

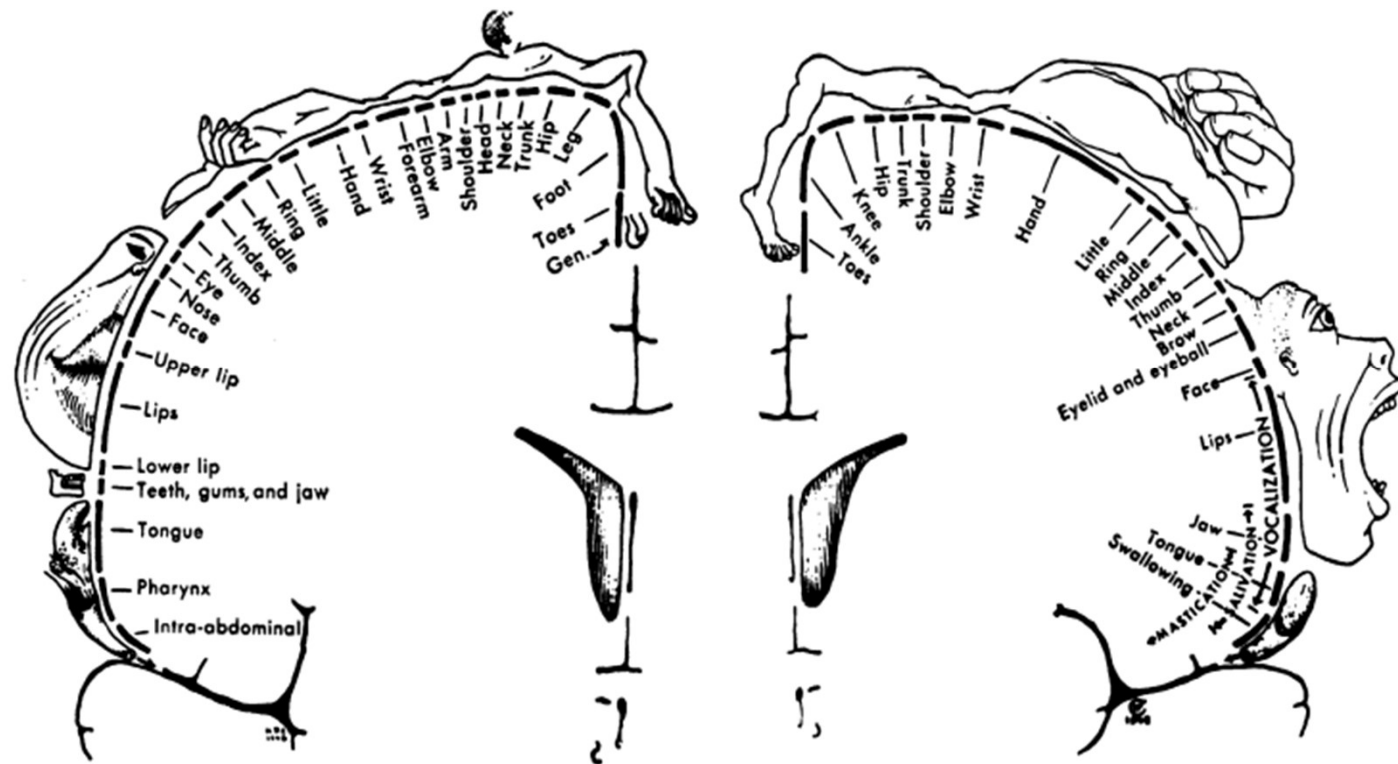
“FISIOLOGIA DELL’ESERCIZIO FISICO – IL METABOLISMO MUSCOLARE”



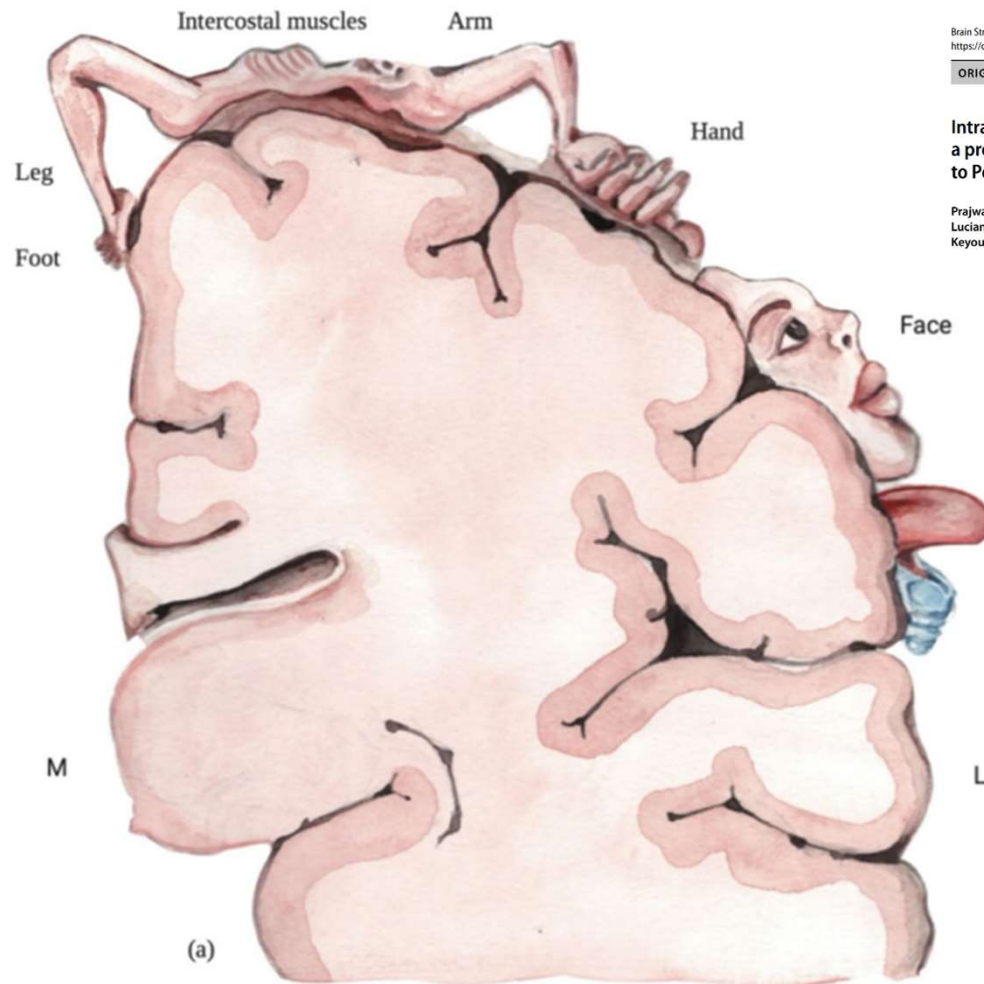
DOCT. MARCELLO TENCE
Medico Specialista in Medicina dello Sport

AREA MOTRICE PRIMARIA

Figure 2 The motor and sensory homunculus: the first map. Penfield and Rasmussen, 1950. Reprinted with permission of Macmillan Publishing Company from *The cerebral cortex of man* by Wilder Penfield and Theodore Rasmussen. Copyright 1950 Macmillan Publishing Company; copyright renewed 1978 Theodore Rasmussen.



AREA MOTRICE PRIMARIA



Brain Structure and Function (2021) 226:1601–1611
<https://doi.org/10.1007/s00429-021-02274-z>

ORIGINAL ARTICLE



**Intraoperative mapping of pre-central motor cortex and subcortex:
a proposal for supplemental cortical and novel subcortical maps
to Penfield's motor homunculus**

Prajwal Ghimire¹ · Jose Pedro Lavrador¹ · Asfand Baig Mirza¹ · Noemia Pereira³ · Hannah Keeble³ · Marco Borri² · Luciano Furlanetti¹ · Christian Brogna¹ · Jozef Jarosz² · Richard Gullan¹ · Francesco Vergani¹ · Ranjeev Bhangoo¹ · Keyoumars Ashkan¹

Primary Motor Cortex

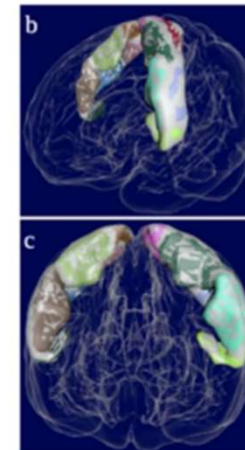
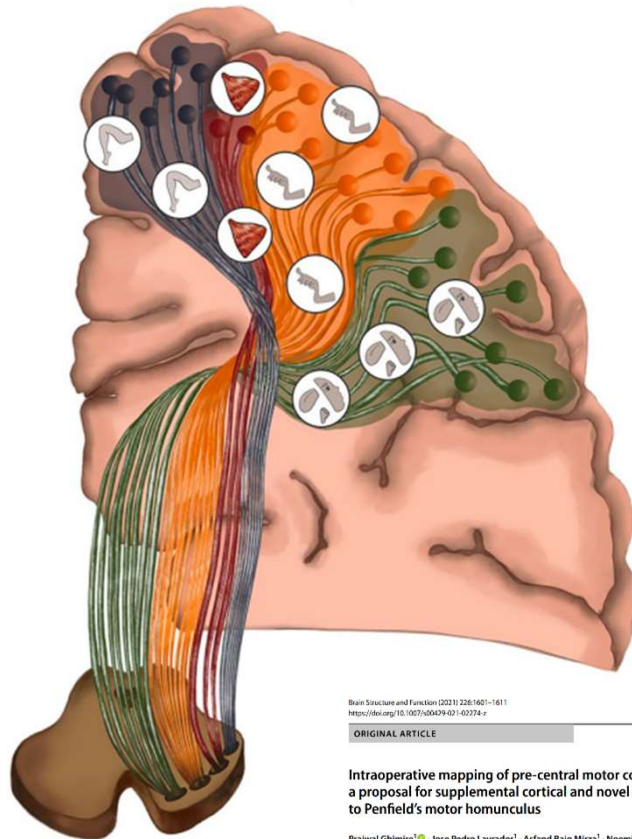
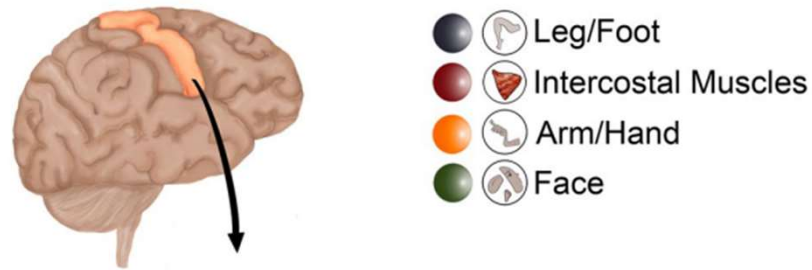


