reinervació i de transformació de la unitat motora[91].

Després d'un entrenament lleuger de velocitat dissenyat per millorar les

característiques anaeròbiques es troben canvis en les fibres, però aquests no són constants ni uniformes. Normalment es troba un augment del numero i àrea de les fibres de tipus I[92,93] encara que Jansson i cols[94] van trobar una reducció en les de tipus I a favor de les tipus IIA. Respecte a les fibres de tipus IIA, no està clara la seva evolució. Alguns autors troben que resten invariables[92,95] mentre que altres troben un augment en el seu numero[93,96]. Sembla ser que el numero de fibres IIB baixa[93] encara que la seva àrea augmenti[92]. L'augment de l'àrea de la fibra és una adaptació desitjada ja que s'ha descrit una bona relació entre diàmetre de la fibra i força generada[97].

Tenint present tots els tipus de fibra trobaríem interconversió entre totes elles de la manera:

$$IIB \leftrightarrow IIA \leftrightarrow IIC \leftrightarrow I$$

Sembla doncs que depenent del treball físic es pot fer variar el sentit de l'adaptació muscular.

EFECTE DE L'ENTRENAMENT EN EL RENDIMENT ESPORTIU

El fet que un entrenament anaeròbic produeixi millora en el rendiment esportiu no està encara del tot clar a la bibliografia científica. Alguns treballs han mostrat millora clara del rendiment, acompanyat per canvis bioquímics en el mecanisme anaeròbic d'obtenció d'energia tant en entrenaments curts de poques setmanes[11,78] com en entrenaments llargs d'una temporada.[43] Per altre banda, altres autors no troben canvis del rendiment anaeròbic després de sotmetre als voluntaris a varies setmanes d'entrenament[42,95], però troben microlesió muscular, abocament de marcadors musculars en sèrum i una tendència al canvi de la cadena pesada de la miosina IIb cap a la IIa. En aquest cas, l'hipòtesi de la lesió muscular podria ser la responsable d'una absència de millora. A la mateixa línia, Houston i cols[98] van trobar augments en la producció de lactat i en l'activitat d'alguns enzims sense millora del rendiment.

Molts factors poden alterar l'evolució programada del rendiment durant un període d'entrenament. La presència de fatiga deguda a un sobreesforç i/o una mancança de descans que impedeix recuperar les condicions adequades per dur a terme la contracció és un dels casos més comuns. La lesió muscular per excés d'entrenament i per tant la reducció del numero de fibres capaces de realitzar contracció provocaran també una disminució del rendiment.

L'activitat muscular produeix lesió de la fibra. El dany produït després de l'exercici és reparable i durant el procés de reparació té lloc una adaptació que dóna al múscul resistència a les lesions en les properes repeticions de l'exercici[99,100]. Malauradament, es desconeixen els mecanismes concrets d'aquesta adaptació.

CONCLUSIONS

La dificultat alhora d'establir possibles relacions de causa-efecte entre entrenament anaeròbic i la millora en el rendiment ve determinada per la complexitat dels mecanisme bioquímics que hi participen. La subtilesa necessària per eludir el sobreentrenament i la millora aeròbica fan extremadament complexe el seu disseny.

Per tractar d'estandaritzar el màxim possible tant els protocols com els resultats, ha estat necessària la utilització d'entrenaments de laboratori, especialment en cicloergometre que permeten eliminar gran quantitat de variabilitat ambiental.

De tota manera les conclusions sobre la millora del rendiment anaeròbic muscular com a conseqüència d'un entrenament encara no són del tot clares, però factors com la distribució de les càrregues o el paper dels descans comencen a esdevenir claus alhora de dissenyar l'entrenament més correcte per a cada individu.

Els estudis vinculats a l'interpretació de la fatiga específica en exercicis anaeròbics, així com la determinació dels mecanismes de restauració musculars i de seguiment de les microlesions induïdes per l'entrenament són els propers punts d'interès per poder comprendre els mecanismes musculars que envolten la millora del rendiment anaeròbic induït per l'entrenament.

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ORIGINAL ARTICLE

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A short training programme for the rapid improvement of both aerobic and anaerobic metabolism

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Abstract The aim of this study was to evaluate the changes in aerobic and anaerobic metabolism produced by a newly devised short training programme. Five young male volunteers trained daily for 2 weeks on a cycle ergometer. Sessions consisted of 15-s all-out repetitions with 45-s rest periods, plus 30-s all-out repetitions with 12-min rest periods. The number of repetitions was gradually increased up to a maximum of seven. Biopsy samples of the vastus lateralis muscle were taken before and after training. Performance changes were evaluated by two tests, a 30-s all-out test and a maximal progressive test. Significant increases in phosphocreatine (31%) and glycogen (32%) were found at the end of training. In addition, a significant increase was observed in the muscle activity of creatine kinase (44%), phosphofructokinase (106%), lactate dehydrogenase (45%), 3-hydroxy-acyl-CoA dehydrogenase (60%) and citrate synthase (38%). After training, performance of the 30-s all-out test did not increase significantly, while in the maximal progressive test, the maximum oxygen consumption increased from mean (SD) 57.3 (2.6) ml · min^{-1} · kg^{-1} to 63.8 (3.0) ml · min^{-1} · kg^{-1}, and the maximum load from 300 (11) W to 330 (21) W; all changes were significant. In conclusion, this new protocol, which utilises short durations, high loads and long recovery periods, seems to be an effective programme for improving the enzymatic activities of the energetic pathways in a short period of time.

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Key words Human skeletal muscle · Oxidative enzymes · Oxygen uptake · Anaerobic performance · Lactate

Introduction

Exercise-induced muscle changes can be modulated by the structure of a training programme (Abernethy et al. 1990). An endurance protocol produces major adaptations in aerobic metabolism (via oxidative enzymes, oxygen uptake ($\dot{V}O_2$) and performance of endurance tests (Henriksson 1996), while sprint training increases the concentration of energetic substrates and the activity of anaerobic-metabolism-related enzymes (Thorstensson et al. 1975; Roberts et al. 1982; Cadefau et al. 1990). However, in most cases, the goal of athletic preparation is to improve a subject's aerobic and anaerobic characteristics. Usually, an initial endurance phase is undertaken, followed by a second phase of high-intensity or sprint training. Training programmes that are capable of increasing aerobic or anaerobic metabolism (with continuous exercise or interval training) are based mainly on periods of at least 6 weeks (Costill et al. 1979; Jacobs et al. 1987), although there are shorter programmes lasting no more than 1 week. Such programmes are usually based on continuous endurance training, which produces some metabolic and haemodynamic changes, but which do not improve performance, increase the maximum oxygen consumption ($\dot{V}O_{2max}$) or produce great enzymatic changes (Green et al. 1992; Cadefau et al. 1994; Phillips et al. 1996; Shoemaker et al. 1996).

Often, however, athletes require a training programme in order to achieve fitness in a short period of time, particularly after periods of inactivity due to injury, illness or personal problems, or when it is necessary to make sudden changes in the training schedule. In these cases, cycle ergometer training has several advantages: the lower cost and size of the equipment required, the large number of muscles involved, the easy and

accurate design of loads and rests, indoor practice unaffected by the weather, and compatibility with some upper-limb injuries. However, each sport will require its own specific training.

Taking into account that the more intense the stimuli, the more intense the adaptations (or overtraining when the training protocol exceeds the capability of muscle adaptation), we applied a training programme that was characterised by high loads and daily application, for a 14-day period to produce the biggest response in this short period. The aim of the present study was thus to describe the physiological and biochemical changes produced in aerobic and anaerobic metabolism through a new incremental training programme of "all-out" loads, repeated daily for 2 weeks, and with long recovery periods.

Methods

Subjects

Five healthy male student volunteers agreed to take part in this study. Their mean (SD) age, height and body mass were 20.8 (2.9) years, 171 (5) cm and 68.1 (4.2) kg, respectively. All were active, but none were currently participating in a regular training programme. During the study period, all volunteers stopped their normal physical activity (recreational) and only exercised during the training sessions as part of the experiment. Prior to the experiment, the volunteers underwent a medical check-up to ensure that they were fit and healthy.

The experiment was conducted in accordance with the code of ethics of the World Medical Association (Declaration of Helsinki), and approval was given by the Human Experimentation Ethical Committee of the Pi i Sunyer Biomedical Research Institute of Barcelona (Hospital Clínic i Provincial, University of Barcelona). All subjects were informed before recruitment as to the purpose of the study, known risks and possible hazards associated with the experimental protocol, and each gave their written consent to participate.

Training protocol

Familiarisation with the equipment, sprint cycling and testing procedures took place prior to the experiment, until we were confident that volunteers would reach all-out effort from a stationary start. The programme involved fourteen training sessions, and subjects trained every day for 2 weeks (Fig. 1). The sessions without warm-up consisted of a number of repetitions of 15-s cycling with 45-s rest-periods, and a number of 30-s all-out cycling repetitions with 12-min rest-periods. The number of repetitions was modified and the total load increased during training. The first three sessions consisted of two bouts of 15-s, and two bouts of 30-s all-out cycling sprints. In the following sessions, the number of 15-s and 30-s bouts was increased by one every two training sessions. The last three sessions consisted of seven bouts of 15-s and seven bouts of 30-s all-out cycling sprints. As in the performance tests, subjects were instructed to remain seated during the cycle sprints in the training period. The flywheel tension was set at 0.075 kg · kg body mass^{-1}, and remained constant for the duration of the training programme. The maximum pedal revolutions reached by each volunteer in every 30-s bout were recorded. All subjects were highly motivated and verbally encouraged during training, and were instructed to cycle with maximum effort in every session. The purpose of this training protocol was to evaluate whether progressively higher loads that cannot be tolerated for long periods are able to elicit a rapid improvement in the metabolic pathways.

Performance tests

In order to quantify any training-induced alterations to anaerobic and aerobic capacities, volunteers performed a 30-s all-out test and a maximal progressive test before and after training (1 and 5 days before and after training, respectively, see Fig. 1).

The 30-s all-out test was performed against a constant tension. The flywheel tension was set at 0.075 kg · kg body mass^{-1}, and remained constant for the duration of both the test and the entire training programme. After a gentle warm-up, the subjects were comfortably seated with feet secured to the pedals by toe clips. They were requested to pedal as fast as possible from the start and were encouraged to maintain maximum pedalling speed throughout the 30-s period. A friction-loaded cycle ergometer (Monark model 814E, Valberg, Sweden) interfaced with a microcomputer was used to attain high-frequency logging of the flywheel angular velocity. The flywheel velocity was monitored every 0.5 s and was

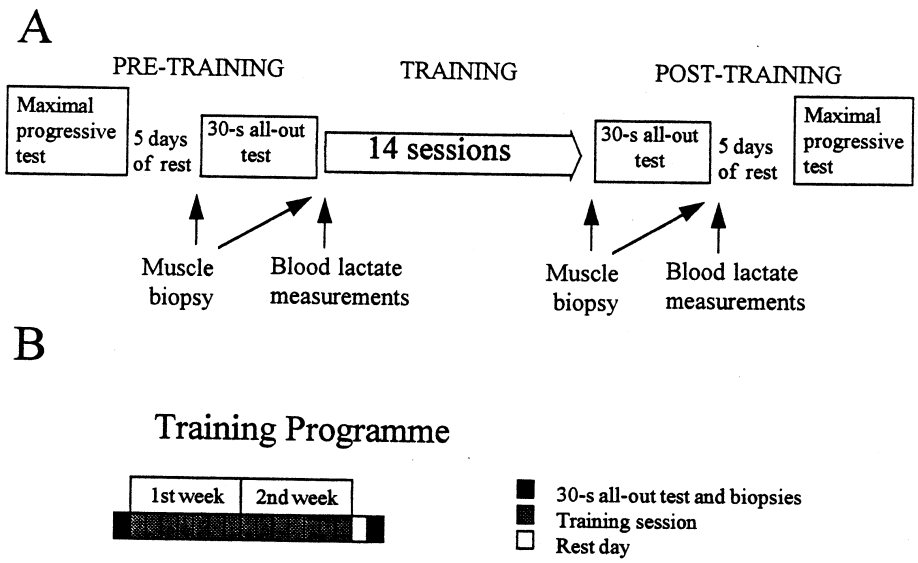

Fig. 1 **A** Schematic representation of the study procedures. **B** Design of the sprint-training programme illustrating the distribution of training sessions, test-biopsy sampling and rest days

displayed by the computer. Two parameters were determined: peak power, which referred to the power output corresponding to the maximum velocity reached during the test, and mean power, which referred to the average work performed during the test.

The progressive test started with a 3-min warm-up at 25 W. The exercise intensity was then increased by 25 W every minute until exhaustion (the moment when the subjects were unable to bear the imposed load), which was detected by a fall in the fixed pedalling rate of 60 rpm.

The subjects were monitored for ventilatory parameters and three-lead electrocardiogram in both tests. Heart rate (beats · min^{-1}) was obtained using a CM-5 precordial lead. The tests were performed under laboratory conditions of controlled temperature and humidity (temperature: 22–24 °C, and humidity: 55–65%). Subjects were requested to rest for 24 h prior to each test and to eat a light meal without alcohol or stimulants at least 3 h before tests, which is a usual procedure in tests for physical evaluation (Wasserman 1987).

Ventilatory parameters and blood lactate

To obtain ventilatory parameters during both tests, the participants breathed through a mask (Rudolph 2700), and the gaseous exchange was determined and monitored by an automatic open-circuit system (Oxycon 4). The system was calibrated before each test in relation to both volume and flow by means of a 3-l capacity syringe (Hans Rudolph), and with gases obtained from a tank of mixtures of oxygen and carbon dioxide of a known composition. The following parameters were recorded: pulmonary ventilation (l · min^{-1} BTPS), $\dot{V}O_2$ (ml · min^{-1} STPD) and expired carbon dioxide (ml · min^{-1} STPD). The respiratory values were obtained from an average of 30 s.

