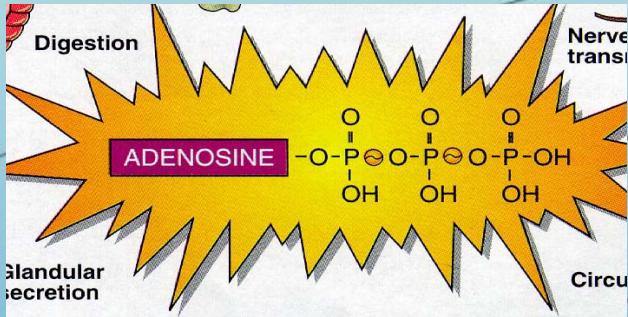


High Intensity Interval Training

Adenosin-tri-Fosfato
la moneta di scambio energetico



Marco Bernardi



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Ministero della Salute
Istituto Superiore di Sanità
Convegno Nazionale
**LA TUTELA DELLA SALUTE NELLE
ATTIVITÀ SPORTIVE
E LA LOTTA AL DOPING**
Istituto Superiore di Sanità
Viale Regina Elena, 299 - Roma

17 maggio 2012



COMITATO SCIENTIFICO

P. Calà, L. Caprino, M. Casciello, A. D'Andrea,
P. Della Porta, M. Frameglio, L. Fumagalli, A. Mandelli,
R. Pacifici, G. Santilli, A.E. Sberna, L. Simonetto,
C. Tomino, C. Tranquilli

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I Sessione

LA TUTELA DELLA SALUTE NELLE ATTIVITÀ SPORTIVE
Moderator: C. Tomino, A.E. Sberna

10.30 *I progetti di ricerca della CVD*
L. Caprino

10.50 *Le campagne informative/formative della CVD*
A. Mandelli

II Sessione

L'ATTIVITÀ DI CONTRASTO AL DOPING
Moderator: L. Fumagalli, G. Santilli

11.10 *I controlli antidoping della CVD e il Reporting System Doping Antidoping*
R. Pacifici

11.30 *I Carabinieri dei NAS e il contrasto al doping*
C. Piccinno

11.50 *Nuovi modelli organizzativi e operativi per la tutela della salute e la lotta al doping*
P. Fiorella

12.10 *L'attività antidoping del CONI-NADO*
G. Curli

12.30 Discussione

13.00 Intervallo

III Sessione

Moderatori: C. Tranquilli, M. Frameglio

14.00 *Biomarcatori genomici nel doping genetico*
E. Giardina

14.15 *Effetti benefici dell' "High intensity interval training"*
M. Bernardi

14.30 *I materiali a supporto dei progetti di prevenzione del doping*
P. Zuccaro

14.45 *Siamo sportivi, giochiamo pulito*
D. Rossi

Effetti Benefici dell'Allenamento
Intervallato di Alta Intensità

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Upper Body High Intensity Short Term Interval Training: Effects on both Aerobic and Anaerobic Fitness And Body Composition

Marco Bernardi^{1,2}, Luigi Fattorini¹, Silvia Carucci^{1,2}, Maria Rosaria Squeo^{1,2}, Riccardo Lanzano^{1,2}, Paolo Emilio Adami^{1,2}, Alessandro D'Alessio¹, Yagesh Bhambhani³, Andrea Lenzi⁴, Davide Francomano⁴, Silvia Migliaccio^{4,5}, Angelo Rodio⁶, Serena Bianchini⁵, Paolo Sgro^{’5}, Luigi Di Luigi^{2,5}, Vincenzo Castellano⁷, Giuseppe De Vito⁸.

¹Dept. Physiology and Pharmacology, “Sapienza”, University of Rome.

²Italian Federation of Sports Medicine, Rome, Italy.

³University of Alberta, Edmonton, AB, Canada.

⁴ Medical Physiopathologic Dpt, “Sapienza” University of Rome.

⁵ Dpt. Health Sciences, Foro Italico University of Rome.

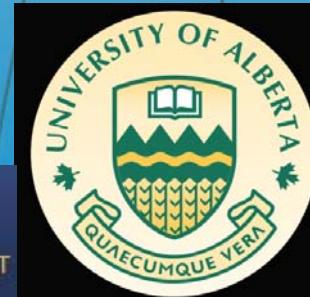
⁶ University of Cassino

⁷ Scientific Dept., Santa Lucia Foundation, Rome, Italy

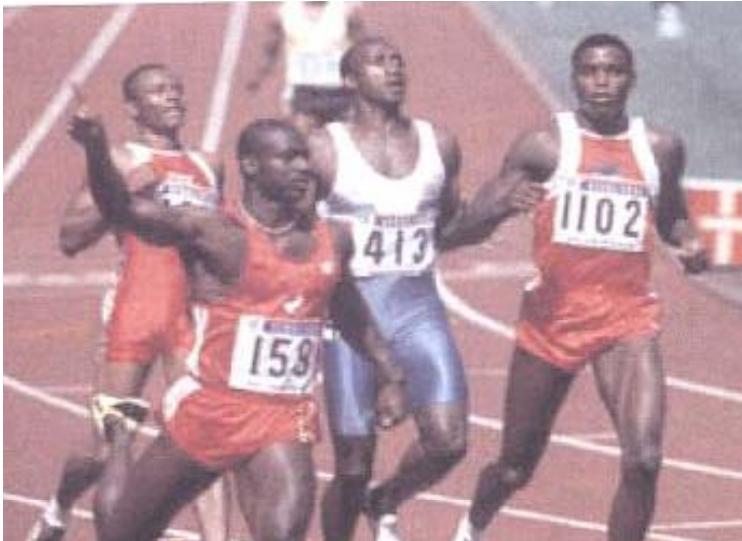
⁸



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Allenamento Intervallato di Alta Intensità per migliorare la prestazione nella Velocità



- Finale dei 100 metri di Seul 1988



- Finale dei 100 metri di Pechino 2008

A. Ross and M. Leveritt Long-Term Metabolic and Skeletal Muscle Adaptations to Short-Sprint Training: Implications for Sprint Training and Tapering. Sports Medicine: 200 - 31-15, 1063-1082

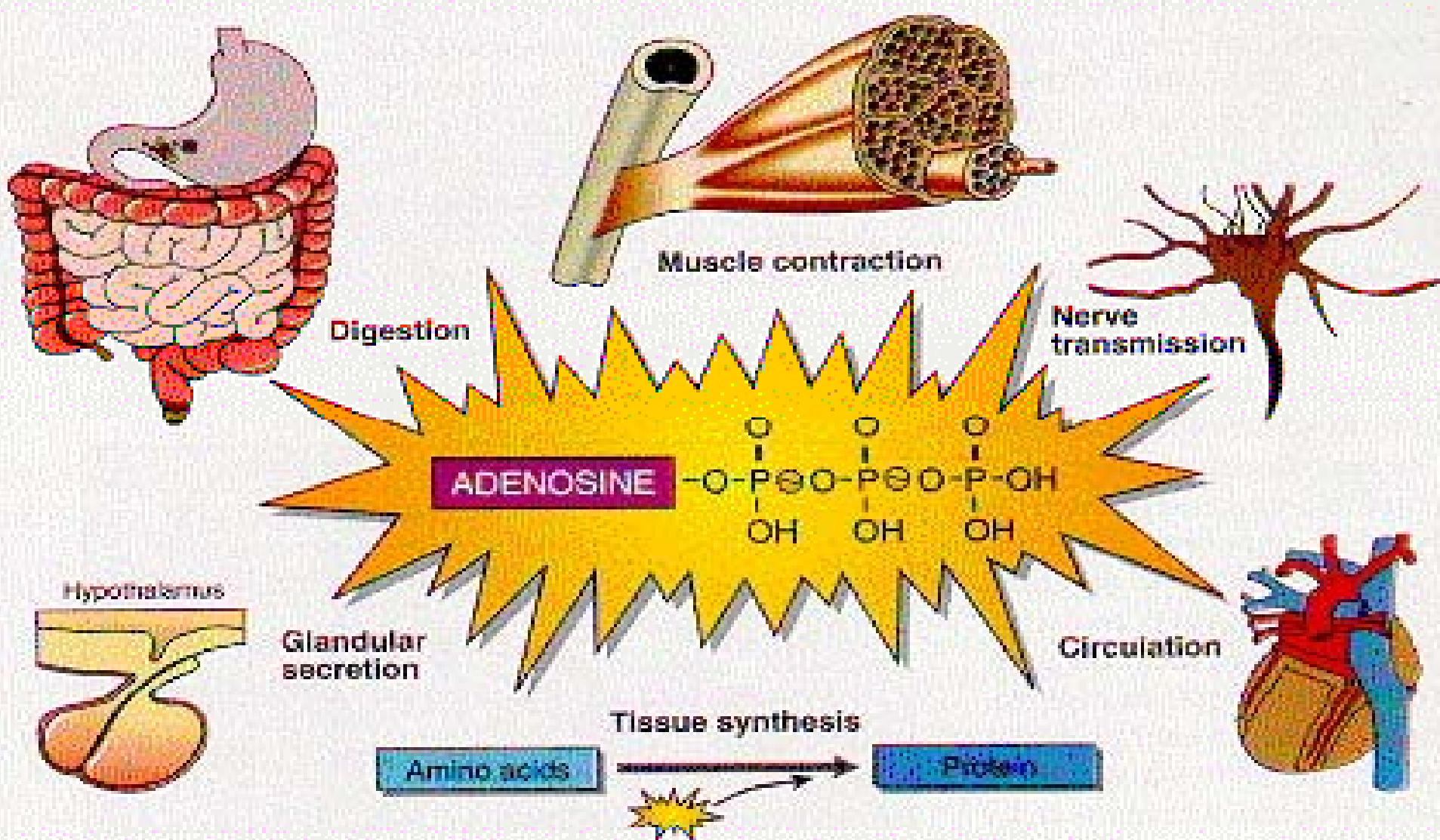
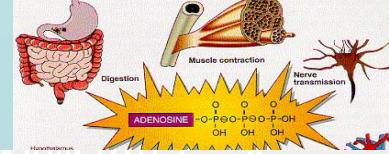
A. Ross and M. Leveritt. Long-Term Metabolic and Skeletal Muscle Adaptations to Short-Sprint Training: Implications for Sprint Training and Tapering. Sports Medicine: 200 - 31-15, 1063-1082

- Morphological and Enzyme adaptations of muscles

- Morphological adaptations to sprint training include changes in muscle fibre type, sarcoplasmic reticulum, and fibre cross-sectional area. An appropriate sprint training programme could be expected to induce a shift toward type IIa muscle, increase muscle cross-sectional area and increase the sarcoplasmic reticulum volume to aid release of Ca²⁺.
- Enzyme adaptations of all three energy systems represent a major metabolic adaptation to sprint training. Myokinase and creatine phosphokinase have shown small increases as a result of short-sprint training. Glycolytic enzyme activity (notably lactate dehydrogenase, phosphofructokinase and glycogen phosphorylase) has been shown to increase after training consisting of either long (>10-second) or short (<10-second) sprints. Mitochondrial enzyme activity also increases after sprint training, particularly when long sprints or short recovery between short sprints are used as the training stimulus.



Visione d'insieme del Metabolismo Energetico



1 Il glucosio deriva dal glicogeno epatico o dalla dieta.

2 Gli acidi grassi possono essere catabolizzati solo tramite il metabolismo aerobico.

3 L'acido lattico prodotto dal metabolismo anaerobico può essere convertito in glucosio dal fegato.

Immediate Energy Sources



Hydrolysis of ATP



Role of Creatine Phosphate (CP)



Coupled Reactions

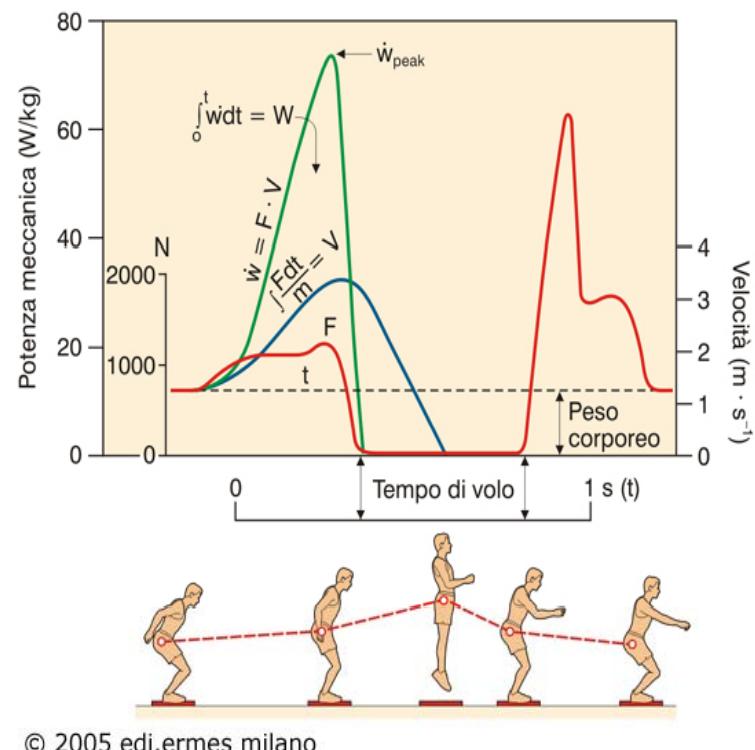


Resynthesis of CP



ATP - Energy Liberation

Hydrolysis of ATP



Human body stores \simeq 20-25 mmol/Kg dry muscle (dm)

Maximal turnover rate \simeq
15mmol/Kg dm/sec

Gaitanos et al, J Appl Physiol 1993; 75(2):712-9

Bogdanis et al, Acta Physiol Scand 1998; 163: 261-72

Parolin et al, Am J Physiol 1999; 277:E890-900

PHOSPHOCREATINE

Resynthesis of ATP from Phosphocreatine



Human body stores \approx 70-80 mmol/Kg dm

Maximal turnover rate \approx 9 mmol/Kg dm/sec

Gaitanos et al, J Appl Physiol 1993; 75(2):712-9

Bogdanis et al, Acta Physiol Scand 1998; 163: 261-72

Bangsbo et al, Am J Physiol 2001; 280 (6): E956-64

Hultman et al, J Physiol 1983;345:525-32

ANAEROBIC GLYCOLYSIS

Resynthesis of ATP from Anaerobic Glycolysis



Human body ATP stores \approx

$\approx 300 \text{ mmol/kg dm}$

Peak rates $\approx 6\text{-}9 \text{ ATP mmol/Kg dm/sec}$
after $\sim 5 \text{ seconds}$

Parolin et al, Am J Physiol 1999; 277:E890-900

Hultman et al, J Physiol 1983;345:525-32

Hultman et al, Human Kinetics 1990:73-92

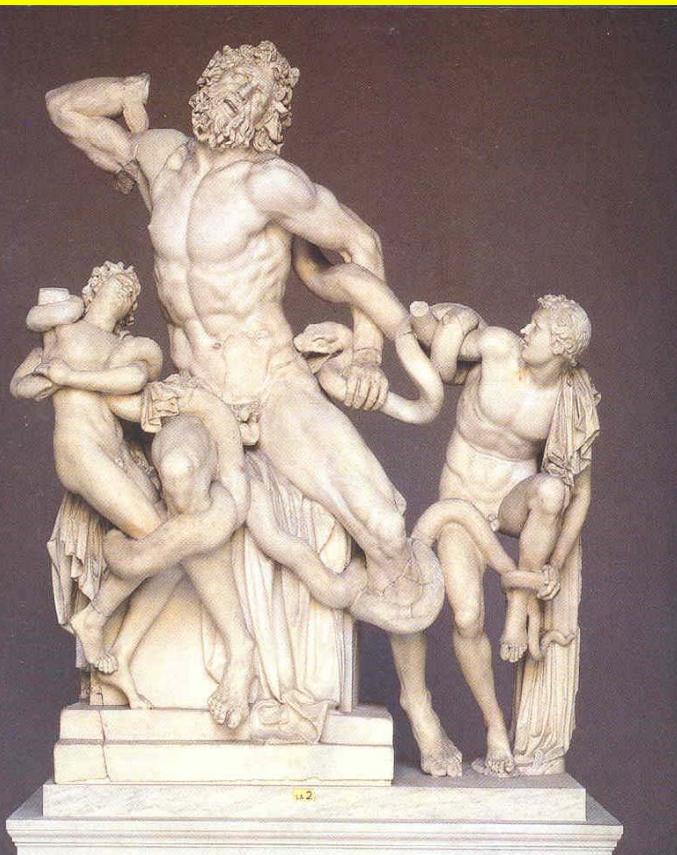
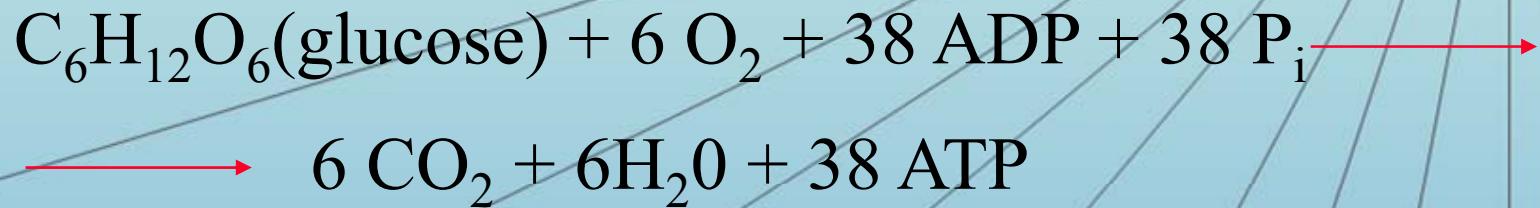
Jones et al, J Appl Physiol 1985; 59 (1):132-6

Gastin et al, Sports Med 2001; 31 (10) :725-41

Greenhaff et al, Human Kinetics 1996; 216-42

Aerobic Metabolism during exhaustive maximal exercise

Aerobic ATP resynthesis



In a 30-second long maximal exercise, mean turnover rates in the first 6 seconds $\simeq 1.32 \text{ mmol ATP/Kg dm/sec}$ equal to $\simeq 9\%$ of total energy supplied

Parolin et al, Am J Physiol 1999;
277:E890-900

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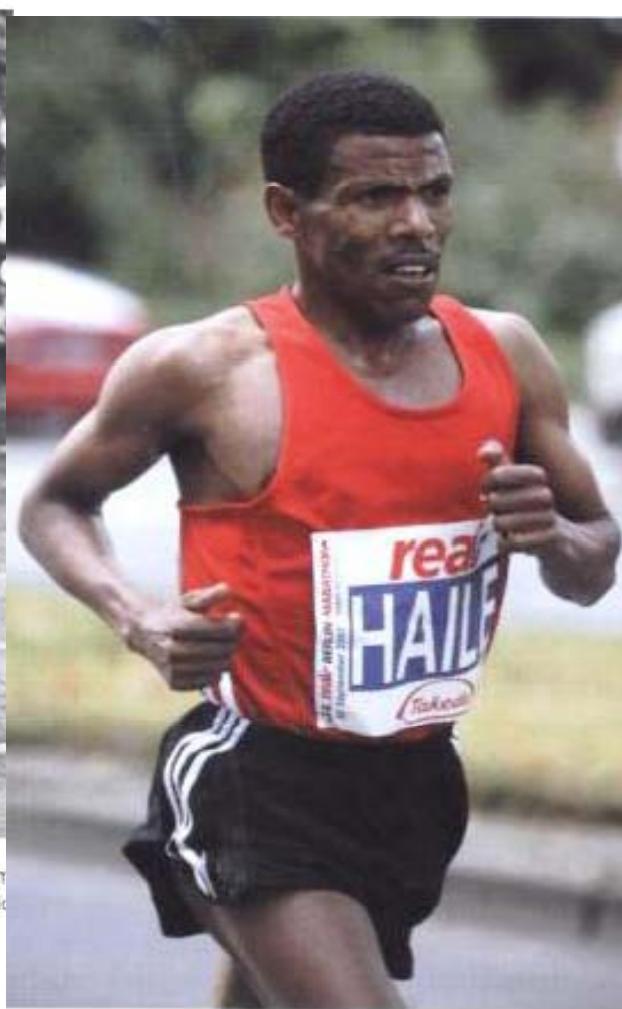
Allenamento Intervallato di Alta Intensità per migliorare la prestazione nel Mezzofondo e nel Fondo



Paavo Nurmi domina negli anni '20 mezzofondo e fondo e comprende l'importanza dell'alta intensità (elevata velocità) per migliorare la prestazione. Negli anni '50 e '60 nasce, grazie a A. Lydiard che inventa l'allenamento frazionato, la grande scuola Neozelandese ed Australiana.



Emil Zatopek in un momento della gara dei 10.000 m Giochi Olimpici di Londra nel 1948 (© International Olympic Committee/Olympic Museum Collection).

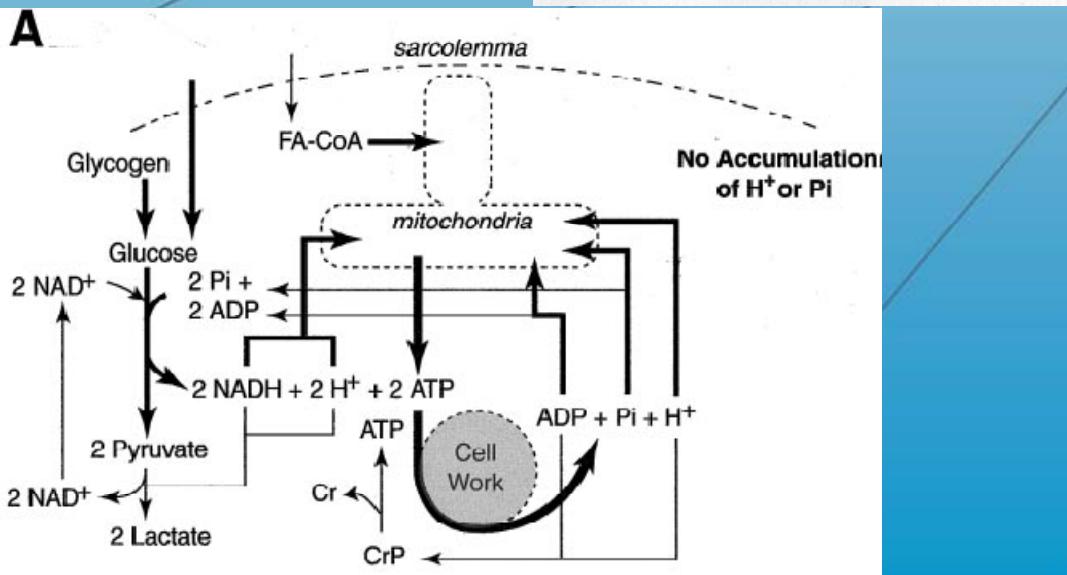
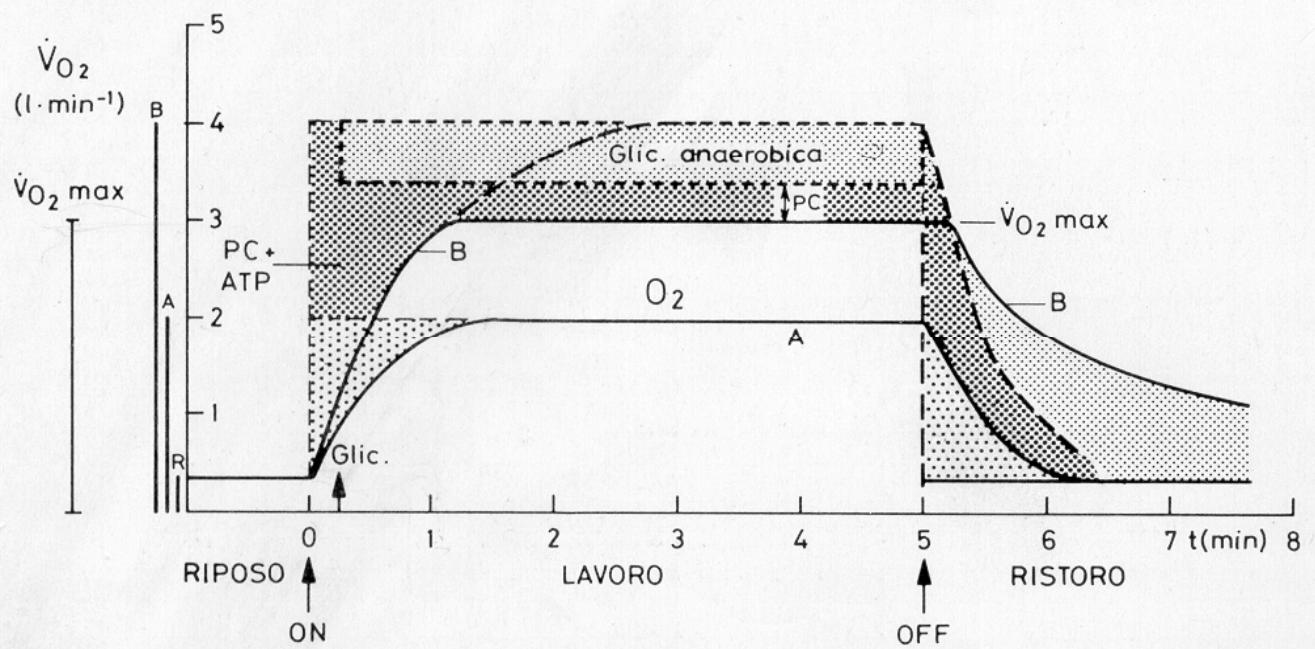


Haile Gebreselassie, vincitore dei 10.000 m

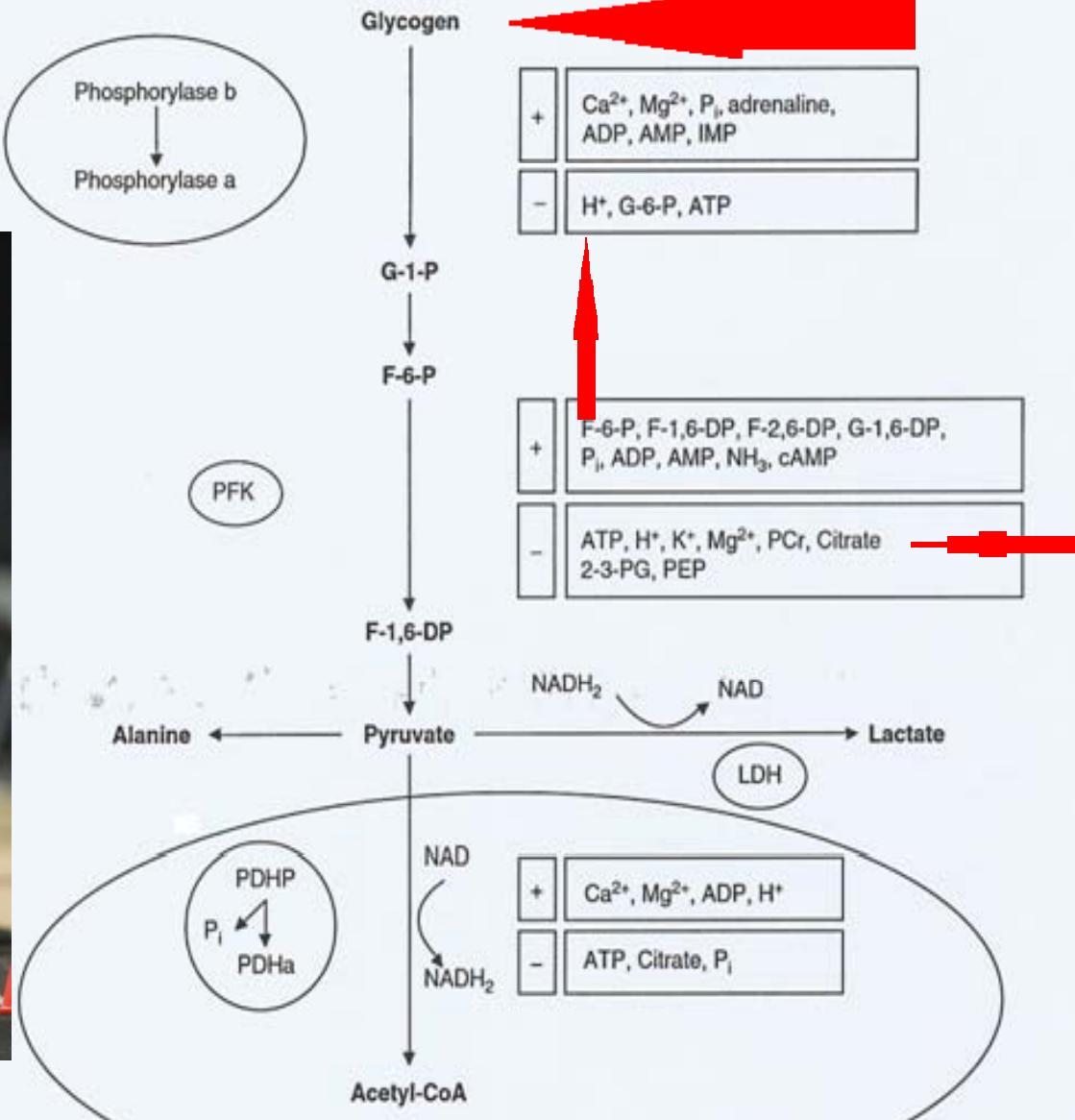
Saltin B, Blomqvist G, Mitchell JH, Johnson RL Jr, Wildenthal K, Chapman CB. **Response to exercise after bed rest and after training.** *Circulation*. 1968 Nov;38(5 Suppl):VII1-78.