Fig. 3 Proposed supplemental cortical motor homunculus including the cortical somatotopy of intercostal muscles: **a** cortical motor representation. **b, c** Primary motor cortex reconstructed with Mesh-

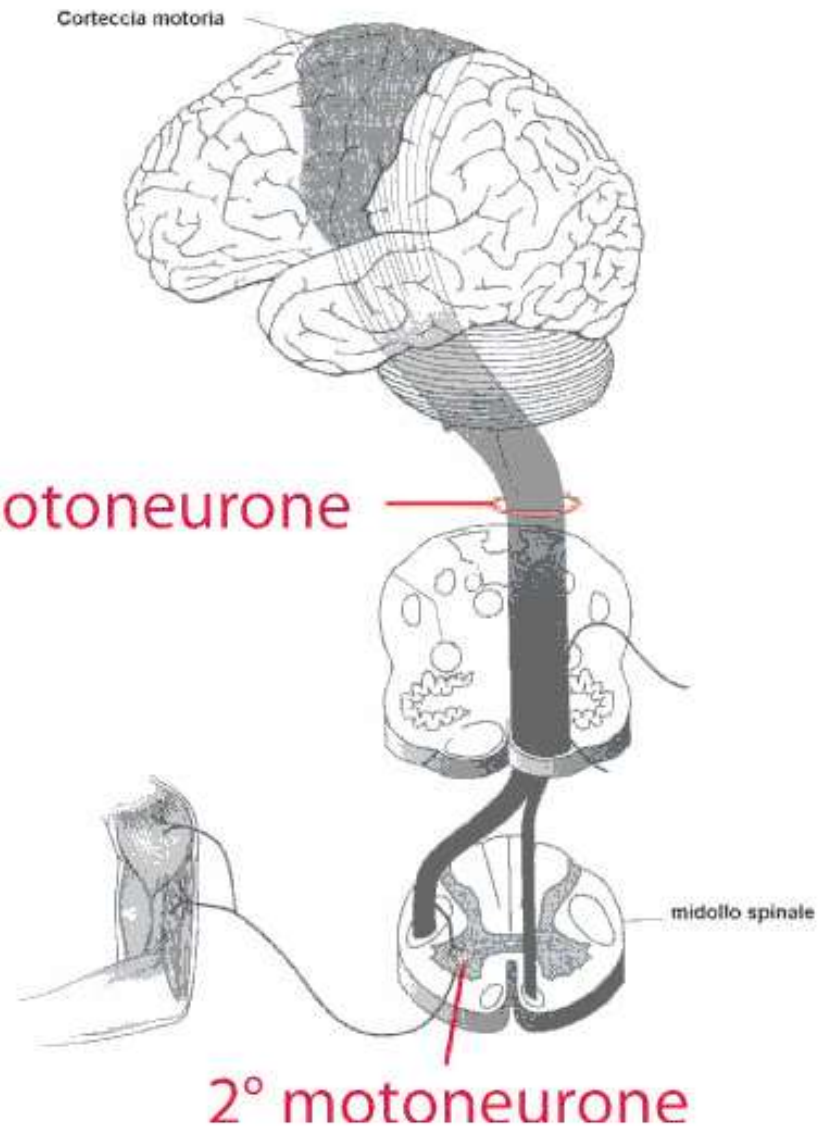
lab opensource software (Cignoni et al. 2008) with the data available from The Human Brainnectome Atlas (Fan et al. 2016)

AREA MOTRICE PRIMARIA

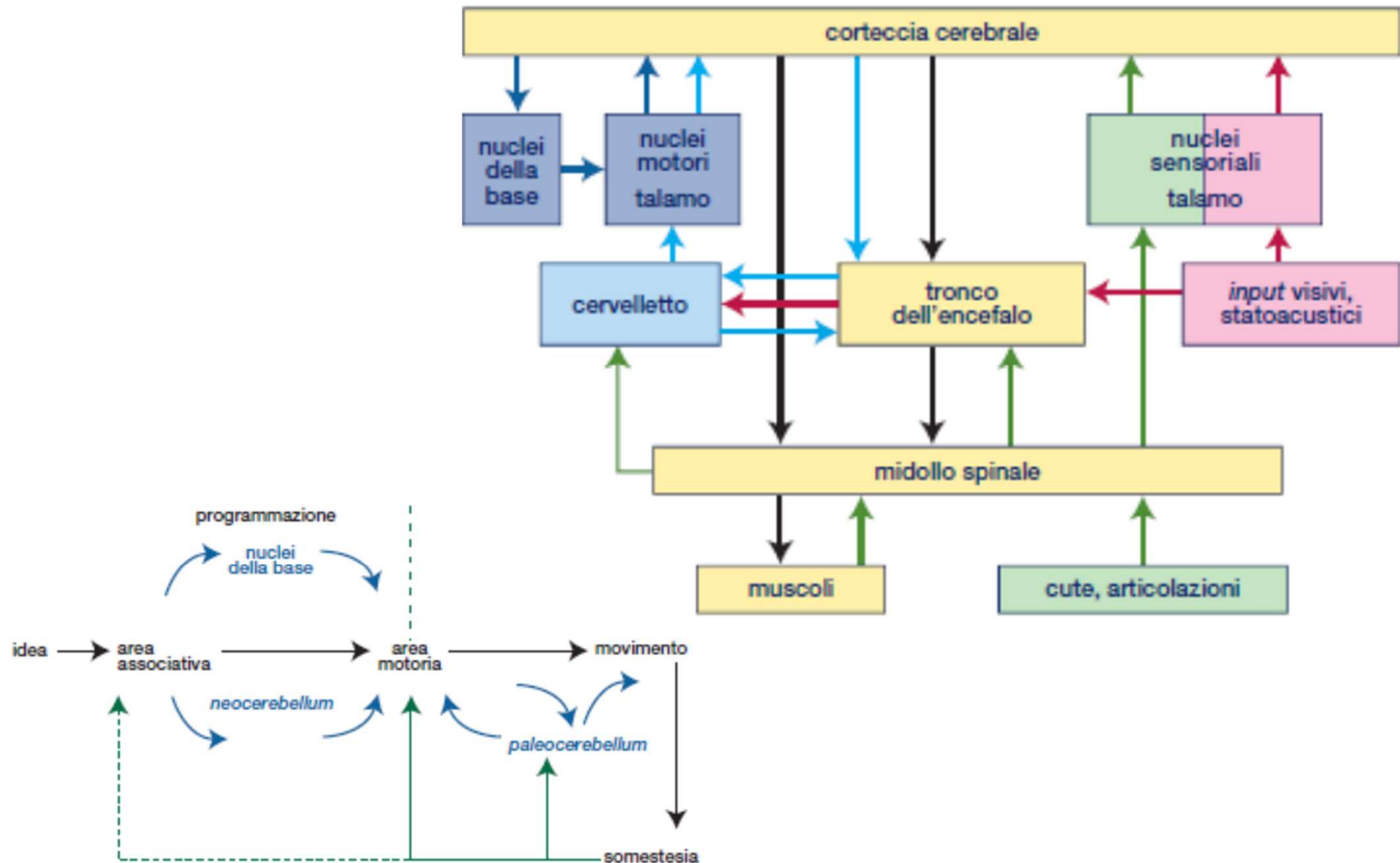


Intraoperative mapping of pre-central motor cortex and subcortex:
a proposal for supplemental cortical and novel subcortical maps
to Penfield's motor homunculus