Capillary tubes containing heparin, sodium fluoride, and sodium nitrite were used to obtain blood samples from the ear lobe immediately before, and after 3, 5, 7 and 10 min of the 30-s all-out test, both before and after training. The lactate concentration (mmol · l^{-1}) of each sample was determined by an electroenzymatic method (Micro Stat PLM4, Analox Instruments). The reagent used was a standard lactate solution (ref. GMRD 090/091/092, Analox Instruments).

Muscle biopsy sampling

On the days of the all-out tests, subjects sat quietly on an examination table while small incisions were made in both legs through the skin and fascia, and the first muscle biopsy sample was obtained from the left leg (Rest). The needle biopsy technique was used to sample muscle tissue. Muscle biopsy samples (30–50 mg) were taken under local anaesthesia (mepivacaine 2%) from the mid-region of the quadriceps femoris muscle (vastus lateralis) from both legs, 15 cm above the top edge of the patella on the 1st day, and 5 cm above it on the next day. Subjects then performed the test, and the second biopsy from the right leg (30 s) was taken on completion (less than 2 s), while they were still seated on the cycle ergometer. The same protocol was performed before (pre-) and after (post-) training. The samples were immediately frozen, removed from the biopsy needles under liquid nitrogen and stored at −80 °C until they were lyophilised and analysed.

Biochemical analyses

Freeze-dried samples were dissected free of visible blood and connective tissue and then powdered. A part of each sample (4–6 mg) was treated with 0.5 M $HClO_4$, centrifuged at 15,000 *g* at 4 °C for 15 min, and the supernatant was neutralised with 2.1 M $KHCO_3$. The neutralised extract was assayed for phosphocreatine (PCr), pyruvate and lactate. All muscle metabolites were assayed enzymatically by fluorometric analysis (Lowry and Passonneau 1972). Glycogen concentration was measured both in the neutralised extract and in the pellet after acid hydrolysation. Then, the amount of free glucose produced was determined by the method described by Lowry and Passonneau (1972).

For the enzymatic analyses, a part of the dried muscle sample (4–6 mg) was homogenised in 30 volumes of ice-cooled extraction medium, using a special Potter (Teflon pellet pestle, Kontes). The extraction medium contained 50 mM HCl-Tris (pH 7), 4 mM ethylenediaminetetraacetic acid, 50 mM KF, and 30 mM β-mercaptoethanol. The preparation was centrifuged at 15,000 *g* and 4 °C for 15 min. The following enzymes were immediately measured spectrophotometrically in the supernatant: creatine kinase (CK), phosphofructokinase (PFK), and lactate dehydrogenase (LDH) using the methods described in Cadefau et al. (1990); citrate synthase (CS) and 3-hydroxyacyl-CoA dehydrogenase (HADH) using the methods described by Essen-Gustavsson and Henriksson (1984).

The pH values were calculated with the lactate and pyruvate concentration using the equation of Sahlin (Sahlin 1978) for dry tissue, when concentration is expressed in mmol · kg^{-1}:

$$pH = 7.06 - 0.00413 \times ([lactate] + [pyruvate]) \quad (1)$$

where [lactate] and [pyruvate] are the concentrations of lactate and pyruvate, respectively.

Statistics

Data are expressed as the mean (SD) unless stated otherwise. The statistical significance of the differences between two mean values was assessed by the nonparametric Wilcoxon rank-sum test for paired values. The level of statistical significance was set at $P < 0.05$.

Results

Muscle metabolites in the pre-training all-out test

One day before the beginning of the training protocol, subjects undertook a 30-s all-out test. Some metabolites were measured from the biopsy samples taken immediately before and after the test (Table 1). PCr and glycogen concentrations were significantly decreased (in all cases $P < 0.01$). The increase in lactate concentration was higher than ten-fold, and the increase in pyruvate reached five-fold ($P < 0.01$ in all cases). After the test, as lactate and pyruvate concentrations increased, the muscle fibre pH fell sharply.

Muscle metabolites in the post-training all-out test

The training programme caused a significant increase in PCr (31%, $P < 0.05$) and glycogen (32%, $P < 0.05$) concentrations before the post-training test. After the test, PCr and glycogen concentrations decreased in the post-training test as before. The increase in lactate concentration after the all-out test was more than nine-fold after training ($P < 0.01$), although it was significantly smaller than the lactate accumulation in the pre-training test. However, the blood lactate generated after the post-training all-out test increased by more than 25% over the blood lactate produced after the pre-training all-out test. The pre-test concentrations were 1.38 (0.26) mM pre-training and 1.64 (0.22) mM

Table 1 Muscle metabolite concentrations in muscle biopsies at rest and after the 30-s all-out test (*30 s*), before (*Pre-training*) and after (*Post-training*) training. Values are given as the mean (SD), expressed in mmol · kg dry tissue^{-1}

Metabolite	Pre-training		Post-training	
	Rest	30 s	Rest	30 s
Phosphocreatine	53.2 (6.3)	20.0 (8.5)**	69.8 (2.0)***	29.6 (8.8)**
Glycogen	251 (19)	178 (26)**	332 (22)***	281 (25)**,***
Pyruvate	0.30 (0.03)	1.49 (0.70)**	0.58 (0.16)	1.29 (0.33)**
Lactate	8.7 (0.8)	103.5 (15.2)**	9.4 (2.2)	87.0 (17.3)**,***
pH	7.02 (0.02)	6.63 (0.04)*	7.02 (0.03)	6.70 (0.04)*

*Significant difference ($P < 0.05$) between rest and 30 s in the same training status; **significant difference ($P < 0.01$) between rest and 30 s in the same training status; ***significant difference ($P < 0.05$) between values of the same parameter before and after training

post-training, and at 3, 5, 7 and 10 min of recovery the values reached 10.3 (1.6), 11.8 (1.4), 13.2 (1.2), 11.9 (0.8) mM pre-training and 14.6 (1.2), 16.2 (1.3), 16.2 (1.1), 15.3 (1.2) mM post-training ($P < 0.05$ in all the recovery cases), respectively.

Enzymatic adaptations to sprint training

Several enzymatic activities were modified as a response to training (Table 2). CK activity showed a significant increase of 44% ($P < 0.01$). LDH increased by a significant 45% ($P < 0.01$) and PFK increased by a full 100% ($P < 0.01$). Enzymatic activities connected to oxidative metabolism also increased significantly ($P < 0.05$) after training: CS by 38% and HADH by 60%.

Performance evolution

The increase in peak and mean power output in the 30-s all-out test was slight and nonsignificant (3% and 3%, respectively). However, a significant improvement in maximum pedalling rate was observed during training (Fig. 2), which disappeared when subjects were requested to increase the number of repetitions to seven (last three sessions). The last training session showed a marked decline in performance. In contrast, performance in the maximal progressive test improved after training and the subjects were able to increase their maximum power output by 10% and their $\dot{V}O_{2max}$ by 11%, both increases were significant ($P < 0.05$; Table 3). It is worth noting that the post-training maximal progressive test was carried out 5 days after the 30-s all-out test.

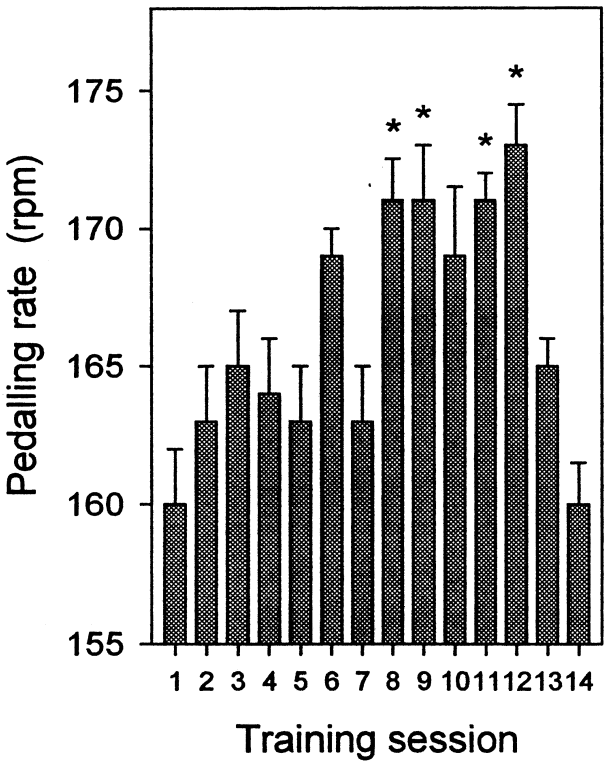

Fig. 2 Time course of performance in the 30-s all-out cycling during training. Each point represents the average of the best individual repetition. Values are presented as the mean ± SD for five subjects. *Significant differences from the first session ($P < 0.05$)

Table 2 Enzymatic activities in muscle biopsy samples taken Pre- and Post-training. Values are given as the mean (SD) for five subjects in each group, expressed in μmol · min^{-1} · g dry tissue^{-1}. (*CK* Creatine kinase, *PFK* phosphofructokinase, *LDH* lactate dehydrogenase, *HADH* hydroxyacyl-CoA dehydrogenase, *CS* citrate synthase)

Enzyme	Pre-training	Post-training
CK	10847 (1686)	15608 (1873)*
PFK	75.3 (6.6)	155.5 (12.4)**
LDH	886 (89)	1283 (124)*
HADH	19.3 (2.7)	30.9 (3.1)*
CS	28.1 (2.4)	38.8 (1.6)*

*Significant difference ($P < 0.05$) between values before and after training; **significant difference ($P < 0.01$) between values before and after training

Discussion

It is well established that aerobic metabolism can be improved by repeated bouts of intense exercise or by

Table 3 Functional parameters at maximum effort in the maximal progressive and the 30-s all-out test, Pre-training and Post-training. Values are given as the mean (SD). ($\dot{V}O_2$ oxygen consumption, f_c heart rate)

Functional parameters	Pre-training	Post-training
Progressive test		
$\dot{V}O_2$ (ml · min^{-1} · kg^{-1})	57.3 (2.6)	63.8 (3.0)*
f_c (beats · min^{-1})	189 (5)	195 (4)
Power (W)	300 (11)	330 (21)*
30-s all-out test		
$\dot{V}O_2$ (ml · min^{-1} · kg^{-1})	28.3 (4.3)	36.2 (2.1)*
f_c (beats · min^{-1})	150 (4)	153 (7)
Peak power output (W)	723 (33)	744 (29)
Mean power output (W)	578 (30)	595 (24)

*Significant difference ($P < 0.05$) between values of the same parameter before and after training

continuous exercise (endurance). However, continuous muscle activity might not produce any improvement in anaerobic metabolism (Holloszy 1975), whereas repeated bouts of intense exercise may (Linossier et al. 1993). Each training programme affects muscle differently depending on recovery time and exercise intensity (Dudley et al. 1982). However, in general, short periods of high muscle load with long recovery periods induce an adaptive response of anaerobic metabolism such as increases in PCr kinase activity (Thorstensson et al. 1975; Costill et al. 1979), while longer periods of effort have no effect on PCr metabolism, but induce a greater adaptive response in glycolytic metabolism (Sahlin 1978; Cadefau et al. 1990).

The most important finding of this study is that a very short, daily high-intensity training programme over fourteen sessions (with a maximum of 5 min per day of total exercise) can increase the enzymatic activities related to both aerobic and anaerobic metabolism in just 2 weeks. The training protocol proposed in this study significantly increases energetic compound concentrations (PCr and glycogen) and enzymatic activity of aerobic (CS and HADH), anaerobic alactic (CK) and anaerobic lactic pathways (PFK and LDH) in muscle.

The changes in aerobic metabolism were similar to those observed with other longer training protocols (Simoneau et al. 1986). This improvement in the aerobic pathway with intermittent maximum cycling probably occurs as aerobic metabolism becomes an important source of ATP during repeated 30-s all-out efforts (Bogdanis et al. 1996; Trump et al. 1996). When muscle work increases in intermittent exercises, the oxidative pathway is challenged. Therefore, the rest period intervals play a significant role, since during this time alactacid reserves are replenished and lactate accumulation is reduced (Fox et al. 1989) through oxidative phosphorylation (Sahlin et al. 1979; Gaesser and Brooks 1984).

With regard to anaerobic alactic metabolism, special attention was paid to the time required for PCr resynthesis, which was 12 min for the 30-s all-out repetitions (Bogdanis et al. 1995). This recovery period could be one of the keys to the particular muscle adaptation induced by this high-intensity training protocol, and might be connected with the increment in PCr concentration that is observed after training. Increased PCr concentrations following training have been described in other studies (Eriksson et al. 1973; McDougall et al. 1977), although they are not always found (Thorstensson et al. 1975; Nevill et al. 1989; Stathis et al. 1994). In contrast, PCr values at rest were lower than reported in other studies with biopsy samples (Green et al. 1992; Trump et al. 1996). This was probably due to the warm-up period that was given prior to muscle biopsy sampling, which was not included in other studies. In our case, the light unloaded warm-up was carried out in both tests before and after the training programme.

All of these adaptations, together with the increase in $\dot{V}O_{2max}$, indicate a considerable improvement in both aerobic and anaerobic enzymatic activities.

Alterations induced by training led to a decrease in glycogen breakdown and reduced accumulation of lactate in muscle during the post-training all-out test. This reduction in anaerobic energy production should correlate with the increase in the activity of the oxidative pathway, which plays a considerable role in this type of all-out test (Granier et al. 1995).