Metabolismo utilizzato dipende dall'intensità e dalla durata dell'esercizio

Da Cerretelli P.:
Manuale di
fisiologia dello
sport e del lavoro
muscolare, 1985-
Società Editrice
Universo

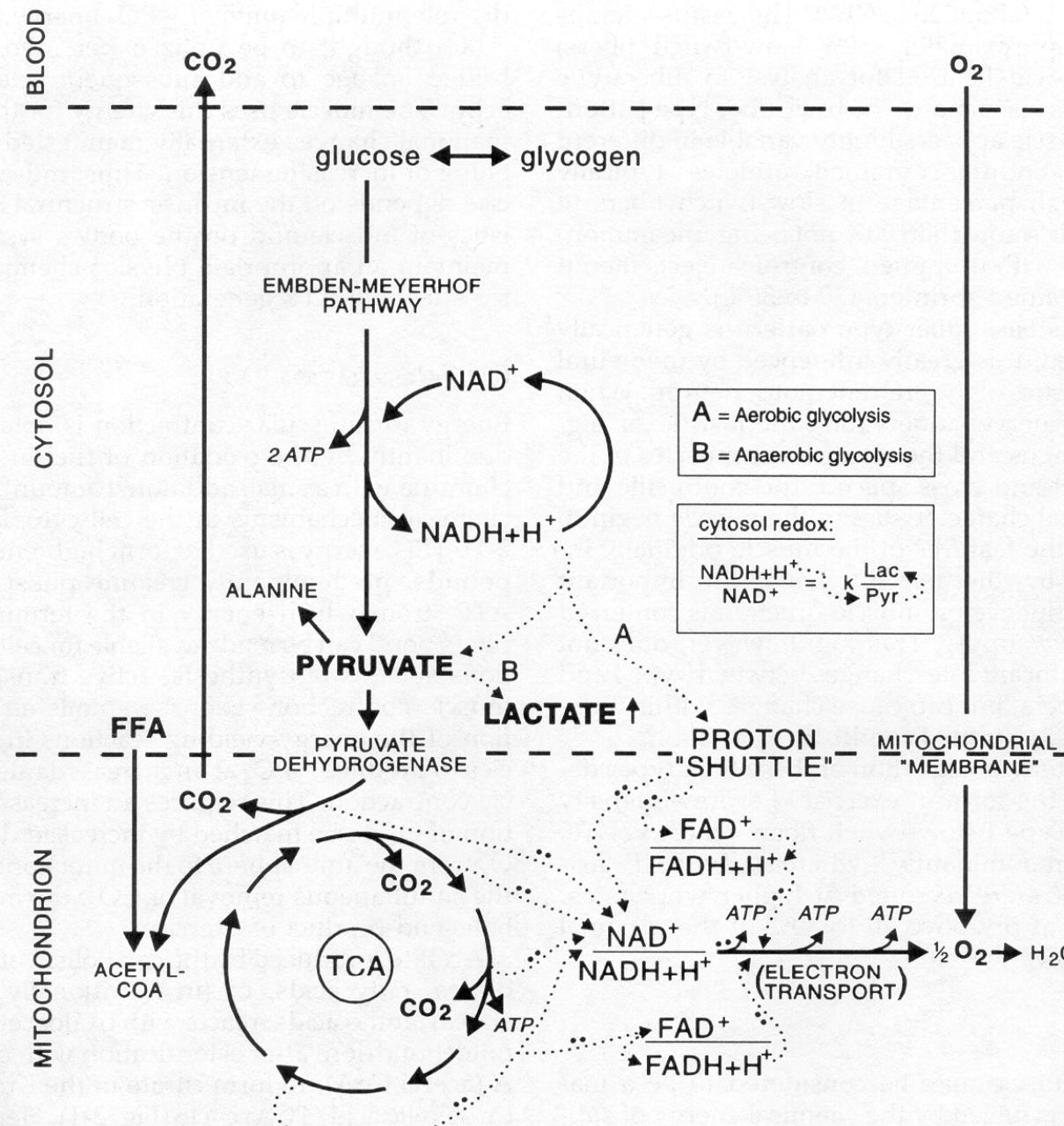


Glycolysis



Schematic representation of the anaerobic metabolic pathways of glycogenolysis/glycolysis and a number of potential regulators (Bangsbo Et al, Human Kinetics 1996; 261-75). 2-3-PG = 2-3-phosphoglycerate; ADP = adenosine diphosphate; AMP = adenosine monophosphate; ATP = adenosine triphosphate; cAMP = cyclic adenosine monophosphate; CoA = coenzyme A; F-1,6-DP = fructose-1,6-diphosphate; F-2,6-DP = fructose-2,6-diphosphate; F-6-P = fructose-6-phosphate; G-1,6-DP = glucose-1,6-diphosphate; G-1-P = glucose-1-phosphate; G-6-P = glucose-6-phosphate; IMP = inosine monophosphate; LDH = lactate dehydrogenase; NAD = nicotinamide-adenine dinucleotide; NADH₂ = the reduced form of NAD; PCr = phosphocreatine; PDHa = active form of pyruvate dehydrogenase; PDHP = pyruvate dehydrogenase phosphatase; PEP = phosphoenolpyruvate; PFK = phosphofructokinase; P_i = inorganic phosphate; + indicates positive regulators; - indicates negative regulators.

Vie metaboliche per la produzione di ATP durante esercizio fisico mediante metabolismo Aerobico ed Anaerobico Lattacido



Wasserman,
Hansen,
Sue,
Whipp,
Casaburi:
Principles
of exercise
testing and
interpretatio
n, 1994 -
Lea &
Febiger

Il complesso integrato “CUORE - POLMONE – MUSCOLO”

Massimo Consumo di Ossigeno

$$\dot{V}O_2 = \dot{Q} \times (C_a - C_v)O_2$$



La massima quantità di ossigeno che l'organismo è in grado di consumare esprime la massima potenza aerobica cioè la massima resistenza del sistema cardiovascolare

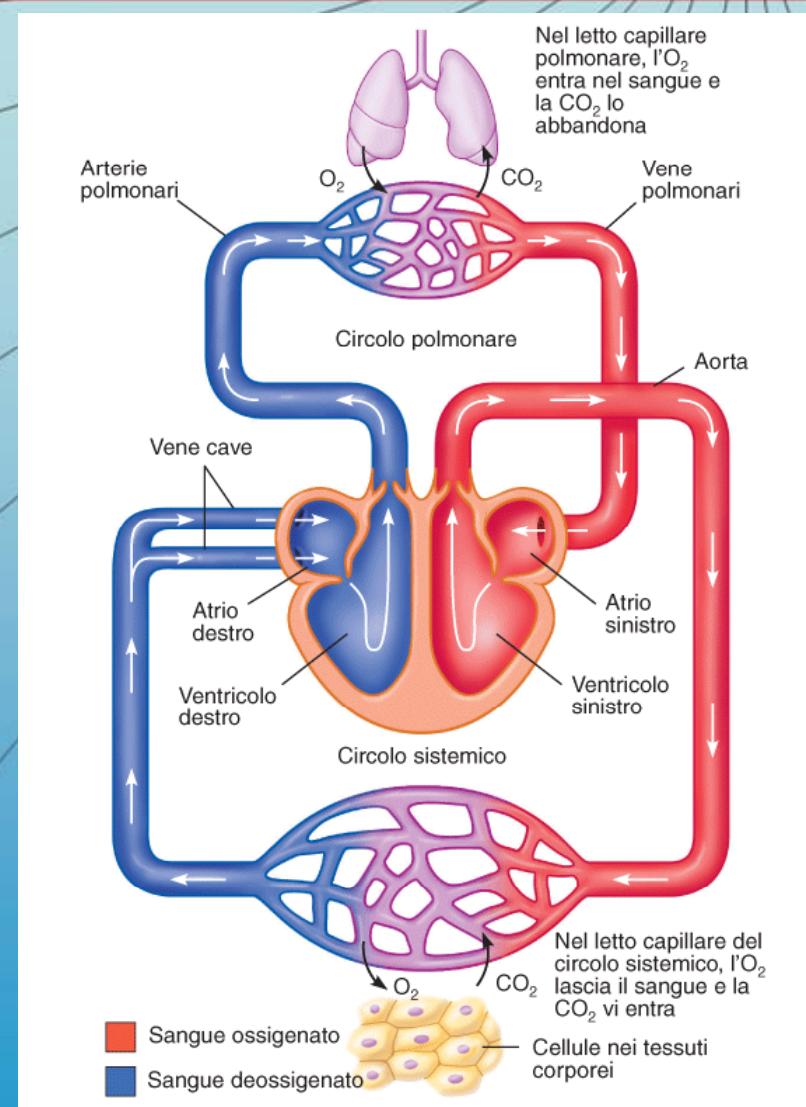
VO_2 = consumo di ossigeno (l/min)

\dot{Q} = gettata cardiaca (l/min)

CaO_2 = concentrazione arteriosa di O_2
($mlO_2/100 ml$ sangue arterioso)

CvO_2 = concentrazione di ossigeno

Nel sangue venoso misto ($mlO_2/100 ml$ sangue venoso)



The New England Journal of Medicine

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VOLUME 346

MARCH 14, 2002

NUMBER 11



EXERCISE CAPACITY AND MORTALITY AMONG MEN REFERRED FOR EXERCISE TESTING

JONATHAN MYERS, PH.D., MANISH PRAKASH, M.D., VICTOR FROELICHER, M.D., DAT DO, M.D., SARA PARTINGTON, B.Sc., AND J. EDWIN ATWOOD, M.D.

ABSTRACT

Background Exercise capacity is known to be an important prognostic factor in patients with cardiovascular disease, but it is uncertain whether it predicts mortality equally well among healthy persons. There is also uncertainty regarding the predictive power of exercise capacity relative to other clinical and exercise-test variables.

Methods We studied a total of 6213 consecutive men referred for treadmill exercise testing for clinical reasons during a mean (\pm SD) of 6.2 ± 3.7 years of follow-up. Subjects were classified into two groups: 3679 had an abnormal exercise-test result or a history of cardiovascular disease, or both, and 2534 had a normal exercise-test result and no history of cardiovascular disease. Overall mortality was the end point.

Results There were a total of 1256 deaths during the follow-up period, resulting in an average annual mortality of 2.6 percent. Men who died were older than those who survived and had a lower maximal heart rate, lower maximal systolic and diastolic blood pressure, and lower exercise capacity. After adjustment for age, the peak exercise capacity measured in metabolic equivalents (MET) was the strongest predictor of the risk of death among both normal subjects and those with cardiovascular disease. Absolute peak exercise capacity was a stronger predictor of the risk of death than the percentage of the age-predicted value achieved, and there was no interaction between the use or non-use of beta-blockade and the predictive power of exercise capacity. Each 1-MET increase in exercise capacity conferred a 12 percent improvement in survival.

Conclusions Exercise capacity is a more powerful predictor of mortality among men than other established risk factors for cardiovascular disease. (N Engl J Med 2002;346:1117-25)

La Fitness aerobica protegge dal rischio cardiovascolare



Fattori di Rischio Malattia Aterosclerotica

OBESITA'

IPERTENSIONE ARTERIOSA

IPERCOLESTEROLEMIA

IPERGLICEMIA

ELEVATO RAPPORTO LDL/HDL

FUMO

La SEDENTARIETÀ costituisce il principale fattore di rischio per lo sviluppo di malattie cardiovascolari e si consiglia a tutta la popolazione di praticare attività fisica e, nel caso questo già avvenisse, è consigliato di praticarla più spesso e ad una maggiore intensità.

NIH Consens Statement. Physical activity and cardiovascular health. 1995

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Physiological adaptations to low-volume, high-intensity interval training in health and disease

J Physiol 590.5 (2012) pp 1077–1084

Martin J. Gibala¹, Jonathan P. Little², Maureen J. MacDonald¹ and John A. Hawley³

¹Department of Kinesiology, McMaster University, Hamilton, Ontario L8S 4K1, Canada

²School of Arts and Sciences, University of British Columbia Okanagan, Kelowna, British Columbia V1V 1V7, Canada

³Exercise Metabolism Group, RMIT University, Bundoora, Victoria 3083, Australia



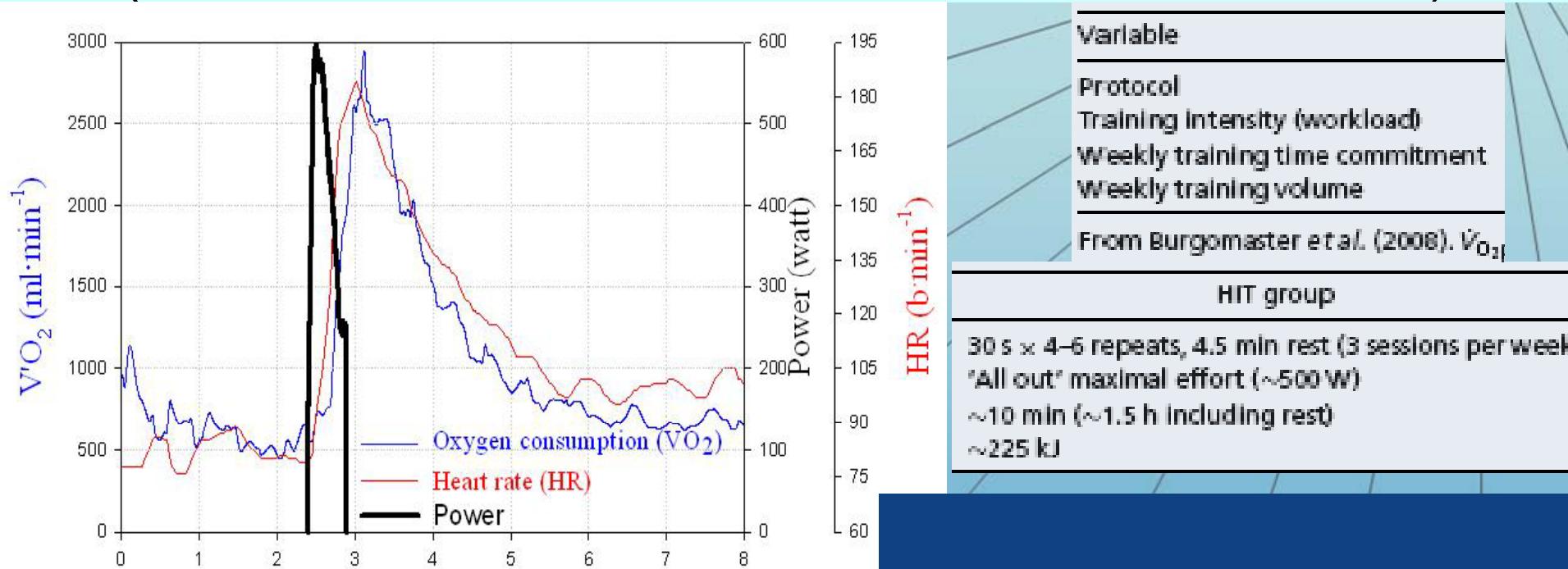
Abstract Exercise training is a clinically proven, cost-effective, primary intervention that delays and in many cases prevents the health burdens associated with many chronic diseases. However, the precise type and dose of exercise needed to accrue health benefits is a contentious issue with no clear consensus recommendations for the prevention of inactivity-related disorders and chronic diseases. A growing body of evidence demonstrates that high-intensity interval training (HIT) can serve as an effective alternate to traditional endurance-based training, inducing similar or even superior physiological adaptations in healthy individuals and diseased populations, at least when compared on a matched-work basis. While less well studied, low-volume HIT can also stimulate physiological remodelling comparable to moderate-intensity continuous training despite a substantially lower time commitment and reduced total exercise volume. Such findings

Table 1. Summary of protocols in studies from our laboratory that directly compared 6 weeks of either high-intensity interval training (HIT) or traditional endurance training

Variable	HIT group	Endurance group
Protocol	30 s × 4–6 repeats, 4.5 min rest (3 sessions per week)	40–60 min cycling (5 sessions per week)
Training Intensity (workload)	'All out' maximal effort (~500 W)	65% of $\dot{V}_{O_2\text{peak}}$ (~150 W)
Weekly training time commitment	~10 min (~1.5 h including rest)	~4.5 h
Weekly training volume	~225 kJ	~2250 kJ

From Burgomaster et al. (2008). $\dot{V}_{O_2\text{peak}}$, peak oxygen uptake.

Risposta Cardiaca e Metabolica al Wingate Test (test fuori tutto della durata di 30 secondi)



Metabolic Adaptations to Short-term High-Intensity Interval Training: A Little Pain for a Lot of Gain?

Martin J. Gibala,¹ and Sean L. McGee²

¹Exercise Metabolism Research Group, Department of Kinesiology, McMaster University, Hamilton, Ontario, Canada; and ²Department of Physiology, University of Melbourne, Melbourne, Victoria, Australia

GIBALA, M.J., and S.L. MCGEE. Metabolic Adaptations to Short-term High-Intensity Interval Training: A Little Pain for a Lot of Gain? *Exerc. Sport Sci. Rev.*, Vol. 36, No. 2, pp. 58–63, 2008. High-intensity interval training (HIT) is a potent time-efficient strategy to induce numerous metabolic adaptations usually associated with traditional endurance training. As little as six sessions of HIT over 2 wk or a total of only approximately 15 min of very intense exercise (~600 kJ), can increase skeletal muscle oxidative capacity and endurance performance and alter metabolic control during aerobic-based exercise. Key Words: exercise, skeletal muscle, mitochondria, oxidative capacity, substrate metabolism, cell signaling

Physiological adaptations to interval training in health

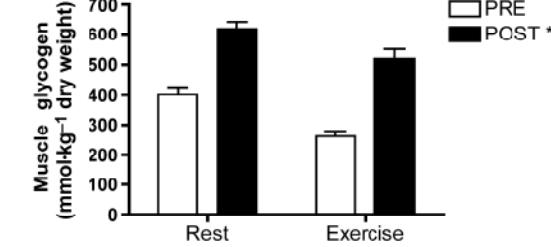
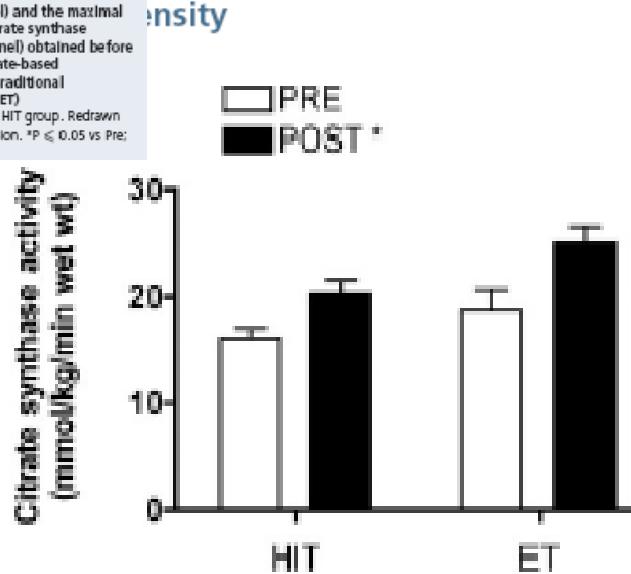
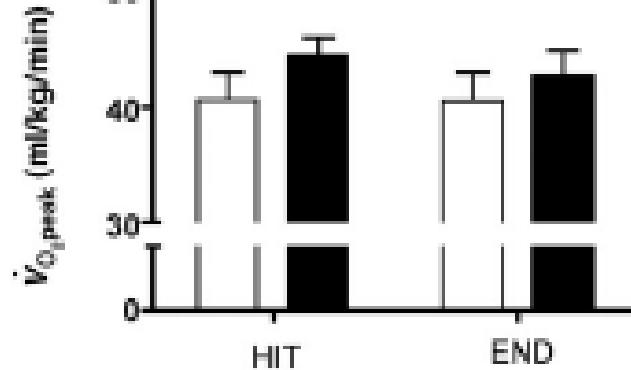


Figure 2. Potential intracellular signalling mechanisms involved in HIT-induced mitochondrial biogenesis
Low-volume HIT has been shown to activate 5'-AMP-activated protein kinase (AMPK) and p38 mitogen-activated protein kinase (MAPK). Both of these exercise-responsive signalling kinases are implicated in direct phosphorylation and activation of PGC-1 α . Increased nuclear abundance of PGC-1 α following HIT is hypothesized to co-activate transcription factors (TF) to increase mitochondrial gene transcription, ultimately resulting in accumulation of more mitochondrial proteins to drive mitochondrial biogenesis.

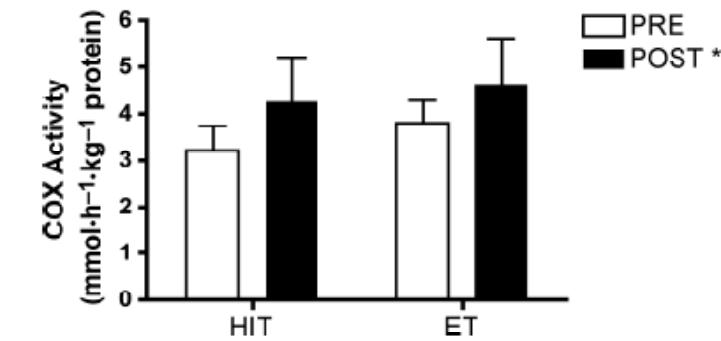
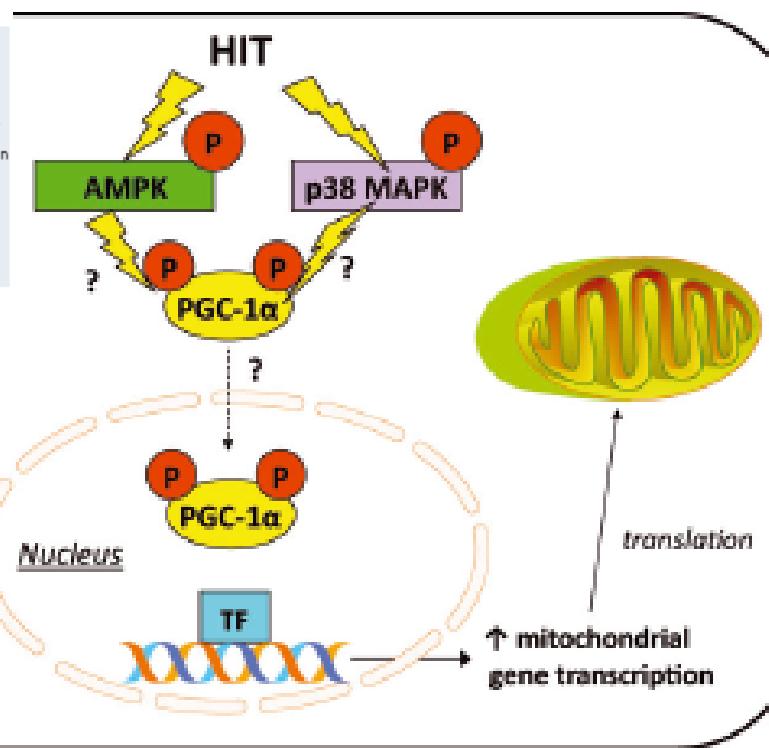


Figure 2. Maximal activity of cytochrome c oxidase (COX) measured in resting human skeletal muscle biopsy samples obtained before (PRE) and after (POST) six sessions of high-intensity interval training (HIT) or continuous moderate-intensity training (ET) over 2 wk. Total training time commitment was approximately 2.5 and 10.5 h for the sprint and endurance groups, respectively, and total exercise volume was approximately 90% lower for the HIT group. Values are means \pm SE for eight subjects in each group. *P < 0.05 versus PRE (main effect for time). [Adapted from Gibala, M.J., J.P. Little, M. van Essen, G.P. Wilkin, K.A. Burgomaster, A. Safdar, S. Raha, and M.A. Tarnopolsky. Short-term sprint interval versus traditional endurance training: similar initial adaptations in human skeletal muscle and exercise performance. *J. Physiol.* 575(Pt 3):901–911, 2006. Copyright © 2006 Blackwell Publishing. Used with permission.]

Scopo dello Studio (Ministero Salute, Conv. 2007-03)



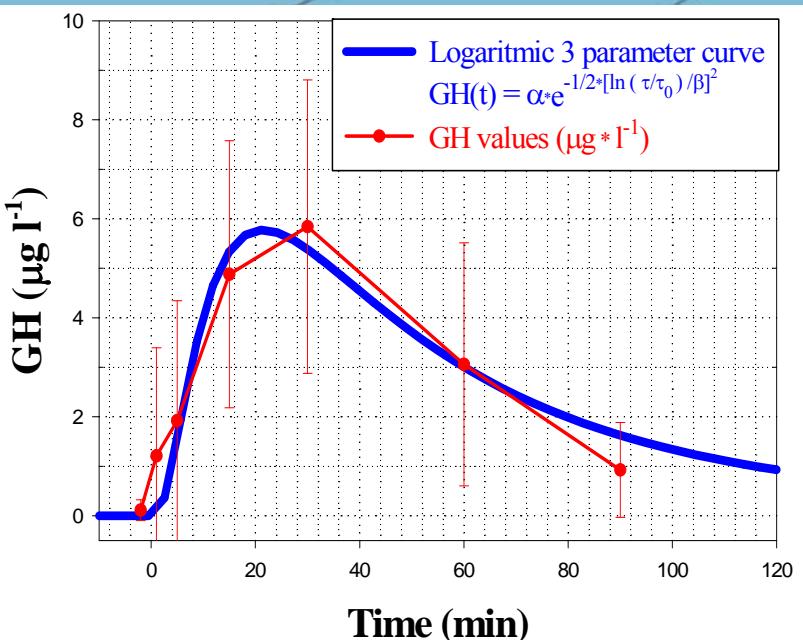
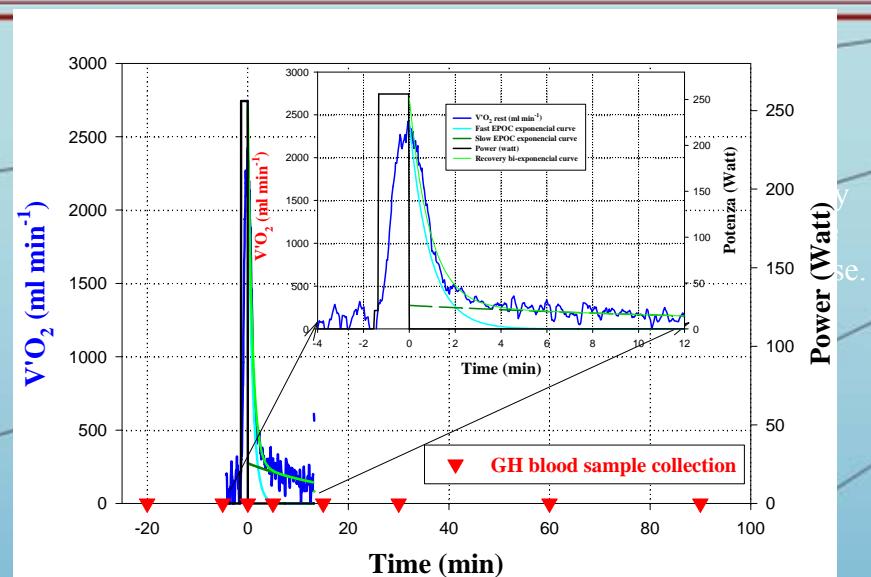
To study the effects of a short term (total time about 20 minutes) high intensity (higher than maximal aerobic power) interval (start every 5 minutes) training (HI-IT) based on 5 repetitions of an “up to exhaustion upper body exercises on Health.



Hypothesis: An Upper limb HI-IT is effective in improving, not only aerobic and anaerobic fitness, but also Body Composition

Growth Hormone Response to High Intensity Exercise (one bout up to exhaustion)

Acknowledgements: Funds from Italian Ministry of Health (Ministero Salute, Convenz. 2004-11)



Kinetics of GH response (time “0” corresponds to the end of exercise). GH mean value (red points) with standard deviation (vertical red line) and best fit logarithmic curve (blue line)

GH basal value $0,29 \pm 0,44 \mu\text{g min l}^{-1}$ - GH peak value $7,09 \pm 3,55 \mu\text{g min l}^{-1}$ - GH increment 113 ± 91 times - Total production $404 \pm 230 \mu\text{g min l}^{-1}$

L'ormone della Crescita stimolato dall'allenamento di Alta intensità incide sulla composizione corporea?



Rapporto fra massa in kg ed altezza
(metri) al quadrato =

BMI (Kg/m²) = Indice di Massa Corporea

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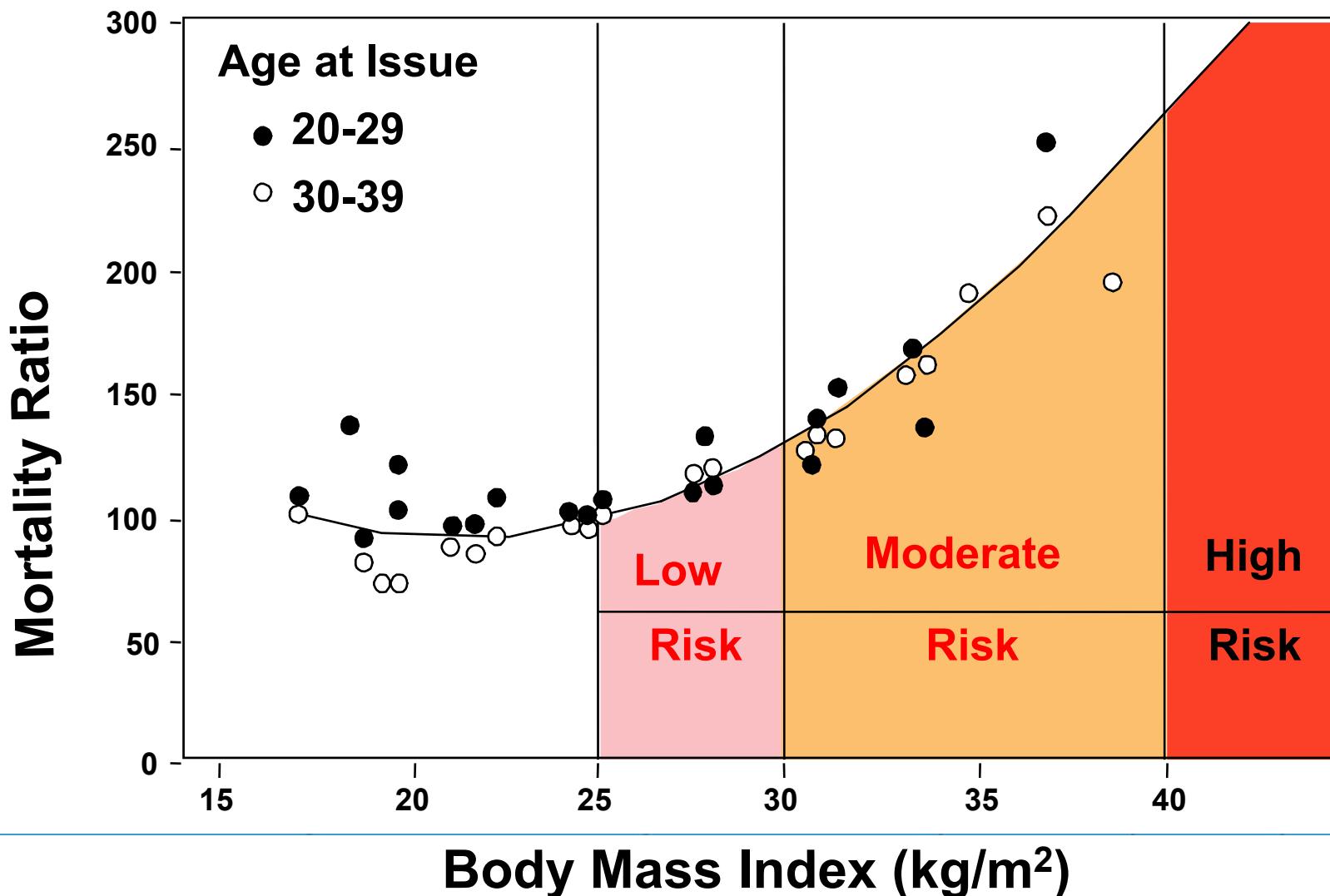


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Rischio cardiovascolare

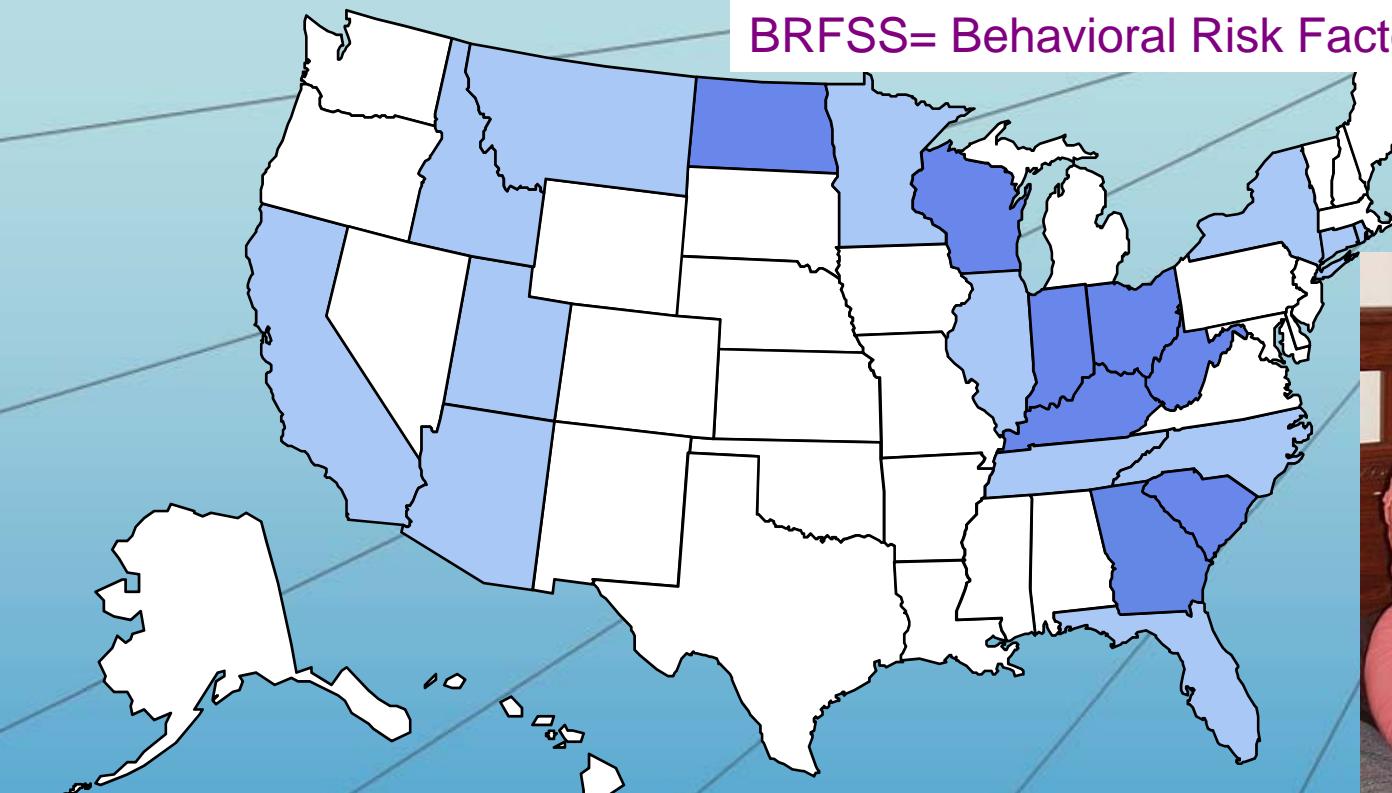
		Rischio di malattia rispetto alla circonferenza vita		
Tipo	BMI (Kg/m ²)	Classe d'obesità	Uomini ≤ 102 cm Donne ≤ 88 cm	≥ 102 cm ≥ 88 cm
Sottopeso	<18,5			
Normale	18,5 – 24,9			
Soprappeso	25 – 29,9		Aumentato	Alto
Obeso	30 – 34,9	I	Elevato	Molto elevato
Obeso grave	35 – 39,9	II	Molto elevato	Molto elevato
Estremamente grave	≥ 40	III	Estremamente elevato	Estremamente elevato