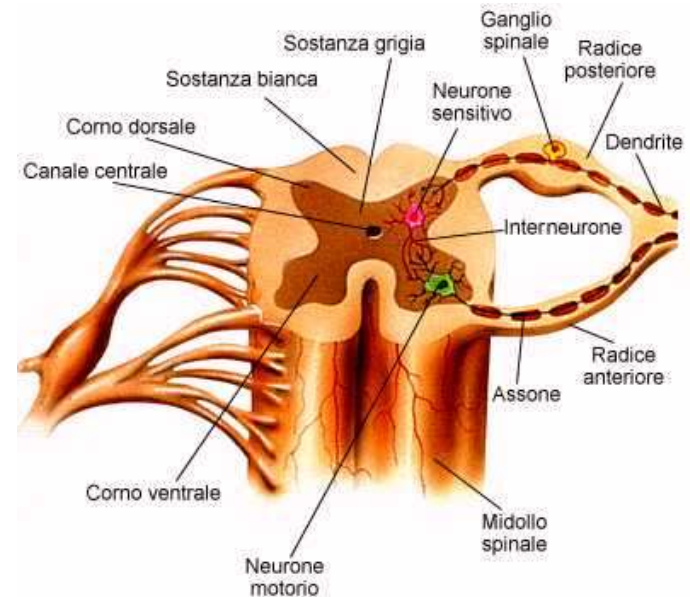
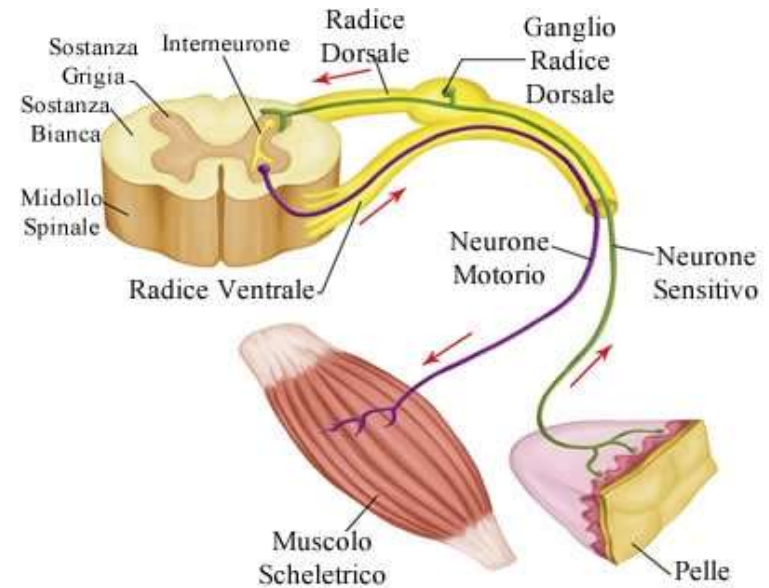
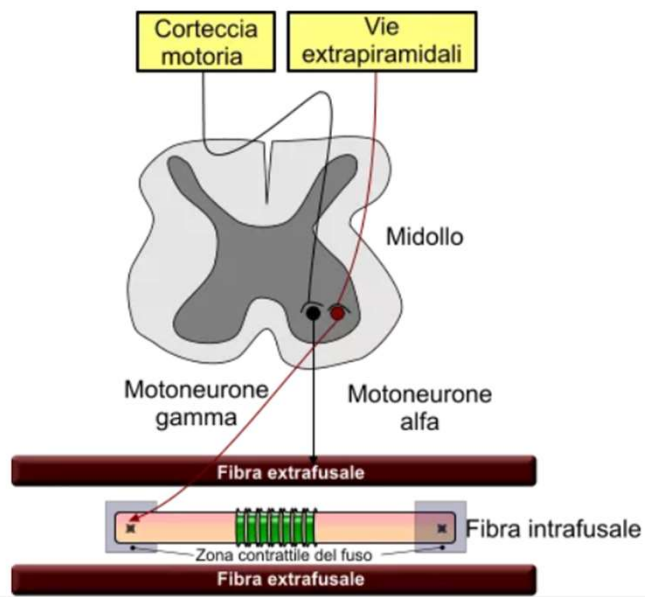
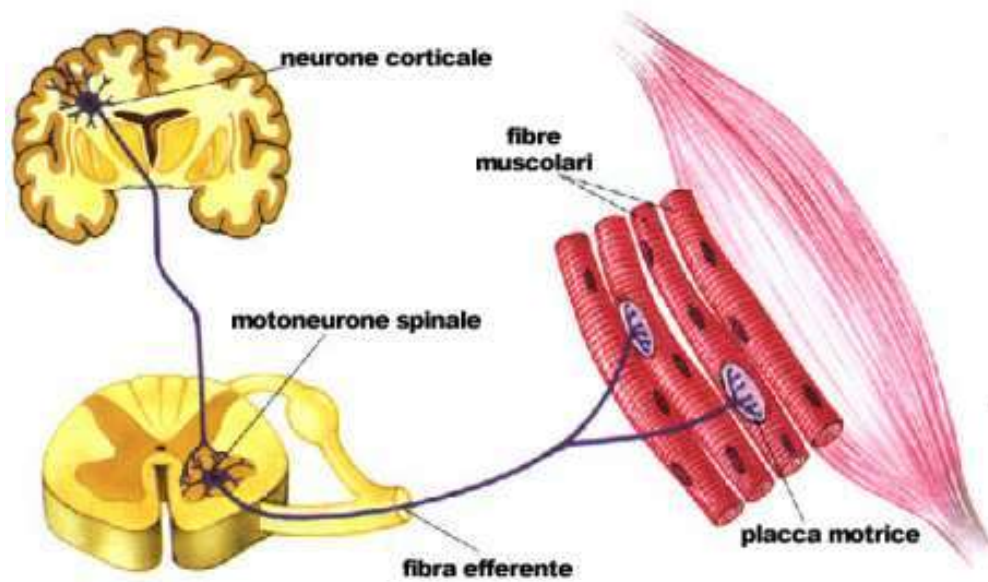
Prajwal Ghimire¹ · Jose Pedro Lavrador¹ · Asfand Baig Mirza¹ · Noemia Pereira² · Hannah Keeble³ · Marco Borri² ·
Luciano Furlanetti¹ · Christian Brogna¹ · Jozef Jarosz² · Richard Gullan¹ · Francesco Vergani¹ · Ranjeev Bhargoo¹ ·
Keyumars Ashkan¹



CONTROLLO DEL MOVIMENTO



1° e 2° MOTONEURONE



2° MOTONEURONE

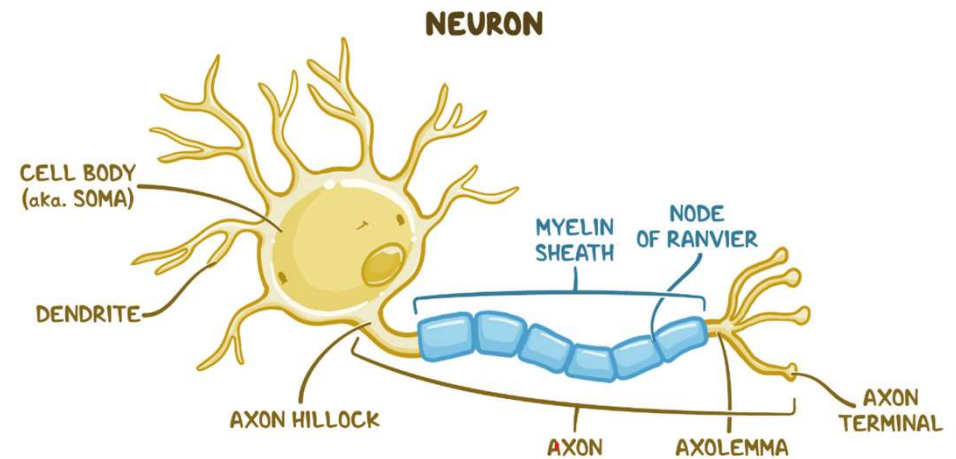
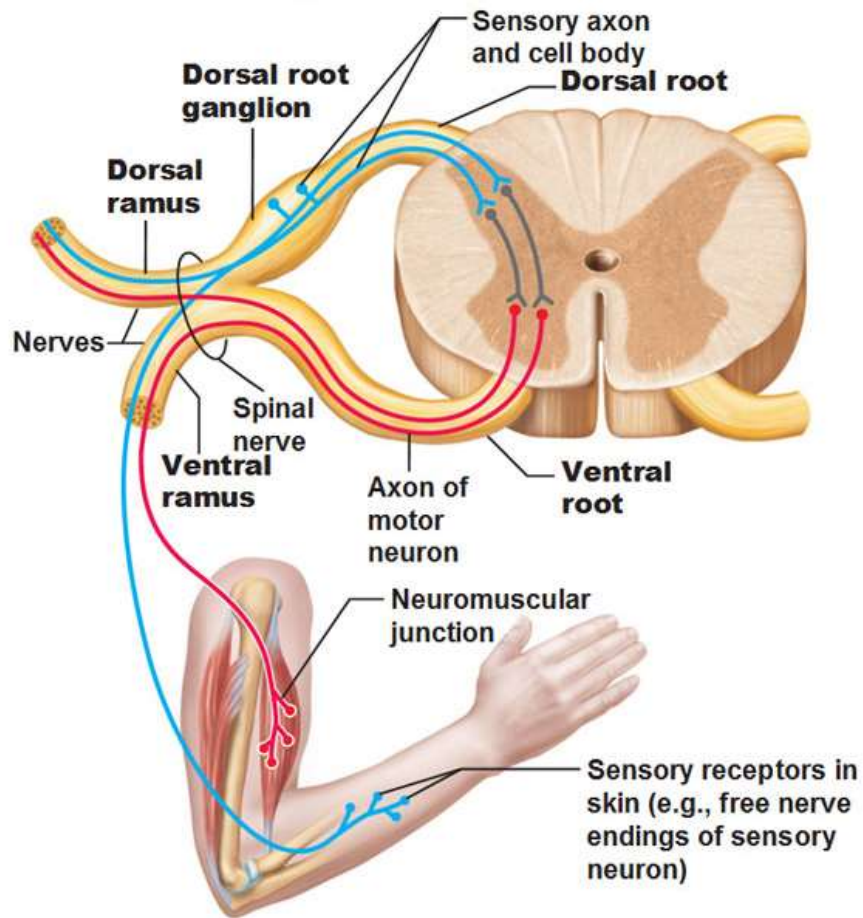
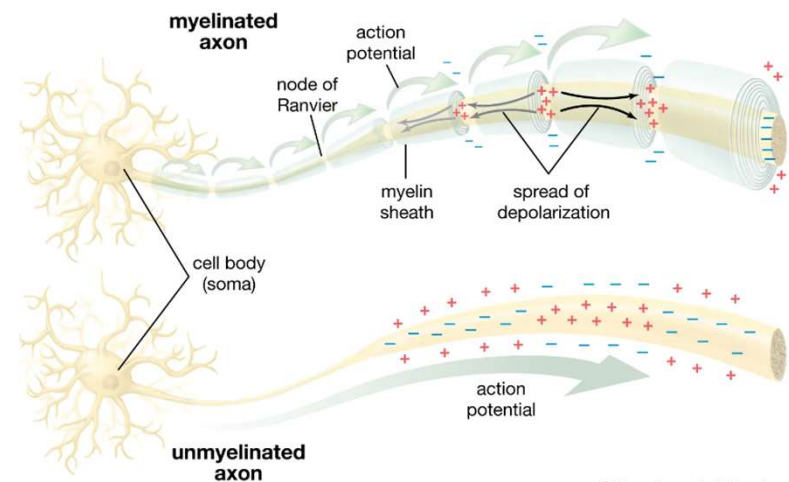
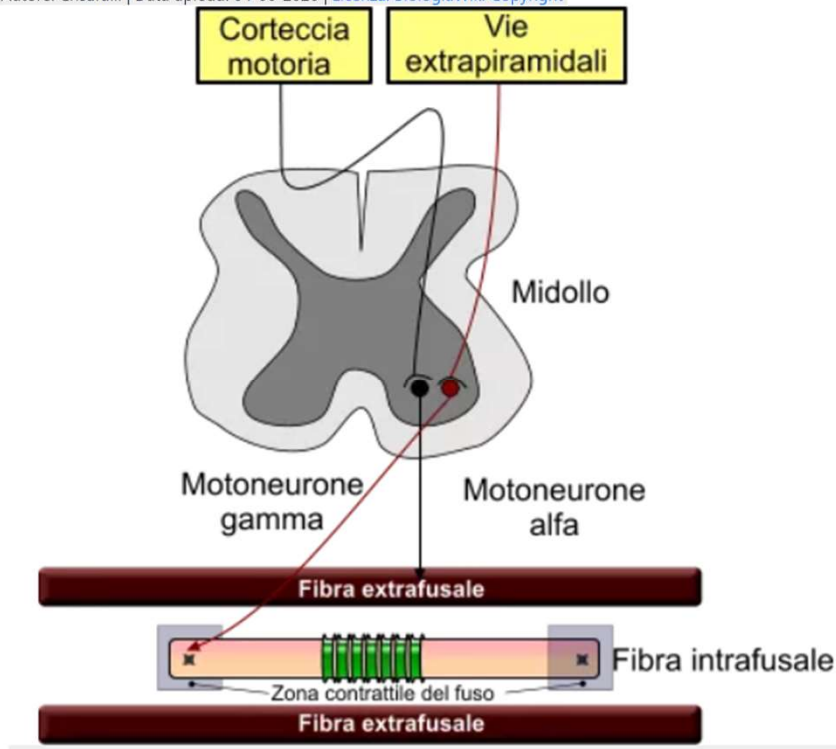


Figure 50.2 Structure of a neuron.