The reduced intramuscular lactate concentration at the end of the 30-s post-training all-out test compared with the initial test could be due to decreased glycolysis activation, an increased efflux of lactate to the blood (Fox et al. 1989), or to increased lactate utilization in the muscle fibre. It is also worth considering that training can induce an increased use of lactic acid as a metabolic fuel for the aerobic system. The use of lactate as a muscle energy source has been described as being training-sensitive, and short endurance programmes appear to be able to increase the metabolic clearance rate of lactate (Phillips et al. 1995).

In contrast, we found that blood lactate concentrations increased during the all-out test recovery period after training. The kinetics of lactate extrusion from skeletal muscle to the blood by a lactate transporter are not fully understood. However, this mechanism could be affected by training, in this case permitting an increased efflux of lactate to the blood, thereby producing increased blood lactate concentrations for a given amount of muscle lactate (Bonen et al. 1998). Wilson et al. (1998) have described a new monocarboxylate transporter (MCT3), suggested to be responsible for efflux of glycolytically derived lactic acid from white skeletal muscle, although its regulation has not been determined. Our results indicate that to some extent, lactate transport from the muscle cell is independent of muscle lactate concentration.

Although the all-out test performance improvement (carried out 1 day after the end of training) was slight, a significant increase in pedalling rate during training

was observed (Fig. 2). The decrease in maximum pedalling rate during the last 2 days of training, compared with the previous days, could suggest some symptoms of fatigue. However, the fact that a higher $\dot{V}O_{2max}$ and higher PCr and glycogen muscle concentrations were found after training, and lower lactate concentration and acidosis were found after the test, suggests that this failure to improve performance was not energetic in origin. It was probably attributable to neuromuscular fatigue, as has been demonstrated to occur after heavy exercises, producing an impairment in action potential propagation (Strojnik and Komi 1998). When this fatigue disappears, the 30-s all-out test performance will probably increase greatly owing to the improved biochemical parameters. The fact that the progressive test (in which a significant performance improvement was detected) was carried out 5 days after the 30-s all-out test, should be taken into account, together with the great number of studies in which a direct relationship between metabolic markers of aerobic and anaerobic pathways and performance was found (e.g. Roberts et al. 1982; Linossier et al. 1997; MacDougall et al. 1998).

In conclusion, the training method described in the present study is a very short and convenient procedure, and is of particular interest when an increase in aerobic and anaerobic energetic pathways is required within a short period of time.

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The distribution of rest periods affects performance and adaptations of energy metabolism induced by high-intensity training in human muscle

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ABSTRACT

The effect of the distribution of rest periods on the efficacy of interval sprint training is analysed. Ten male subjects, divided at random into two groups, performed distinct incremental sprint training protocols, in which the muscle load was the same (14 sessions), but the distribution of rest periods was varied. The 'short programme' group (SP) trained every day for 2 weeks, while the 'long programme' group (LP) trained over a 6-week period with a 2-day rest period following each training session. The volunteers performed a 30-s supramaximal cycling test on a cycle ergometer before and after training. Muscle biopsies were obtained from the *vastus lateralis* before and after each test to examine metabolites and enzyme activities. Both training programmes led to a marked increase (all significant, $P < 0.05$) in enzymatic activities related to glycolysis (phosphofructokinase – SP 107%, LP 68% and aldolase – SP 46%, LP 28%) and aerobic metabolism (citrate synthase – SP 38%, LP 28.4% and 3-hydroxyacyl-CoA dehydrogenase – SP 60%, LP 38.7%). However, the activity of creatine kinase (44%), pyruvate kinase (35%) and lactate dehydrogenase (45%) rose significantly ($P < 0.05$) only in SP. At the end of the training programme, SP had suffered a significant decrease in anaerobic ATP consumption per gram muscle ($P < 0.05$) and glycogen degradation ($P < 0.05$) during the post-training test, and failed to improve performance. In contrast, LP showed a marked improvement in performance ($P < 0.05$) although without a significant increase in anaerobic ATP consumption, glycolysis or glycogenolysis rate. These results indicate that high-intensity cycling training in 14 sessions improves enzyme activities of anaerobic and aerobic metabolism. These changes are affected by the distribution of rest periods, hence shorter rest periods produce larger increase in pyruvate kinase, creatine kinase and lactate dehydrogenase. However, performance did not improve in a short training programme that did not include days for recovery, which suggests that muscle fibres suffer fatigue or injury.

Keywords anaerobic exercise, enzyme activities, glycogen, glycolysis, lactate, recovery, skeletal muscle metabolism, sprint training.

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Muscle adaptation owing to exercise or physical training seems to be correlated with the amount, intensity, distribution and duration of muscle loads (Dudley *et al.* 1982). The combination of these factors is especially important when interval high-intensity training is designed because biochemical responses depend on the protocol of contractile activity to which the muscle is subjected (summarized in MacDougall *et al.* 1998). Hence, a direct relationship between adaptations and the components of the sprint training has been hard to find. However, certain desirable biochemical adaptations and sprint performance improvement seem to be associated with high-intensity training. Increases in enzymatic activities related to glycolysis, including phosphofructokinase, lactate dehydrogenase or glycogen phosphorylase and changes in metabolite concentration, including phosphocreatine or glycogen, have been recorded, although the extent of these changes varies from one sprint training programme to another (Thorstensson *et al.* 1975, Costill *et al.* 1979,

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Roberts *et al.* 1982, Cadefau *et al.* 1990, Linossier *et al.* 1993, 1997, Dawson *et al.* 1998, MacDougall *et al.* 1998).

The duration of each bout affects the adaptations induced by interval high-intensity training, as described by Costill *et al.* (1979), comparing 6 s in the left leg with 30 s in the right leg of maximal isokinetic exercise of the same subject. Bouts lasting less than 10 s are considered more anaerobically dependent (Hellsten-Westing *et al.* 1993, Linossier *et al.* 1993) than longer bouts, which are more demanding and during which power output decreases before the end (Bogdanis *et al.* 1995, 1996).

The recovery periods between bouts are also decisive and the time ratio between recovery and exercise phases has to be taken into account (Linossier *et al.* 1997). Intense and brief muscle loads with long recovery periods are proposed to induce an adaptative response in phosphocreatine metabolism (Thorstensson *et al.* 1975), while an increased training load has no effect on this process, but seems to produce a greater adaptative response in lactate metabolism (Roberts *et al.* 1982, Cadefau *et al.* 1990). The distribution of rest periods between days of training is usually less carefully planned than the ratio of recovery and work in each session and its effects on the adaptations induced by sprint training have not been studied. The rest periods between sessions prevent fatigue, which could appear when rest periods are insufficient and/or muscle load is exhausting.

In order to analyse the effect of rest distribution on muscle adaptations, we designed two high-intensity training cycle protocols with identical daily muscle loads but different distribution of rest periods. Enzymatic activities related to glycolysis, glycogen and creatine metabolism and aerobic metabolism were measured together with adenine nucleotides, glycolytic intermediates and creatine concentrations. We also evaluated the effect of the two training protocols on performance and muscle metabolic response by a 30-s all-out cycling test before and after training.

METHODS

Subjects

Ten healthy male student volunteers agreed to take part in this study. Their age, height and body mass were (mean ± SD) 23.6 ± 2.4 years, 171.1 ± 3.4 cm and 70.2 ± 4.8 kg, respectively. All were active, but none was currently participating in a regular training programme. During the experiment, all volunteers stopped their normal physical activity and only exercised within the experiment. Before the commencement of the experiment the volunteers underwent a medical check-up to verify that they were healthy and fit. They were divided at random into two groups called 'short programme' (SP) and 'long programme' (LP).

The experiment was conducted in accordance with the code of ethics of the World Medical Association (Declaration of Helsinki) and approval was given by the Ethical Committee of Human Experimentation from the Pi i Sunyer Biomedicine Research Institute of Barcelona (Hospital Clínic i Provincial – University of Barcelona). All subjects were informed before recruitment about the purpose of the study, known risks, and possible hazards associated with the experimental protocol and each gave his written consent.

Performance test

In order to evaluate the anaerobic capacity of volunteers and possible improvement owing to training, subjects were required to perform a supramaximal cycling test (30 s) the day before the initiation of training and 48 h after finishing 14 training sessions. The test was performed on a friction-loaded cycle ergometer (Monark 814 E, Varberg, Sweden). A microprocessor, interfaced with the cycle ergometer, counted flywheel revolutions every second for 30 s of supramaximal cycling sprinting against a constant resistance of 0.075 kg × (kg body mass)$^{-1}$. With the flywheel progression per pedal revolution and the elapsed time, the following variables were calculated: peak power (the highest power output) and mean power (the average power output during the 30 s). The subjects were comfortably seated with feet secured to the pedals by toe clips. They were requested to pedal as fast as possible from the start and were encouraged to maintain maximum pedalling speed throughout the 30-s period.

Training protocol

Familiarization with the equipment, sprint cycling and testing procedures took place before the experiment started, until there was complete confidence in reaching an all-out effort from a stationary start. Each group (SP and LP) participated in a different high-intensity programme designed to improve performance in high-intensity tests. These programmes involved the same 14 training sessions but with different duration of rest. The SP group trained every day for 2 weeks while the LP group trained for 6 weeks resting for 2 days between each session. The sessions comprised a number of warm-up repetitions of 15 s maximal cycling with 45 s rest-periods and a number of training repetitions of 30 s maximal cycling with 12 min rest-periods. The number of repetitions was modified and the total muscle load increased during training. The first three

sessions comprised of two bouts of 15 s sprints and two bouts of 30 s supramaximal cycling sprints. In the following sessions, the number of 15- and 30-s bouts were increased by one every two training sessions. The last three sessions consisted of seven bouts of 15 s and seven bouts of 30 s. As in the performance tests, subjects were instructed to remain seated during the cycle sprints in the training period. The flywheel tension was set at 0.075 kg (kg body mass)$^{-1}$ and remained constant for the duration of the training programme. The maximum number of pedal revolutions reached by each volunteer in every 30-s bout was recorded. All subjects were highly motivated and verbally encouraged during training and instructed to cycle with maximum effort in every session.

Muscle biopsies

The needle biopsy technique was used to sample muscle tissue. Muscle biopsy samples (30–50 mg) were taken under local anaesthesia (mepivacaine 2%) from the mid-region of the *quadriceps femoris* muscle (*vastus lateralis*) from both legs, 15 cm above the top edge of the patella on the first day and 5 cm above it next day (48 h after finishing training). On the day of the performance test, volunteers reported to the laboratory at least 3 h after their last meal. After a light warm-up, they sat quietly on an examination couch while small incisions were made in both legs through the skin and fascia and the first muscle biopsy was obtained from left leg (rest). Subjects then performed the test and the second biopsy from right leg (30 s) was taken immediately after, while they were still seated on the cycle ergometer. The same protocol was performed 1 day before (pre) and 2 days after (post) training. The samples were directly frozen, removed from the biopsy needles under liquid nitrogen and stored at –80 °C until they were lyophilized and analysed.

Biochemical studies

Freeze-dried samples were dissected free of blood and connective tissue and powdered. A part (20 mg) of the dry tissue was treated with 0.5 M $HClO_4$ and centrifuged at 13 000 × *g* at 4 °C for 15 min. The supernatant was neutralized with 2.1 M $KHCO_3$. The neutralized extract was assayed for phosphocreatine (PCr), ATP, creatine (Cr), free glucose, glucose 1-phosphate (G-1-P), glucose 6-phosphate (G-6-P), fructose 6-phosphate (F-6-P), fructose 1,6-bisphosphate (F-1,6-P_2), pyruvate (Pyr) and lactate (Lac). All muscle metabolites were assayed enzymatically by fluorometric analysis (Lowry & Passonneau 1972). Glycogen concentration was measured both in the neutralized extract and in the pellet, by prior 1 M HCl hydrolysis extraction. Then the free glucose produced was determined by the method described above. From the neutralized extraction, IMP, ATP, ADP and AMP were measured using the HPLC method (Ingebretsen *et al.* 1982). Muscle metabolite concentrations were adjusted to the individual mean total creatine (PCr + Cr) because this mean should be kept constant during exercise (Harris *et al.* 1976). The adjustment to total creatine content enabled any variability in solid non-muscle constituents of the biopsies to be corrected.

For the enzymatic analyses, a portion of the muscle biopsies (10 mg) taken before the tests was homogenized in 30 volumes of ice-cooled extraction medium. The extraction medium contained 50 mM HCl–Tris (pH 7), 4 mM EDTA, 50 mM KF and 30 mM β-mercaptoethanol. The preparation was centrifuged at 15 000 × *g* at 4 °C for 15 min. The following activities were immediately measured in the supernatant: glycogen synthase (GS), glycogen phosphorylase (GPh), creatine kinase (CK), phosphofructokinase (PFK), aldolase (ALD), lactate dehydrogenase (LDH) and pyruvate kinase (PK), as described in Cadefau *et al.* (1990); hexokinase (HK), citrate synthase (CS), phosphoglucoseisomerase (PGI) and 3-hydroxyacyl-CoA dehydrogenase (HAD) as described in Essen-Gustavsson & Henriksson (1984) and myokinase (MK) as described in Oliver (1955).

Calculations

Values of pH in the muscle, before and after the test, were calculated from changes in lactate and pyruvate concentration [expressed as mmol (kg dry tissue)$^{-1}$] as reported by Sahlin (1978):

$$\mathrm{pH} = 7.06 - 0.00413 \cdot ([\mathrm{Lac}] + [\mathrm{Pyr}])$$

ATP consumption [mmol (kg dry tissue)$^{-1}$] during the tests, before and after training, was also calculated using a specific equation (Katz *et al.* 1986), where increments in ATP, ADP, PCr, Lac and Pyr were obtained from each value before and after the 30 s all-out cycling test.

$$\text{ATP consumption} = 2(-\Delta\mathrm{ATP}) - \Delta\mathrm{ADP} - \Delta\mathrm{PCr} + 1.5\Delta(\mathrm{Lac}) + 1.5(\Delta\mathrm{Pyr})$$

No corrections were made for lactate or pyruvate efflux during sprints or for anaerobic-produced ATP as a result of pyruvate oxidation.