Relation of BMI to mortality



Obesity Trends* Among U.S. Adults BRFSS, 1985

BRFSS= Behavioral Risk Factor Surveillance System



No Data <10% 10%-14%



(* $BMI \geq 30$, or ~ 30 lbs. overweight for 5' 4" person)

BMI (Kg/m^2) = Indice di Massa Corporea

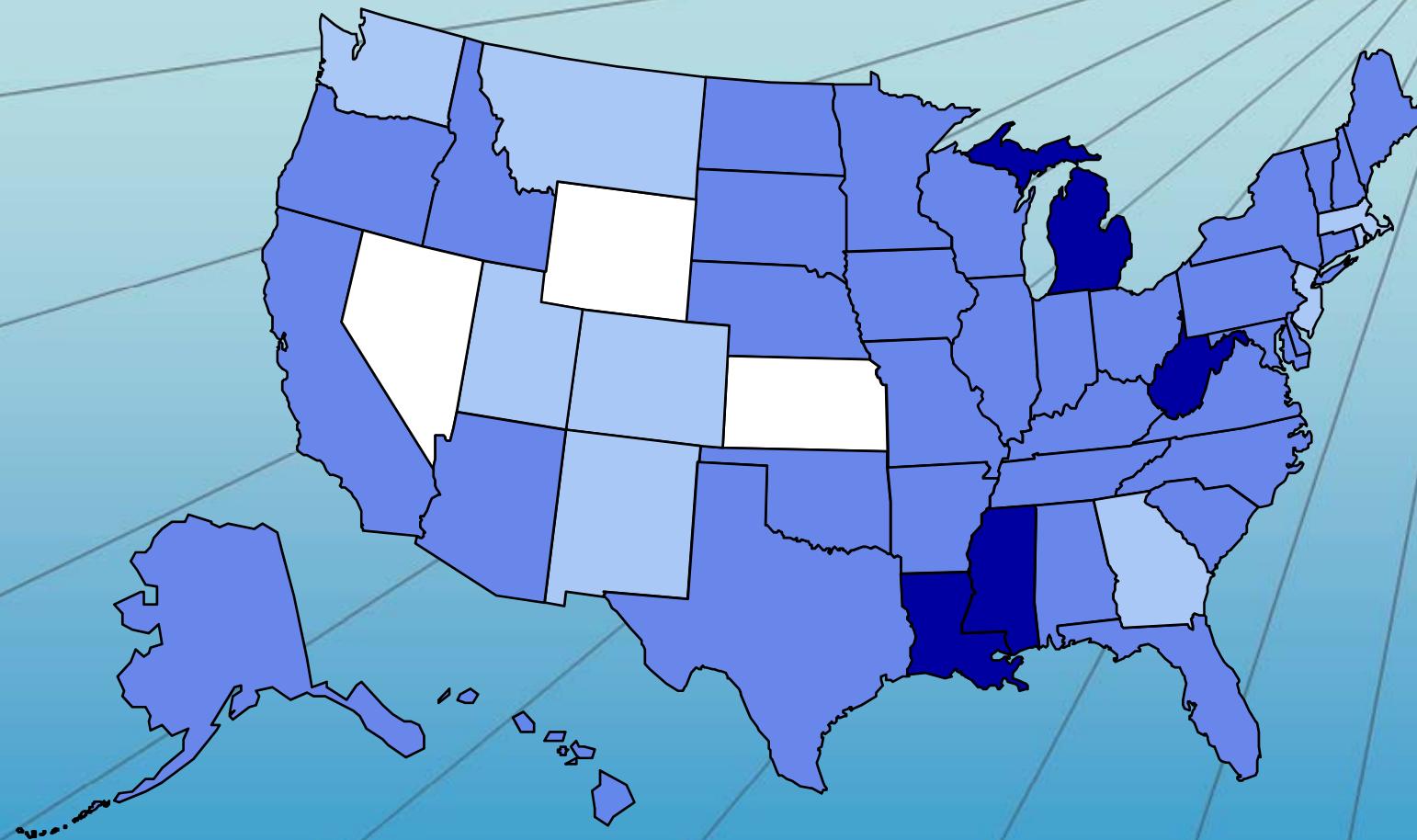
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Obesity Trends* Among U.S. Adults

BRFSS, 1991



[No Data] [Light Blue] [Medium Blue] [Dark Blue]

BMI (Kg/m^2) = Indice di Massa Corporea

(* $\text{BMI} \geq 30$, or ~ 30 lbs. overweight for 5' 4" person)

Marco Bernardi

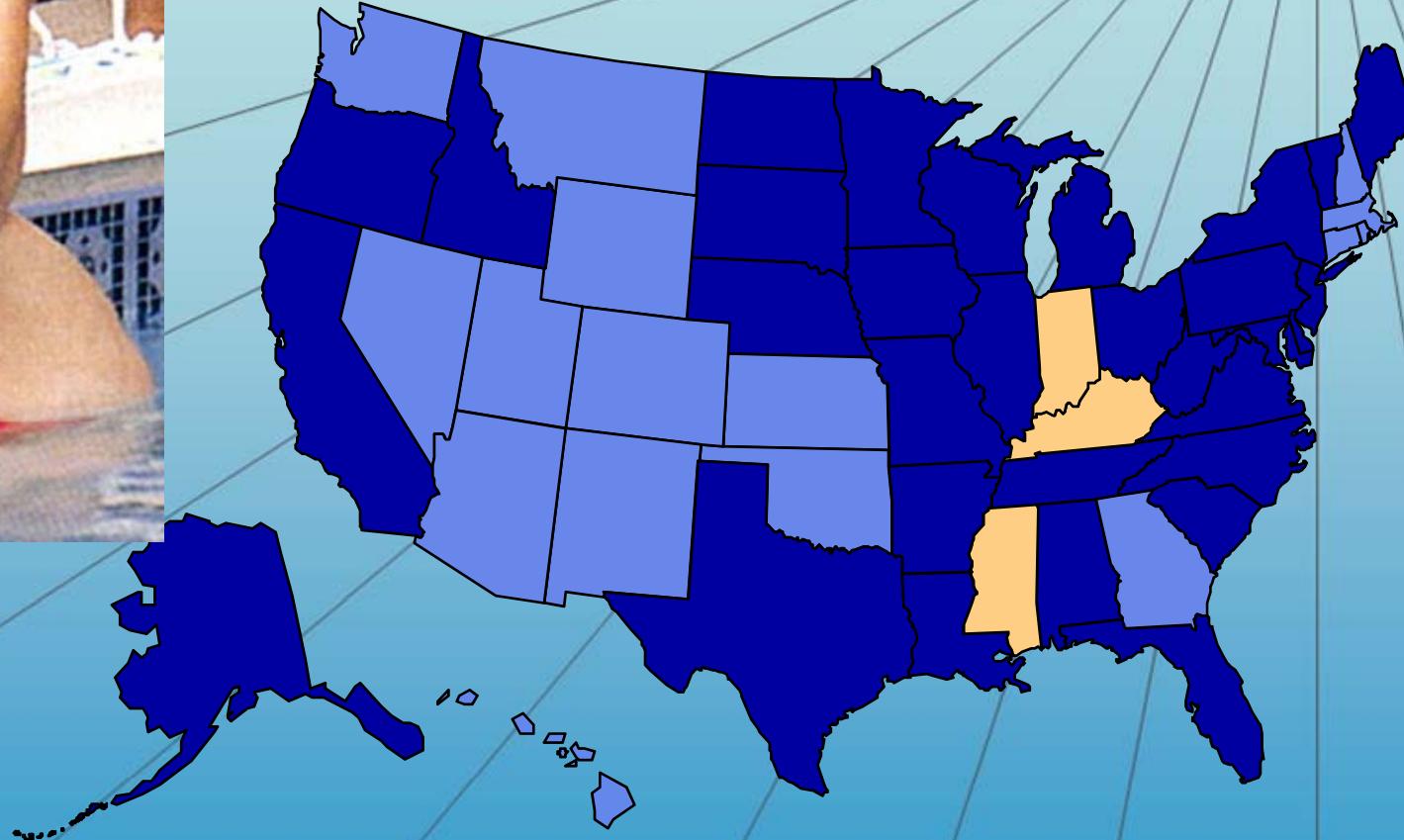


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Obesity Trends* Among U.S. Adults

BRFSS, 1997



BMI (Kg/m^2) = Indice di Massa Corporea



(* $\text{BMI} \geq 30$, or ~ 30 lbs. overweight for 5' 4" person)

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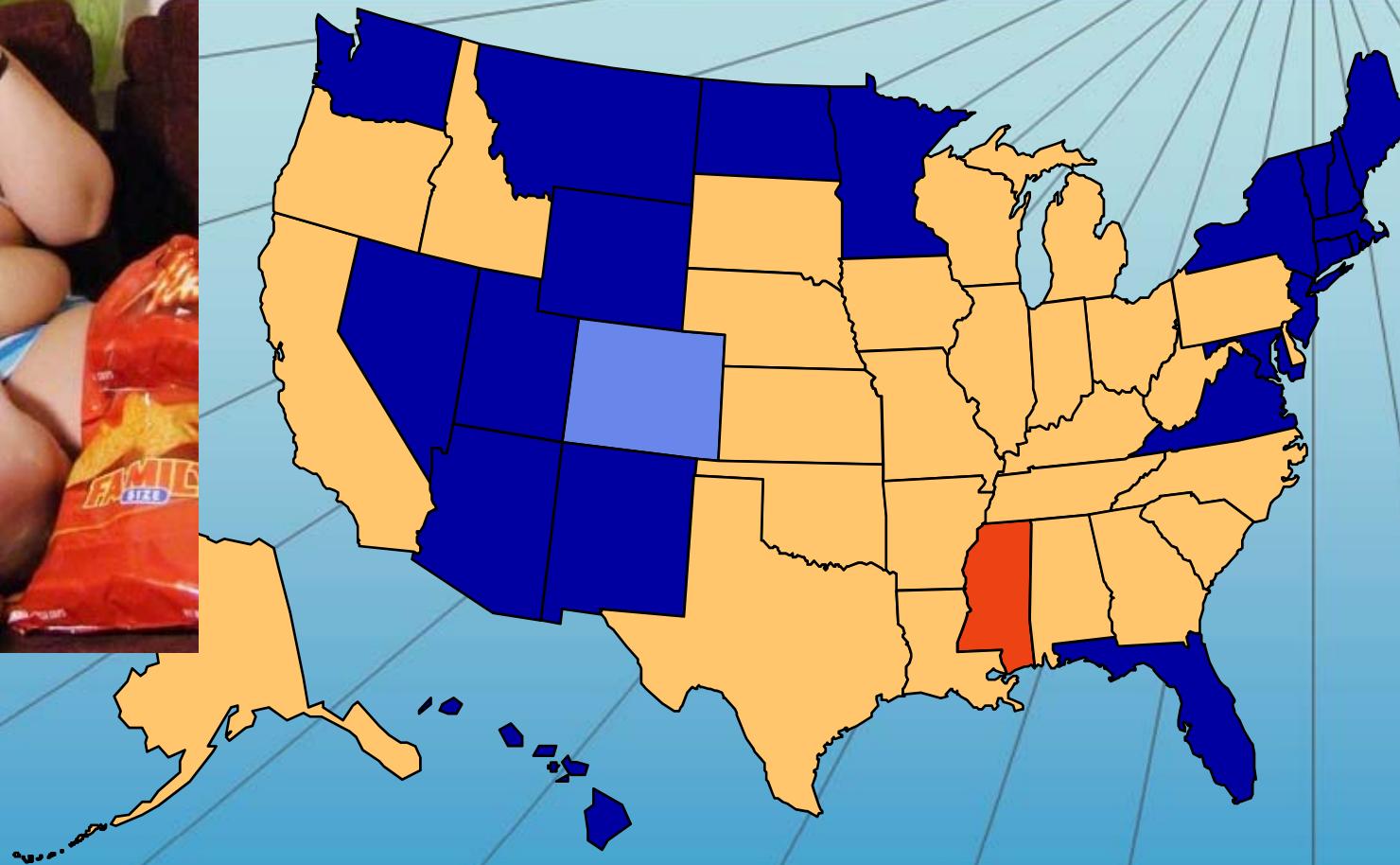


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Obesity Trends* Among U.S. Adults

BRFSS, 2001



BMI (Kg/m^2) = Indice di Massa Corporea



(*BMI ≥ 30 , or ~ 30 lbs. overweight for
5' 4" person)

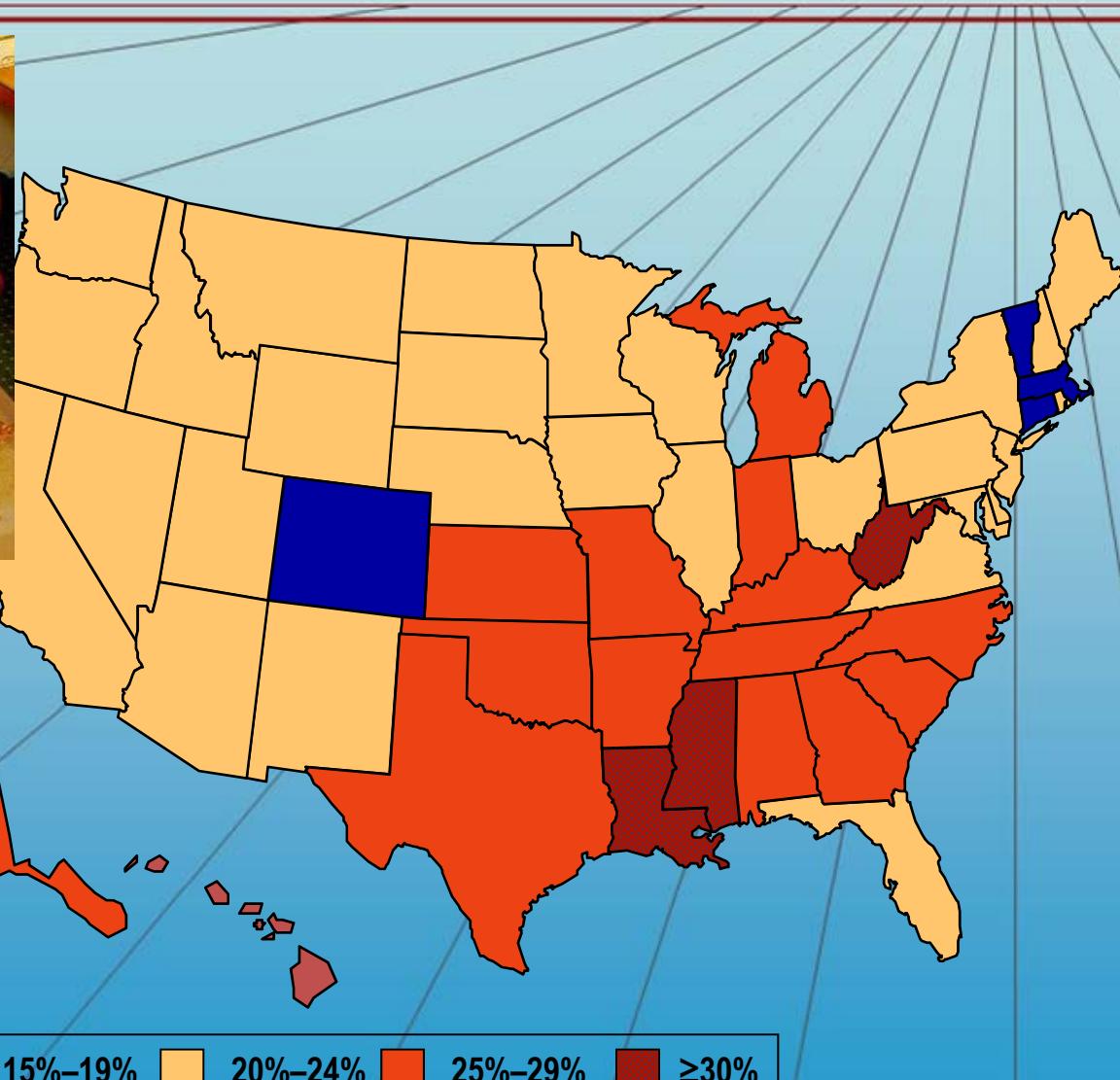
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Obesity Trends* Among U.S. Adults

BRFSS, 2005



BMI (Kg/m^2)
= Indice di
Massa
Corporea

	No Data		<10%		10%-14%		15%-19%		20%-24%		25%-29%		≥30%
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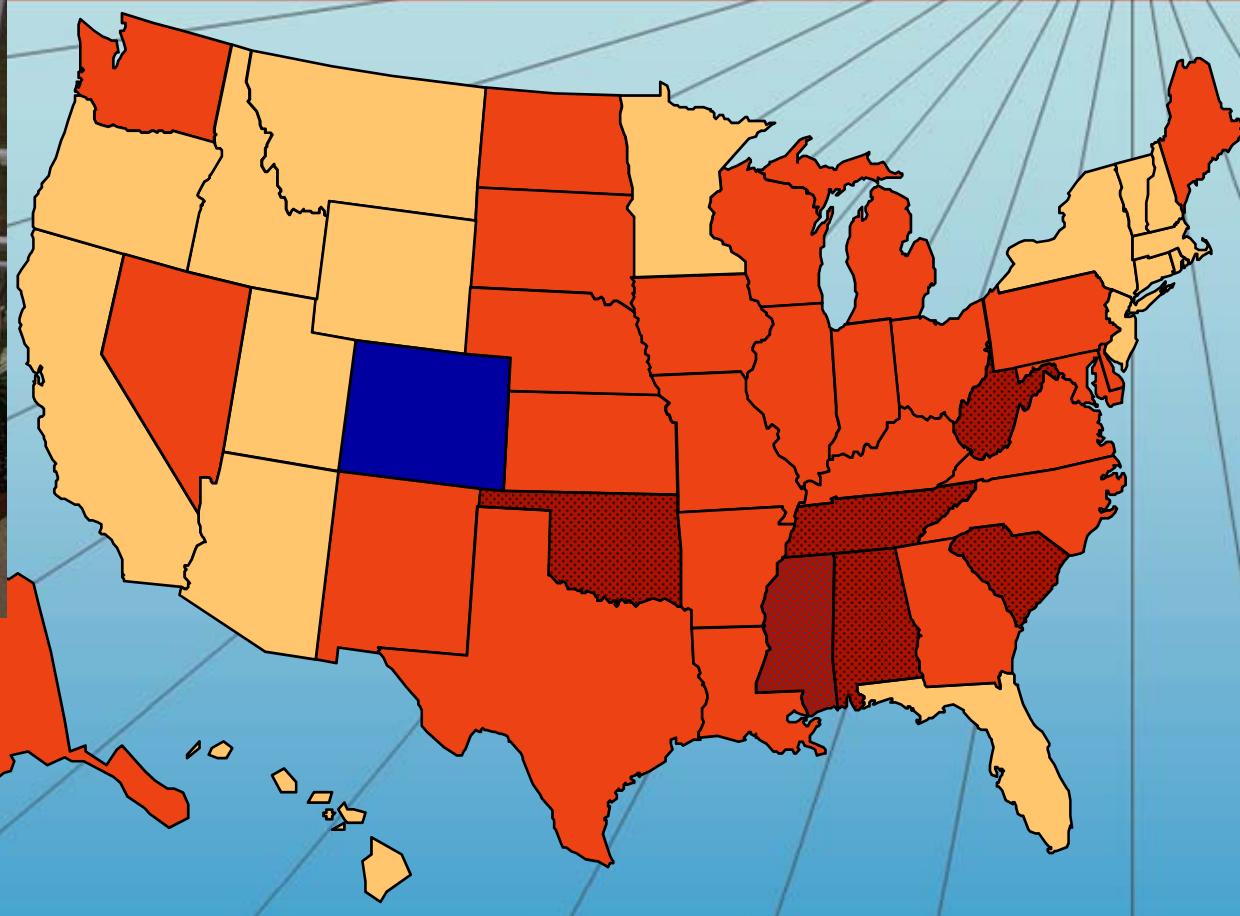
(*BMI ≥ 30 , or ~ 30 lbs. overweight for 5' 4" person)

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Obesity Trends* Among U.S. Adults BRFSS, 2008



(*BMI ≥ 30 , or ~ 30 lbs. overweight for 5' 4" person)

BMI (Kg/m^2) = Indice di Massa Corporea

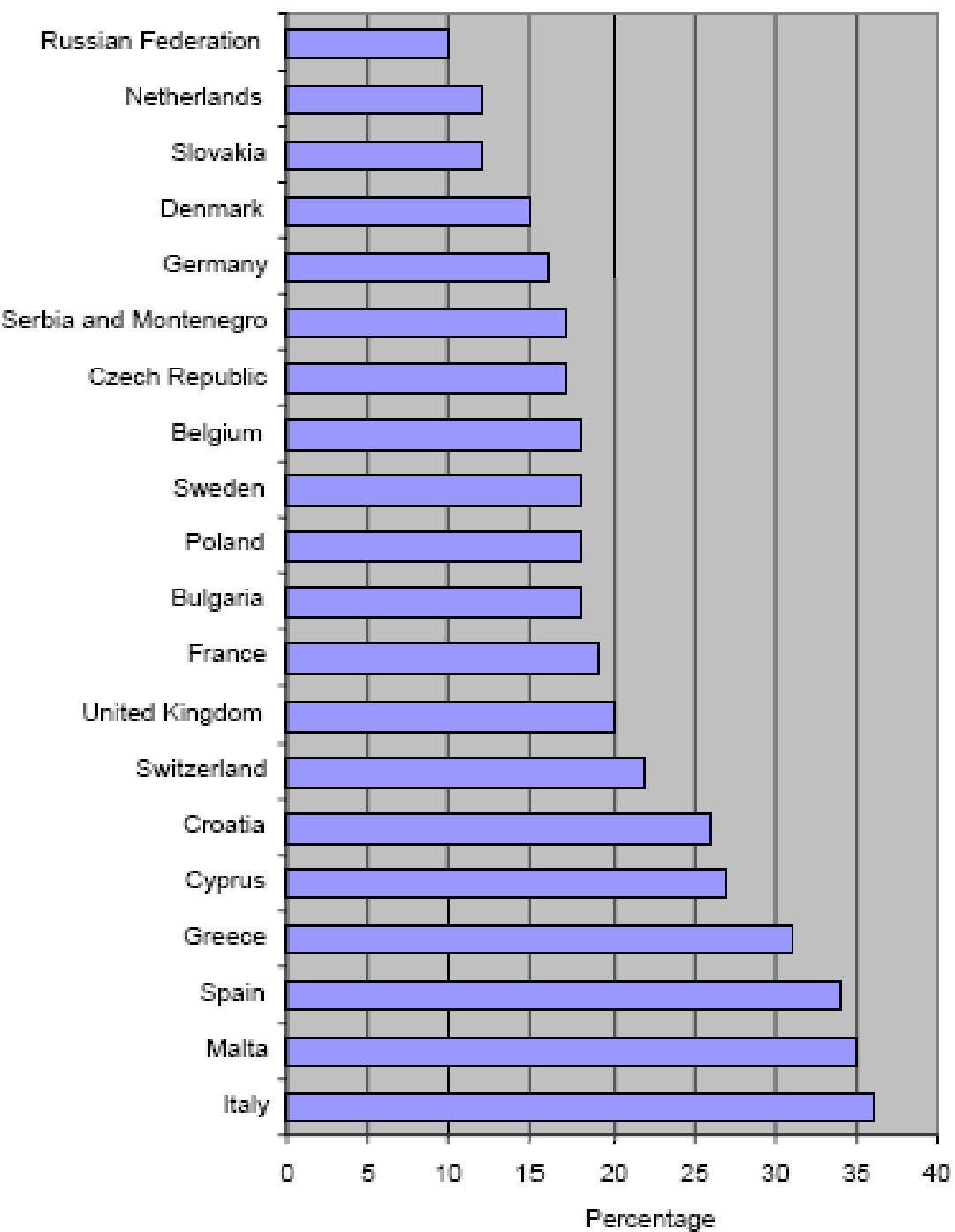
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Prevalenza di bambini (7-11anni) in sovrappeso o obesi in alcune Nazioni Europee



Metodi: Soggetti dello studio

- Sottoposti a “screening” iniziale:
- Hanno completato l’allenamento:

Donne	Uomini	Totale
32	23	55

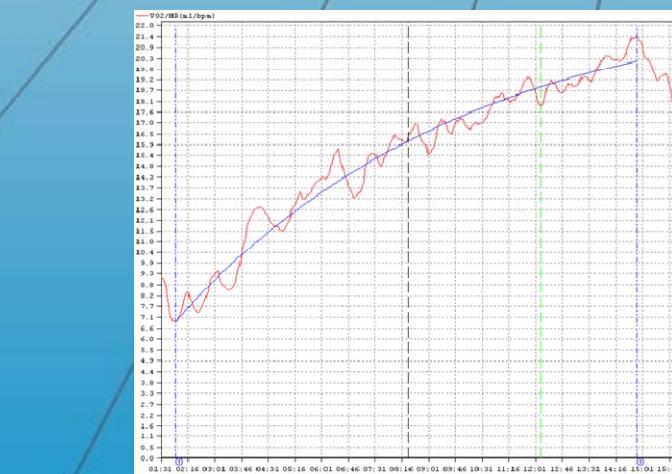
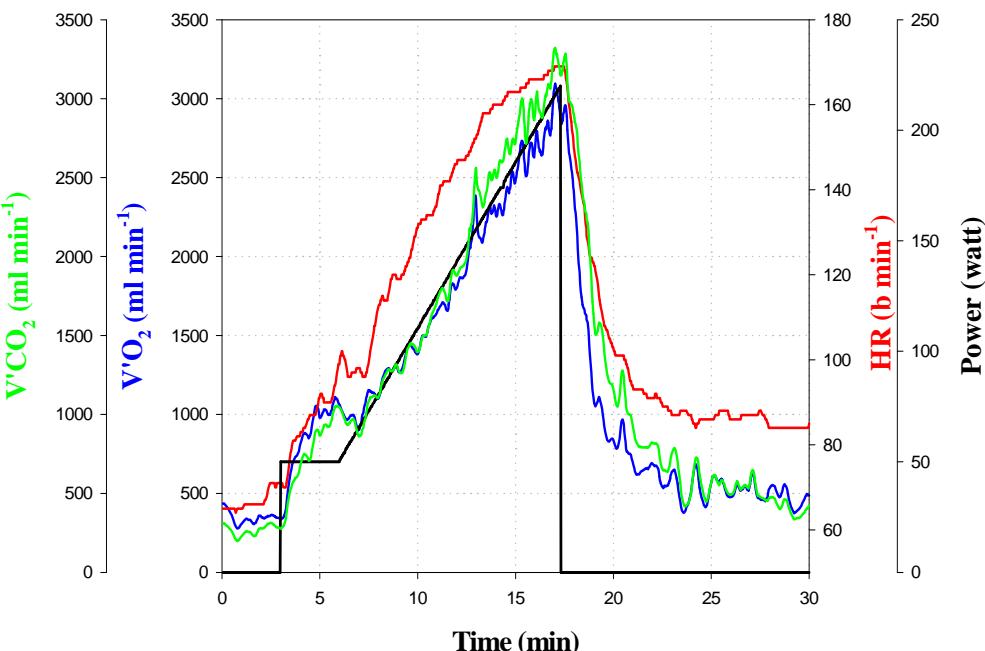
	Donne	Uomini
Numero	6	14
Età	$28,8 \pm 6,37$	$37,4 \pm 10,42$
Massa corporea	$57,9 \pm 1,70$ kg	$77,4 \pm 8,97$ kg
Altezza	$1,66 \pm 0,051$ m	$1,78 \pm 0,058$ m
BMI	$21,3 \pm 1,42$	$24,3 \pm 2,11$



• Tutti i soggetti sono stati sottoposti ad accurata anamnesi, esami di laboratorio di routine, visita medica ed elettrocardiogramma a riposo e sotto sforzo durante test incrementale massimale al cicloergometro, per escludere ogni possibile rischio per la salute derivante dal tipo di allenamento proposto. Nel caso di dubbi, specifici indagini di “secondo livello” quali ecocardiogramma color-doppler ed elettrocardiogramma delle 24 ore secondo Holter.

Materials and Methods: Maximal incremental test for Oxygen Consumption peak ($\dot{V}'\text{O}_{2\text{peak}}$) measurement

The subjects underwent to an arm cranking ergometer -ACE- (ER800, Cosmed, Italy) maximal incremental exercise test under metabolic and electrocardiogram monitoring for cardiovascular health evaluation to assess the aerobic fitness and the power to carry out a following exhaustion test at high intensity (130% of the peak power previously reached).