CONDUZIONE SALTATORIA

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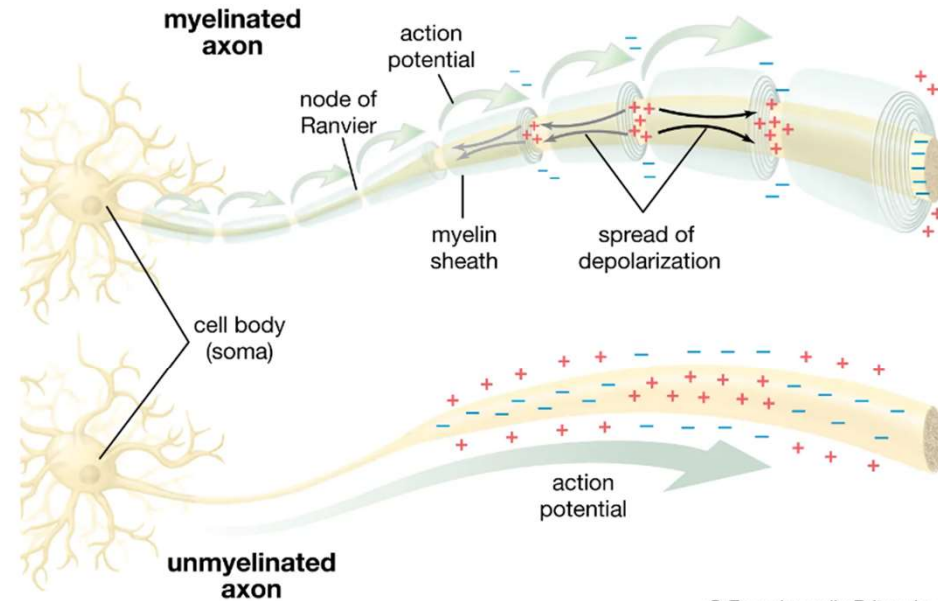


I **motoneuroni alfa** (α) sono i più grandi del corpo umano e per questo presentano la velocità di conduzione del segnale nervoso più elevata, con un valore medio di circa 90 metri al secondo; essi sono specializzati nella conduzione del segnale nervoso alle cellule muscolari scheletriche.

I **motoneuroni gamma** (γ) invece sono più piccoli e per questo possiedono una velocità di trasmissione del segnale nervoso inferiore, con un valore medio di circa 25 metri al secondo. Questi ultimi neuroni sono coinvolti nel riflesso miotatico generato dai **fusi neuromuscolari** (che analizzeremo a breve), in quanto svolgono una funzione di innervazione delle fibre intrafusali di questo fondamentale recettore muscolare.

RIFLESSO DA STIRAMENTO O MIOTATICO -> FUSO NEUROMUSCOLARE

RIFLESSO MIOTATICO INVERSO -> ORGANO TENDINEO DEL GOLGI



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<https://www.projectinvictus.it/motoneurone/>

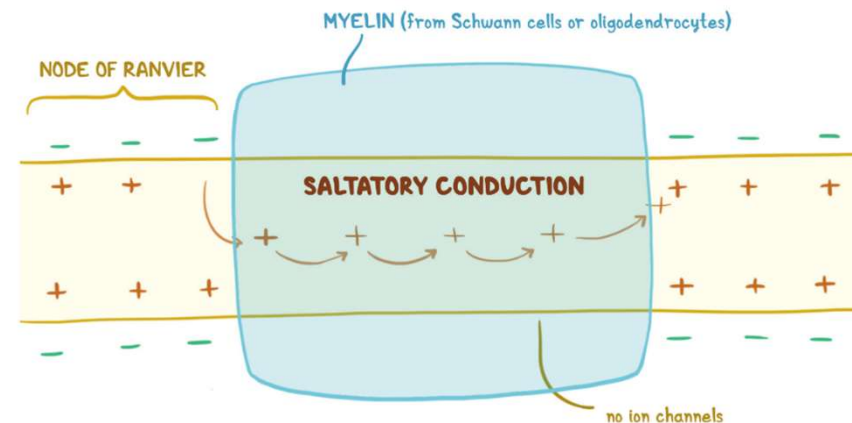


Figure 50.15 Saltatory conduction through myelinated areas of an axon increases the speed of signal conduction down the axon.

POTENZIALE D'AZIONE

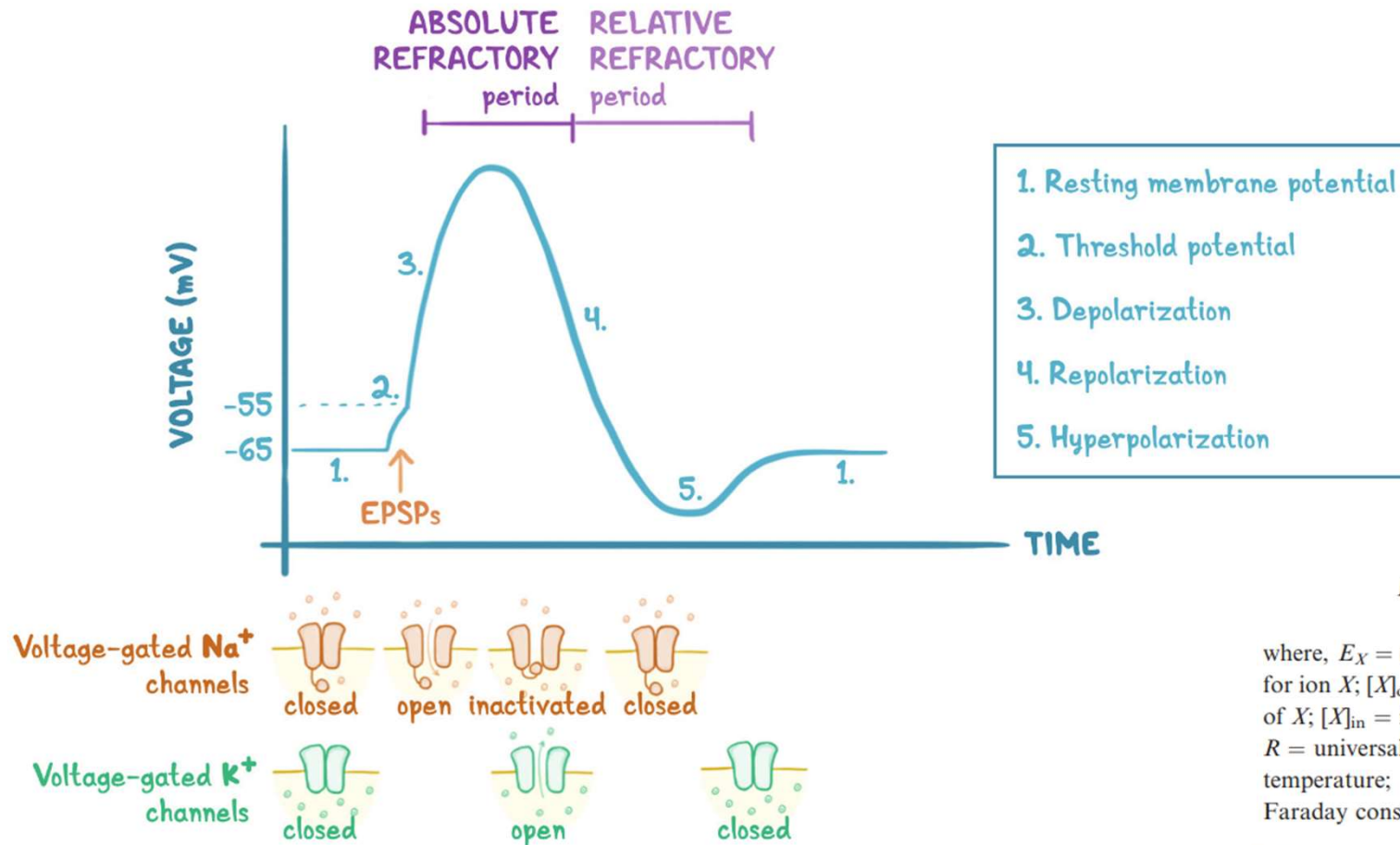


Figure 50.14 Graphical summary of the voltage changes that occur during a neuron action potential and the accompanying states of voltage-gated sodium and potassium channels. The action potential is initiated by a net influx of excitatory postsynaptic potentials (EPSPs). Not shown above are sodium/potassium pumps, which help to maintain the resting membrane potential, as well as help to return to that resting potential through repolarization.