The flux in glycogenolytic and glycolytic pathways [mmol glucosyl units (kg dry tissue)$^{-1}$] was calculated from a combination of the variation in concentration of several metabolites, as described by Spriet *et al.* (1987):

$$\text{Glycogenolytic rate} = \Delta\text{G-1-P} + \Delta\text{G-6-P} + \Delta\text{F-6-P} + 0.5(\Delta\mathrm{Lac} + \Delta\mathrm{Pyr})$$

$$\text{Glycolytic rate} = 0.5(\Delta\mathrm{Lac} + \Delta\mathrm{Pyr})$$

Table 1 Nucleotides and creatine concentration in muscle biopsies of LP and SP groups at rest and after the 30-s cycle ergometer sprint test before (pre) and after (post) training

	SP Group				LP Group			
	Pre-training		Post-training		Pre-training		Post-training	
	Rest	30 s	Rest	30 s	Rest	30 s	Rest	30 s
ATP	24.4 ± 0.9	16.8 ± 0.9*	22.6 ± 0.7	19.4 ± 1.0	24.2 ± 0.9	16.6 ± 0.7*	22.6 ± 0.8	19.8 ± 0.7
ADP	2.38 ± 0.20	2.76 ± 0.60	1.96 ± 0.20	2.66 ± 0.20	2.34 ± 0.49	2.85 ± 0.32	2.08 ± 0.27	2.30 ± 0.26
AMP	0.25 ± 0.05	0.42 ± 0.03*	0.30 ± 0.07	0.40 ± 0.03	0.26 ± 0.04	0.42 ± 0.08*	0.24 ± 0.05	0.34 ± 0.03*
IMP	0.63 ± 0.10	6.65 ± 1.10**	0.57 ± 0.02	1.03 ± 0.20[a]	0.76 ± 0.12	7.15 ± 1.21**	0.52 ± 0.13	2.55 ± 0.53*[a]#
TAN	27.3 ± 0.8	19.8 ± 0.9*	24.9 ± 1.0	22.5 ± 1.0	26.8 ± 0.8	20.9 ± 0.9*	24.9 ± 0.8	22.4 ± 0.8
TAN + IMP	27.9 ± 0.9	26.5 ± 1.3	25.5 ± 1.2	23.5 ± 1.3	27.6 ± 0.7	27.0 ± 0.8	25.4 ± 0.9	25.0 ± 0.7
PCr	55.9 ± 6.6	19.0 ± 8.1**	68.4 ± 2.0[a]	30.2 ± 8.9**	64.8 ± 8.1	35.5 ± 7.9*	64.2 ± 7.0	19.9 ± 4.8**[a]
Cr	52.1 ± 10.7	88.1 ± 9.8*	53.7 ± 5.2	93.1 ± 12.5*	44.8 ± 8.7	74.3 ± 11.7*	46.7 ± 4.9	91.1 ± 16.5**[a]
Total creatine	107.9 ± 13.2	107.9 ± 14.9	123.0 ± 10.2[a]	123.0 ± 11.5[a]	109.8 ± 11.4	109.8 ± 12.7	111.0 ± 8.7	111.0 ± 8.7

Values are means ± SD for five subjects in each group expressed in mmol (kg dry tissue)$^{-1}$. *,** Significant difference ($P < 0.05$, $P < 0.01$) between the rest and after the test in the same training status. [a] Significant difference ($P < 0.05$) between values of the same parameter before and after training. # Significant difference ($P < 0.05$) between values of the same parameter on different group.

The mean rate was calculated by dividing the absolute values by the time of the test (30 s).

Statistics

Differences in the same groups before and after training were analysed by non-parametric Wilcoxon test for paired values. Differences between the two groups were evaluated by non-parametric Mann–Whitney test for unpaired values. Differences were considered significant at $P < 0.05$ and values were expressed as means ± SD.

RESULTS

Muscle metabolites in the pre-training test

One day before the beginning of the training period, subjects undertook a supramaximal test (pre-training test). Several metabolites were measured from biopsies taken immediately before and after the test for both groups (Tables 1, 2). Before training, neither group showed any significant differences in metabolite concentration at rest and the changes produced owing to the test were similar. ATP and PCr concentration decreased significantly in both groups (in all cases $P < 0.05$). IMP increased significantly during the test in both groups. Despite the extent of changes in total adenine nucleotide (TAN = ATP + ADP + AMP), the amount of TAN + IMP remained unchanged after 30 s of supramaximal cycling. Glycogen concentration decreased significantly in both groups ($P < 0.05$) and to a similar extent (SP 29%; LP 26%). As a result of glycogen degradation, G-1-P concentration increased significantly, about 3–4-fold ($P < 0.01$ both groups). G-6-P concentration increased more than 10-fold (both groups $P < 0.01$). F-6-P increased (both groups $P < 0.01$) in a similar manner to G-6-P. Although glucose concentration increased in both groups, no significant differences were found. The increase in lactate concentration was higher than 10-fold in both groups and the increase in pyruvate reached 5-fold ($P < 0.01$ in all cases). As lactate and pyruvate concentrations increased, the muscle fibres became acidic. The calculated pH fell sharply after the 30-s-test in both groups.

Enzymatic adaptations to sprint training

There were no significant differences in enzymatic activities between the SP and LP groups before the start of the training programmes. However several enzymatic activities were modified in response to training (Table 3). Myokinase, glycogen synthase and glycogen phosphorylase did not vary significantly in either group, but the percentage of change was different between groups ($P < 0.05$).

Creatine kinase activity showed significant increase in the SP group (44%, $P < 0.05$), but only a slight variation in the LP group (9%). Pyruvate kinase and lactate dehydrogenase increased significantly ($P < 0.05$) and to a considerable extent (35 and 45%, respectively) in the SP group, but not in the LP group. All these enzyme activities (CK, PK and LDH) showed a different ($P < 0.05$) percentage of change between both SP and LP group.

Table 2 Muscle metabolite concentration in biopsies of LP and SP groups at rest and after the 30-s cycle ergometer sprint test before (pre) and after (post) training

	SP Group				LP Group			
	Pre-training		Post-training		Pre-training		Post-training	
	Rest	30 s	Rest	30 s	Rest	30 s	Rest	30 s
Glycogen	251 ± 19	178 ± 26*	332 ± 22[a]	281 ± 25*[a]	246 ± 25	181 ± 16*	321 ± 29[a]	242 ± 19*[a]
G-1-P	0.07 ± 0.01	0.35 ± 0.07**	0.11 ± 0.02	0.29 ± 0.08	0.06 ± 0.02	0.35 ± 0.17**	0.06 ± 0.01	0.36 ± 0.07*
Glucose	3.84 ± 0.98	6.47 ± 1.49	2.87 ± 0.14	4.81 ± 0.91	3.68 ± 0.49	5.78 ± 0.88	3.44 ± 0.76	4.73 ± 0.54
G-6-P	0.72 ± 0.24	15.9 ± 1.8**	1.82 ± 0.39	6.77 ± 3.40[a]	1.08 ± 0.26	12.67 ± 3.65**	0.73 ± 0.09	19.20 ± 1.85**#
F-6-P	0.48 ± 0.05	3.40 ± 0.35**	0.73 ± 0.07	2.41 ± 0.53*	0.62 ± 0.14	3.23 ± 0.93**	0.75 ± 0.14	3.42 ± 0.69**
F-1,6-P_2	0.20 ± 0.05	0.28 ± 0.07	0.15 ± 0.03	0.33 ± 0.04*	0.18 ± 0.06	0.27 ± 0.07	0.17 ± 0.02	0.27 ± 0.06
Pyr	0.30 ± 0.03	1.49 ± 0.70**	0.58 ± 0.16	1.29 ± 0.33*	0.37 ± 0.11	1.52 ± 0.68**	0.29 ± 0.06	2.27 ± 0.33**
Lac	8.7 ± 0.8	103.5 ± 15.2**	9.4 ± 2.2	87.0 ± 17.3**	9.0 ± 1.8	105.2 ± 16.6**	6.9 ± 1.0	102.5 ± 19.7**
pH	7.02 ± 0.02	6.63 ± 0.04*	7.02 ± 0.03	6.70 ± 0.04*	7.02 ± 0.02	6.62 ± 0.04*	7.03 ± 0.02	6.63 ± 0.05*

Values are means ± SD for five subjects in each group expressed in mmol (kg dry tissue)$^{-1}$. *,** Significant difference ($P < 0.05$, $P < 0.01$) between the rest and after the test in the same training status. [a] Significant difference ($P < 0.05$) between values of the same parameter before and after training. # Significant difference ($P < 0.05$) between values of the different group.

Phosphofructokinase, aldolase, citrate synthase and 3-hydroxyacyl-CoA dehydrogenase increased in both groups (all cases $P < 0.05$). Although for the PFK and HAD activities, the increases were more extended in SP group ($P < 0.05$).

Hexokinase and phosphoglucose isomerase activities remained unchanged or showed only slight variations.

Muscle metabolites in the post-training test

After training and before the post-training test, both groups showed similar metabolite concentration at rest. However, the SP training caused a slight variation in total creatine (14%), together with increases in PCr (39%, $P < 0.05$) and glycogen (32%, $P < 0.05$) concentration, while only glycogen (30%, $P < 0.05$) increased in the LP group. In contrast, ATP and TAN concentration showed a slight change after 14 training sessions in both groups (not significant).

After the post-training test, IMP and AMP varied as during the pre-training test in both groups. Whereas the change was smaller and not significant in the SP group, the increase in the LP group was significant ($P < 0.05$). In both cases, the variation was less pronounced than that previously produced in the pre-training test. ATP and PCr concentration changed in both groups, but in this second test only PCr decreased significantly ($P < 0.01$, in both groups). Glycogen concentration decreased significantly in both groups ($P < 0.05$). The increase in lactate concentration after the post-training test was more than 9-fold in both groups ($P < 0.01$). Long programme group attained values similar to those in the pre-training test, while the SP group showed a slightly lower lactate concentration than in the pre-training test.

Performance evaluation

The test gave a different result for the two groups after training (Fig. 1). The LP group significantly improved their maximum peak power (20%) and mean power (14%), while the variation in the same parameters of the SP group was smaller and not significant (3 and 3%, respectively). The difference in performance between SP and LP groups was significant ($P < 0.05$).

The SP group improved the maximum peak power during the first session of training, reaching 10% on the 10th day of training (data not shown). However, in the last three sessions, their performance

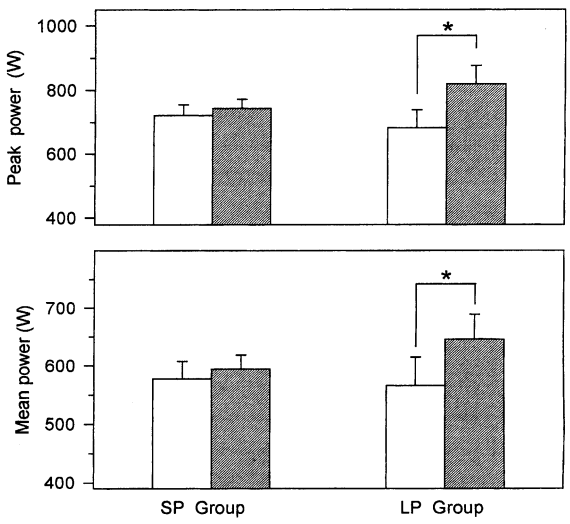

Figure 1 Mean and peak power values of SP and LP group during the 30 s tests, pre- (open bars) and post-training (striped bars). Values are means ± SD for five subjects each group. * Significant difference ($P < 0.05$) between values pre- and post-training.

Table 3 Enzymatic activities in muscle biopsies of SP and LP groups before (pre) and after (post) training

	SP Group			LP Group		
	Pre-training	Post-training	% of change	Pre-training	Post-training	% of change
MK	2788 ± 454	2711 ± 377	−2.8 ± 1.4	2463 ± 323	2910 ± 445	+18.1 ± 3.5#
CK	10847 ± 1686	15608 ± 1873*	+43.9 ± 4.8	12434 ± 1823	13571 ± 1990	+9.1 ± 2.1#
GS	7.42 ± 0.72	6.80 ± 0.62	−8.4 ± 1.7	8.41 ± 1.21	8.81 ± 0.60	+4.8 ± 0.9#
GPh	108.9 ± 8.3	99 ± 13.8	−9.1 ± 2.7	117 ± 19.5	120.2 ± 19.3	+2.7 ± 1.1#
HK	21.2 ± 0.8	22.3 ± 0.7	+5.2 ± 0.4	21.7 ± 0.8	23.3 ± 0.6	+7.4 ± 1.1
PGI	841 ± 30	976 ± 90	+16.0 ± 2.1	814 ± 95	861 ± 79	+5.8 ± 2.1
PFK	75.3 ± 6.6	155.5 ± 12.4**	+106.5 ± 8.2	89.9 ± 15.6	150.7 ± 14.9**	+67.6 ± 6.2#
ALD	317 ± 27	463 ± 52*	+46.1 ± 3.8	411 ± 41	526 ± 84*	+27.9 ± 4.1
PK	1384 ± 45	1872 ± 101*	+35.3 ± 2.7	1587 ± 117	1719 ± 156	+8.3 ± 1.9#
LDH	886 ± 89	1283 ± 124*	+44.8 ± 3.1	807 ± 97	876 ± 109#	+8.6 ± 1.6#
CS	28.1 ± 2.4	38.8 ± 1.6*	+38.1 ± 2.0	33.1 ± 3.6	42.5 ± 2.8*	+28.4 ± 2.1
HAD	19.3 ± 2.7	30.9 ± 3.1*	+60.1 ± 4.3	25.3 ± 3.1	35.1 ± 1.9*	+38.7 ± 3.3#

Values are means ± SD for five subjects in each group expressed in U (g dry tissue)$^{-1}$. *,** Significant difference ($P < 0.05$, $P < 0.01$) between values before and after training. (Unit = μmol min^{-1}). # Significant difference ($P < 0.05$) between values of the same parameter on different group.

deteriorated to the same extent as in the post-training test.