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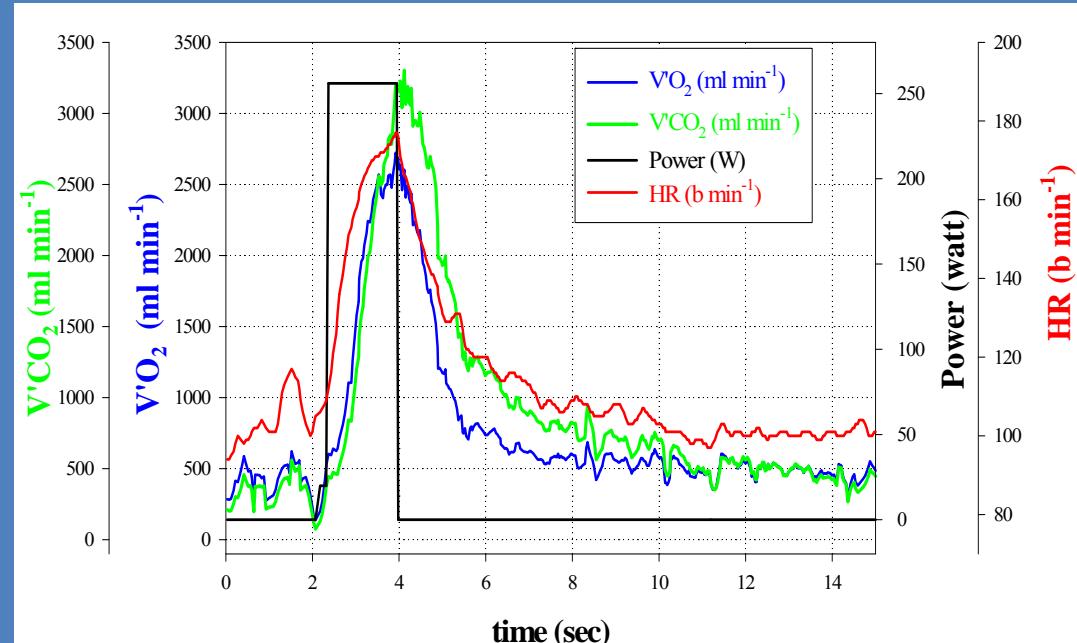
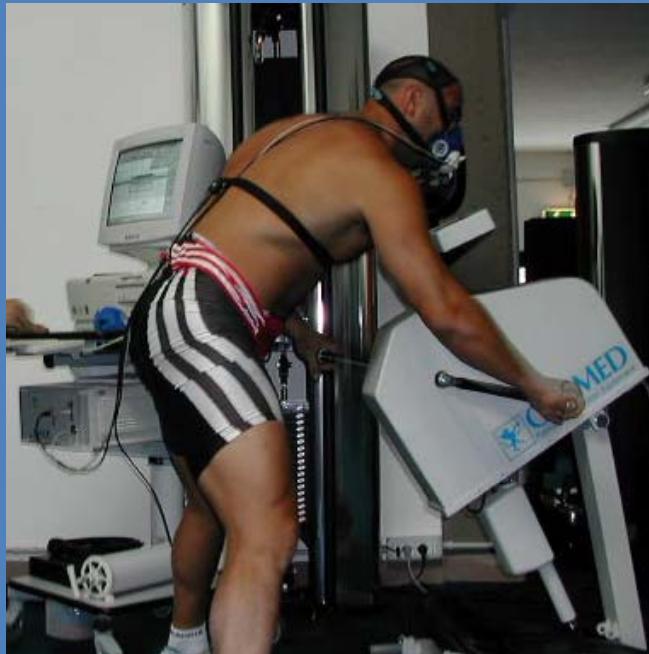
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Materials and Methods:

Anaerobic test up to exhaustion (AnT)

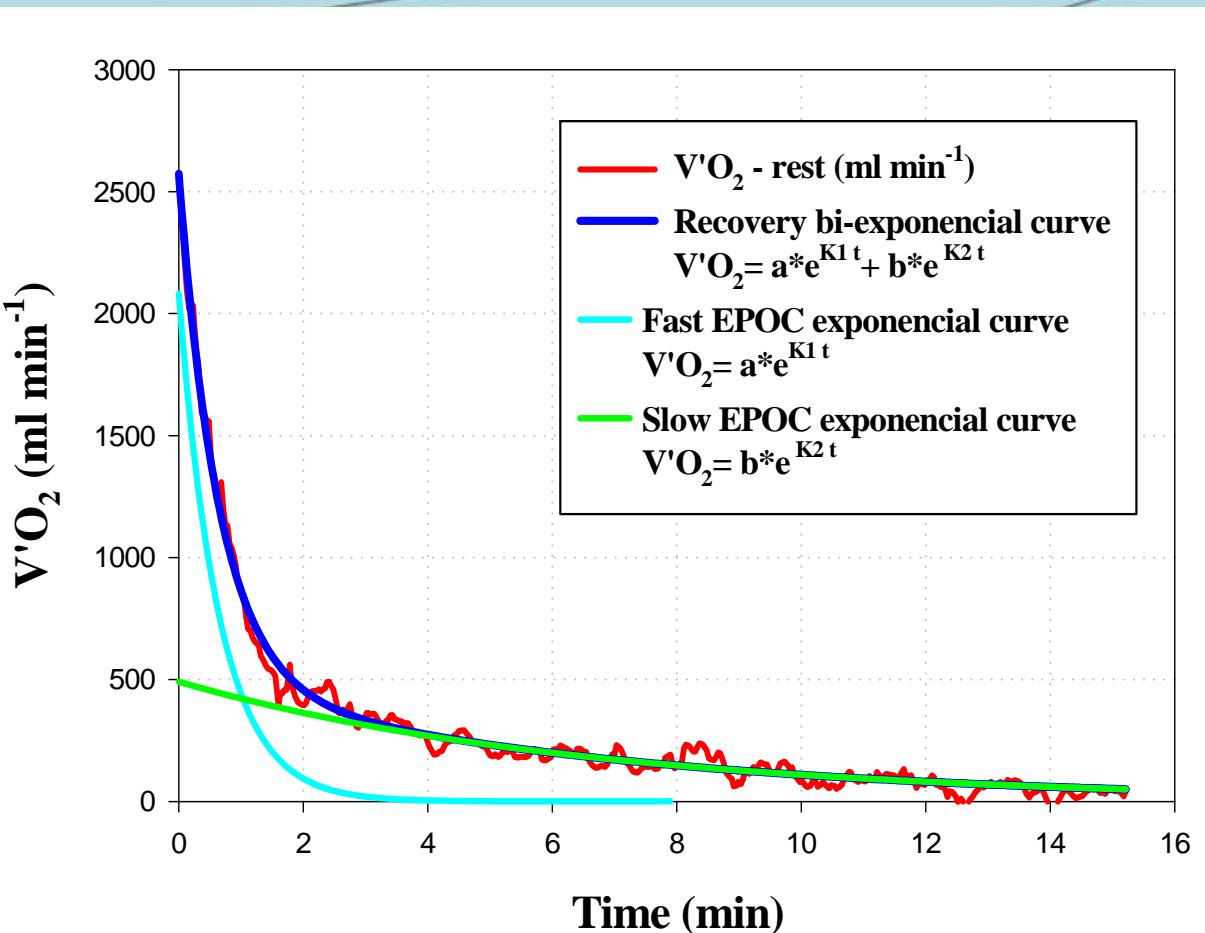
The AnT was carried out with the same ACE of the $\dot{V}O_2$ peak test and performed up to exhaustion under metabolic and cardiac monitoring (Quark b2, Cosmed, I; LactatePro, ARKRAI, Japan).

The relative intensity was the same for all subjects and equal to 130% of the Peak Power of the previous Test. The AnT was carried out up to exhaustion and therefore duration of exercise (total mechanical work) was different for each subject and dependent on his/her aerobic and anaerobic capacities.



Materials and Methods

V' O₂ curve processing before, during and after (Excess of oxygen consumption post exercise – EPOC) the effort to estimate relative energy contributions to perform exercise



$V' O_2$ curve processing during the recovery phase. The best fit curves of the O_2 flow that describe **Fast EPOC** and **Slow EPOC** during recovery.

The bi exponential equation:

$$V' O_2 = \alpha \cdot e^{K1*t} + \beta \cdot e^{K2*t}$$

Lactate Peak at the end of exercise to assess Lactacid anaerobic Energy Contribution

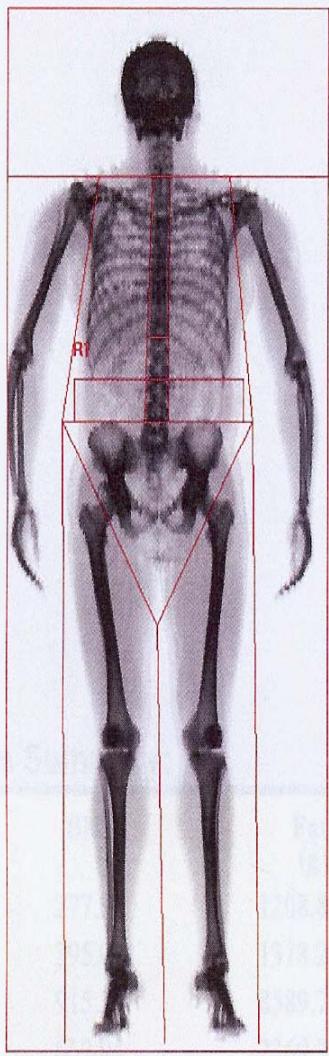
Beneke et al., Eur Appl Physiol. 87:338-392, 2002

Di Prampero PE., Rev Physiol Biochem Pharmacol. 89:143-222, 1981

Di Prampero & Ferretti, Respiration Physiology. 118:103–115, 1999.

Materials and Methods: DEXA

Dual Energy absorbometry X-Ray



$k = 1.161$, $d0 = 41.3$
318 x 150



Laboratori di Endocrinologia del Dipartimento
di Fisiopatologia Medica, I Facoltà di Medicina
e Chirurgia della Sapienza Università di Roma,
Diretti dal Prof. Andrea Lenzi



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Materiali e Metodi

Sono stati eseguiti prelievi ematici a digiuno, prima e dopo le 6-8 settimane di allenamento per dosare:

	v.n.
Col Tot	120-200 mg/dl
HDL	40-80 mg/dl
Trig	< 150 mg/dl
LDL	70-150 mg/dl
CPK	60-90 UI/L
Gli	60-100 mg/dl
gammaGT	< 45 UI/L
GOT	< 40 UI/L
GPT	3-40 UI/L

A cura del Gruppo del Prof Marcello Arca. Dept.

Ormoni e citochine	Unità di misura
HGH	mcg/L
IGF-1	ng/mL
C-PEPTIDE	ng/mL
GH-RELINA	ng/mL
GIP	ng/mL
GLP-1	ng/mL
GLUCAGONE	ng/mL
INSULINA	ng/mL
LEPTINA	ng/mL
PAI-1	ng/mL
RESISTINA	ng/mL
VISFATINA	pg/mL
ADIPONECTINA	μg/mL
ADIPSINA	ng/mL

A cura del
Gruppo del Prof
Luigi Di Luigi.
Dpt. Health
Sciences, Foro
Italico
University of
Rome.



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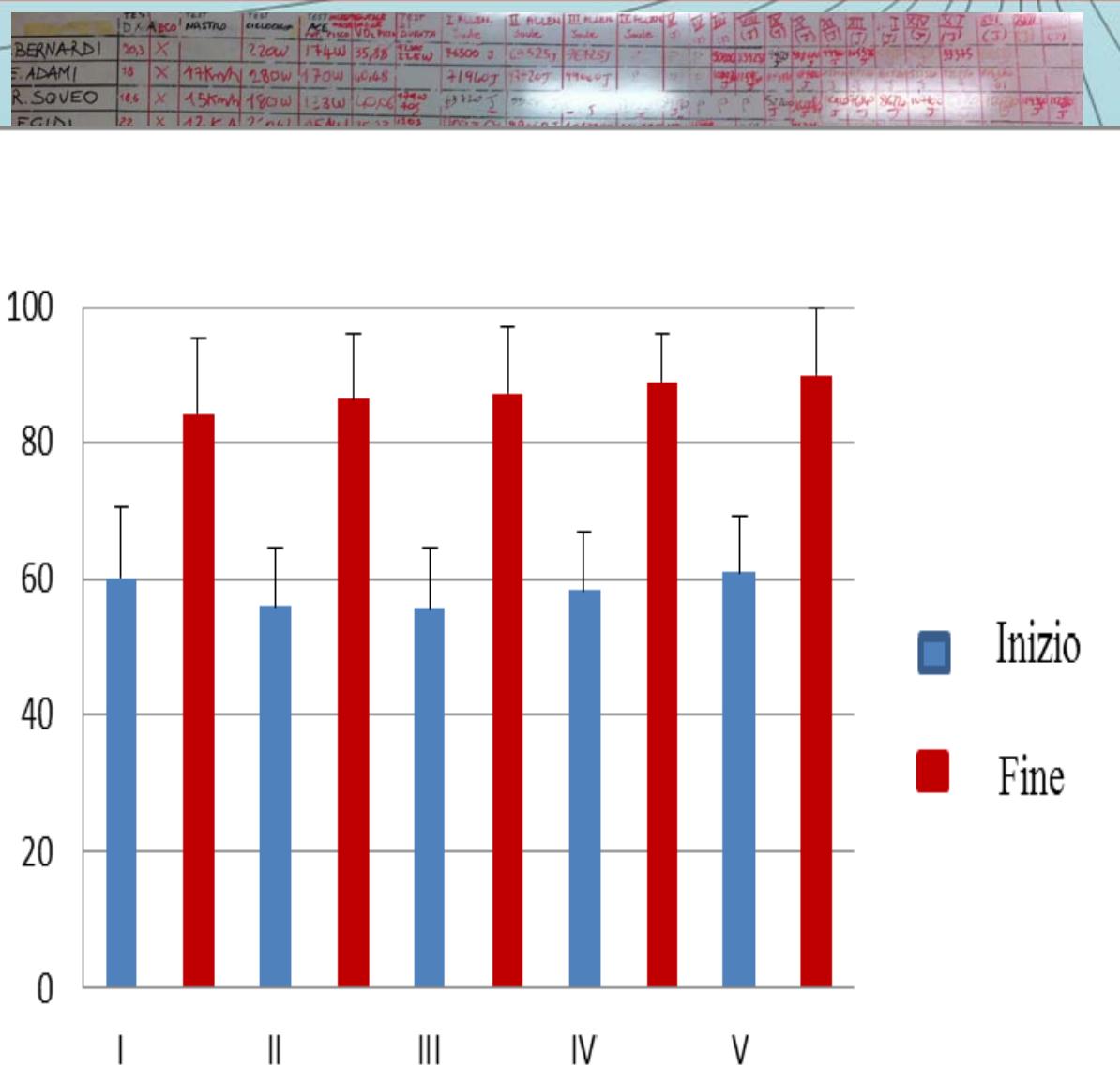
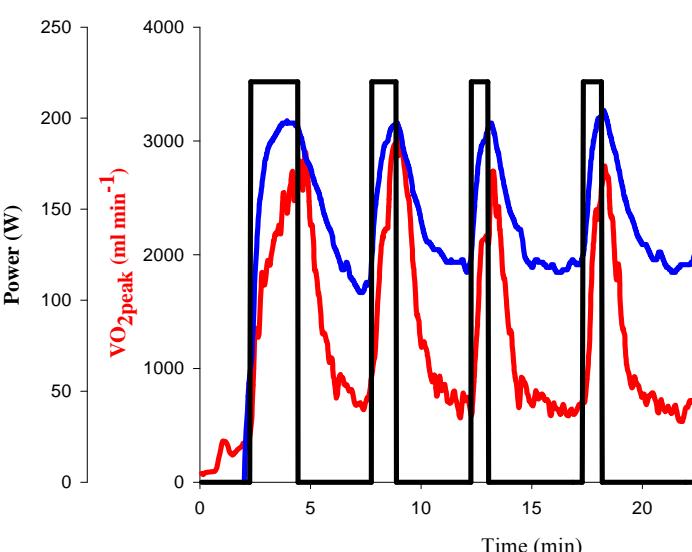
Risposta di ormoni e citochine prima e dopo una singola sessione di allenamento



Prelievi dalla vena antecubitale, mediante ago lasciato in sede e tolto solamente per il periodo dell'esercizio. Per i prelievi basali, i soggetti erano a riposo in posizione semireclinata e sono stati effettuati tre prelievi (10 ml di sangue ciascuno, -30, -15, -1 min, dall'inizio del test). Gli altri campioni di sangue sono stati prelevati subito dopo la fine dell'esercizio rispettivamente a 1, 5, 15, 30, 60 e 90 min dopo l'esercizio.



Allenamento Intervallato di Alta Intensità



Tipica risposta cardiaca e metabolica ad un allenamento di alta intesità -intervallato

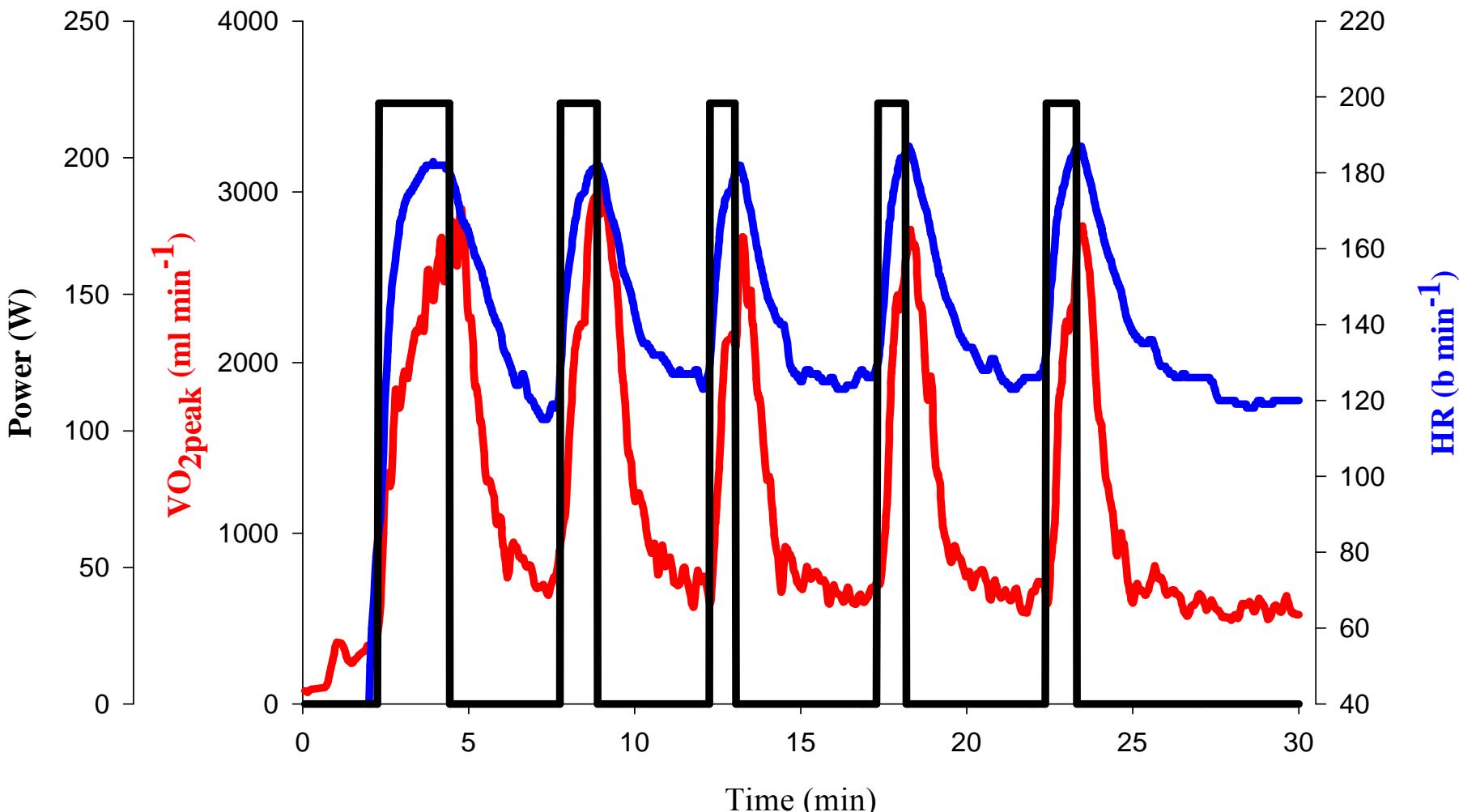
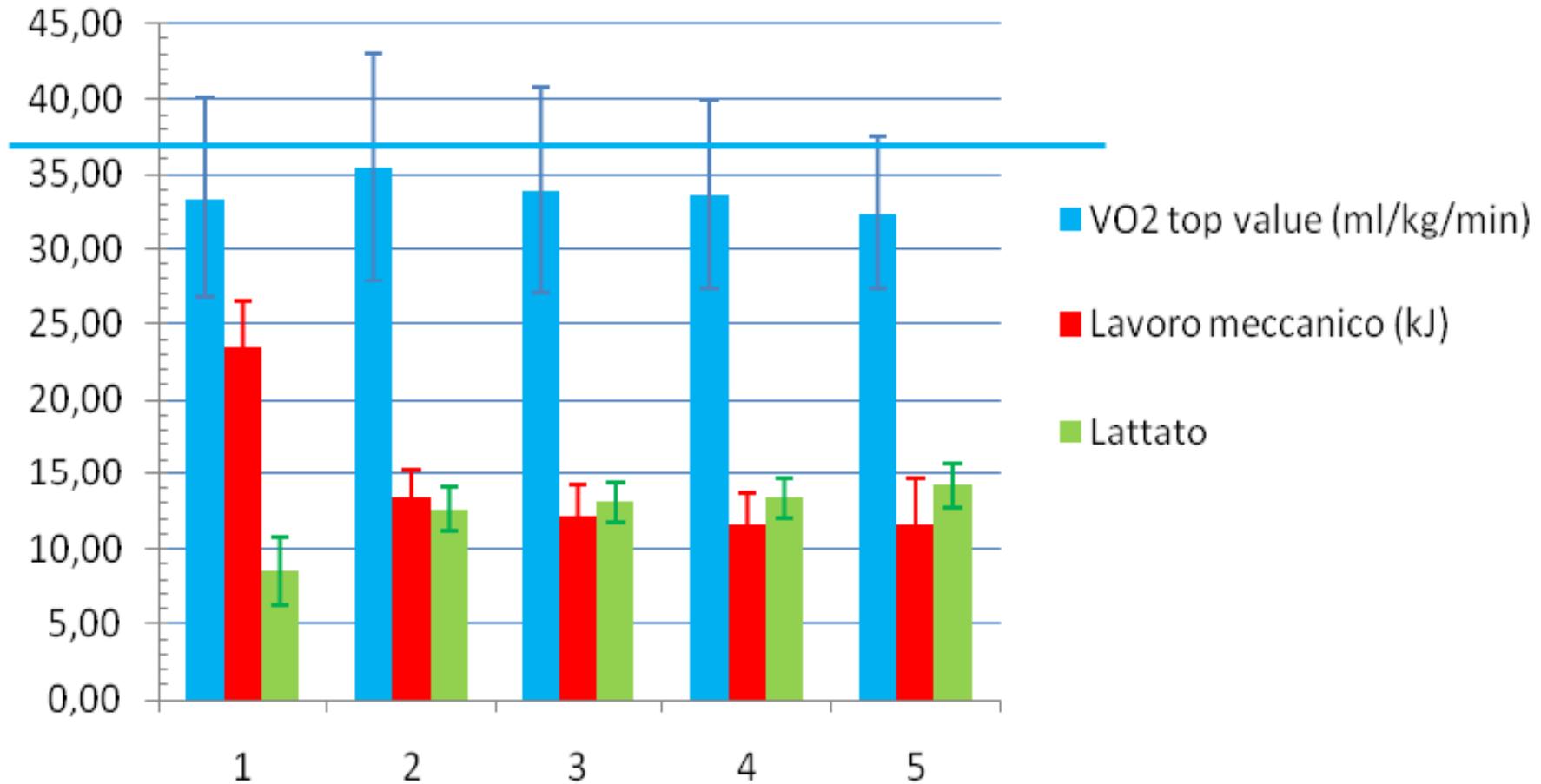


Figure 3:Heart rate (HR in blue), oxygen uptake ($\dot{V}O_2$ in red) and mechanical power (in black) during an ACE interval training of 5 bouts of a high intensity constant power exercise

Risposta meccanica e metabolica alla Singola sessione di allenamento

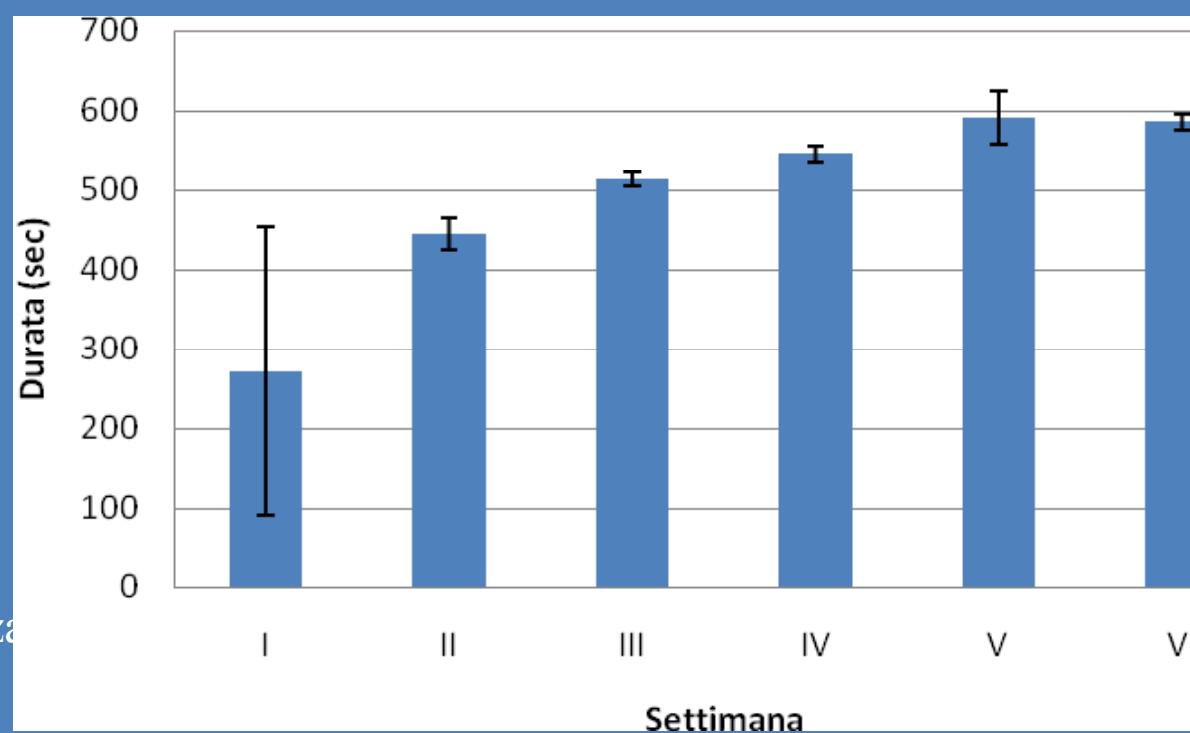


Lavoro meccanico (inizio allenamento): kJ

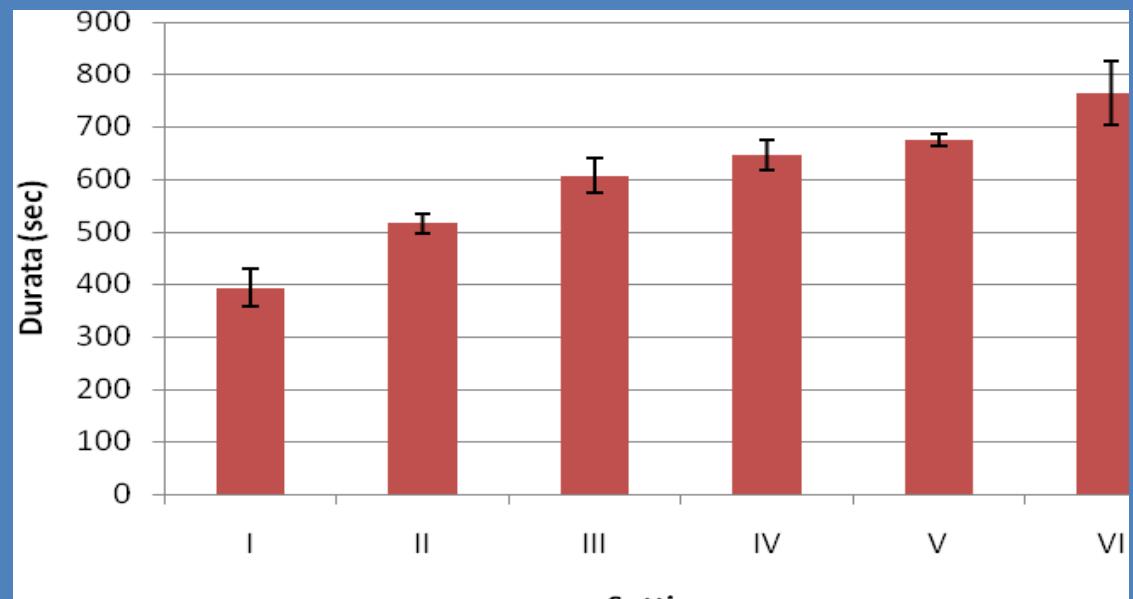
Dispensio Energetico: kJ - kcal

Andamento settimanale dei tempi di lavoro (somma dei tempi delle cinque ripetizioni di cui è composta ciascuna sessione)

Una singola ripetizione
dell'allenamento intervallato (Potenza
media: $215 \pm 23,9$ watt negli uomini)
aveva una durata di $68,6 \pm 12,81$ sec
(durata complessiva delle 5 rip:
 $349,9 \pm 36,69$ sec) e $127,2 \pm 40,09$ sec
(durata complessiva $636 \pm 116,25$ s),
rispettivamente all'inizio ed alla fine
dell'allenamento,



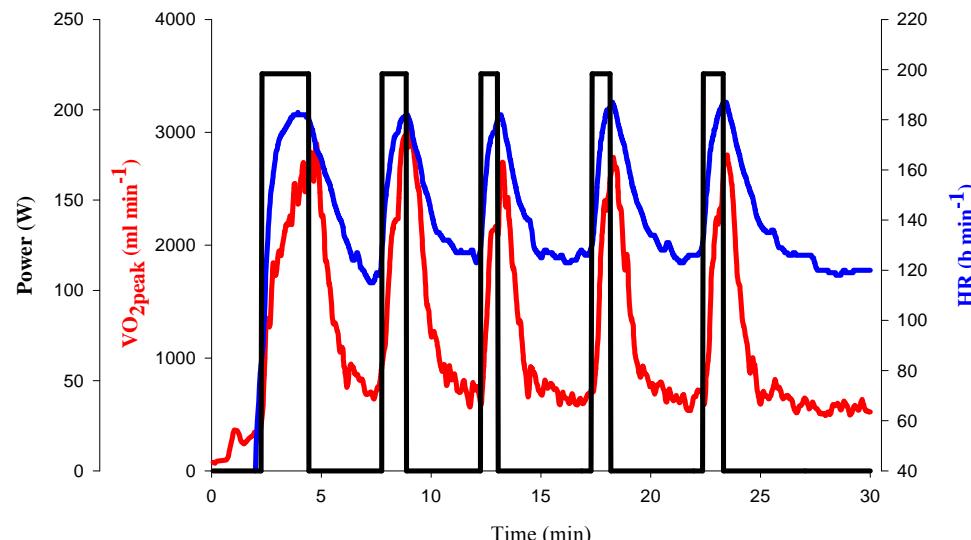
- In blu gli uomini e in rosso le donne



Lavoro meccanico (TWmech) e dispendio energetico per singola sessione di allenamento all'inizio e alla fine del periodo d'allenamento e valori totali complessivi di tutto il periodo di attività (6-8 settimane)

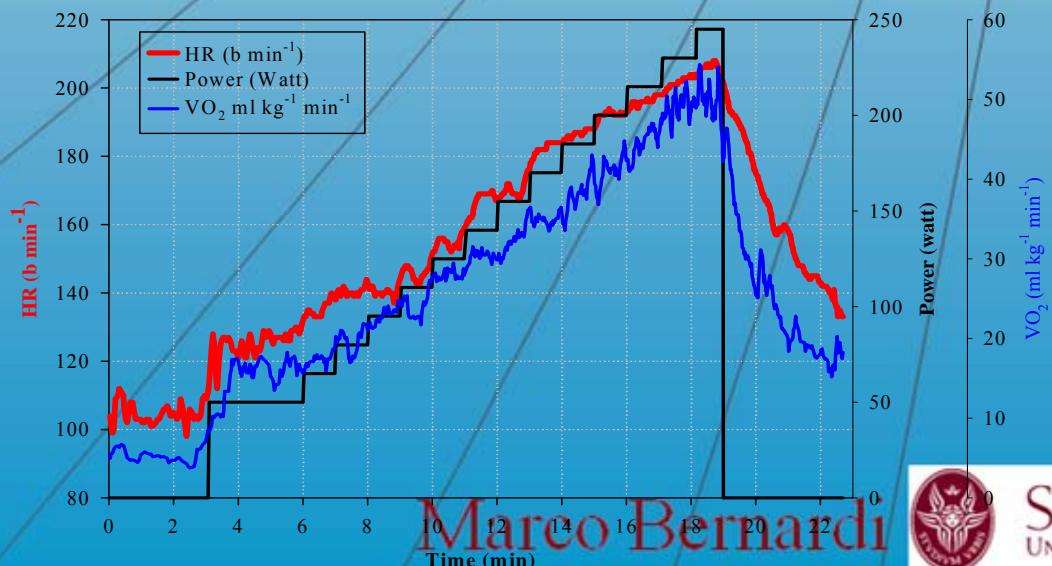
	TWmech	Total Energy
	(kJoule)	(kJoule)
1st session	$73,3 \pm 10,30$	$483 \pm 67,8$
Last session	$135,4 \pm 19,32$	771.8 ± 110.13

L'HI-IT comportava un lavoro metabolico totale nelle 24 sessioni di allenamento pari a $5691,9 \pm 1904,05$ kcal.