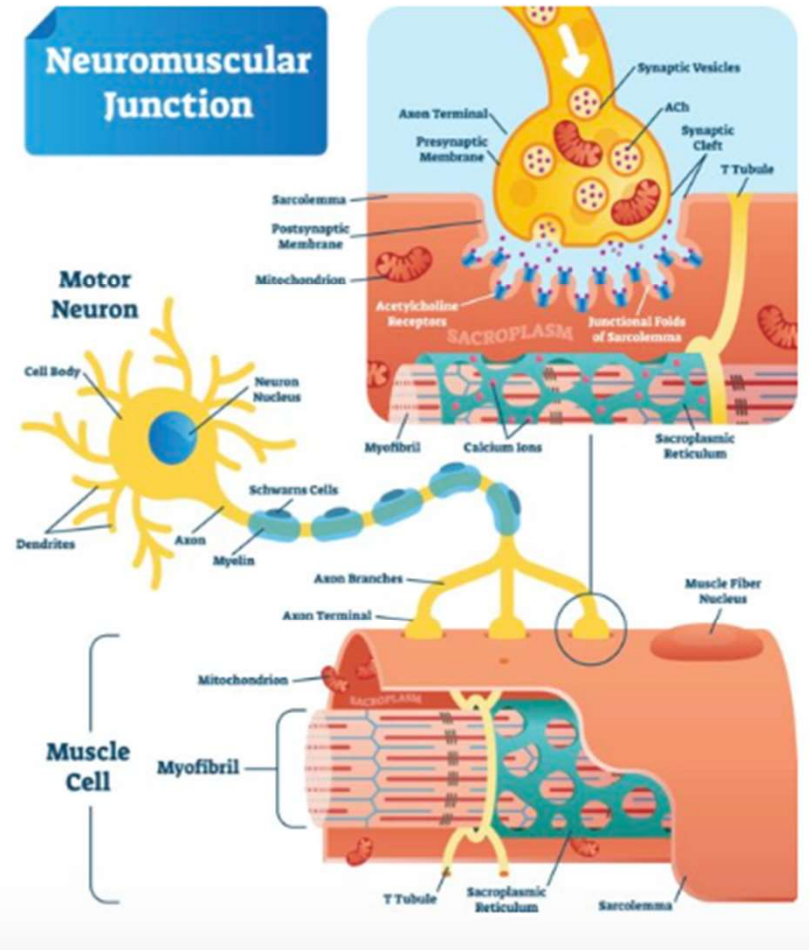
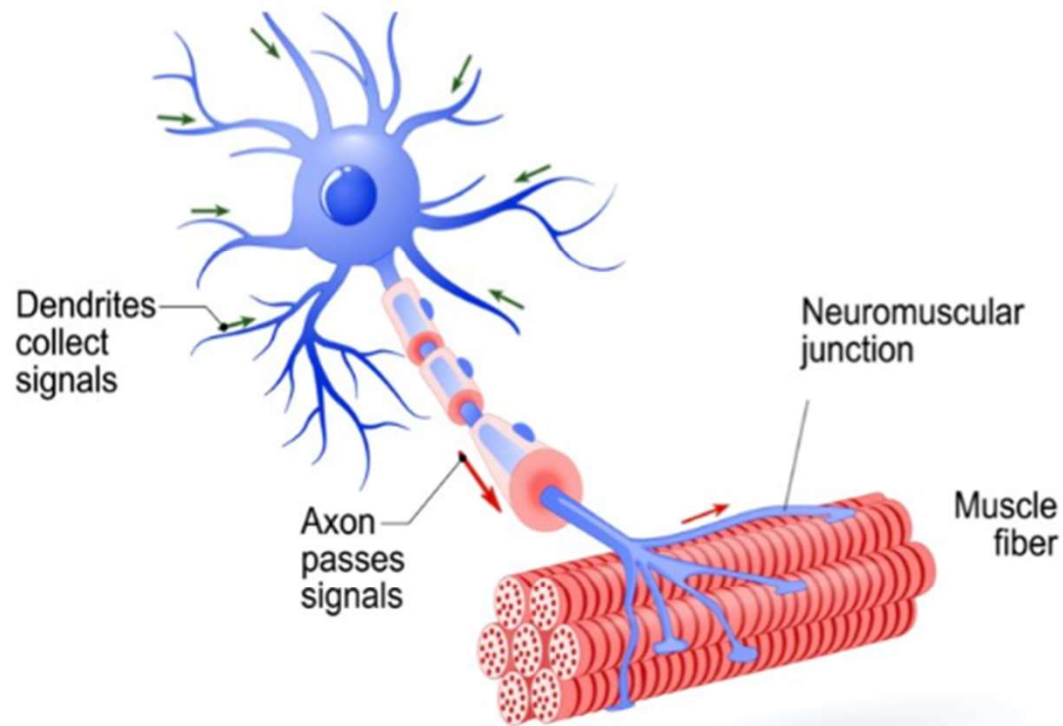
$$E_X = \frac{RT}{zF} \ln \frac{[X]_{\text{out}}}{[X]_{\text{in}}}, \quad \text{Eq. 1}$$

where, E_X = equilibrium or Nernst potential for ion X ; $[X]_{\text{out}}$ = extracellular concentration of X ; $[X]_{\text{in}}$ = intracellular concentration of X ; R = universal gas constant; T = absolute temperature; z = valency of ion; and F = Faraday constant.

Table 1 Plasma and cytoplasmic concentrations of various ions and the resulting Nernst potential (E_X)

Ion	Plasma (mM)	Cytoplasm (mM)	E_X (mV)
Na ⁺	145	12	+67
K ⁺	4	140	-95
Ca ²⁺	1.2	10 ⁻⁴	+125
Mg ²⁺	1.5	0.8	+8
Cl ⁻	115	4	-90

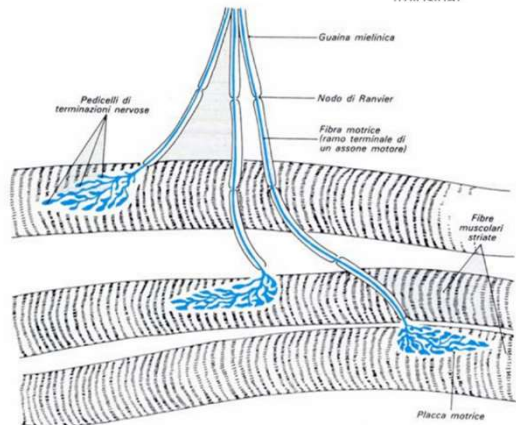
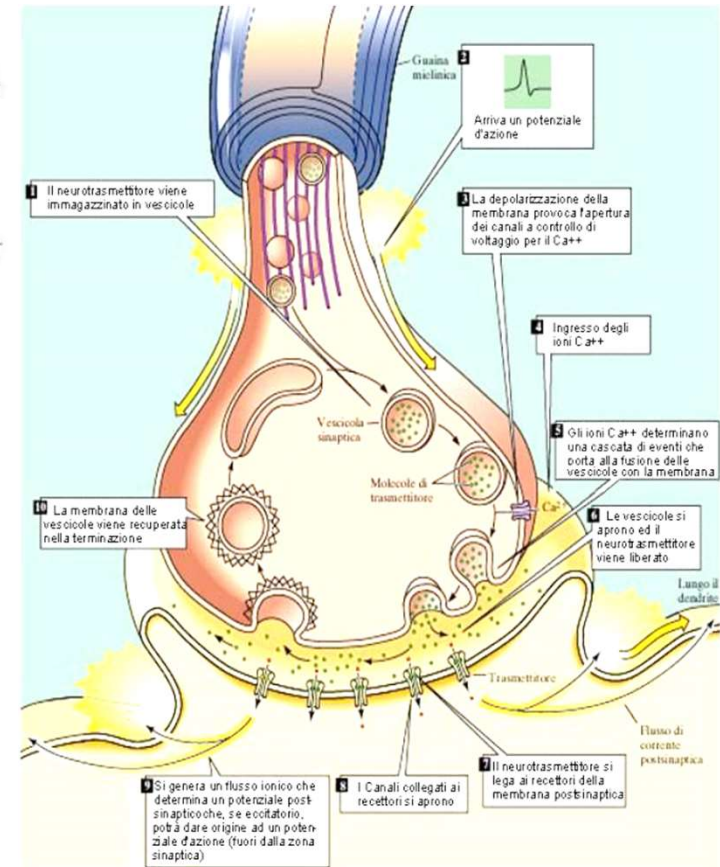
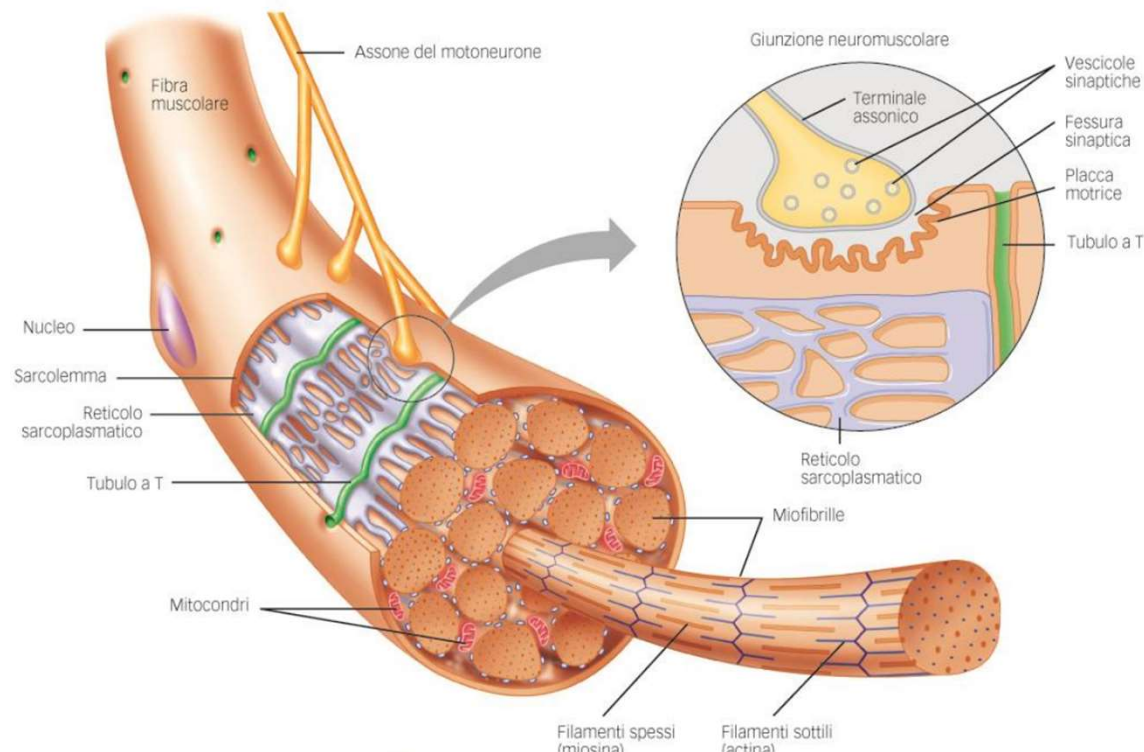
GIUNZIONE NEURO-MUSCOLARE