ATP consumption and glycogenolysis and glycolysis rates during test

ATP consumption and glycogenolysis and glycolysis rates during the pre-training test was similar in both groups, however, differences in those rates were observed after training (Table 4). Short programme showed a decrease in ATP consumption (16%, $P < 0.05$) as a consequence of a reduction in the glycolysis (18%, $P < 0.05$) and glycogenolysis (30%, $P < 0.05$) rates. Long programme showed a slight but non-significant variation in ATP consumption, probably produced by an increase in the glycogenolytic rate (11%).

The SP group showed lower values of ATP consumption and glycogenolysis and glycolysis rates than the LP group. However only the glycogenolysis rate was significantly different ($P < 0.05$) between groups after training.

DISCUSSION

Muscle metabolic response to the pre-training test

The muscle concentration of metabolites in the volunteers was within the same range reported in the literature (Bogdanis *et al.* 1995). Likewise, muscle metabolite response during the pre-training 30 s all-out test was in line with that described previously for normal active volunteers (Nevill *et al.* 1989, Bogdanis *et al.* 1995), with no differences being recorded between groups. However, it is worth noting the variability in glycolytic intermediate concentrations depending upon the experimental protocol (with or without warm-up) and the effect of previous training status of the volunteers.

The pre-training test led to a sharp reduction in ATP and PCr concentration after 30 s supramaximal cycling, which is consistent with other findings (Stathis *et al.* 1994, Bogdanis *et al.* 1995, 1996). A large increase in IMP concentration was found in muscle after 30 s of supramaximal exercise in both groups. This increase in IMP muscle concentration is a consequence of a greater

Table 4 ATP consumption and glycolgenolysis and glycolysis rates of SP and LP groups during tests, before (pre) and after (post) training

	SP Group			LP Group		
	Pre-training	Post-training	% Variation	Pre-training	Post-training	% Variation
ATP consumption	193.0 ± 14.7	161.6 ± 11.5*	−16.3 ± 4.8	188.6 ± 17.4	194.4 ± 17.1	+3.1 ± 1.1#
Glycogenolysis rate	65.8 ± 5.5	45.6 ± 5.1*	−30.7 ± 4.2	62.6 ± 5.8	69.2 ± 5.9#	+10.5 ± 2.8#
Glycolysis rate	47.4 ± 3.8	38.8 ± 3.9*	−18.1 ± 3.1	48.1 ± 4.1	47.8 ± 4.1	−0.6 ± 0.2#

Values are means ± SD for five subjects in each group expressed in mmol (kg dry tissue)$^{-1}$. * Significant difference ($P < 0.05$) between values before and after training. # Significant difference ($P < 0.05$) between values of the same parameter on different group.

requirement of the adenine nucleotide metabolism, often described after high intensity exercise (Sahlin *et al.* 1978, Stathis *et al.* 1994).

Although changes in ATP, ADP and IMP were considerable, the addition of TAN + IMP remained constant in both groups after the test, which suggests that IMP was not dephosphorylated during the 30 s of sprint cycling and there was no loss of purine nucleotides.

During the 30-s test, approximately 30% of the glycogen was broken down. We found an average rate of 1.6 mmol glucosyl units (kg dry muscle)$^{-1}$ s^{-1} after 30 s of sprint cycling, while Gaitanos *et al.* (1993) reported a glycolysis rate of 2.2 mmol glucosyl units (kg dry muscle)$^{-1}$ s^{-1} after the first 6 s and Jacobs *et al.* (1983) reported that after 10 s of maximal cycling the production of lactate was 60% of the total lactate produced during 30 s. All these results suggest that the expenditure of glucose via anaerobic glycolysis is not constant during a 30-s all-out cycling test.

Biochemical changes caused by sprint training

Sprint training produced changes in muscle metabolite concentration which seemed unaffected by rest period distribution, as variations were similar in both groups. Resting values of glycogen concentration increased after training (30%, $P < 0.05$) in both groups. Increases in glycogen storage after high-intensity training protocols has been described, although the amount depends on the programme design, as found elsewhere (Boobis *et al.* 1983, Cadefau *et al.* 1990).

TAN and ATP concentration at rest were lower (although not significantly) after training than before in both groups. Such a reduction of TAN owing to high-intensity muscle contractile activity has been previously reported (Hellsten-Westing *et al.* 1993, Stathis *et al.* 1994). Moreover, this fall has been related to an insufficient resting-time between bouts for PCr resynthesis and to a parallel loss of muscle IMP by catabolism (Stathis *et al.* 1994). In our experiment, the recovery periods between bouts were 12 min, long enough for total PCr resynthesis (Bogdanis *et al.* 1995), but not long enough for the resynthesis of IMP to AMP. However, the later sessions probably generated sustained high IMP concentration because of the intensity of training (seven bouts of 30 s) and IMP could then be catabolized. Indeed, increased catabolism of IMP has been described after intense exercise repetition (Bangsbo *et al.* 1992). Thus, the recovery periods between bouts and the exercise intensity seem to be important in the adaptation of ATP and IMP metabolism.

Before training, enzyme activities were similar in both groups. However, after training, enzyme activities were clearly varied. Creatine kinase activity showed a significant increase in the SP group. This enzyme usually shows only slight variations, probably because of its abundance (Cadefau *et al.* 1990). This study points out the possible importance of rest periods between sessions of training in order to produce an increase in the muscle CK activity.

In the glycolytic pathway, PFK and ALD activities increased in both groups. An increase in PFK activity is expected after sprint training, but its extent seems to depend on the training procedure (Cadefau *et al.* 1990, Linossier *et al.* 1993). PK and LDH activity increased significantly only in the SP group. These data indicate that the more concentrated the protocol, the greater the changes in glycolytic enzymes (PFK, PK and LDH).

Aerobic metabolism improvement, represented by CS and HAD activities, is an unusual adaptation to sprint training. However, high production of lactate following repeated bouts might induce an aerobic adaptation by improving the metabolism of the excess of pyruvate through pyruvate dehydrogenase (MacDougall *et al.* 1998). If we consider this possibility, both programmes were intensive enough to induce aerobic adaptation, although shorter rest periods induced larger increases in HAD activity ($P < 0.05$).

It is of particular interest to note the lack of variation in the HK activity of both groups, while increases of HK activity has been found after other sprint training protocols (Linossier *et al.* 1997, MacDougall *et al.* 1998). These authors described an increase in HK activity when recovery/work ratio was 8 (4 min of recovery between repetitions of 30-s bouts, MacDougall *et al.* 1998) or 11 (55 s of recovery between repetitions of 5 s bouts, Linossier *et al.* 1997). Our sessions had a recovery/work ratio of 24 in both groups. Thus, this ratio of recovery periods between bouts could be involved in the adaptation of the external glucose use through HK.

Muscle metabolic response to the post-training test

Muscle metabolic response to the post-training test was different in the two groups. Glycogen consumption during the post-training test reached 25% of the rest concentration in the LP group but only 15% in the SP group. Consequently, glycolysis and glycogenolysis rates during the post-training tests were significantly higher in LP group. However, part of this difference was a consequence of a significant decrease in rates of the SP group. These suggestions are proposed when related to the weight of muscle and they could differ in the case when related to the whole body because the recruited muscle mass may be increased after training.

The increase in performance of the 30 s all-out test in LP group was associated with an increase in

enzymatic activities related to muscle energy metabolism. The relationship between muscle enzymes and performance has been suggested by Linossier *et al.* (1997) and MacDougall *et al.* (1998). However, the lack of correlation between the improved performance (maximum power output appears at 5 s) in the 30-s test and the glycolytic rate averaged over the 30-s period is a question which invites further studies, specially focused on a better understanding of the muscle metabolism during the first few seconds of high-intensity exercise.

With shorter rest periods, the SP group consumed less glycogen and anaerobically generated ATP and produced less lactate during the post-training test than before training. In contrast, performance was similar to the pre-training value. Hence, we suggest a decreased involvement of anaerobic metabolism and an enhanced involvement of aerobic metabolism through the increase in CS and HAD activities.

However, the reduction in anaerobic ATP consumption and the failure to improve performance in the post-training 30 s test were unexpected, because changes of enzyme activities in SP group were greater than in the LP group. A possible explanation for this might be that their muscles were suffering fatigue or injury (Allemeier *et al.* 1994). Repeated exercise at high intensity may cause a loss of K^+ from the contracting muscle (McKenna *et al.* 1993) and a decrease in the gradient regulated by muscle Na^+-K^+-ATPase has been related to fatigue (Lindinger & Heigenhauser 1991). This K^+ homeostasis in exercising humans has been connected with rest duration (Kowalchuk *et al.* 1988). Another critical point to explain fatigue is the intracellular Ca^{2+} exchange (Williams & Klug 1995). Possible training-induced alterations in Ca^{2+} uptake and alterations in the sarcoplasmic reticulum could be rest-dependent, given the different responses of the two groups. More than 48 h of recovery, after the last training session, had in all probability avoided part of the remaining negative effects of the last training session.

It can be concluded that sprint cycling training may produce major enzyme activity changes in human muscle such as PFK, ALD, CS and HAD activities, together with increases in glycogen concentration. However, part of these modifications depend on rest distribution. We suggest that some adaptations are better induced by shorter rest periods, such as increases in PFK, HAD, PK and CK activities or in PCr concentration, although LDH activity is the most sensitive to rest distribution.

Furthermore, in spite of the fact that shorter rest periods during high-intensity training induce greater biochemical adaptation in human muscle than a more restful training programme with the same muscle load, less rest hinders the improvement of short-time performance, probably because of fatigue.

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ASSESSMENT METHODOLOGY ON THE EFFECTIVENESS OF PSYCHOLOGICAL TRAINING[1]

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INTRODUCTION

Psychological training in high level athletes has developed widely during the last decade showing its usefulness in controlling precompetitive anxiety as well as in enhancing competitive performance (Capdevila and Cruz, 1991, 1992; Vealey, 1988). However, since psychological training includes several

psychological techniques, it is often difficult to assess whether all these techniques are useful or not. More than this, and because of methodological difficulties to test the effectiveness of psychological training in natural settings, some studies offered unclear results about the usefulness of different psychological training methods (see Greenspan and Feltz, 1989, for a review). Thus, the correct assessment of the effects of psychological training in aspects such as precompetitive anxiety or competitive performance has not yet been established. In order to achieve these methodological issues, we are following the main guidelines established by ourselves (Capdevila and Cruz, 1991, 1992) and authors such as Prapavessis, Grove, McNair and Cable (1992), and we are developing some strategies addressed to obtain an adequate assessment of the efficacy of different techniques used widespread in psychological training in competitive sport.

METHODOLOGY

We consider that single case design is the most appropiate strategy to assess correctly the psychological demands expressed by the athlete as well as the possible benefits obtained by psychological training on self-report, physiological and behavioral measures. In order to achieve these goals, four steps are followed:

1. Step 1: Pretest levels

Using interviews and questionnaires, such as CSAI (Maertens, 1977), Rating of Perceived Exertion (Borg, 1985), or others

questionnaires and rating scales developed by ourselves, athletes explain their problems and feelings in front of practice and competition settings. At the same time, physiological measures are recorded, when possible, with techniques such as lactate tests and heart rate telemetry. Videotape recordings of the athlete prior to and during a practice or competition session are also registered. These informations are analyzed by psychologists providing them a profile of the psychological demands and problems of the athlete, which can be related (by analyzing videotape records) with the behaviors developed by the individual on practice and competitive settings.

2. Step 2: Laboratory sessions

At laboratory setting, different performances are demanded to the athlete by using bycicle ergometer or treadmill run. Psychophysiological recordings of electromyography, heart rate or respiratory frequency, are obtained, as well as biochemical indicators (lactate tests) and psychological indexes (by using questionnaires and rating scales similar to those described on step 1). Once obtained, these measures will be used as a baseline which will be considered as a criterion to establish the usefulness of the psychological training which will be applied on the next step.

3. Step 3: Application of the psychological training techniques

In this step, the athlete is instructed individually in the use of a technique which is suspected to be adequate to control his/her psychological competition-related anxiety and/or

performance. The technique we are now using is the heart rate biofeedback, addressed to enhance cardirespiratory efficiency. Since this is the first technique we are testing within our general research design, only preliminary aspects and results will be described. Once assessed the usefulness of this technique, other strategies such as imagery or control of the respiratory rhythm will be also tested in a similar way.

We have chosen maximal tests to obtain the baseline and visual biofeedback of heart rate as training strategy. Because of technical reasons, we have tested this technique in bycicle ergometer only, and not yet in the treadmill run, although the general research design will be the same in both apparatus. In the preliminary designs developed, the athlete participates in two laboratory sessions. In the first one, a maximal effort test is developed and is followed, 30 minutes later, by a 6-minutes submaximal effort test at 50% of his/her maximum cardiorespiratory efficiency. This session is considered as baseline. One week later, a second session, identical to the first one, is developed, although heart-rate biofeedback is included at the submaximal test. The heart-rate biofeedback is provided in different ways, in order to find some guidelines which can allow us to the best schedule to provide optimal results. As an example, Figure 1 shows the results obtained by a subject who received heart-rate biofeedback at the first 2-minutes and at the last 2-minutes of the submaximal test.