VO_2 picco (valori assoluti e relativi) e potenza picco
al test incrementale massimale all'ergometro a manovella.

	$\text{VO}_{2\text{peak}}$ ($\text{l}\cdot\text{min}^{-1}$)	$\text{VO}_{2\text{peak}}$ ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$)	Peak power (watt)
Before training	2.9 ± 0.31	37.4 ± 6.98	167.7 ± 20.93
After training	3.3 ± 0.32	43.1 ± 6.65	224.7 ± 17.38

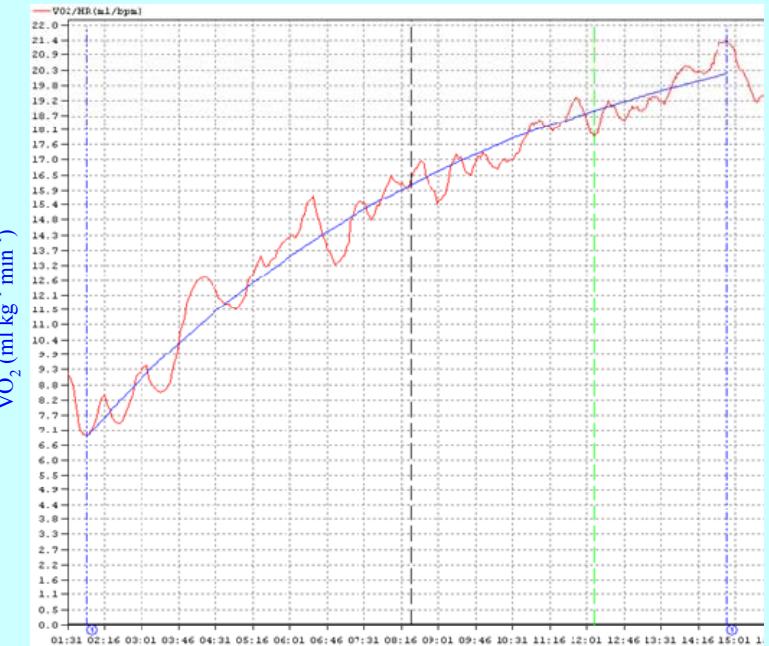
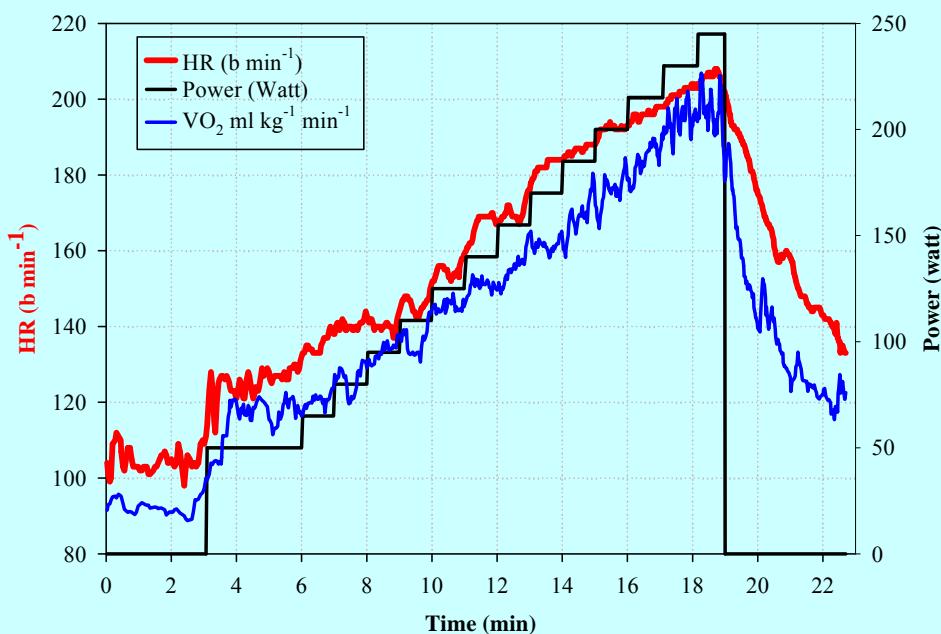


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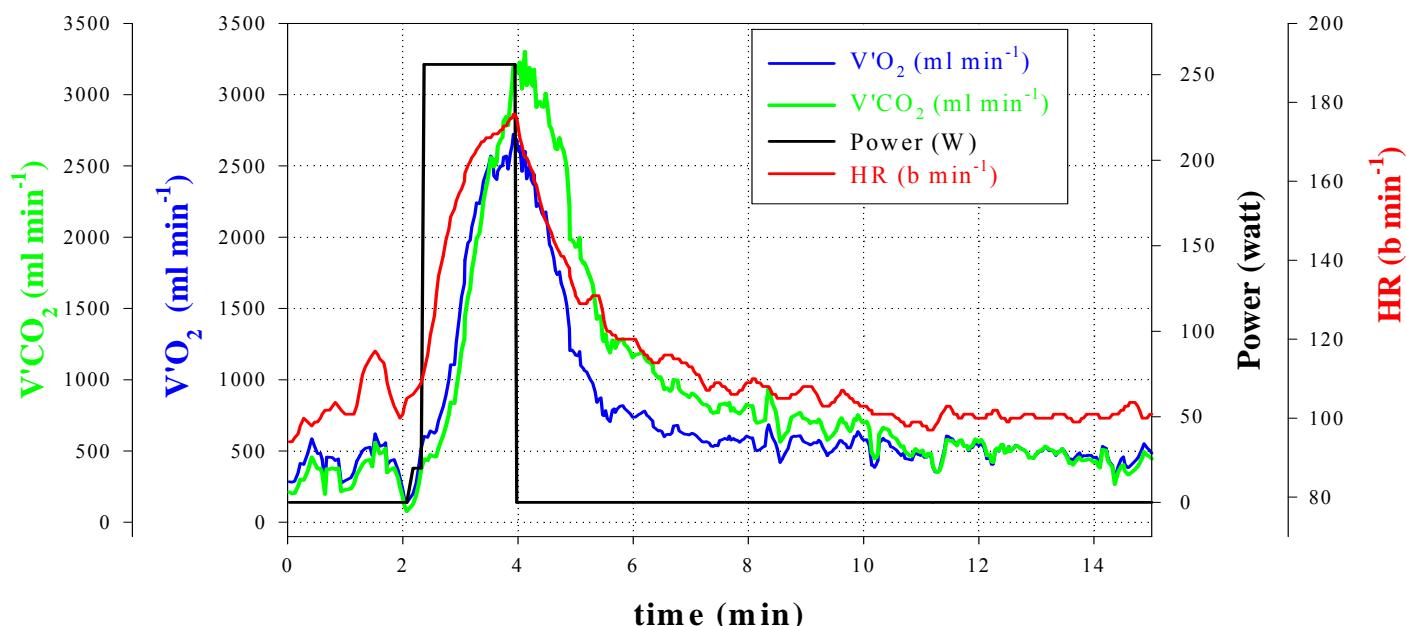
Picco di Polso d'Ossigeno



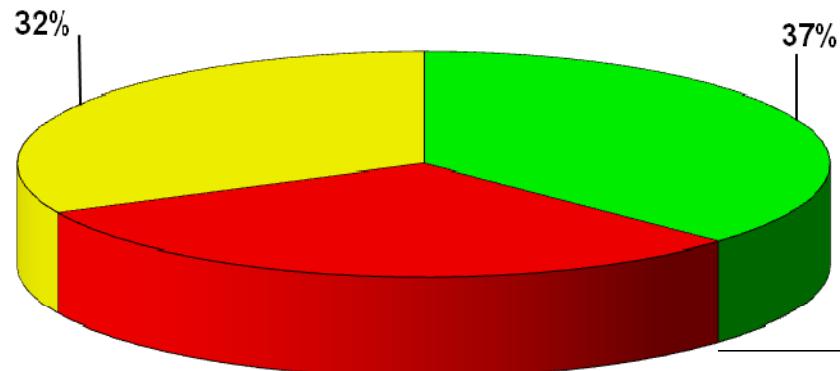
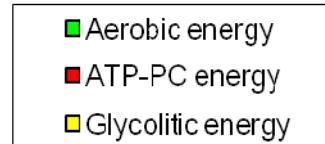
- Il picco del polso di ossigeno misurato alla fine del test aerobico ha dimostrato un incremento statisticamente significativo passando da $14,9 \pm 1,75$ ml battito⁻¹ prima dell'allenamento a $17,6 \pm 1,98$ dopo l'allenamento ($p=0,00089$). Questo risultato, tenuto conto dei dati ecocardiografici, dimostra un miglioramento della contrattilità cardiaca in condizioni di esercizio.

Lavoro meccanico totale e picco di lattato ematico valutato dopo test ad esaurimento

	TMW (kJ)	Blood lactate (mMol)
Before training	22.9 \pm 6.10	11.7 \pm 1.75
After training	33.5 \pm 6.76	13.3 \pm 2.77



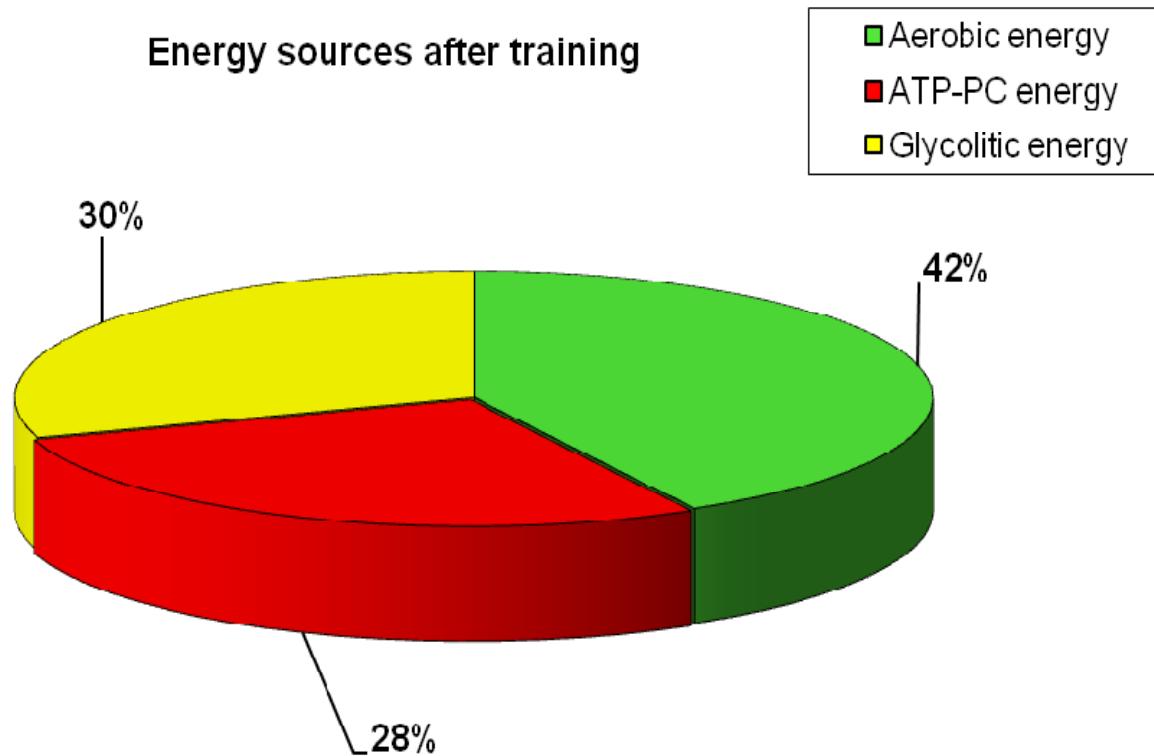
Energy sources before training



Sorgenti Energetiche del Test ad esaurimento

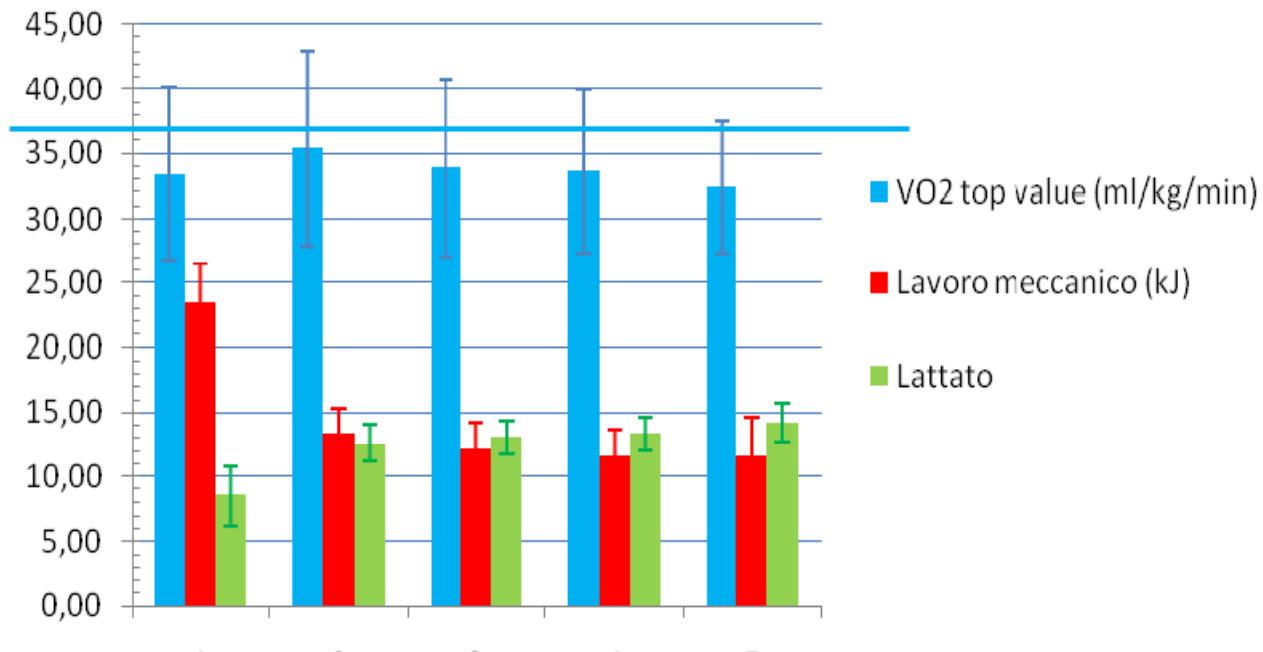
Prima
e dopo
l'allenamento

Energy sources after training

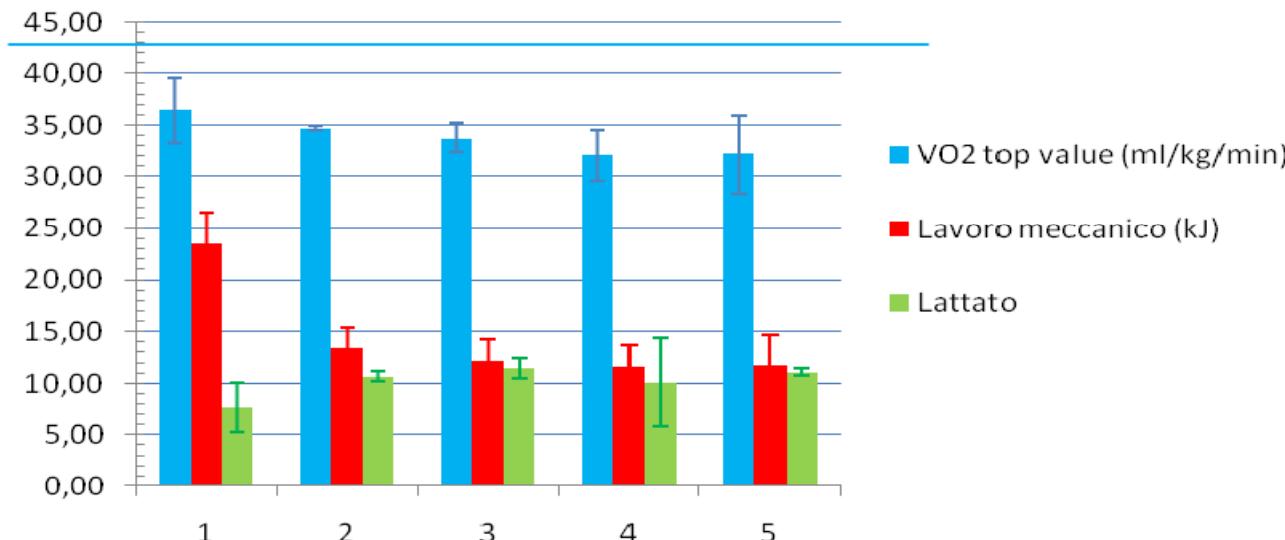


Risposta Metabolic a allo stesso carico di lavoro

Allenamento PRE uomini



Allenamento POST uomini



Total Fat Mass

**Before
training**

(kg)

Man 14.3±2.57

12.3±3.11

Women 12.0±1.62

10.7±1.31

Total 13.4±2.43

11.7±2.59

Abdominal Fat Mass

**Before
training**

After training

(g)

Man 1063.7±296.09

(g)

879.6±228.75

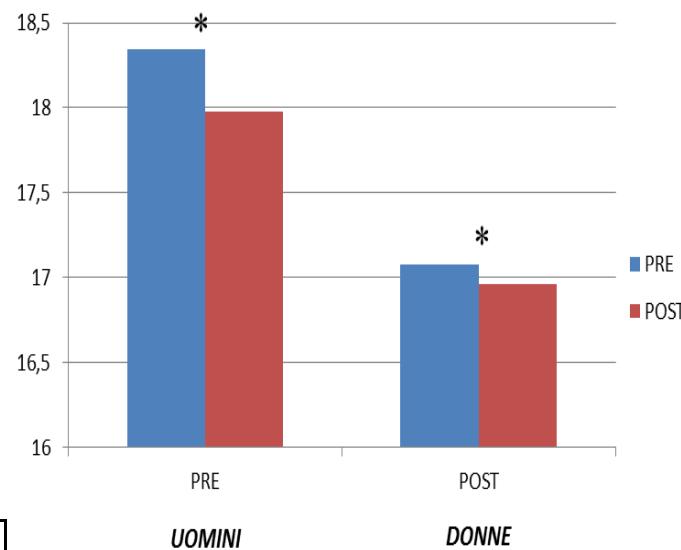
Women 654.8±149.60

551.0±140.54

Total 910.3±318.24

756.4±338.10

Percentuale di Massa grassa prima e dopo l'allenamento



Massa grassa in sottoregione addominale prima e dopo l'allenamento



Prelievi ematici prima e dopo l'allenamento

	v.n.	PRE		POST		P
		Media	Dev. St.	Media	Dev. St.	
Col Tot	120-200 mg/dl	194,4	54,49	171,8	31,52	0,094
HDL	40-80 mg/dl	52,8	15,88	53,8	12,54	0,393
Trig	<150 mg/dl	164,6	123,59	99,8	37,84	0,151
LDL	70-150 mg/dl	108,8	42,67	98	19,77	0,198
CPK	60-90 UI/L	87,6	37,69	70	28,83	0,094
Gli	60-100 mg/dl	87,2	12,34	90,6	10,31	0,282
gammaGT	< 45 UI/L	27	25,25	21	9,33	0,230
GOT	< 40 UI/L	17,4	3,36	16,2	1,48	0,294
GPT	3-40 UI/L	16	5,15	16,6	6,19	0,317

A cura del Gruppo del Prof Marcello
Arca. Dept.



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Valori medi e deviazioni standard di ormoni e citochine prima e dopo l'allenamento

Ormoni e citochine	Unità di misura	Pre-allenamento	Post-allenamento	p-value
HGH	mcg/L	0.25±0.34	0.56±0.86	0.15
IGF-1	ng/mL	260.53±42.20	281.73±16.15	0.02*
C-PEPTIDE	ng/mL	14.7±8.8	13.3±4	0.19
GH-RELINA	ng/mL	38.3±65.15	47.7±83.5	0.07
GIP	ng/mL	2±0.8	3±2	0.11
GLP-1	ng/mL	2±1	2.3±1.5	0.09
GLUCAGONE	ng/mL	26±1.9	26.3±2	0.1
INSULINA	ng/mL	2.5±2.3	2.1±1.8	0.28
LEPTINA	ng/mL	9.3±8.4	5.2±4.3	0.04*
PAI-1	ng/mL	233.8±57	176.3±7	0.03*
RESISTINA	ng/mL	230±228	108.2±110.2	0.02*
VISFATINA	pg/mL	864±520	975±424	0.18
ADIPONECTINA	μg/mL	5.56±2.8	5.3±2.1	0.25
ADIPSINA	ng/mL	698.96±708	673±694	0.44

A cura del Gruppo del Prof Luigi Di Luigi.
Dpt. Health Sciences, Foro Italico University of Rome.

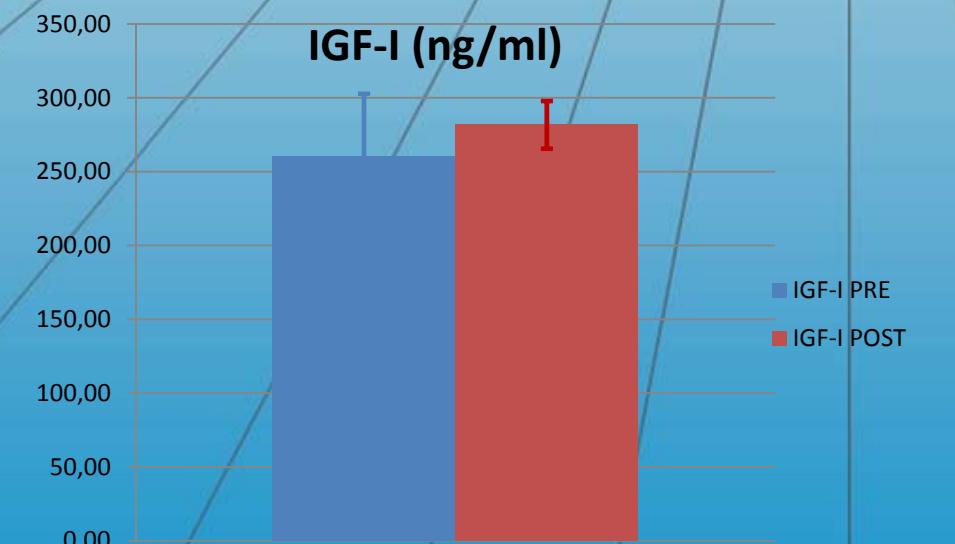
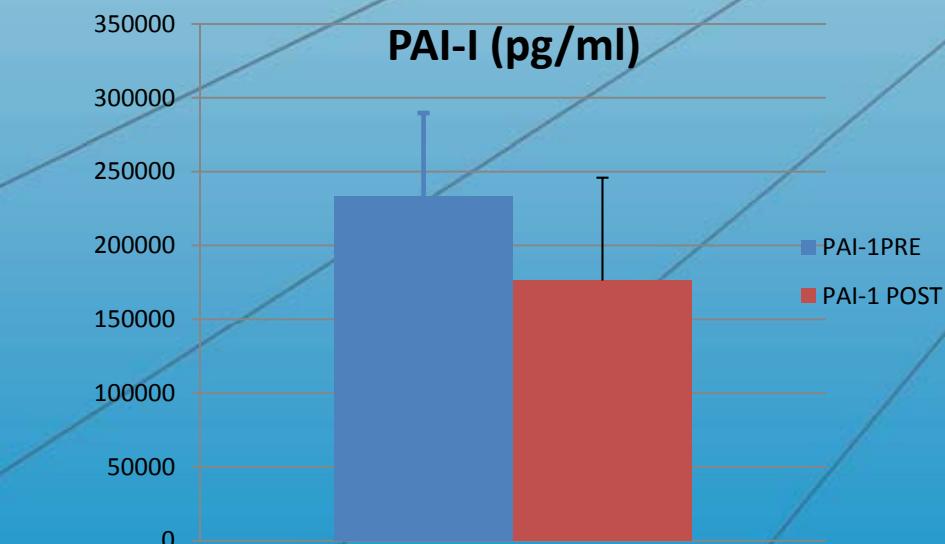
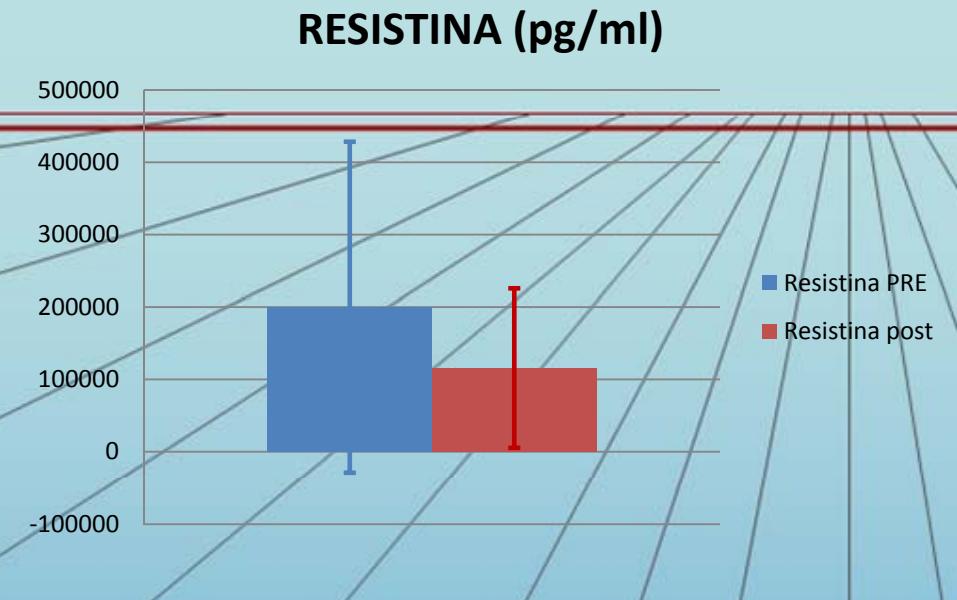
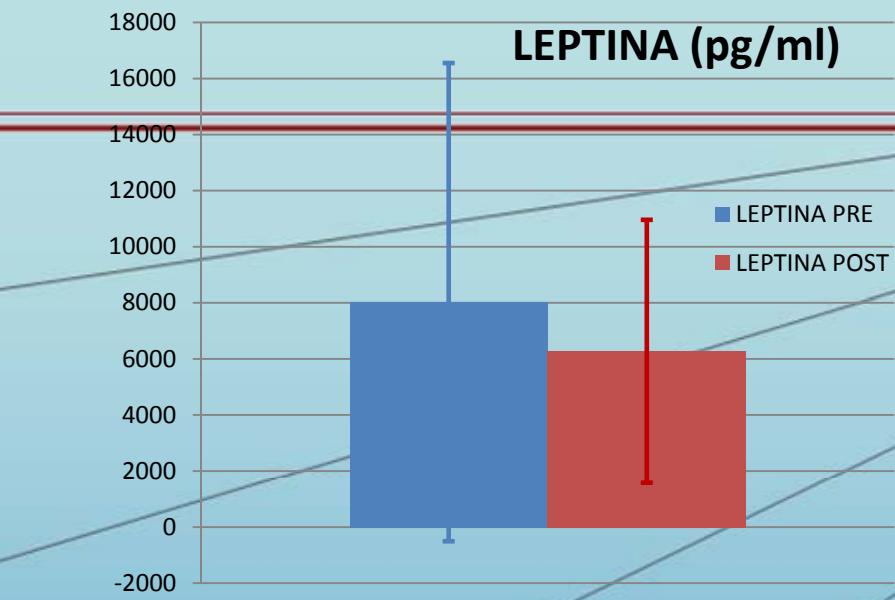


Table 1 - Adipose tissue-produced neuronal and neuroendocrine factors.

Neuropeptides

- Agouti protein (2, 3)
- Neuropeptide tyrosine (NPY) (9, 12)
- Calcitonin gene-related peptide (10)
- Adrenomedullin (10)
- Somatostatin (11)
- Insulin-like growth factor (12)
- Substance P (13)
- Kisspeptin (14)
- Neuromedin B (a bombesin-like peptide) (15)
- Neurotensin (16)
- Mineralocorticoid-releasing factors (21)
- Corticotropin-releasing hormone (CRH) (22)
- Stresscopin and urocortin (CRH-like peptides) (22)
- Atrial natriuretic peptide (35)
- Nesfatin-1 (36)

Neurotrophic factors

- Nerve growth factor (3, 12, 24, 27)
- Brain-derived neurotrophic factor (12, 23, 27)
- Vascular endothelial growth factor (24, 27)
- Angiopoietin-1 (25)
- Ciliary neurotrophic factor (27)
- Metallothioneins (27)
- Glial cell line-derived neurotrophic factor (27)

Neurotransmitters

- Noradrenaline (2)
- Glutamate (17)
- Gamma-aminobutyric acid (GABA) (17)

Others

- Leptin (2, 3)
- Adiponectin (2, 3)
- Resistin (2, 3)
- Fasting-induced adipose factor (2)
- Apelin (2)
- Semaphorin (Sema3A) (28)
- Neuropilin-1 (28)
- Pantophysin (29)
- Nestin (30)
- Neuronal nuclear antigen (30)
- Glial fibrillary acidic protein (30)
- Neuron-specific enolase (30)

References are indicated in parentheses.