UNITÀ MOTORIA = MOTONEURONE + FIBRE MUSCOLARI INNERVATE

- MUSCOLI PICCOLI 10-20 FIBRE
- MUSCOLI GRANDI MIGLIAIA DI FIBRE

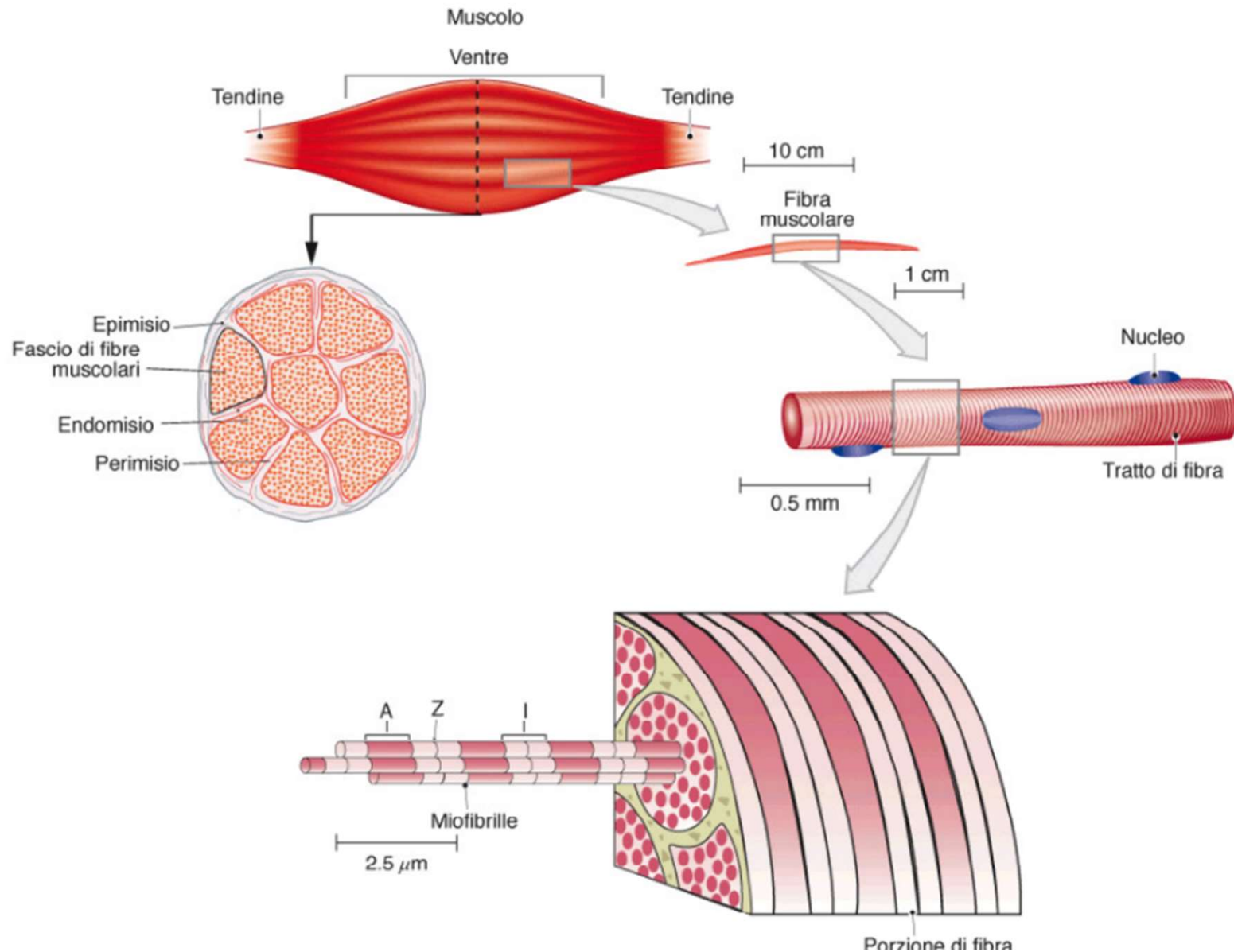
GIUNZIONE NEURO-MUSCOLARE



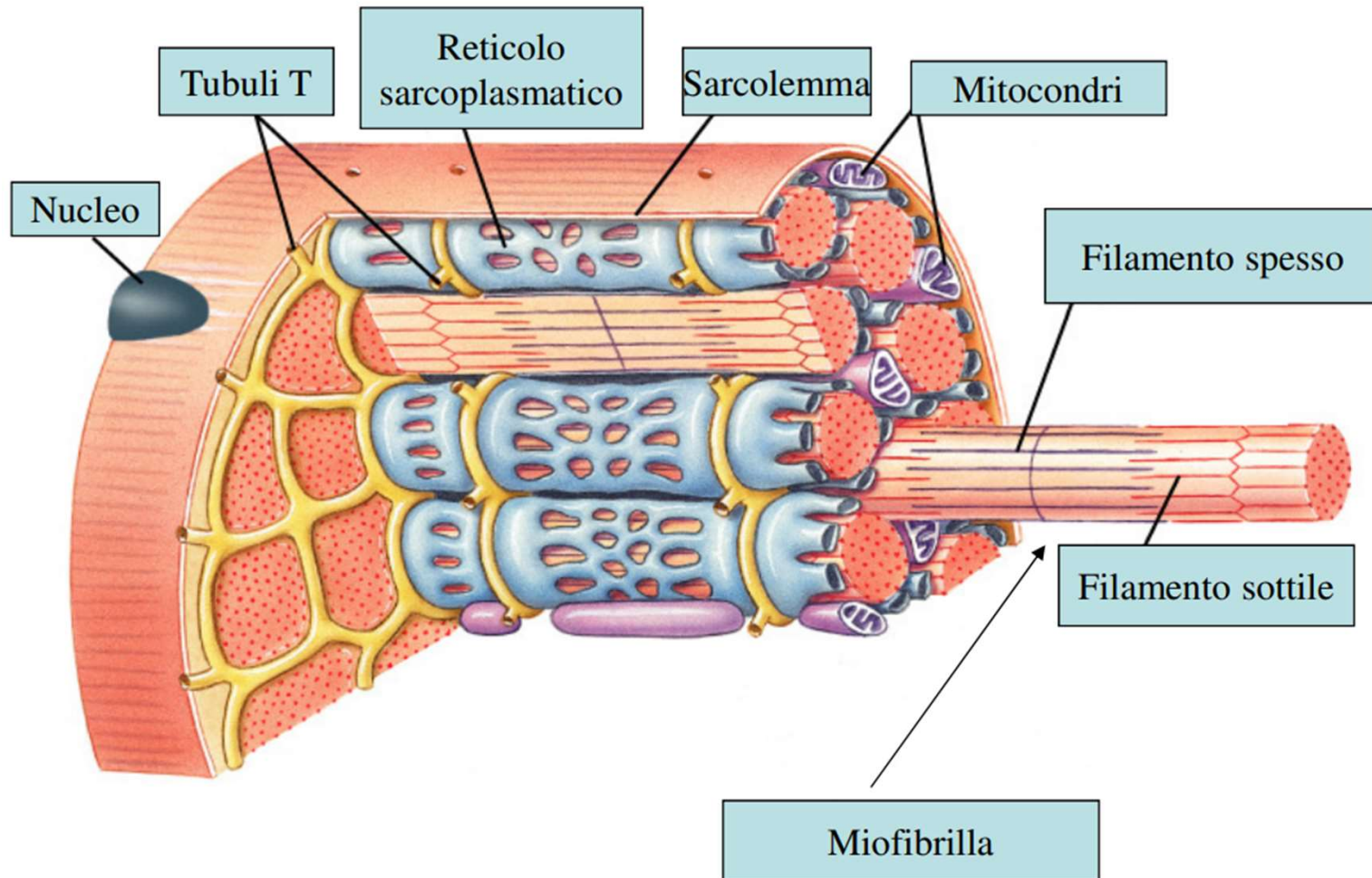
GIUNZIONE NEURO-MUSCOLARE o PLACCA MOTRICE