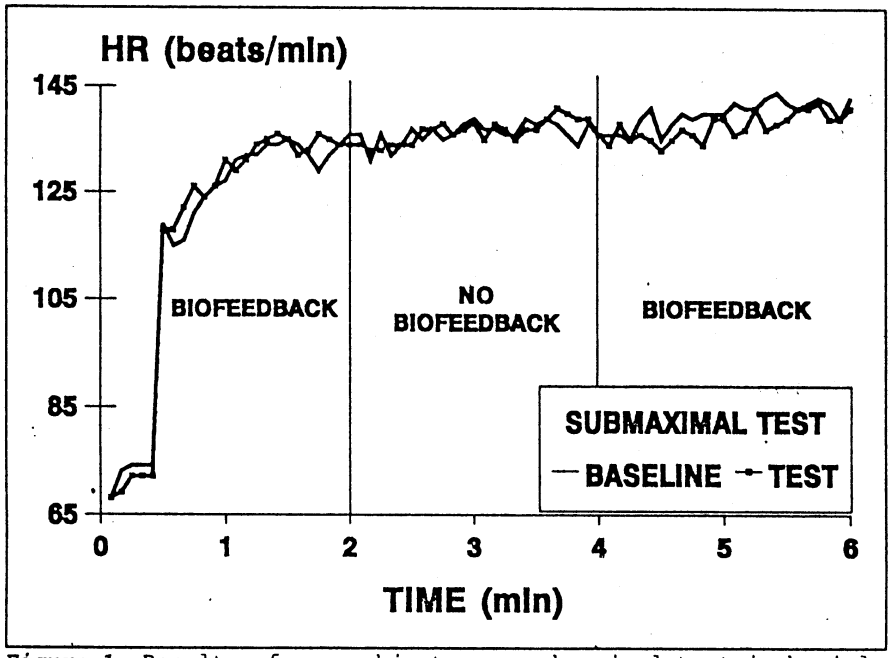

Figure 1. Results of one subject on a submaximal test in bycicle ergometer.

Although a visual analysis suggests that heart rate is lower than baseline data in the second biofeedback period, results are not still clear. Whatever the case, and in order to assess whether biofeedback, or any other psychological technique, produces some changes on cardiorespiratory efficiency, an adapted time-series analysis we have yet developed (Capdevila and Cruz, 1991, 1992), can be applied.

We must point out that we are describing preliminary reports addressed to find the optimal laboratory tests which allow us to the optimal assessment of the efficacy of psychological techniques. Once developed these tests and identified the best psychological techniques for each athlete, different

psychological training programs combaining different strategies and techniques will be developed and tested at the laboratory setting both by treadmill run or bycicle ergometer.

4. Step 4: Assessment on practice and competitive settings

We have not yet developed any study on this phase. Our intention is to obtain data in a similar way as described on step 1, once the athlete has acquired the psychological techniques suggested by the psychologists and its usefulness has been established by the tests described on step 3.

DISCUSSION AND CONCLUSIONS

Our purpose has been to show how to develop a specific assessment methodology about the usefulness of psychological training programs. We have not yet concluding results, but they will be progressively obtained in the near future. These results could imply modifications of some aspects we are now developing, but we consider that the general design (the four steps described) is adequate. Whatever the case, we think that single-case design is always the best strategy to test the efficacy of psychological training methods, both on laboratory or natural settings, when our purpose is to achieve new goals on the application of sport psychology to individual sports.

Furthermore, the single-case designs, as has been stated by Bryan (1987), Smith (1988), Wolman (1986) and Zaichowsky (1980), also have some advantatges over the traditional group designs, such

as: a) fewer number of subjects are required; b) small, l consistent effects, that may be masked in a group design, can detected, and, c) they are more appropiate when working w elite athletes whose performance is unlikely to change clea from baseline levels.

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Effect of prior ingestion of glucose or fructose on the performance of exercise of intermediate duration

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Abstract. The metabolic responses induced by the ingestion of a beverage containing glucose (G), fructose (F) or placebo (W) 30 min before exercise of high intensity and intermediate duration have been investigated; in these conditions the energy processes are mostly dependent on aerobic reactions. A group of 11 male recreational sportsmen ran on a treadmill, at an intensity corresponding to 82% of peak oxygen consumption, until exhaustion on three different occasions (after ingestion of a beverage containing 75 g of G, 75 g of F or W). Plasma glucose, insulin, and lactic acid concentrations were determined just prior to the ingestion of the beverages, 30 min afterwards and 10 and 30 min after completion of the exercise. The mean endurance time was 644 (SD 261) s after the ingestion of G, 611 (SD 227) s after the ingestion of F and 584 (SD 189) s after the ingestion of the W ($P<0.05$ between G and W). No differences in the oxygen uptake, respiratory quotient or lactate concentrations between the three trials were observed. Both plasma glucose and insulin concentrations determined in samples obtained immediately before the onset of exercise were higher when G was ingested than when F ($P<0.05$ and $P<0.05$, respectively) or W ($P<0.001$ and $P<0.005$, respectively) were ingested. These findings would suggest that the ingestion of G prior to an effort of intermediate duration may improve physical performance.

Key words: Blood glucose – Carbohydrates – Endurance

Introduction

Apart from genetic endowment and adequate training, no factor other than nutrition plays a more important role on physical performance (Costill 1988). Diets enriched with carbohydrates ingested during the days prior to a competition are quote often used by athletes to improve physical performance in activities of long duration (Costill 1988; Coyle et al. 1985); such procedures have also been studied in activities of moderate duration of around 1 h (Foster et al. 1979; Hargreaves et al. 1987; MacMurray et al. 1983), as well as in short, strenuous exercise at an intensity corresponding to 100% of the maximal oxygen uptake ($\dot{V}O_{2max}$) (Foster et al. 1979). No studies have been performed on the effect that the administration of glucose, shortly before physical activity, may have on the performance of exercise of intermediate duration, which are the type of activities carried out perhaps by the majority of sportsmen. Such typical activities would be those of races of 1500 m, 3000 m or even 5000 m track and field events, or the 400 m or 1500 m in swimming competitions, to mention just a few. All the above-mentioned activities represent effort that has to be sustained for more than 2 min and are, therefore, mostly dependent on aerobic processes for the regeneration of ATP.

In the initial phases of such exercise, the muscle has been shown to utilize glucose as the main substrate, with a progressive increase in the proportion of fatty acids according to the duration (Costill 1988; Hagenfeld 1979). Due to the fact that the respiratory exchange ratio (R) is higher when glucose is being oxidized, the ratio of CO_2 to O_2 exchange constitutes an indicator of the type of substrate being utilized in the steady state. In this respect, it is necessary to take into account the fact that the efficiency of the oxidative system has been shown to be higher when glucose is consumed – about 12% more adenosine triphosphate (ATP) per volume of O_2 consumed being generated when glucose is oxidized than when fatty acids are being utilized (Fox et al. 1989; Newsholme 1986).

The ingestion of glucose may have negative effects on the supply of energy substrates to the muscle. When the amount of glucose being absorbed in unit time surpasses a certain value, the small intestine liberates a larger amount of the gastric inhibitory peptide, which has a high insulinotropic activity. Further, the secretion of insulin by the pancreas increases markedly, resulting in a lower lipolytic activity in adipose tissue and, thus,

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a smaller increase in the supply of nonesterified fatty acids through the plasma. This favours a higher glucose uptake by all insulinodependent tissues that will compete with the muscles for the circulating glucose. If such conditions prevail during the initial phases of physical exercise, it has been found that the muscle will have to resort more to the glycogen stores to obtain the amount of energy required, accelerating the depletion of muscle glycogen and, thus, precipitating the appearance of fatigue when a sustained effort of 1 h or more duration is being performed (Costill 1988). However, with exercise of shorter duration glycogen depletion has not appeared to be the limiting factor: when the rate of aerobic metabolism is not able to meet the energy demands of the muscle, the acid-basic disturbances induced by the byproducts of the anaerobic pathways have appeared to be the likely cause(s) of exercise limitation (Foster et al. 1979).

Nevertheless, muscle glycogen has been shown to be the primary and initial source of substrate for oxidative phosphorylation in the contracting muscles (Costill et al. 1977; Gollnick et al. 1974). The ingestion of glucose prior to exercise has been shown to increase muscle glycogen utilization and total carbohydrate oxidation (Costill et al. 1977). It has been shown that blood glucose at concentrations below 5.5 $mmol \cdot l^{-1}$ do not appear to affect this process (Cooper et al. 1989).

When fructose, instead of glucose, is ingested before exercise, a smaller increase in the concentrations of glucose as well as of insulin in plasma has been observed and, at the same time, only a small reduction in the fatty acid mobilization has been found to be produced (Crapo and Kolterman 1984; Hargreaves et al. 1987; Koivisto et al. 1981, 1985; MacMurray et al. 1983). Thus, the ingestion of this sugar would offer some advantages over that of glucose, when performing exercise of long duration. However, the studies performed thus far are not conclusive: some have suggested improvement (Okano et al. 1988) and others have found no effect on physical performance (Hargreaves et al. 1987). To our knowledge, no studies have been performed on the effects of fructose ingestion prior to exercises of short or intermediate duration.

We have attempted, with this study, to determine the effect that the ingestion of either glucose or fructose shortly before the exercise of intermediate duration would have on physical performance and to evaluate the metabolic changes produced under these circumstances.

Methods

Subjects. A group of 11 male amateur sportsmen, with different levels of physical fitness agreed to participate in the study after the characteristics and objectives of the trials were explained to them. The mean characteristics of the participants and of the tests performed are as follows: age 25 (SD 5) years, height 173 (SD 5) cm, mass 70 (SD 8) kg, treadmill gradient 5% and treadmill speed 12 (SD 2) $km \cdot h^{-1}$, equivalent to an oxygen uptake ($\dot{V}O_2$) of 82% of $\dot{V}O_{2max}$ [51 (SD 6) $ml \cdot kg^{-1} \cdot min^{-1}$]. All the participants consumed their normal standard diet and performed their habitual amount and type of physical activity on the days previous to the laboratory tests. These tests were performed in the afternoon, a minimum of 3 h after lunch, in a well ventilated room with an ambient temperature of 22–24° C and a relative humidity ranging between 43% and 48%.

Protocol. The participants ingested one of the following beverages 30 min before each exercise test:
1. An amount of 100 ml of water (W) flavoured with orange juice and sweetened with Aspartame to a level similar to that of the other solutions:
2. An amount of 100 ml of a solution containing 75 g of F, flavoured with orange juice and
3. An amount of 100 ml of a solution containing 75 g of glucose (G) flavoured also with orange juice and adjusted with Aspartame.

The different solutions were ingested on three separated occasions in a random, double blind order, and spaced a week apart.

The peak oxygen uptake was determined through a maximal incremental test performed on the treadmill (Ergometrics, Barcelona, Spain) a week before the different trials were started. An exercise test at an intensity corresponding to 82% of the peak oxygen uptake, was performed 30 min after the corresponding ingestion. Each test was initiated by a period of running at 8 $km \cdot h^{-1}$, with a gradient of 5%, for 5 min before reaching the speed assigned to each individual. In each case, the time elapsed until exhaustion was recorded. The individual was monitored for heart rate, $\dot{V}O_2$ and other respiratory parameters, electrocardiographic events, etc., throughout the test and during the first 10 min of the recovery period.

Each participant respired through a mask (Rudolph 2700, Kansas City, Mo, USA), the gaseous exchange being determined and monitored by an automatic open circuit system (Oxycon 4, Mijnhardt, Odijk, The Netherlands). The system was calibrated with mixtures of O_2 and CO_2 of a known composition, before each test. The spirometer was calibrated using a syringe and the treadmill speed using a chronometer.

The expired air was analysed every 30 s during the test and the recovery period. The excess $\dot{V}O_2$ during the recovery phase was estimated by subtracting the $\dot{V}O_2$ in resting conditions (Kinney 1988) from that obtained every minute during the first 10 min of the recovery period: the value obtained in each case was referred to the body mass of the subject and expressed as millilitres of O_2 per kilogram of body mass. Blood samples were obtained from the antecubital vein before the ingestion of the corresponding solution or beverage, 30 min after that and 10 and 30 min after the completion of the test. For each sample the concentrations of glucose, lactate and insulin were determined. In addition, 7 min after the end of each trial a blood sample for determination of lactate concentration was obtained. The plasma concentrations of glucose and lactate were determined using enzymatic methods and that of insulin using radio-immunoassay.

Statistics. The data obtained was subjected to multiple regression analyses (MANOVA) and Student's *t*-test applied to paired values for those more differentiated. A value of P was considered significant when lower than 0.05.

Results

As can be seen in Fig. 1 there is a significant difference in the duration of the sustained effort after ingesting the G solution compared to the ingestion of W. The mean time of endurance after ingesting G was 644 (SD 261) s while after the ingestion of W it was 584 (SD 189) s ($P<0.05$). In the trials performed after the ingestion of a solution containing F an improvement in

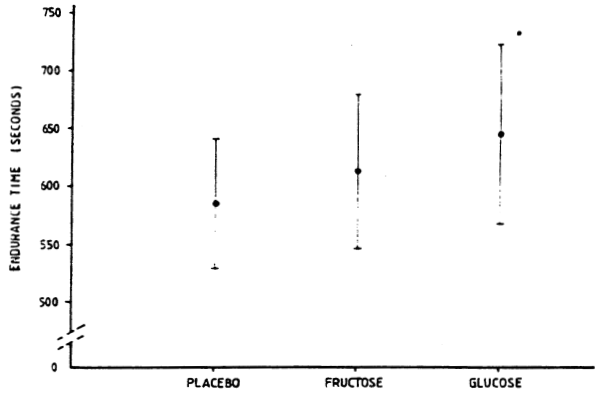

Fig. 1. Mean (standard error) endurance time after the ingestion of beverages of different composition. Value of P between glucose and placebo <0.05

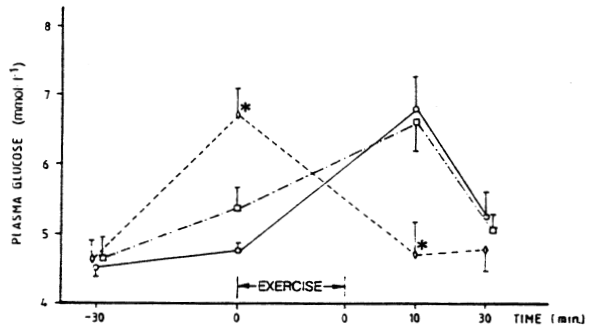

Fig. 2. Mean values (standard error) for plasma concentration glucose during each trial at baseline (−30), pre-exercise (0), 10 min postexercise and 30 min postexercise. ○—○ Placebo; □–·–·–□ fructose; ◇----◇ glucose. * $P<0.05$ glucose group with the other groups

performance, estimated by the endurance time, could also be observed although the difference from W was not statistically significant, 611 (SD 227) s versus 584 (SD 189) s ($P>0.05$) (Fig. 1).