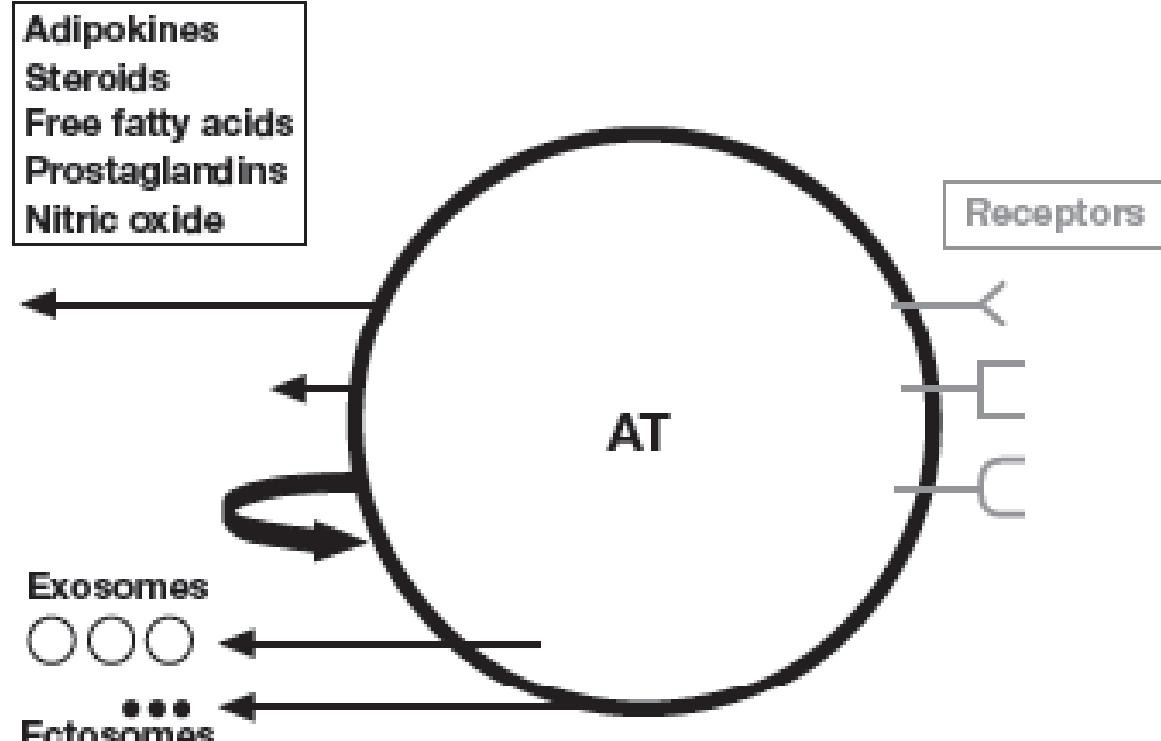
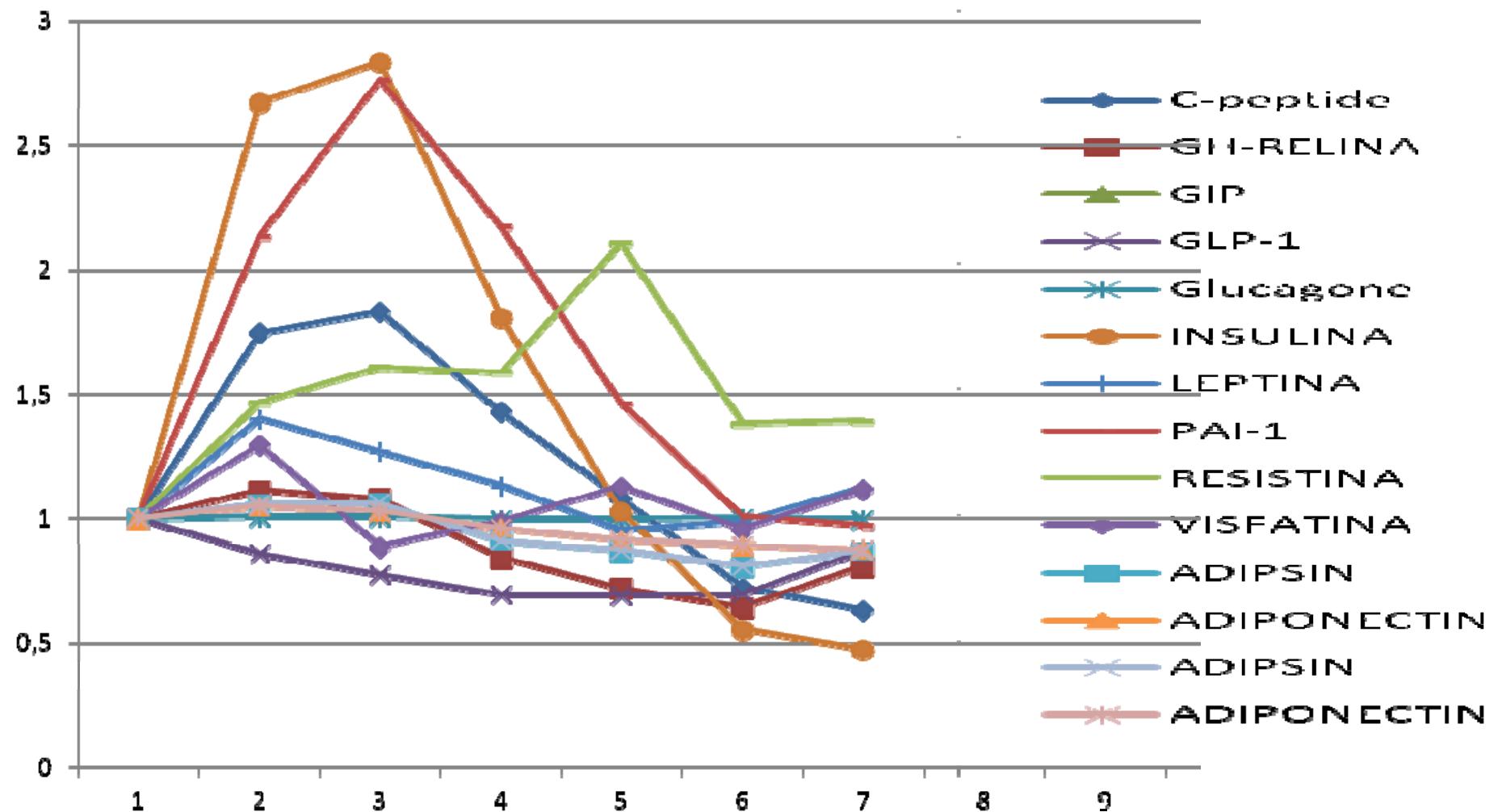


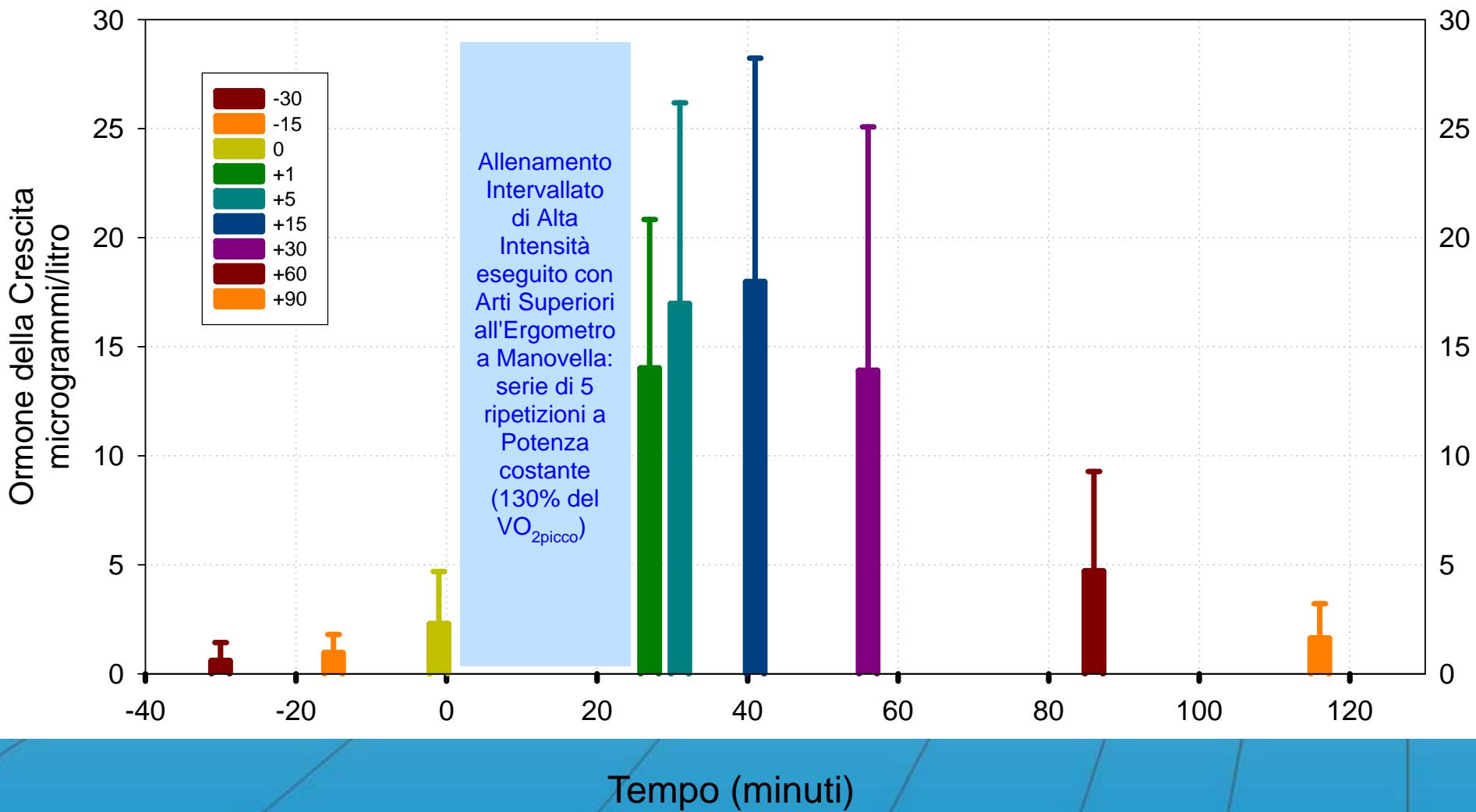
Fig. 1 - A drawing illustrating both secretory and receptive nature of adipose tissue (AT). At secretory level, AT-derived signaling molecules communicate via multiple pathways such as endocrine (arrows 1,4,5, from up-down), paracrine (arrow 2) and autocrine (arrow 3, curved) as well as via exosomes (multivesicular body-derived microvessels) and ectosomes (plasma membrane-shedding microparticles) (see ref. 37). At receptive level, AT possesses receptors for various ligands.

Risposta delle citochine e degli ormoni alla singola sessione di esercizio (parametri normalizzati)

Cinetica parametri ematici



Risposta Acuta (aggiustamenti ormonali) all'allenamento



Valori in k Joule e kcal del DE e del relativo lavoro meccanico

		I ^a settimana	VI ^a settimana	Totale
Lavoro Meccanico	kcal	57.2±6.0	99.5±12.6	505.9±50.4
	kJ	239.3±25.3	416.3±52.7	2117.6±210.7
Dispendio Energetico Metabolico	kcal	575.2±103.8	1009.8±245.9	5121.7±1074.3
	kJ	2407.5±434.4	4226.5±1029.3	21436.9±4496.6

Valori in k Joule e kcal della spesa energetica necessaria per bruciare i lipidi (come desunto dal peso della massa grassa del soggetto)

Dispendio Energetico calcolato su perdita di massa grassa	kc	16452.0±1
	al	5321.6
	kJ	68859.8±6
		4128.4



HI-IT in Athletes



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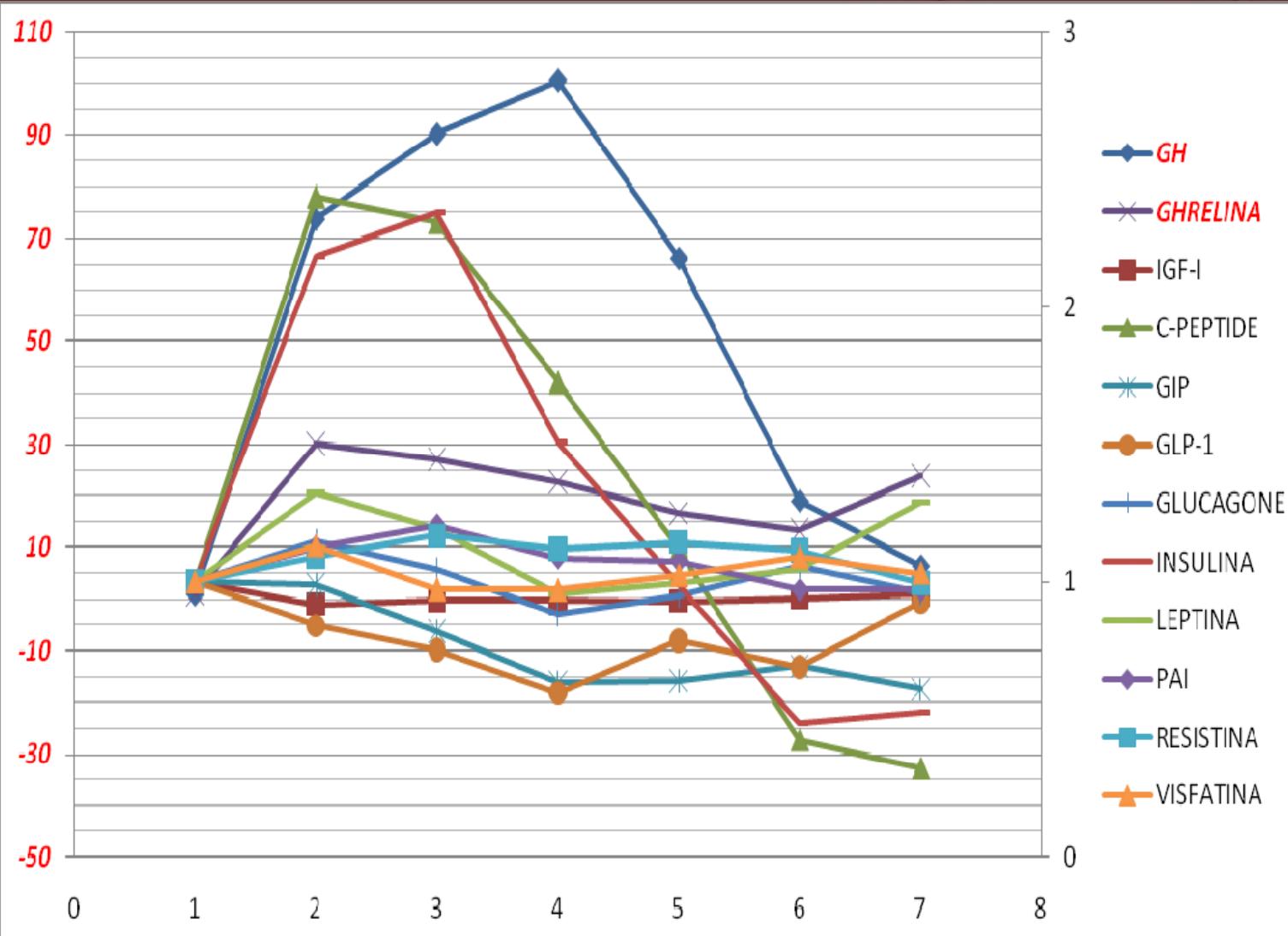
ATP:

La moneta di Scambio Energetico



Il Tempio di
Giunone
Moneta

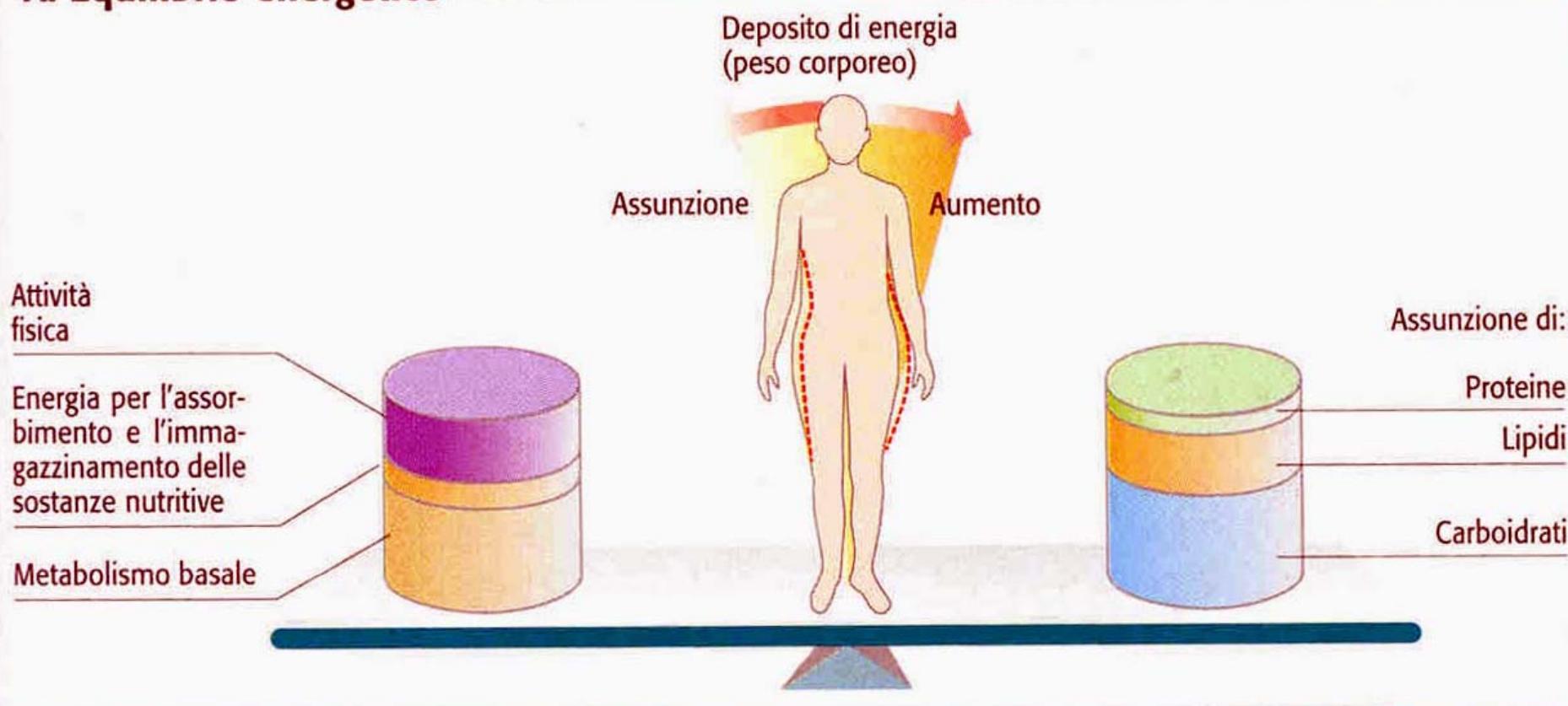
Risposta ormonale e citochinica alla singola sessione di Allenamento



Valori
normaliz-
zati sulla
base dei
dati a
riposo
(resi pari
a zero).

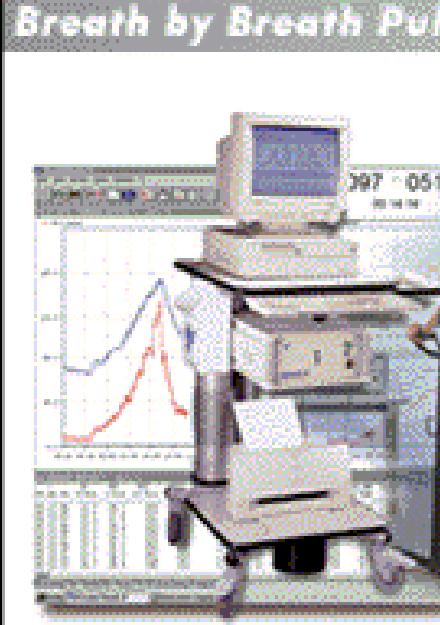
Agire sul bilancio energetico è possibile non solo modificando e riducendo il regime nutrizionale ed aumentando la quantità di esercizio fisico ad intensità moderata ed intensa

A. Equilibrio energetico



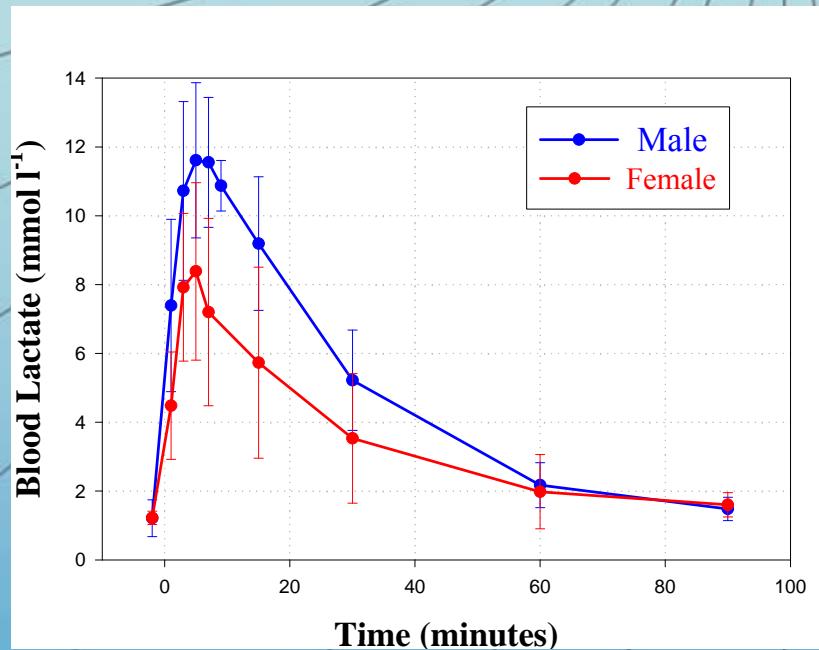
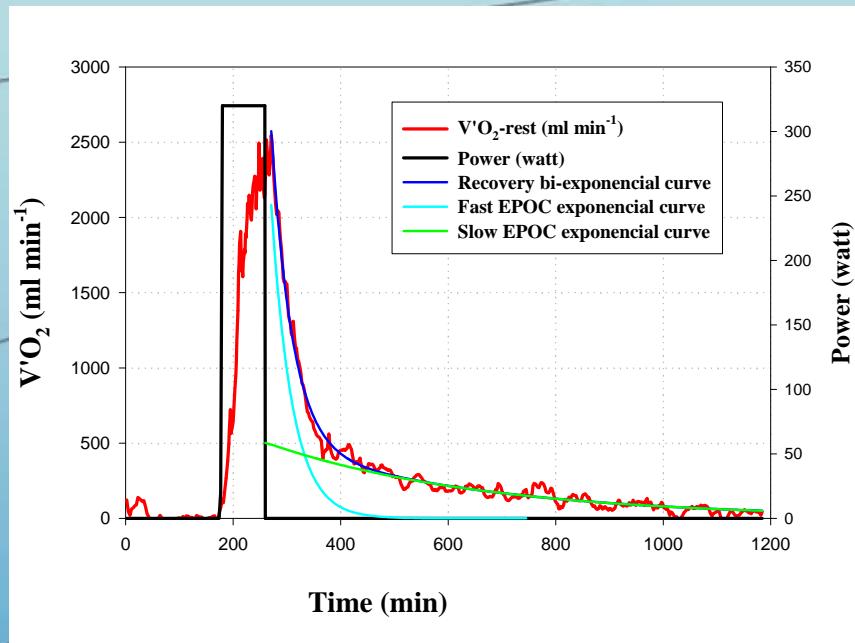
E' possibile incidere sulla spesa energetica riducendo la quantità di tempo passata in condizioni di reale sedentarietà ("stare seduti") ed aumentando la quantità di tempo trascorsa in attività motoria differente dall'esercizio fisico (attività motoria struttura), attività motoria che comunque determina una spesa energetica superiore a quella basale.

Test di Laboratorio per valutare ed allenare la massima potenza aerobica in grinder di Coppa America



Results

Anaerobic test up to exhaustion (AnT - 150%V' O₂ peak)

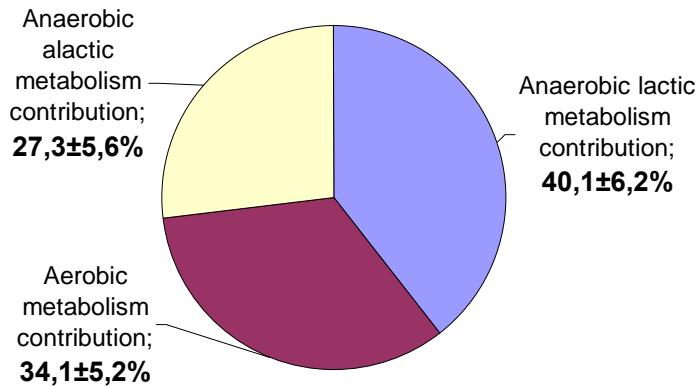


Subjects	Highest $V' O_2$ $\text{ml min}^{-1} \text{kg}^{-1}$	Highest HR $\text{b} \cdot \text{min}^{-1}$	Power watt	AnT duration seconds	Total mechanical work kJ	Lactate peak mmol l^{-1}
Men	31.7 ± 4.86	178 ± 15	277.6 ± 57.9	$91. \pm 19.5$	25.2 ± 7.3	12.3 ± 1.9
Women	28.1 ± 6.3	174 ± 9	170.2 ± 27.5	63.2 ± 11.1	10.9 ± 2.9	8.6 ± 2.7

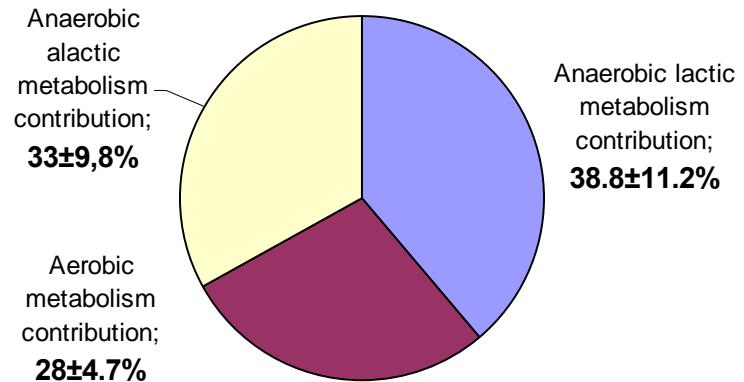
Results: Energy expenditure of the AnT: Metabolic Contributions from the 3 Metabolisms

Aerobic, Lactic Anaerobic, Alactic Anaerobic (high Energy Phosphates) Energy Metabolisms

MEN
Percentages of various metabolisms



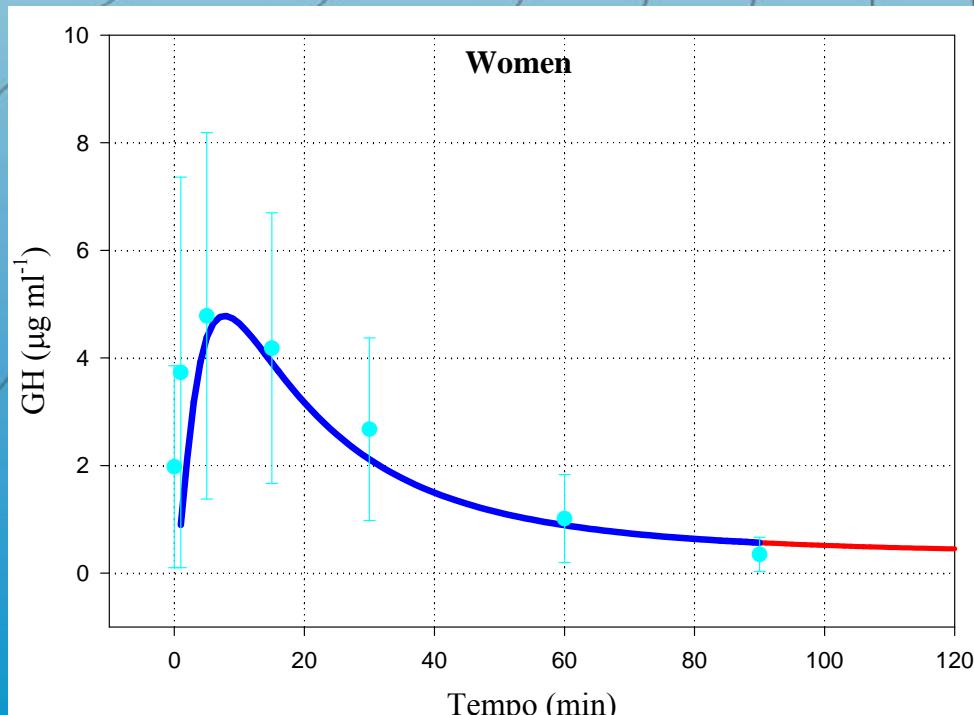
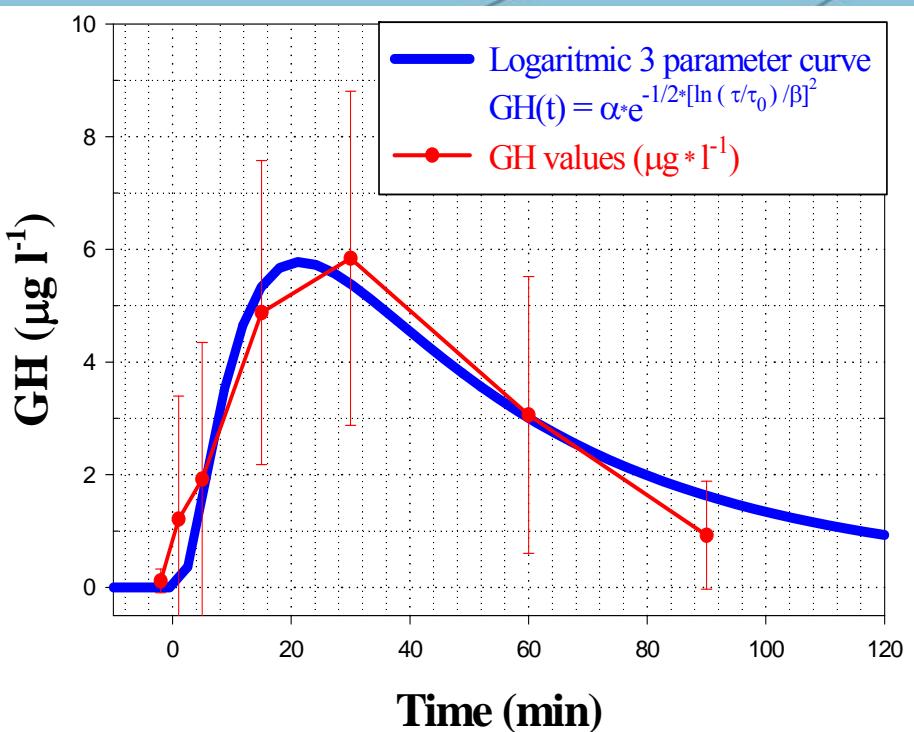
Women
Percentages of various metabolisms



	Aerobic Energy contribution kJ	Anaerobic alactic Energy contribution kJ	Anaerobic lactic Energy contribution kJ	Total Energy expenditure kJ
Men	49.6±17.6	37.7±9.8	56.6±14.4	143.8±36.2
Women	21.3±7.2	23.8±6.6	29.21±11.0	74.4±16.2

Results: GH Response to 150% V' O₂peak Exercise – Men and Women

Men showed after the exercise a much more relevant increment of GH than Women (113 ± 91 vs 5.7 times higher than basal values) with peak values equal to 7.1 ± 3.55 ng/ml vs 4.7 ng/ml



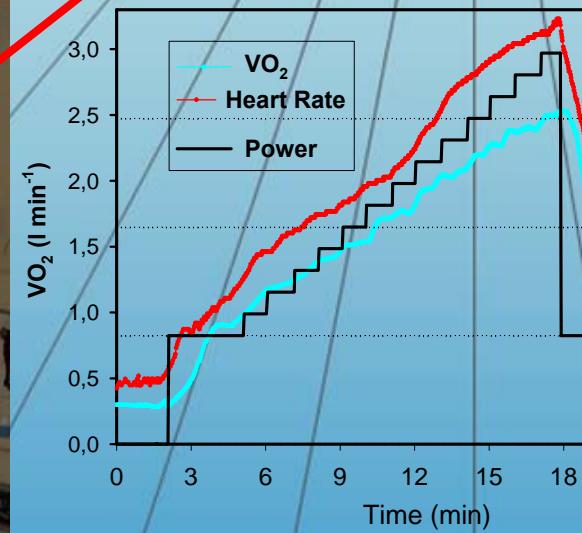
Oxygen consumption peak measurement

Arm Cranking Ergometer (ACE). Continuous multistage exercise test up to exhaustion.