- NERVO PRE-SINAPTICO
- SPAZIO SINAPTICO
- MUSCOLO POST-SINAPTICO

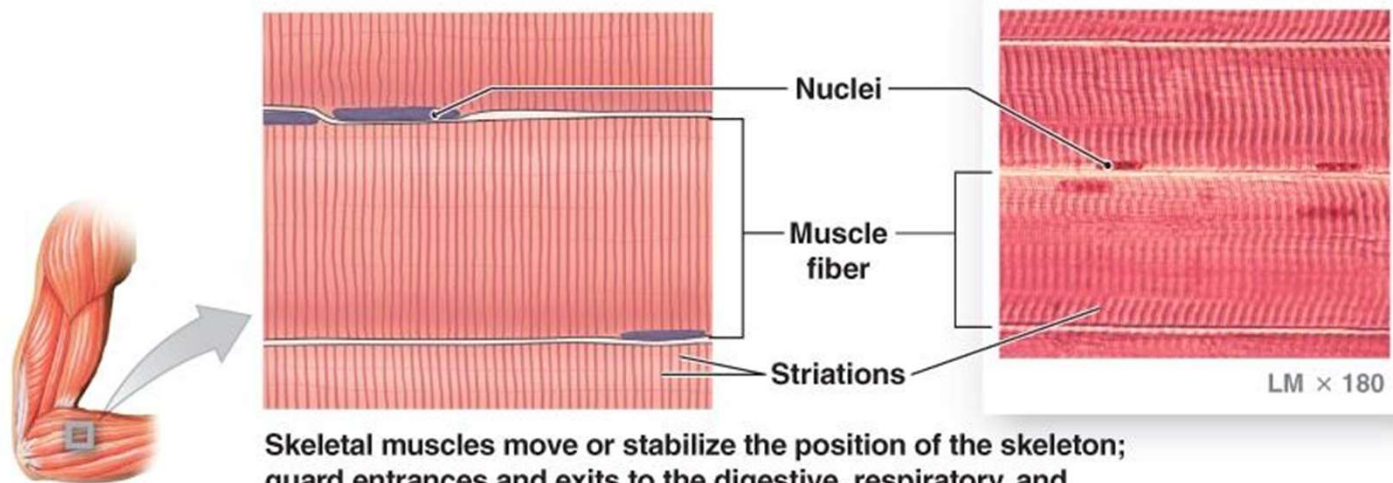
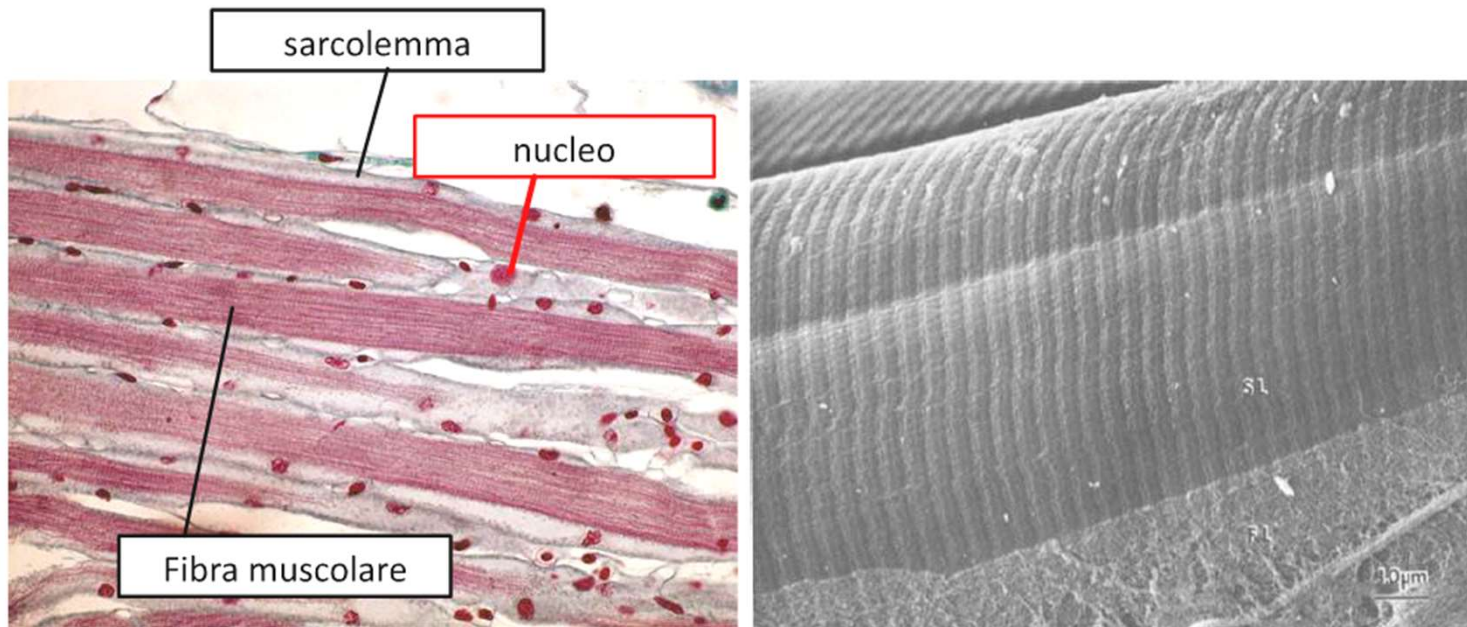
MUSCOLO SCHELETRICO



FIBROCELLULA MUSCOLARE

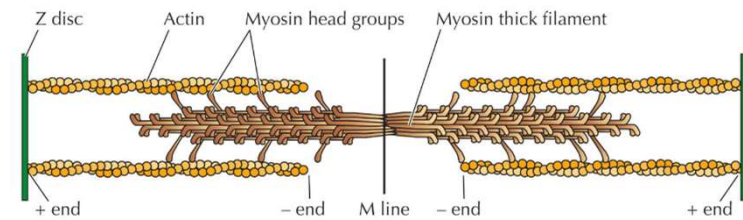
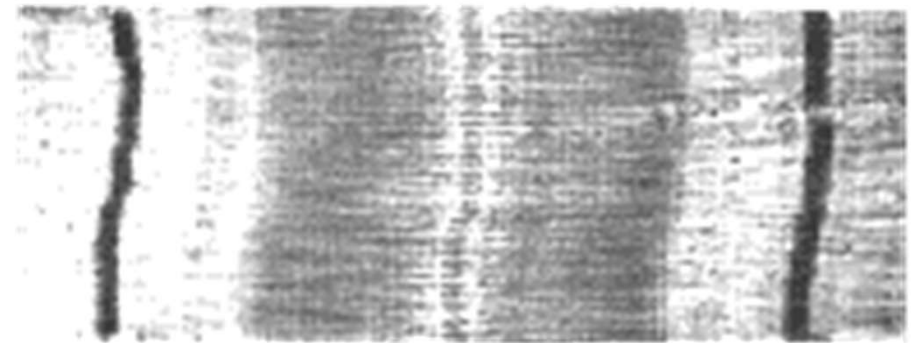
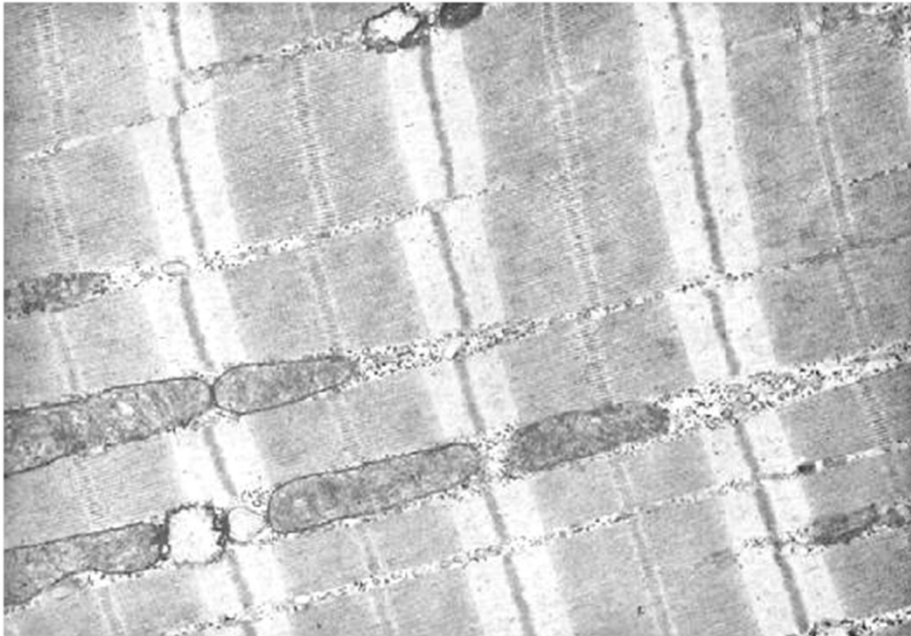


SARCOMERO



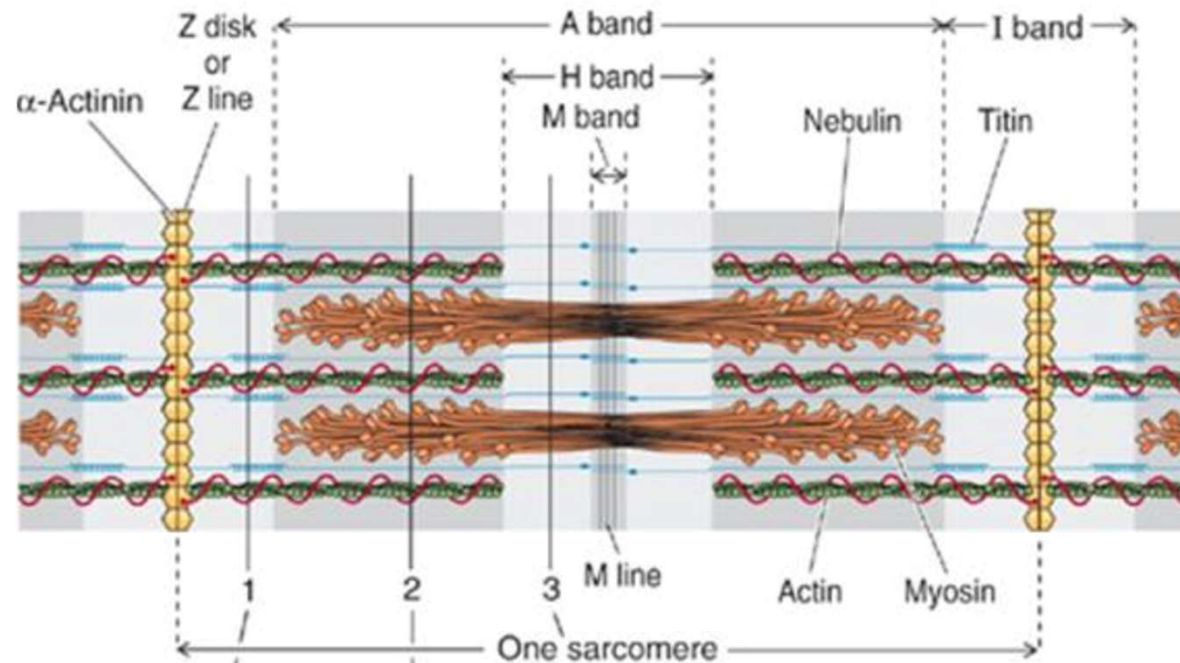
Skeletal muscles move or stabilize the position of the skeleton; guard entrances and exits to the digestive, respiratory, and urinary tracts; generate heat; and protect internal organs.