No differences were noted among the different trials in relation to the increment in the lactate concentrations between basal conditions and the maximal values at the end of the exercise tests ($P=0.53$). The maximal concentrations reached were 7.89 mmol·l^{-1} above the basal levels in the case of G, 7.47 mmol·l^{-1} in the case of F and 7.23 mmol·l^{-1} in the case of W. The excess $\dot{V}O_2$ during the recovery period did not show any difference among the different trials in relation to the type of sugar ingested before the exercise ($P=0.30$). Thus the excess $\dot{V}O_2$ was 87 (SD 15) ml·kg^{-1} when W was ingested, 98 (SD 33) ml·kg^{-1} when G was ingested, and 94 (SD 35) ml·kg^{-1} when F was ingested. The maximal R during the test was very similar in all the trials regardless of the solution ingested before the corresponding exercise [1.07 (SD 0.04) in the case of W ingestion, 1.08 (SD 0.04) in the case of G ingestion and 1.04 (SD 0.06) when F was ingested]. Neither the $\dot{V}O_2$ nor R 5 min after the initiation of the exercise showed any differences of statistical significance among the three trials: 54 (SD 7.1) ml·kg^{-1}·min^{-1} and 1.08 (SD 0.04) for W, 54.9 (SD 6.6) ml·kg^{-1}·min^{-1} and 1.07 (SD 0.07) for F and 54.3 (SD 7.5) ml·kg^{-1}·min^{-1} and 1.07 (SD 0.06) for G.

At 30 min after the ingestion of the different beverages and at 10 min after completion of the exercise the plasma glucose concentrations showed large differences, according to the type of carbohydrate ingested (Fig. 2). At 30 min after the ingestion, there was a significant difference between the G and F trials ($P<0.05$) and between the G and W trials ($P<0.005$) with no differences between the W and F trials. On the other hand, plasma glucose reached much higher concentrations 10 min after the exercise when ingesting F or W, with a highly significant difference for the levels achieved after G ingestion ($P<0.05$ and $P<0.005$, respectively).

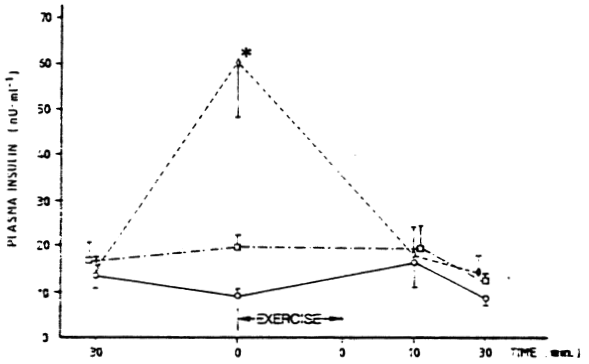

Fig. 3. Mean (standard error) for plasma insulin concentration during each trial at baseline (−30), pre-exercise (0), 10 min postexercise and 30 min post-exercise. For explanations see legend of Fig. 2

The composition of the beverage ingested induced significant variations in the plasma insulin concentrations only 30 min after ingestion of G, when insulin reached its highest values, with a significant difference for the values achieved after F ingestion ($P<0.05$) and after W ingestion ($P<0.005$; Fig. 3).

Discussion

The significant increase in the duration of the effort sustained after ingestion of G in comparison to a placebo not containing carbohydrates, favours the possibility that there is a shift towards a major utilization of glucose, as a metabolic substrate, as happens in exercise of longer duration (Costill et al. 1977). This improvement was also evident when F was ingested shortly before exercise although the difference with W was not statistically significant; one has to take into account that it has been shown that F is absorbed more slowly than glucose and requires its conversion into

glucose, in the liver, before it can be utilized by muscle and the body as a whole (Crapo and Kolterman 1984; Hargreaves et al. 1987; Koivisto et al. 1985; MacMurray et al. 1983). In experiments performed both on animals and humans, contradictory data have been reported, by different groups, on the fate of F absorbed in the gut.

Some have observed a lower activity of glycogen synthase phosphatase after administration of F to rats, with a concomitant reduction in the proportion of the glycogen synthase I active form (Regan et al. 1980). The increase in the proportion of the inactive form would impair storage of glycogen by the liver and thus favour glycogen breakdown and glucose catabolism. On the other hand, others have observed in human volunteers an increase in the amount of muscle and, especially, hepatic glycogen – analysed in appropriate biopsy samples – after ingestion of F (Nilsson and Hultman 1974). Nevertheless it has to be pointed out that, in both cases, the subjects did not perform any physical activity.

The lack of differences in the differential for lactate concentration could be explained by considering that probably with G, the rate of lactate production is lower, but since the exercise was continued for a longer time, the same end-exercise lactate concentration was achieved as with W.

In agreement with findings of other groups (Foster et al. 1979; Hargreaves et al. 1987), we have not observed significant differences in the $\dot{V}O_2$ among the different trials. One possible explanation could be that prior ingestion of glucose, with the associated increase in insulin, led to a transient elevation of muscle glucose content (as both G-6-P and glycogen), and its utilization (Costill et al. 1977), this greater carbohydrate availability leading to the slightly but significantly greater endurance time. With exercise of this duration glycogen depletion has not appeared to be the limiting factor (Foster et al. 1979).

Theoretically, one would expect, for the same exercise intensity, a lower $\dot{V}O_2$ when using a larger proportion of glucose due to the higher energy efficiency of carbohydrates. However, the lack of differences – as far as $\dot{V}O_2$ is concerned – among the different trials could be explained by the lower sensitivity and precision of the O_2 measurement methods in comparison with the measurement of duration. An alternative explanation could be that the higher efficiency reduced not $\dot{V}O_2$ but the production of lactate, probably being lower in the case of G at any moment of the test; to demonstrate this it would have been necessary to determine serially the lactate concentration during the test.

Concerning R both as it relates to its change as well as its maximal values, we have not observed differences between the different trials. As R is determined both by the type of substrate being oxidized and by the concentrations of lactic acid attained, with a rhythm of production probably quite different in the three groups or trials, both the higher energy performance, as well as the different metabolic alternatives could mask any differences that could appear in the R value due to the utilization of the different energy substrates. In addition, it is known that R is a rather insensitive parameter for the quantification of the metabolic changes occurring during effort of short duration (Wasserman et al. 1973).

The plasma insulin concentrations 30 min after the ingestion of the different solutions were lower after ingesting F than after G as has also been observed by other authors (Koivisto et al. 1981, 1985). The other substrate utilized by muscle, the nonesterified fatty acids, shows a dynamic quite different from that of the carbohydrates. At 10 to 15 min after the initiation of an exercise, the plasma concentrations of nonesterified fatty acids have been shown to experience an important reduction in relation to those present at the beginning of the activity (Foster et al. 1979; Hagenfeldt 1979; MacMurray et al. 1983; Sherman et al. 1989). This initial decrease has been shown to be due to an increased utilization by the muscle, not matched by a simultaneous increase in their mobilization from the adipose tissue (Hagenfeldt 1979). Although the proportion of energy obtained through the oxidation of fatty acids diminishes as the intensity of the effort increases, the contribution of this substrate to the regeneration of ATP in our tests was, probably, quite important for it has been estimated that, in similar cases, they furnished more than 30% of the energy transformed by the muscle (Hagenfeldt 1979).

Taking into account the timing of the different processes involved in their mobilization, transport and oxidation and considering that the most important limiting factor has been thought to be the rate of hydrolysis and liberation to the plasma from the adipose tissue (Hodgetts et al. 1991) and that the type of effort performed in our studies was of short to intermediate duration, we have assumed that most of the fatty acids utilized in our trials came from the muscle stores of triglycerides which will furnish the first fatty acids being oxidized. This is similar to the muscle glycogen that constitutes the immediate source of glucose at the beginning of exercise (Costill et al. 1977; Gollnick et al. 1974; Wahren 1979). Although blood glucose at the intensity of our exercise may not be utilized during the initial 10 min with glucose concentrations below 5.5 $mmol \cdot l^{-1}$ (Cooper et al. 1989), in our case we cannot exclude a certain glucose uptake due to the high concentrations achieved in plasma just prior to the beginning of exercise.

On the other hand, the lack of data on the variations of the different parameters during the exercise – as blood samples were not obtained during this period – does not preclude the initial hypothesis, that the improvement in performance by a greater utilization of carbohydrates was due to the fact that, at the beginning of the exercise test, the plasma insulin concentration was more elevated when ingesting G than when ingesting F or W. It is well known that insulin has a long lasting effect that causes a shift in plasma borne fuels from fatty acids to glucose (Coyle et al. 1985).

In conclusion, our findings suggest that the inges-

tion of G prior to exercise of high intensity and intermediate duration may improve performance, as measured by the endurance. However, more work has to be done, with samples obtained at short intervals during exercise as well as with suitable muscle biopsies, to evaluate the time changes in the glycogen and lipid stores according to the type and amount of carbohydrate ingested before exercise.

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909 COMPARISON OF THE BASAL METABOLIC RATE AMONG CHAMPIONS OF TWO NATIONALITIES IN OLYMPIC AND WORLD GAMES.

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The recommendation of the FAO/WHO/UNU committee that the basal metabolic rate (**BMR**) serves as the basis for estimating dietary energy intake has rekindled interest in this fundamental physiological measurement. This study had as objective to compare BMR among champion athletes from Spain (**SPA**) (n=15) and from Brazil (**BRAZ**) (n=15), of the masculine sex , of 4 different sports in even numbers of athletes of each sport. The date of evaluation of BMR is characterized in the Sport Calendar as period of "post competition" that happened in the Olympic and World games. An Mijjnhardt gases analyzer was used for calculation of VO_2 and VCO_2. Routine procedures were strictly followed in the preparation of basal conditions and during its measurements for BMR calculation. The measurement of the oxygen consumption was accomplished along 35 minutes.

Champions' Nationalities	Climate Type	Age years	BMR $LO_2 \cdot min^{-1}$	BMR $mL \cdot kg\ WT^{-1} \cdot min^{-1}$	BMR $mL \cdot kg\ LBM^{-1} \cdot min^{-1}$
Brazil	Tropical	26.23 ± 3.45	0.23±0.07	3.17 ± 0.77	3.44 ± 0.85
Spain	Temperat	25.54 ± 4.32	0.28±0.06	3.82 ± 0.65	4.15 ± 0.71

These results suggest that the Brazilian athletes haver heavier weight (**WT**) and had more higher lean body mass (**LBM**), that could sugest a higher BMR However, the relative percentage was of **less 17.86%** of BMR ($LO_2 \cdot min^{-1}$) and **17.10%** of BMR ($mL \cdot kg\ WT^{-1} \cdot min^{-1}$) and **17.11%** of BMR ($mL \cdot kg\ LBM^{-1} \cdot min^{-1}$) in relation to the Spanish athletes. Conclusion: The champions from Brazil that inhabit a Tropical zone presented significant lower BMR ($p \leq 0,05$) than the Spanish champions that live in a temperate climate.

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Differences between lactate concentration of samples from ear lobe and the finger tip

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J. FELIU, J. L. VENTURA, R. SEGURA, G. RODAS, J. RIERA, A. ESTRUCH, A. ZAMORA and L. CAPDEVILA. *Differences between lactate concentration of samples from ear lobe and the finger tip.* J. Physiol. Biochem., **55** (4), 333-340, 1999.

Blood lactate concentrations in capillary samples obtained from the ear lobe or from the finger tip are used indistinctly, since they are considered equivalents. The aim of the study reported in this paper was to verify whether that assumption is valid due to the practical implications which any possible differences between these two sampling sites would have in the planning and assessing of an athletic training program. Twenty six healthy male athletes competing in different sports at the national level (9 rowers, 7 cyclists and 10 runners) were studied during the performance of a graded exercise test up to the point of exhaustion, on specific ergometers. In each group, capillary blood samples were obtained simultaneously from both the ear lobe and the finger tip at three different times during the test: 1) in resting conditions; 2) when exercising at a submaximal work load and 3) seven minutes after the point of exhaustion. Significant differences were found between the blood lactate concentrations of samples obtained from the ear lobe and from the finger tip ($p < 0.001$). The method error of repeated measurements for lactate concentrations from paired samples obtained in resting conditions was 27%, when exercising at a submaximal work load, 16% and at maximal work load, 3%. Capillary blood samples collected from the finger tip consistently showed higher values in lactate concentration than those obtained, at the same time, from the ear lobe.

Key words: Blood lactate, Ear lobe sample, Finger tip sample.

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In recent years, determination of plasma lactate concentration has become an habitual procedure for assessing physical fitness as well as for the evaluation and monitoring of training programs (1, 2, 5, 6).