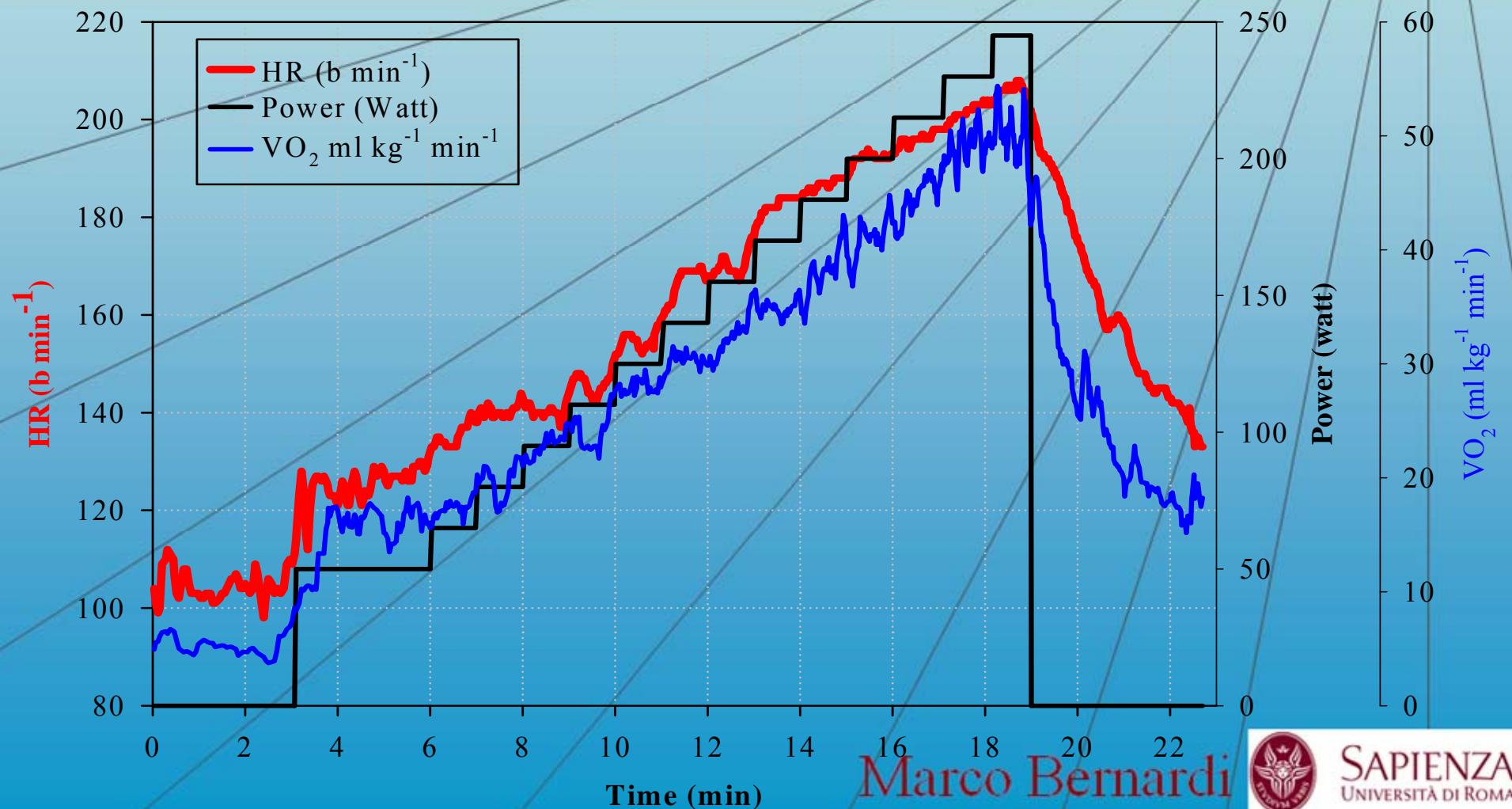
Electrocardiogram:HR

electrodes
monitor



Metabolimeter
(Quark b², Cosmed)
 V_E , $\dot{V}O_2$, $\dot{V}CO_2$

Test Incrementale massimale all'Ergometro a manovella (ACE)

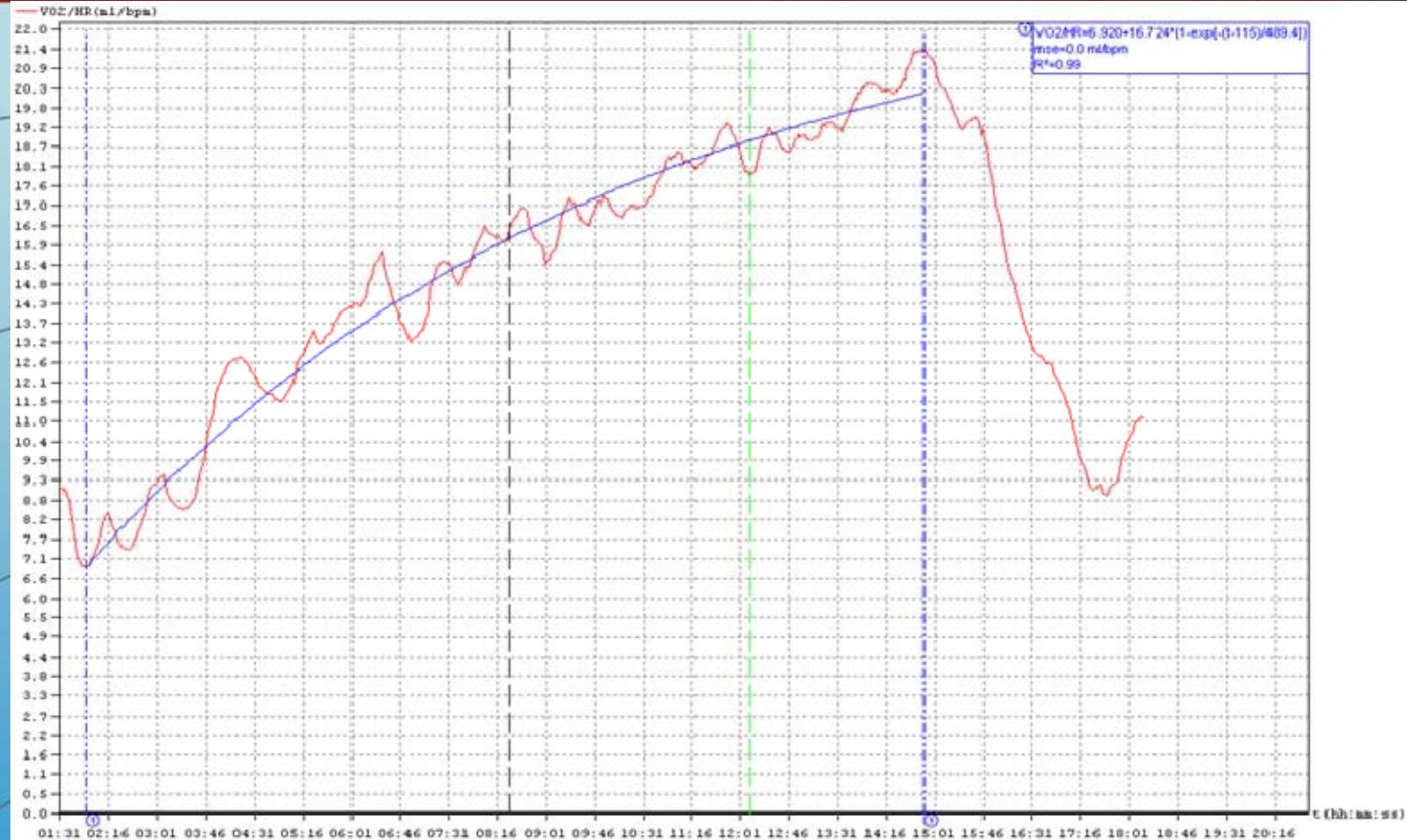


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Polso di Ossigeno



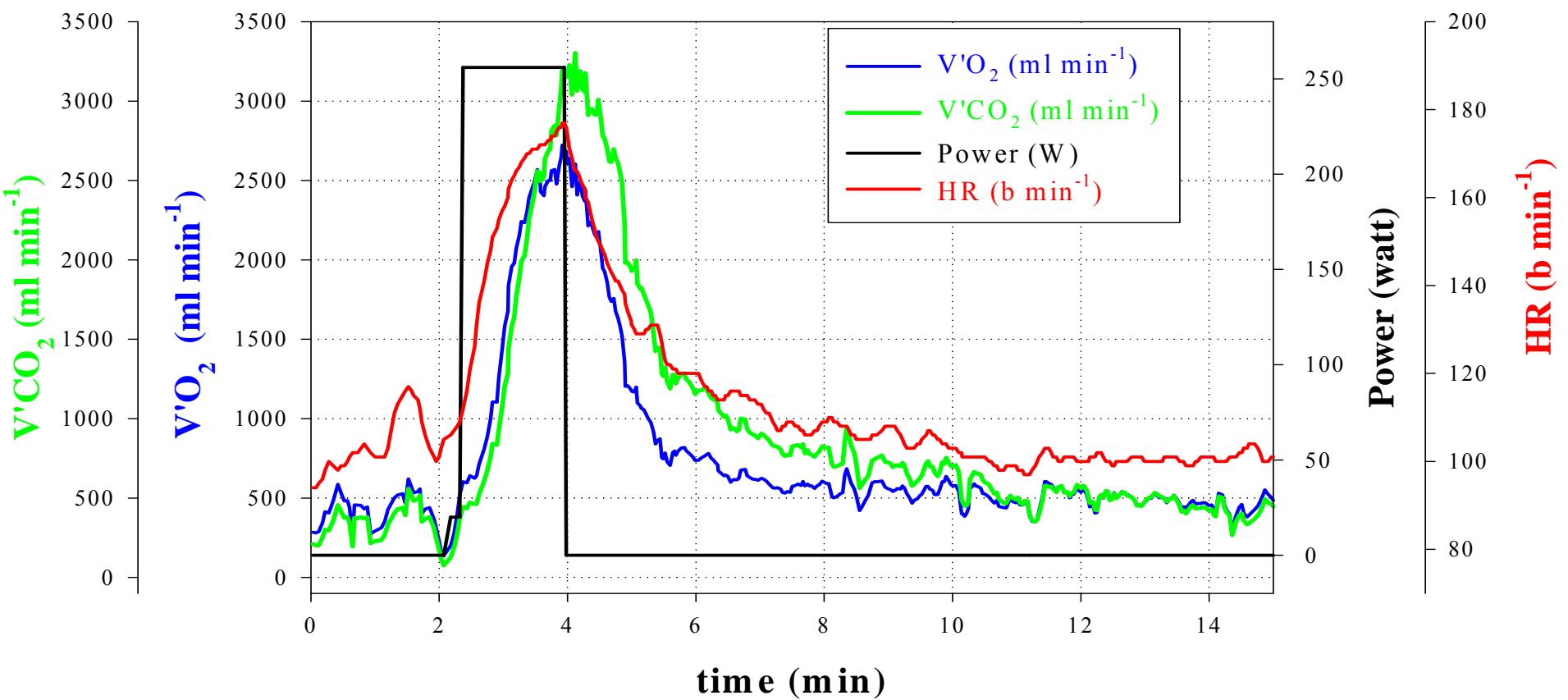
- Andamento del polso di ossigeno durante un test incrementale massimale condotto all'ergometro a manovella (in rosso) e sua curva esponenziale di interpolazione (in blu).

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Tipica risposta cardiaca e metabolica in un test ad Esaurimento al 130% della massima potenza meccanica aerobica.



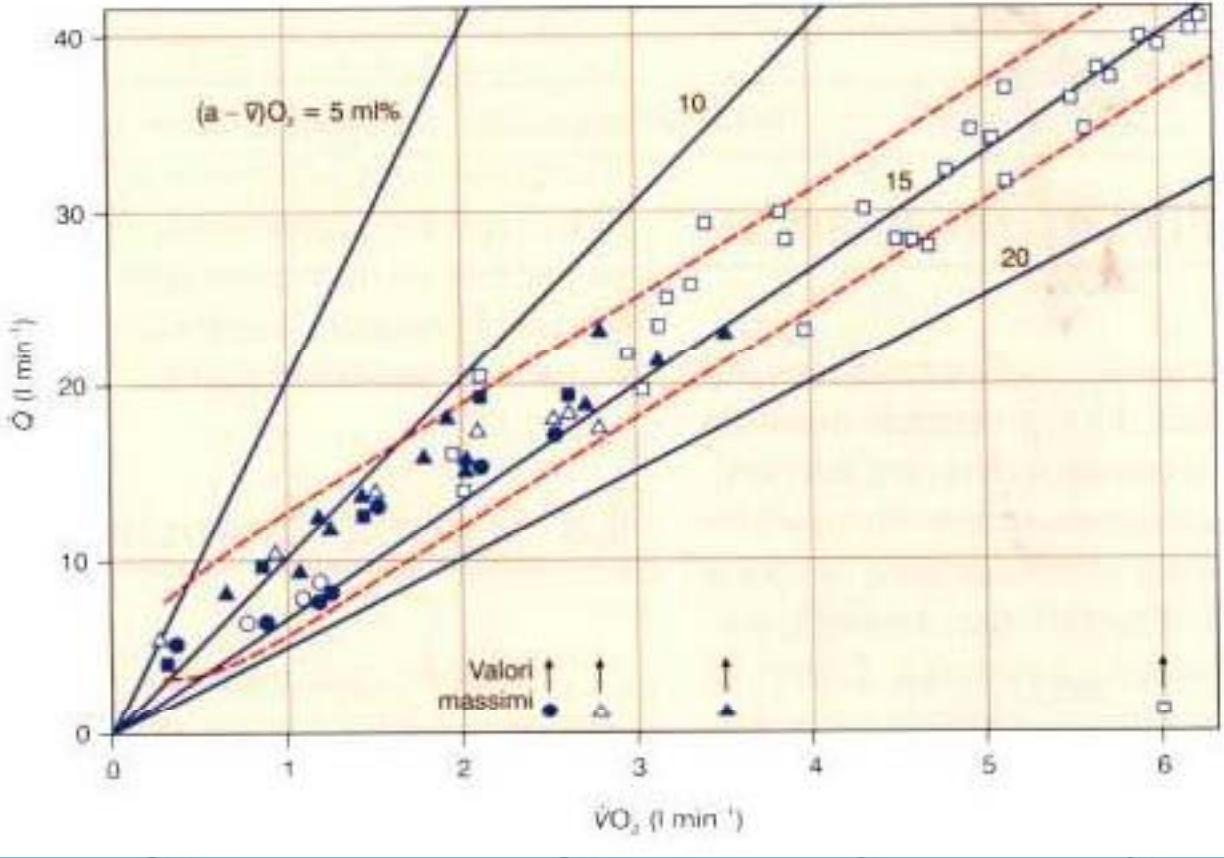
Test all'esaurimento all'ergometro a manovella

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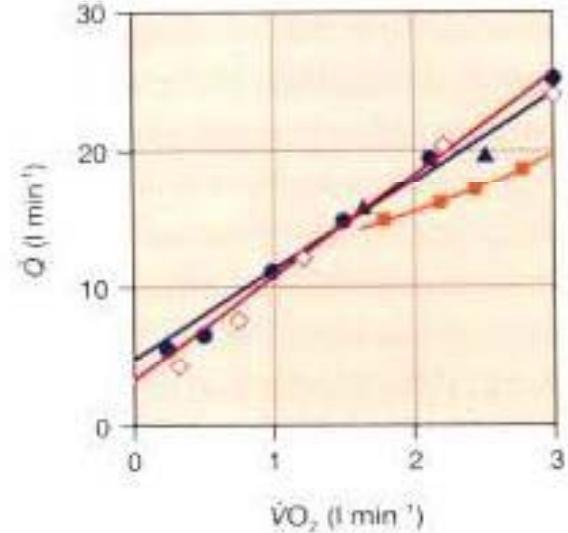
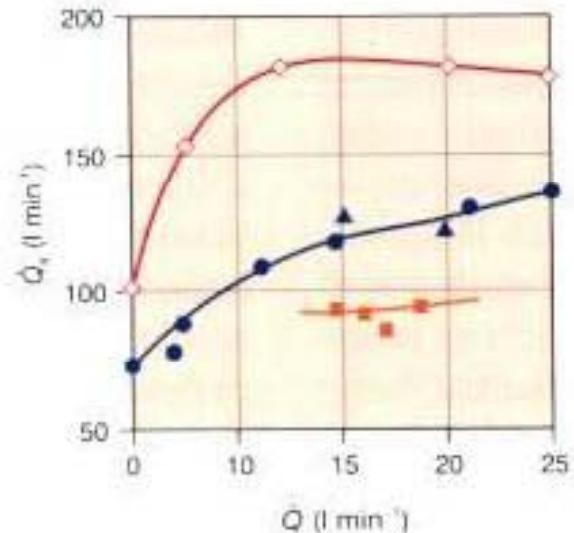
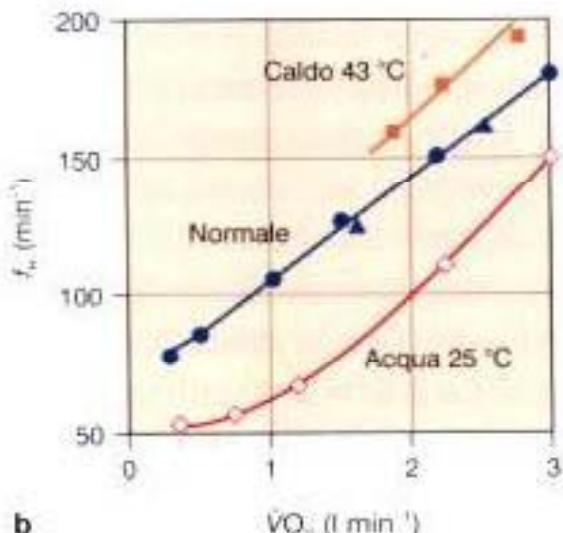


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Gettata cardiaca in funzione del consumo di ossigeno di stato stazionario in esercizi d'intensità crescente

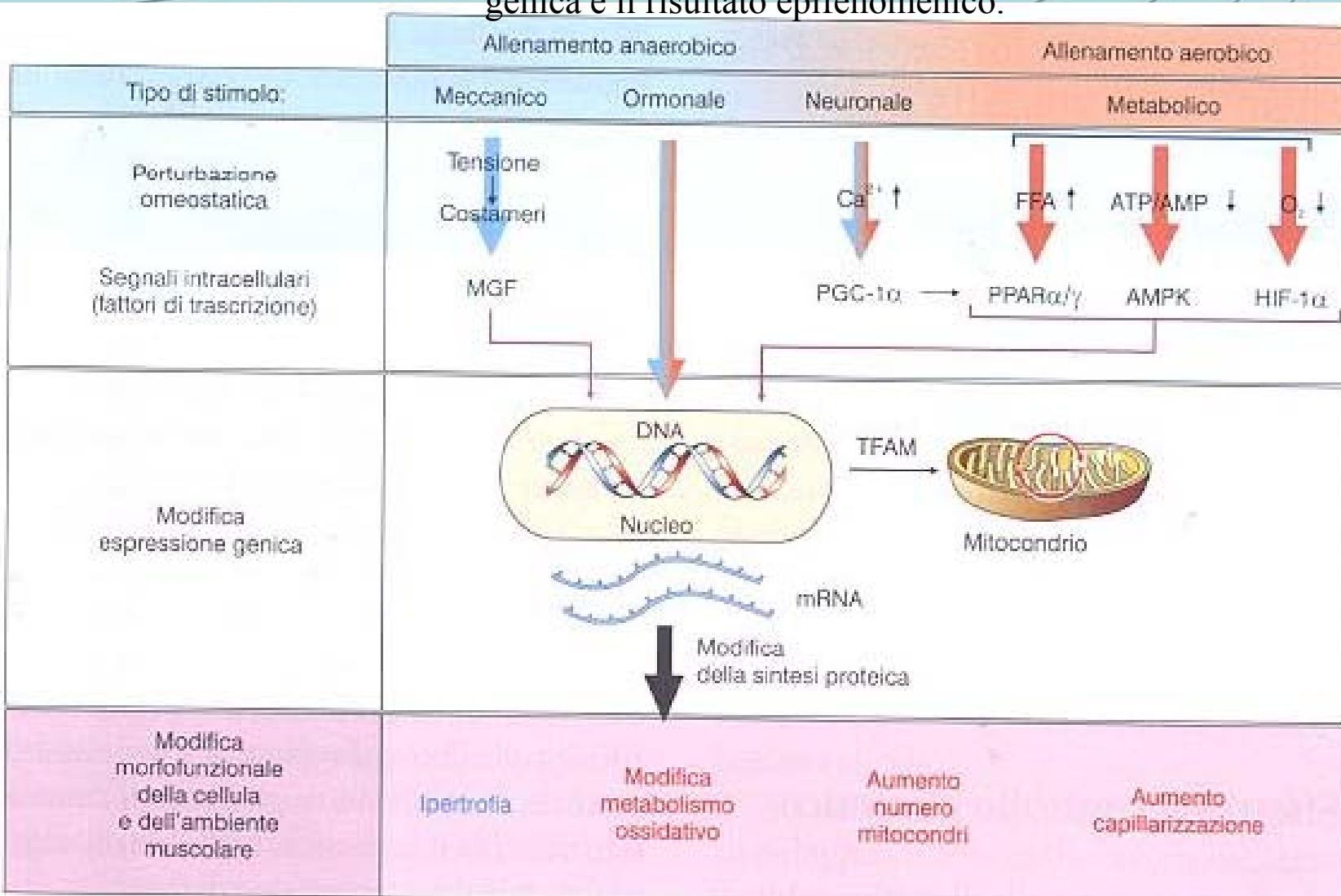


Dimostrazione delle risposte opposte compensatorie della frequenza cardiaca e della gettata pulsatoria



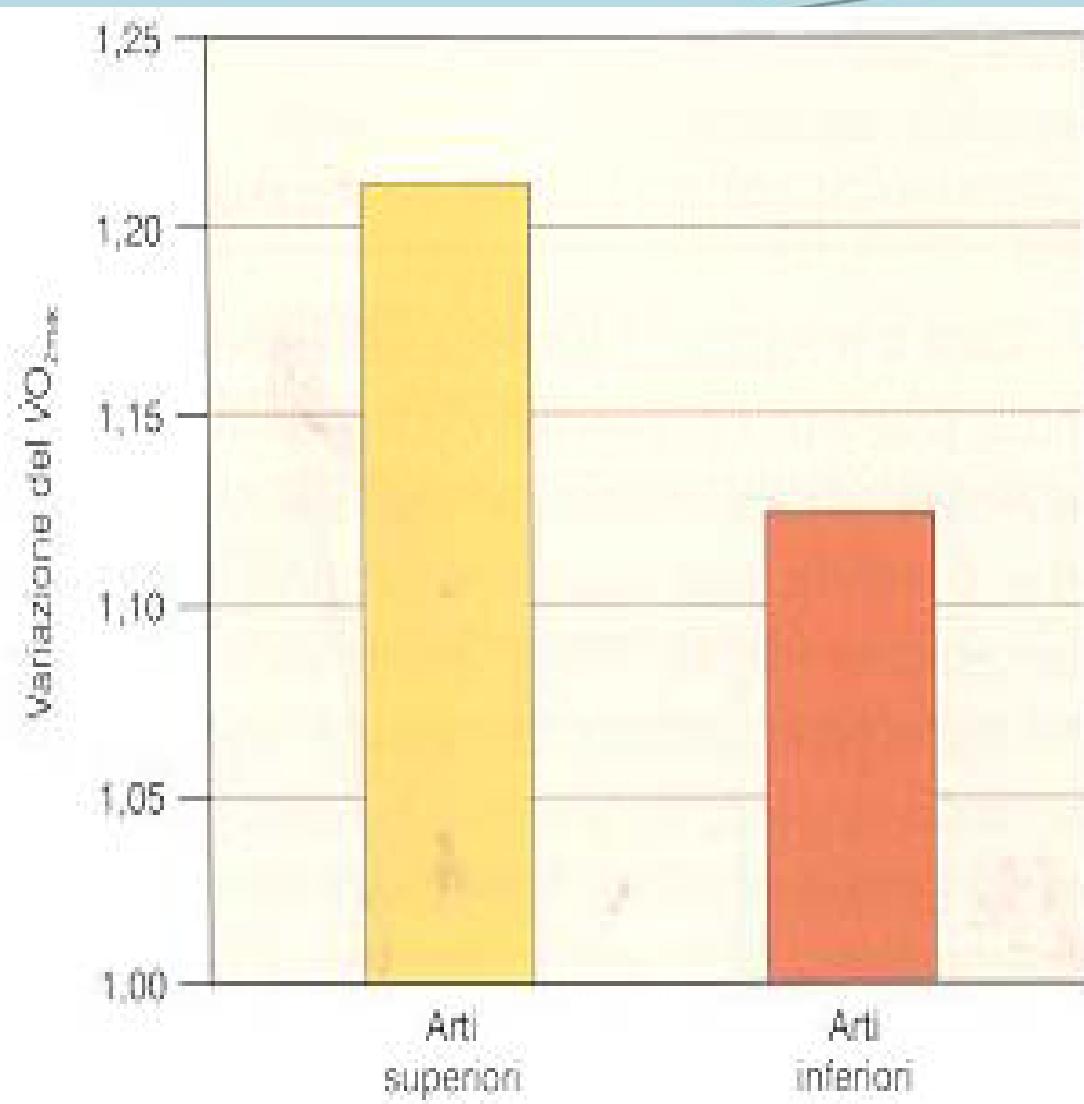
•Sintesi degli effetti molecolari dell'allenamento anaerobico (1) e aerobico (2)

Le frecce blu indicano un effetto specifico del primo, le frecce rosse del secondo. Per ogni stimolo sono indicati la perturbazione omeostatica, i fattori di trascrizione, gli effetti indotti sull'espressione genica e il risultato epifenomenico.



Tratto da Dagli Abissi Allo Spazio, Guido Ferretti – Carlo Capelli. Edi- ermes.

Effetti di sei settimane di allenamento aerobico specifico degli arti superiori e degli arti inferiori di intensità relativa equivalente al rispettivo massimo consumo di ossigeno.

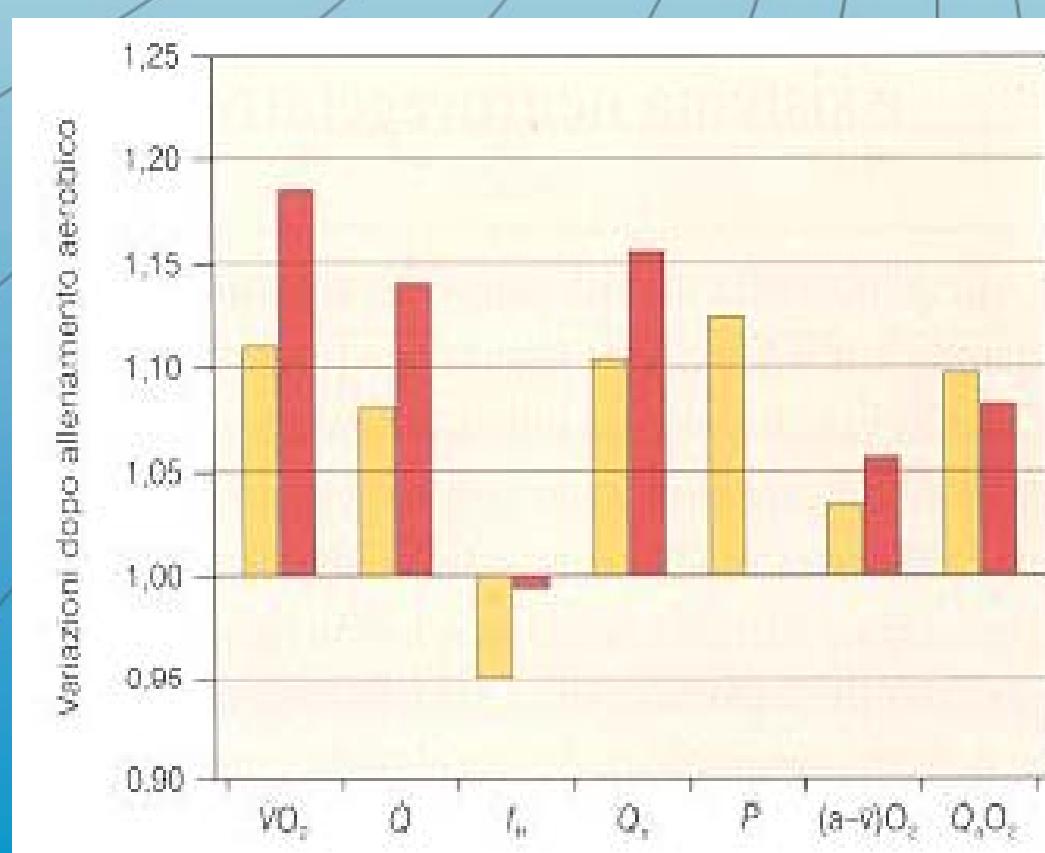


I dati sono espressi come rapporto tra il valore misurato alla fine e quello misurato prima del periodo di allenamento.

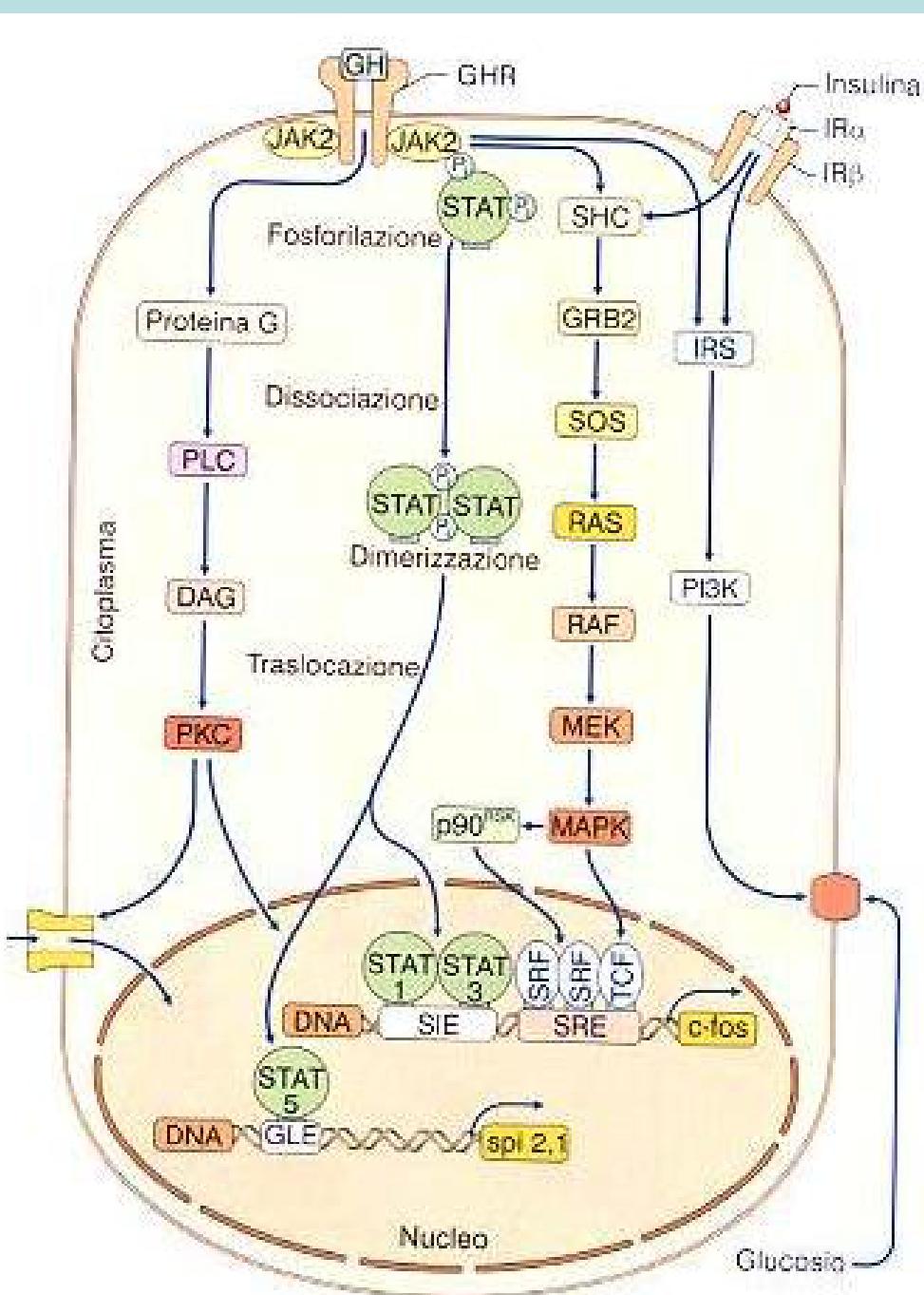
- Dati tratti da Turner et al. (1997). Effects of endurance training on oxidative capacity and structural composition of human arm and leg muscles. Acta Physiol scand 161:459-464.

Effetti allenamento aerobico

- Dati tratti da Ekblom B. et al., (1968) Effect of training on circulatory response to exercise. J Appl Physiol 25:619-625 istogrammi gialli e da Saltin et al., (1968) – istogrammi rossi.