Due to the fact that the concentration of lactate in capillary blood reflects the lactate values in arterial blood quite well, as shown both in laboratory and field studies (9, 10, 11, 18), capillary blood samples are preferred since they are more easily obtained, require a lower degree of technical skills and involve a less invasive procedure. Samples are obtained by micropuncture at the ear lobe or the finger tip. Sampling at the ear lobe is used more often, because blood does not clot so easily, thus allowing for the collection of several consecutive samples. In the evaluation of the athletes, the capillary samples obtained from the ear lobe or from the finger tip are used indistincty as they are considered equivalent (1, 13).

In the present work we report the differences observed in the lactate concentration between blood samples obtained simultaneously from the ear lobe and from the finger tip in a group of subjects who were subjected to physical exercise tests through a wide range of work intensities and on different types of ergometers.

Materials and Methods

Subjects.– Twenty-six male athletes who compete in different sports at a national category agreed to participate in the study after the main characteristics and objectives of the project were explained to them. The personal features of the whole group, expressed as mean and (SD), were: 20 (3) years of age; 70 (9) kg of body mass; 177 (6) cm of height and a peak oxygen uptake of 73 (8) $ml.kg.min^{-1}$. The group was composed of 9 rowers, 7 cyclists and 10 runners. All the subjects were declared to be in good health after receiving a complete check-up.

Test Procedure.– Each subject performed a graded exercise test until exhaustion. All trials took place in the morning in a well-ventilated room at a temperature of 22-24 °C and with a relative humidity ranging between 43 and 48%.

Capillary blood samples were taken simultaneously from the ear lobe and from the finger tip of the left side according to a standarized procedure (3). Both the ear lobe and the finger tip were dried with cotton and then pricked with an hemostylet (Glucojet; A.Menarini Diagnostic, Badalona, Spain) on three consecutive occasions during each trial: before the beginning of the exercise test, between 15 and 30 seconds after reaching a submaximal work load -estimated to correspond to a heart rate of 160 beats/minute-, and seven minutes after having performed an exercise at a maximum work load. The capillary blood samples were collected in 50 µl heparinized glass tubes.

In order to reduce the bias to a minimum, the subjects who collected the blood samples alternated punction or pricking sites in such a way that the person who had obtained the sample from the ear lobe collected the next sample from the finger tip, and viceversa. The lactate concentration of each sample was determined by an electroenzymatic method (Lactate II reagent GM/LM series analyzers, the GMRD-code-90, Analox Instruments LTD, London, United Kingdom), which has been shown to have acceptable precision and accuracy (10).

Although all subjects performed a graded exercise test up to the point of

exhaustion, the type of ergometer used and the kind of protocol followed during the trial was different, according to the sport modality practiced by each athlete.

Runners.– The runners performed the test on a treadmill (Laufergotest JAEGER LF.6, Würzburg-1, Fed.Rep.Germany). After a period of muscle stretching, they started the trial. The speed of the treadmill was increased at a rate of 2 km/h per minute, without variations in the slope, which was maintained at 2.5 %. Two separate trials were performed: one up to the point when heart beat reached 160 beats/min and the second up to the point of exhaustion.

Cyclists.– The group of cyclists performed the trial on a cycloergometer (ERGO-METRICS/900, Bitz, Fed. Rep. Germany), following a triangular protocol, increasing the work load 50 W every three minutes. Two different trials were also performed: one at a submaximal work load (160 beats/min) and another one up to the point of exhaustion.

Rowers.– The members of this group performed the test in a remoergometer (CONCEPT-2, Morrisville, USA). Each individual exercised at a submaximal work load corresponding to rowing a distance of 2000 m at an average speed of 273 $m \cdot min^{-1}$ (16.3 $km \cdot h^{-1}$) during a mean time of 8 minutes. After a resting period of one minute, each subject repeated the test at the maximum possible speed (maximum work load) rowing the same corresponding distance (2000 m) at an average speed of 316 $m \cdot min^{-1}$ (19 $km \cdot h^{-1}$).

Statistical Analyses.- According to the research protocol there are:

a) One independent data factor, the ergometer, with three groups of values in accordance with the different athletes: rowers, runners or cyclists.

b) Two dependent data factors -or repeated measurements-, being the first the exercise level at the moment of sampling and the second the corporal place of extraction -ear lobe or finger tip-.

We analized all these variables by means of analysis of variance (MANOVA) with three factors: ergometer, exercise level and place of extraction. The statistical significance was considered when $p < 0.05$.

In addition, the method error of repeated measurements for paired samples, that may be applied to two trials in a testing session (11), was used. This method may be expressed in units of measurement or as the coefficient of variation (CV), which was calculated for each lactate level and sport modality, between the samples obtained from the ear lobe and from the finger tip.

Results

The most significant result is that lactate levels were always higher in the samples obtained from the finger tip than from the ear lobe ($p < 0.001$), with all ergometers and in all situations (table I). It was also observed different lactate evolution patterns in the rowers' group as compared to those of runners and cyclists: in the rowers' group the difference in the lactate levels between the ear lobe and the finger tip samples were higher at the maximal work load (1.50 versus 1.18 at the submaximal load), while in the runners the differences were 0.7 at the maximal work load versus 1.1 at the submaximal work load and in the cyclists 0.7 at the

Table I.- *Mean (SD) lactate concentration* (mmol.L^{-1}) *of the capillary blood samples obtained, at different moments of the test, from ear lobe or finger tip in differents groups of sportsmen.* All the differences reach statistical significance ($p < 0.001$).

Sport	Rest		Submaximal		Maximal	
	Ear	Finger	Ear	Finger	Ear	Finger
Runners	1.4 (0.2)	2.6 (0.9)	2.6 (1.2)	3.7 (1.3)	9.1 (2.4)	9.8 (2.4)
Rowers	1.5 (0.3)	3.3 (1.3)	3.2 (1.5)	4.3 (2)	17.1(2.1)	18.6 (2.4)
Cyclists	2 (0.3)	3 (0.6)	2.7 (0.6)	4 (0.8)	8 (3)	8.6 (3.2)

maximal work load versus 1.3 at the submaximal work load.

According to method error of repeated measurement of two samples, expressed as the coefficient of variation (CV), the degree of concordance between the values in samples obtained from the two places is lower in the blood samples obtained in resting conditions (mean C.V. 27%) than in the samples obtained when performing against a submaximal work load (mean C.V. 16%) or a maximal work load (mean C.V. 3%).

For both the group as a whole as well as for each individual subgroup, the highest degree of concordance and lowest error method -expressed as CV- was observed for the lactate values of blood samples obtained when performing at a maximum work load, when the concentration of plasma lactate reached the highest values. There does not appear to be a significant difference in the degree of concordance of the lactate values between sampling sites according to the type of exercise or sport performed (rowing, cycling, running).

As can be appreciated in fig. 1, the lactate values present in the samples obtained at different places in all the three sport modalities show higher correlation for the upper levels, the values being always higher for the samples obtained at the finger tip.

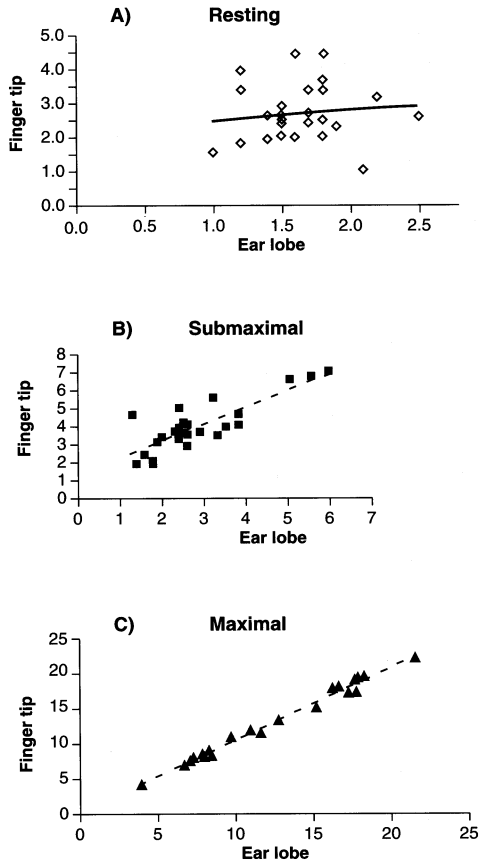

Fig. 1. *Regression line between blood lactate* (mmol.L^{-1}) *obtained from the ear lobe and from the finger tip in three situations (resting, submaximal work load and maximal work load) and sports.*

Discussion

Two research works have studied the correlation between the blood lactate values in samples obtained from the ear lobe and from the finger tip (3, 4), one of them specially designed to test the correlation between a new strip method and the classical enzymatic photometric analysis. Both papers show that the largest differences in blood lactate values in relation to sampling site occur between samples obtained in resting conditions when the largest coefficient of variation has also been observed. Differences in blood lactate concentration depending on sampling site have also been observed when exercising at different intensities although such differences are much smaller than those observed in resting conditions and without a clear trend in the differences. In both cases, further research on this topic was recommended by the authors.

The differences observed in the lactate concentration between blood samples obtained at the same time from the ear lobe and from the finger tip cannot be easily explained. Although the differences in the lactate values between samples obtained from the arterial or the venous bed could be attributed to the presence of different amounts of interposed muscle mass, that may take up or shed lactate from or into the blood (4, 8, 12, 16), this is not the case when the samples are both obtained from the microcirculation bed.

The fact that, in our work, the samples obtained from the finger tip show consistently higher mean values than those obtained from the ear lobe could be due to the presence of sweat, which has a high lactate concentration (up to 60 $mmol.L^{-1}$) (17). Although care is being taken to wipe out the sweat before pricking the corresponding skin site, a certain amount may remain in the fingerprints that, even in minimal quantities, could contaminate the blood being sampled. This would explain the consistently higher values observed in the blood samples obtained from the finger tip when compared with those obtained simultaneously from the ear lobe; at the same time, this would explain also the fact that the differences between the values of samples obtained from both sites are almost constant, from the lowest to the highest lactate concentrations. In any case, it would be difficult to explain the differences in lactate resting values due to the presence of sweat since in these conditions no increase in the activity of the sudoriparous glands takes place; even minimal amounts of sweat can increase the lactate levels and this fact has been adduced as an explanation for the larger resting values in uncleaned finger tips than in the cleaned fingertips and earlobes (3). The different lactate evolution pattern in the rower's group could indicate some degree of grasping effect.

Another factor to be taken into consideration is the amount of blood and the blood flow of each region of the sampling site. The hydrostatic pressure in the capillary bed of the finger tip is higher than that of the ear lobe due to the effect of gravity, although while rowing it does not appear to be so. On the other hand, differences in the participation of both regions in the thermoregulatory processes could determine the existence of a different blood flow rate through these two microcirculatory beds. In that respect, it is worth mentioning that it has been shown that capillary blood samples obtained from the ear lobe were not totally "arterialized", with an arteriovenous shunt rate superior to 6% (7). Should the percentage of "shunting" in the ear lobe be different from that in the finger tip, due to the

lower position of the hand or because of differences in the thermoregulatory processes, this could explain, at least in part, the differences that we have observed in the lactate concentration between samples obtained, simultaneously, from the ear lobe and from the finger tip; a similar explanation has already been proposed by Dietze (3). In this respect, we did not take into consideration the existence of possible temperature differences between the ear lobe and finger tip, and, as it is known (17), temperature levels affect blood flow and, therefore, may have an influence on blood lactate concentration. The differences in the type of tissue- and possibly the different ratio between organic and inorganic ions- in the ear lobe and the finger print also can justify, theoretically, the different results.

In conclusion, lactate concentrations in samples obtained from the ear lobe and from the finger tip are not identical either in resting conditions or when exercising at a submaximal or maximal work level, showing a general trend for lower lactate values in the samples obtained from the ear lobe without significative differences according to the degree of grasping. This fact could lead to different prescription load, depending on the site taken into consideration, making it necessary always to obtain the capillary samples at the same place if different physical tests are to be compared.

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J. FELIU, J. L. VENTURA, R. SEGURA, G. RODAS, J. RIERA, A. ESTRUCH, A. ZAMORA y L. CAPDEVILA. *Diferencias en la concentración de lactato entre muestras obtenidas del lóbulo de la oreja y del pulpejo del dedo.* J. Physiol. Biochem., **55** (4), 333-340, 1999.

Se acepta que los valores de lactato sanguíneo obtenido a partir de muestras capilares del lóbulo de la oreja y del pulpejo del dedo son equivalentes, por lo que se utilizan indistintamente. El objetivo del presente trabajo es comprobar si esta supuesta equivalencia es cierta, debido a las implicaciones que, en la práctica, puede tener en la programación del entrenamiento deportivo.

Veintiséis deportistas varones, sanos, practicantes de diferentes deportes de competición a nivel nacional (9 remeros, 7 ciclistas y 10 atletas), han sido estudiados por medio de pruebas de esfuerzo realizadas en ergómetros específicos para cada especialidad. En cada grupo, se extrajeron simultáneamente muestras de sangre capilar del lóbulo de la oreja y del pulpejo del dedo en tres momentos diferentes del test: 1) en reposo; 2) en carga submáxima y 3) siete minutos después de finalizado el esfuerzo a la máxima intensidad.

Se encuentran diferencias significativas entre la concentración del lactato en sangre en las muestras del lóbulo de la oreja y del pulpejo del dedo ($p<0,001$). El error del método de las mediciones repetidas de la concentración de lactato para muestras apareadas fue del 27% en reposo, del 16% en carga submáxima y del 3% cuando se alcanzaron las concentraciones máximas de lactato. La concentración de lactato de las muestras de sangre obtenidas del pulpejo del dedo es siempre superior a la de las obtenidas del lóbulo de la oreja.

Palabras clave: Lactato sanguíneo, Lóbulo de la oreja, Pulpejo del dedo.

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