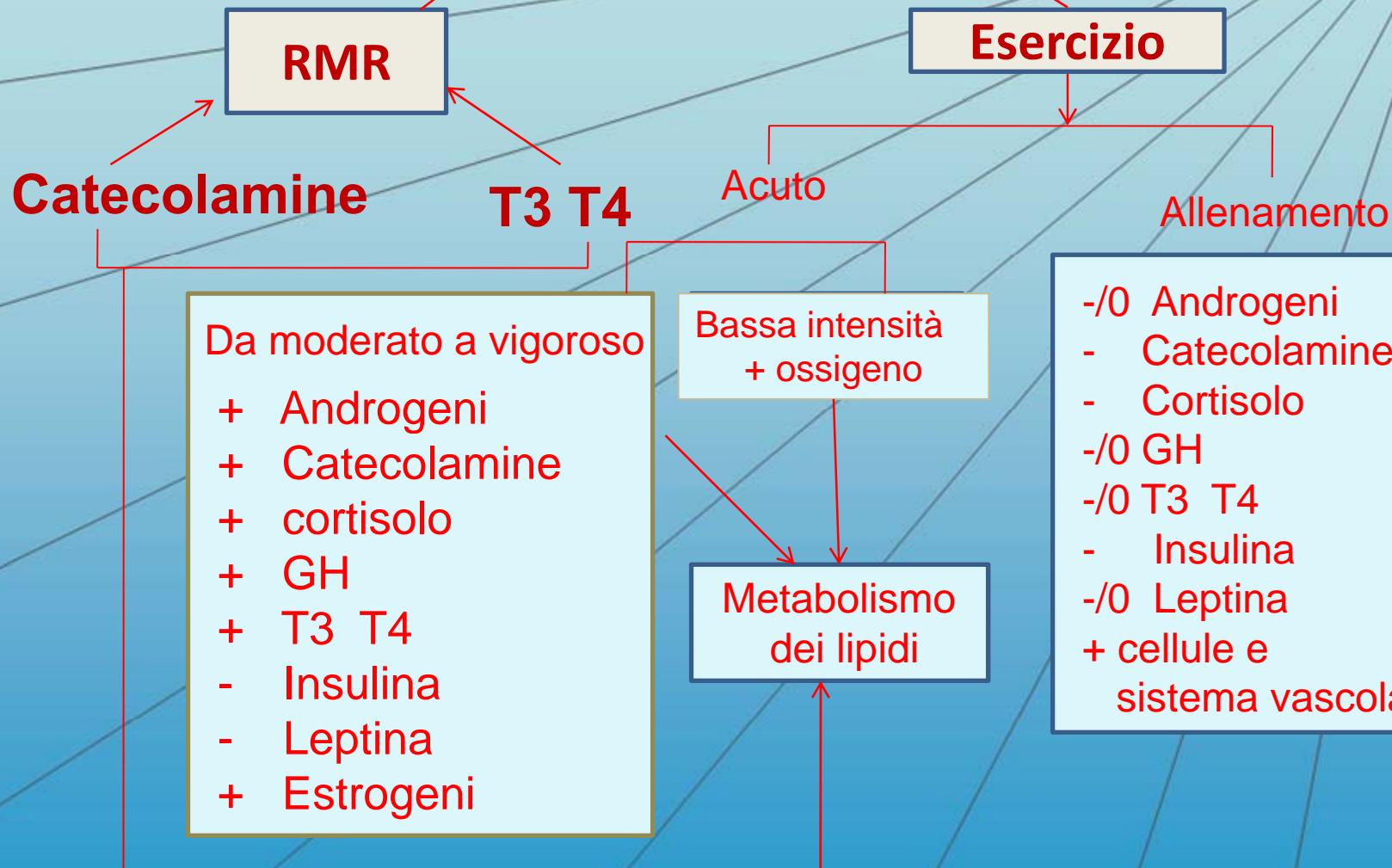


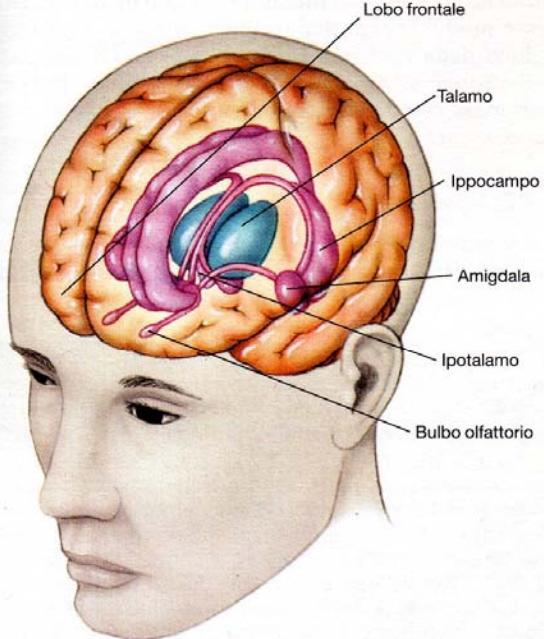
Schema dei meccanismi d'azione intracellulare dell'ormone della crescita (GH)



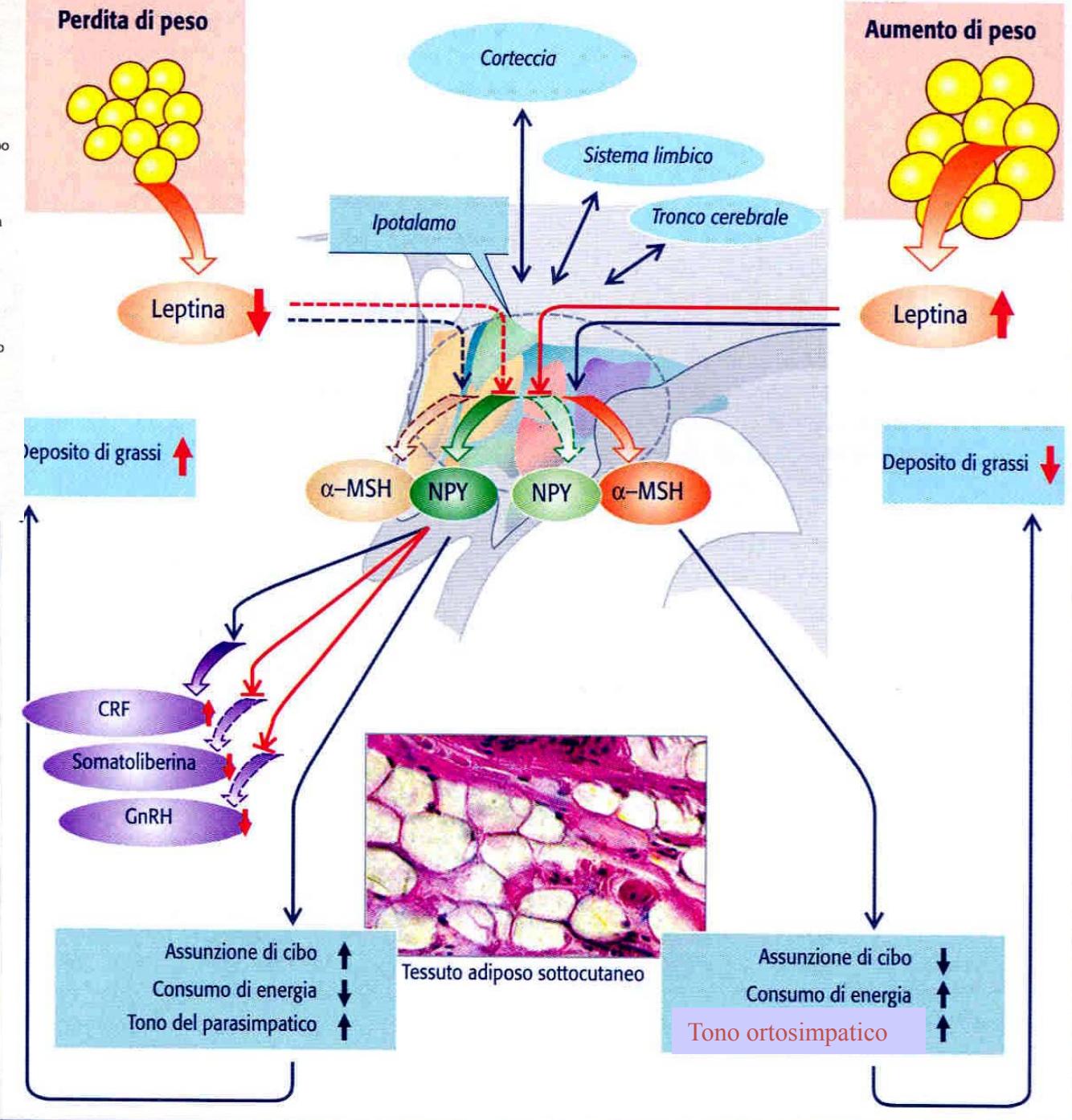
I meccanismi d'azione intracellulare dell'ormone della crescita (growth hormone, GH), il legame dell'ormone con il recettore (GHR) determina attivazione della JAK2, chinasi della famiglia Janus, che a propria volta, fosforila le proteine STAT (trasduttori di segnale e attivatori della trascrizione), ovvero dà origine alla cascata intracellulare della proteinchinasi attivata dai mitogeni (*mitogen-activated protein kinase*, MAPK), ovvero attiva la proteinchinasi C (PKC) o la fosfatidilinositol 3-chinasi (PI3K), DAG, diacilglicerolo; ERK, chinasi regolata da segnale extracellulare; GLE, sequenza attivata dall'interferone γ ; GRB, proteina legante il recettore per il fattore di crescita; GLUT, trasportatore del glucosio; INS, insulina; IR, α e β subunità del recettore dell'insulina; MEK, MAPK-ERK chinasi; PLC, fosfolipasi C; SIE, elemento sis-inducibile; SON, son of sevenless; SRE, elemento di risposta al siero; SRF, fattore di risposta sierico; TCF, fattore complesso ternario (modificata da DeGroot, 2006).

Dispendio energetico

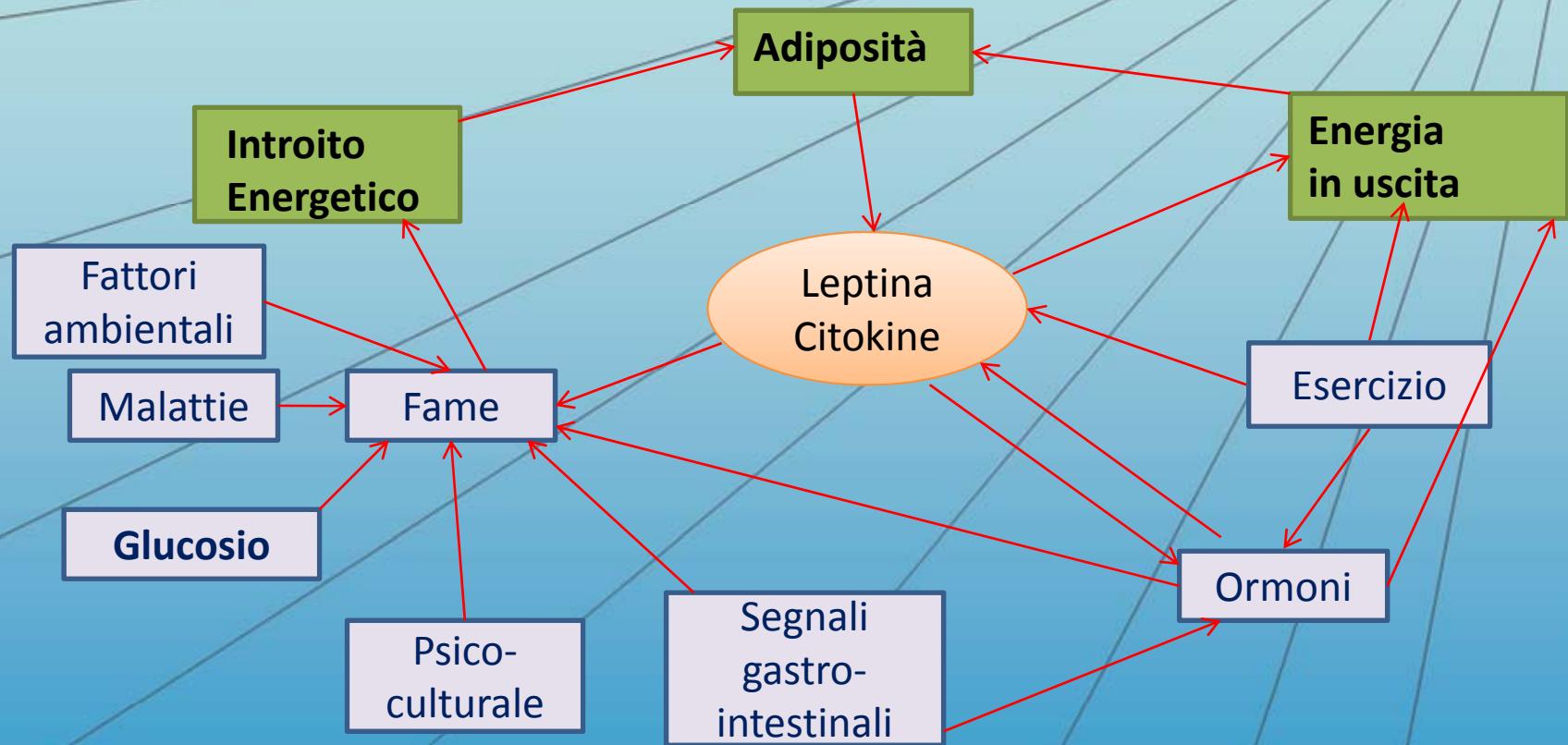




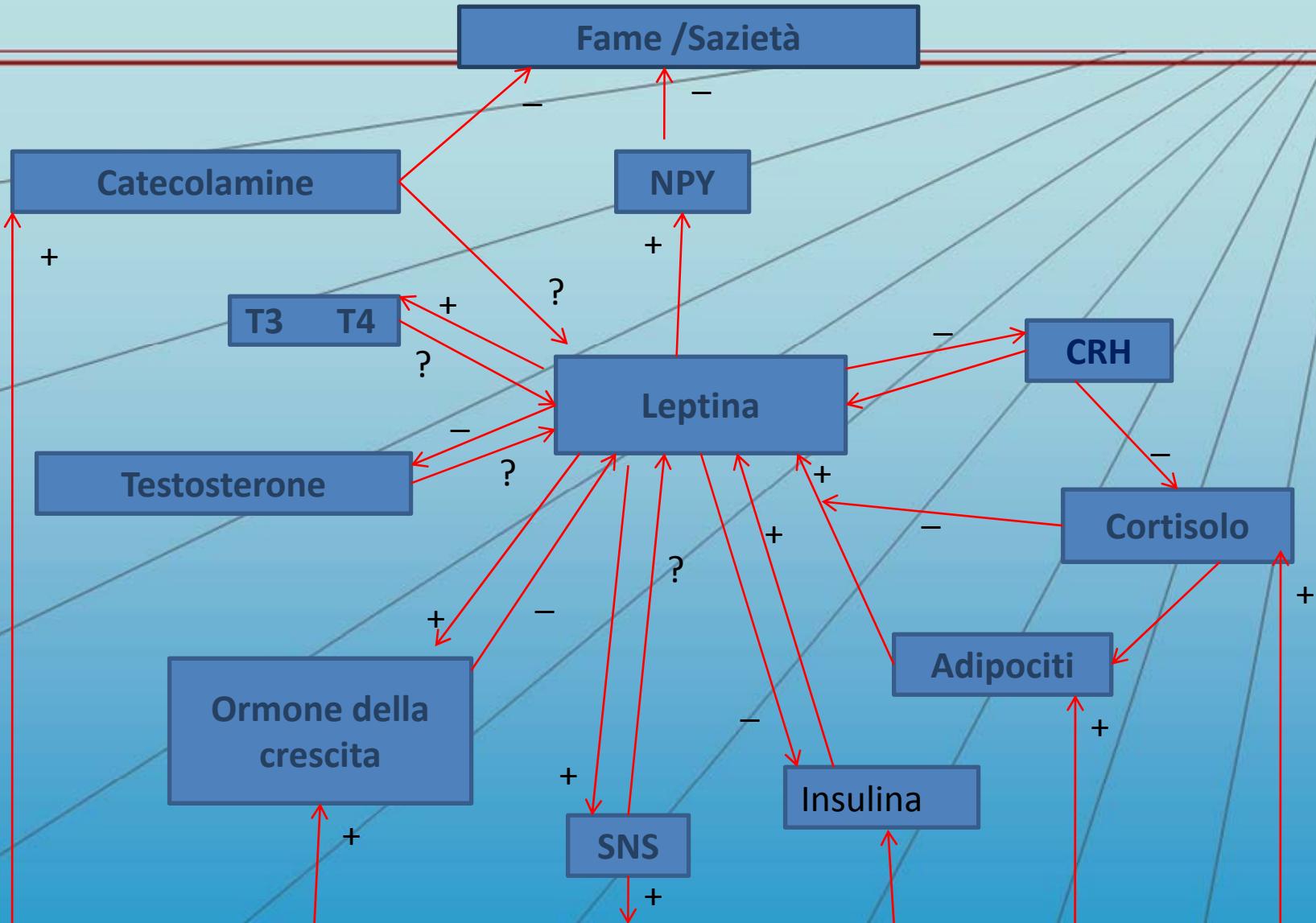
La Teoria Lipostatica:
regolazione della
massa corporea
mediante
Leptina (145
AA),
alpha-MSH e
NPY



Schema generale dei fattori che influenzano l'ingresso e la liberazione di energia e la loro relazione con la quantità di grasso corporeo



Relazione fra ormoni di regolazione del Metabolismo, Leptina e Fame



CRH= Corticotropina NOY= Neuropeptide-Y SNS= Sistema nervoso simpatico

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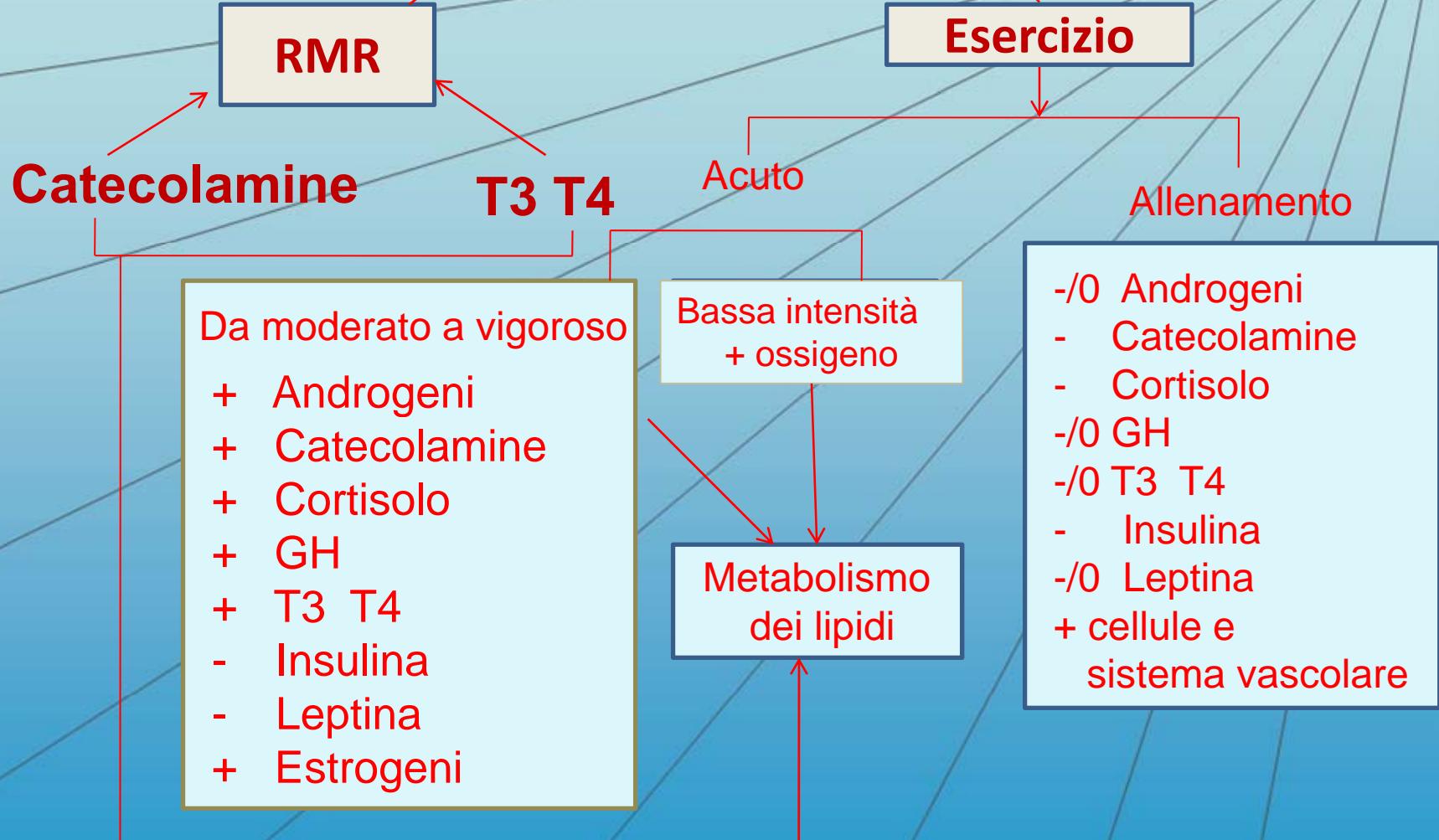


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Analisi dei Campioni Ematici

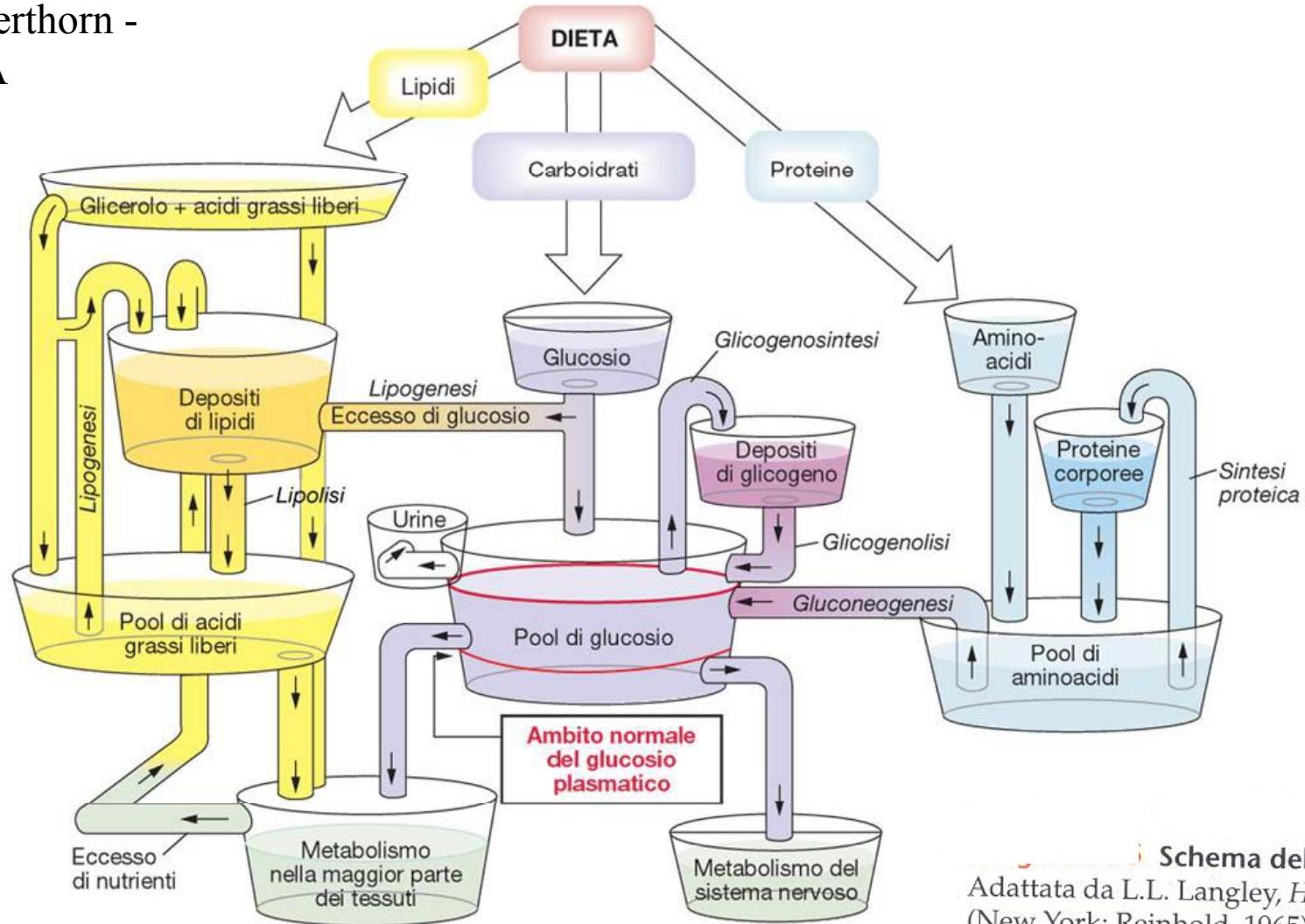
- Dopo centrifugazione per 15 min. a 3000 rpm, i campioni di siero sono stati posti a temperatura di meno 20° C per essere successivamente valutati dopo averli riportati a temperatura fisiologica. I dosaggi sono stati effettuati in duplice. Le concentrazioni sieriche di GH sono state determinate mediante una metodica immunoradiometrica (IRMA GH, IMMUNOTECH SAS, Marseille Cedex, France). La sensibilità analitica è risultata <0.10 mIU/L e il coefficiente di variazione (CV) intra e inter dosaggio è risultato di 1.5% e 14.0% rispettivamente. Le concentrazioni sieriche di IGF-I sono state determinate utilizzando una metodica immunoradiometrica (IRMA IGF-1, IMMUNOTECH SAS, Marseille Cedex, France). La sensibilità analitica è risultata pari a 2 ng/ml, il coefficiente di variazione (CV) intra e inter dosaggio è risultato di 6.3 % e 6.8 % rispettivamente. Le concentrazioni sieriche di chemochine e citochine sono state determinate utilizzando il sistema di sospensione bio-plex (Bio-Rad). Le sostanze analizzate sono c-peptide, ghrelina, GLP-1, GIP, glucagone, insulina, leptina, PAI-1, resistina, visfatinina. La sensibilità analitica e il coefficiente di variazione (CV) intra e inter dosaggio sono risultati rispettivamente 14.5 pg/ml, 5% e 4% per il c-peptide, 1.2 pg/ml 4% e 2% per la Ghrelina, 5.3 pg/ml, 6% e 3% per GLP-1, 0.8 pg/ml, 3% e 4% per il GIP, 4.9 pg/ml, 5% e 6% per il glucagone, 1.0 pg/ml, 3% e 5% per l' insulina, 3.1 pg/ml, 3% e 4% per la leptina, 2.2 pg/ml, 5% e 4% per il PAI-1, 1.3 pg/ml, 3% e 4% per la resistina, 37.1 pg/ml, 4% e 3% per la visfatinina.

Dispendio energetico



La fabbrica metabolica e la Teoria Glicostatica per il Controllo Alimentare

Da
Silverthorn -
CEA



Schema del metabolismo
Adattata da L.L. Langley, *Homeostasis* (New York: Reinhold, 1965).

Metabolismo anaerobico lattacido

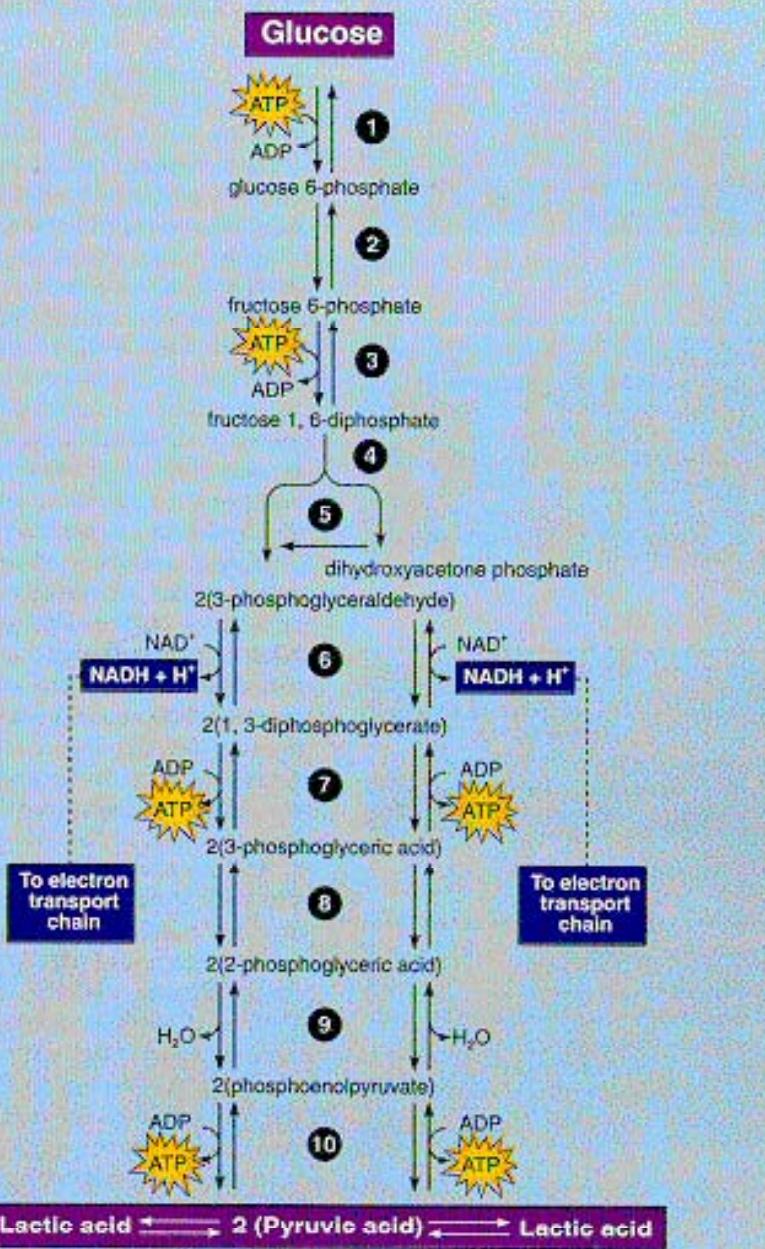


Figure 2-7. Glycolysis: a series of 10, enzymatically-controlled chemical reactions involving the anaerobic breakdown of glucose to two molecules of pyruvic acid. Lactic acid is formed when the oxidation of NADH does not keep pace with its formation in glycolysis.

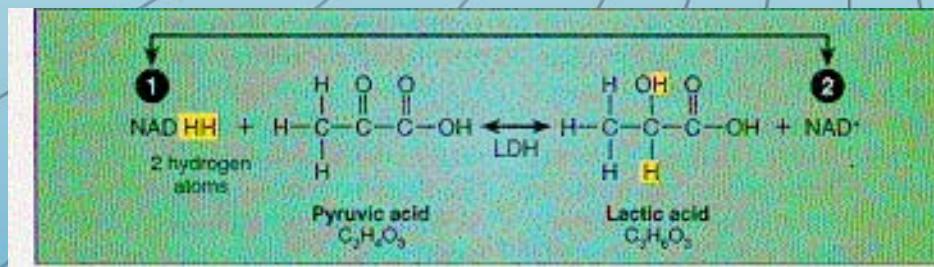


Figure 2-8. The formation of lactic acid occurs when excess hydrogens from NADH combine temporarily with pyruvic acid (1). This frees up NAD⁺ to accept additional hydrogens generated in glycolysis (2). LDH = lactate dehydrogenase.

Tratto da

McArdle, Katch, Katch: Essential of Exercise Physiology, 1994 - LEA & FEBIGER