SARCOMERO

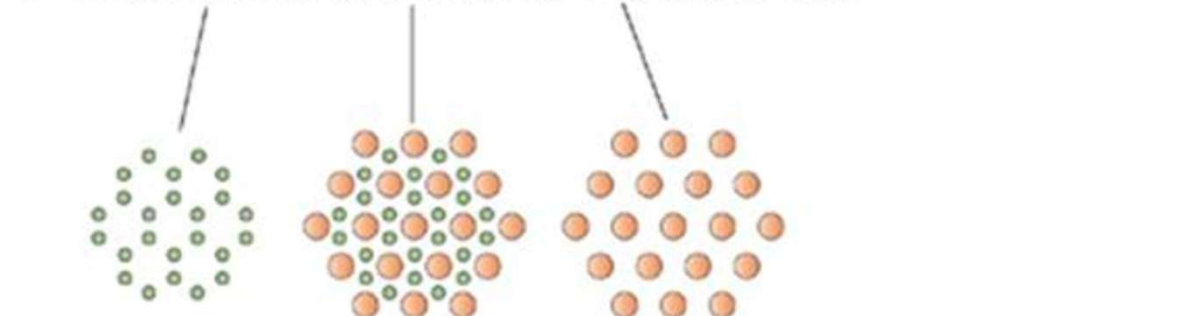


SARCOMERO

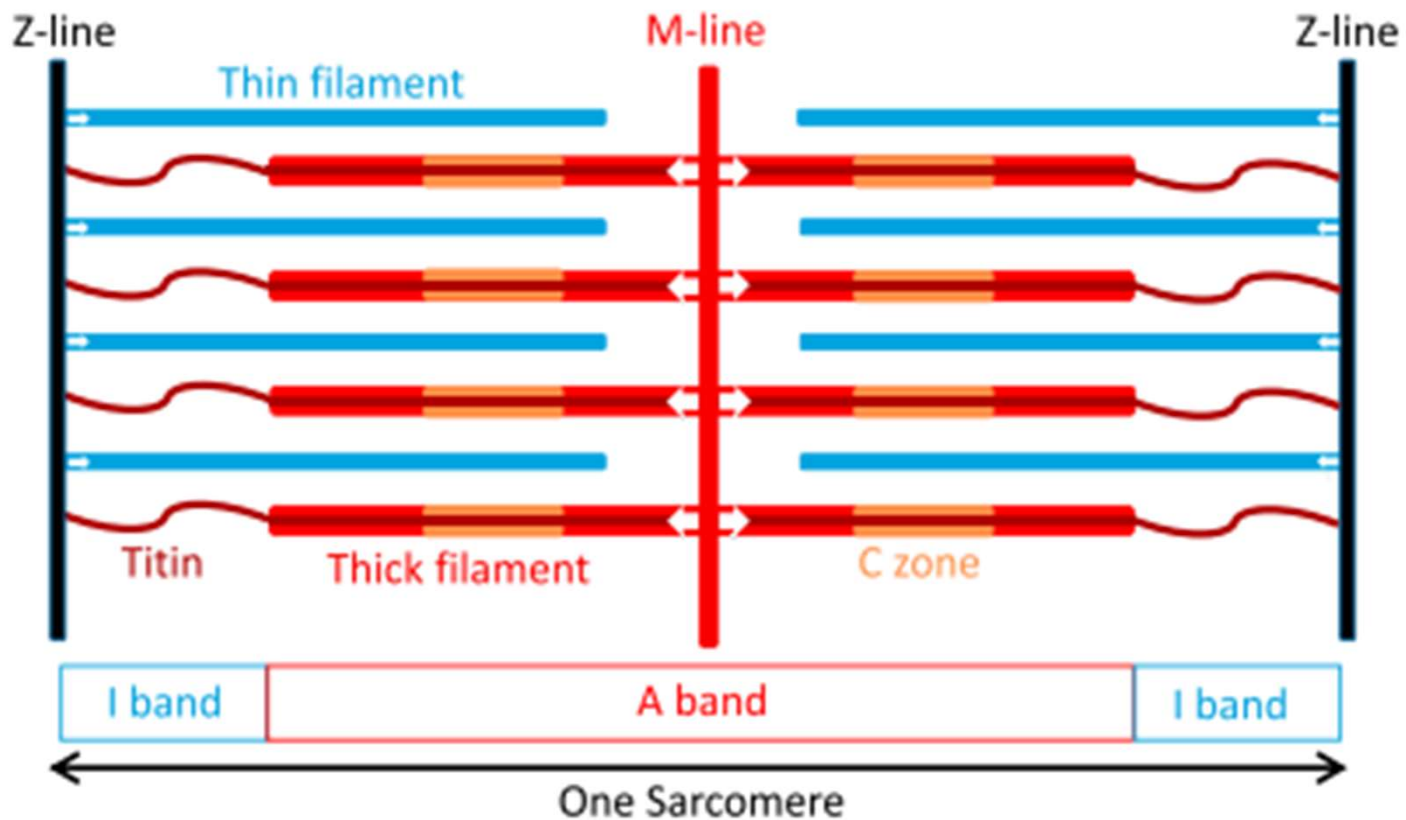
B MODEL OF A SARCOMERE



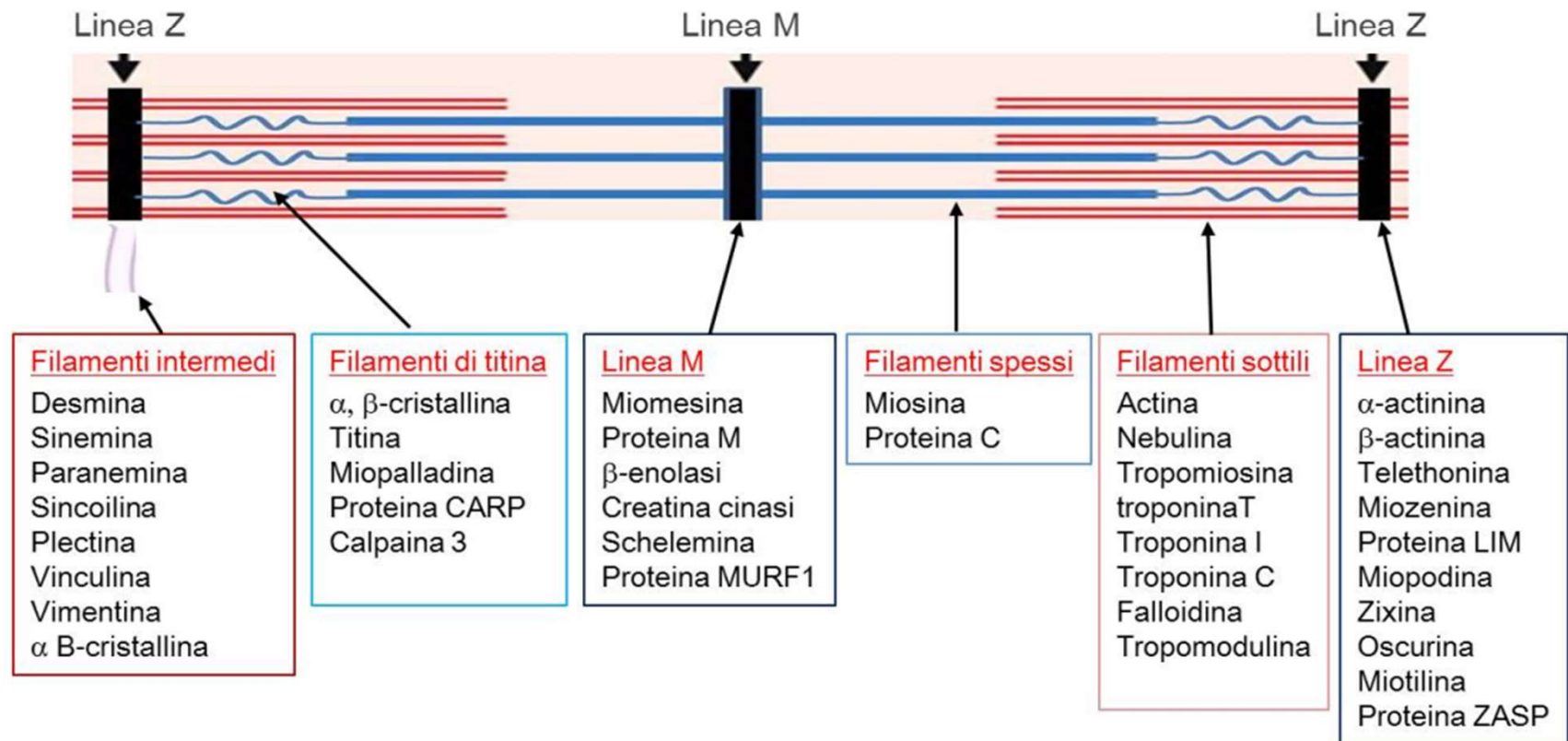
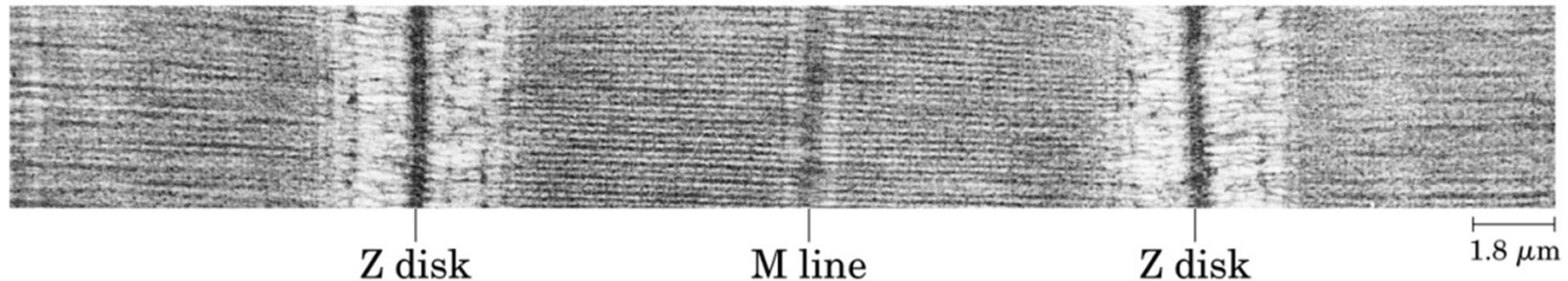
C CROSS SECTIONS OF THIN AND THICK FILAMENTS



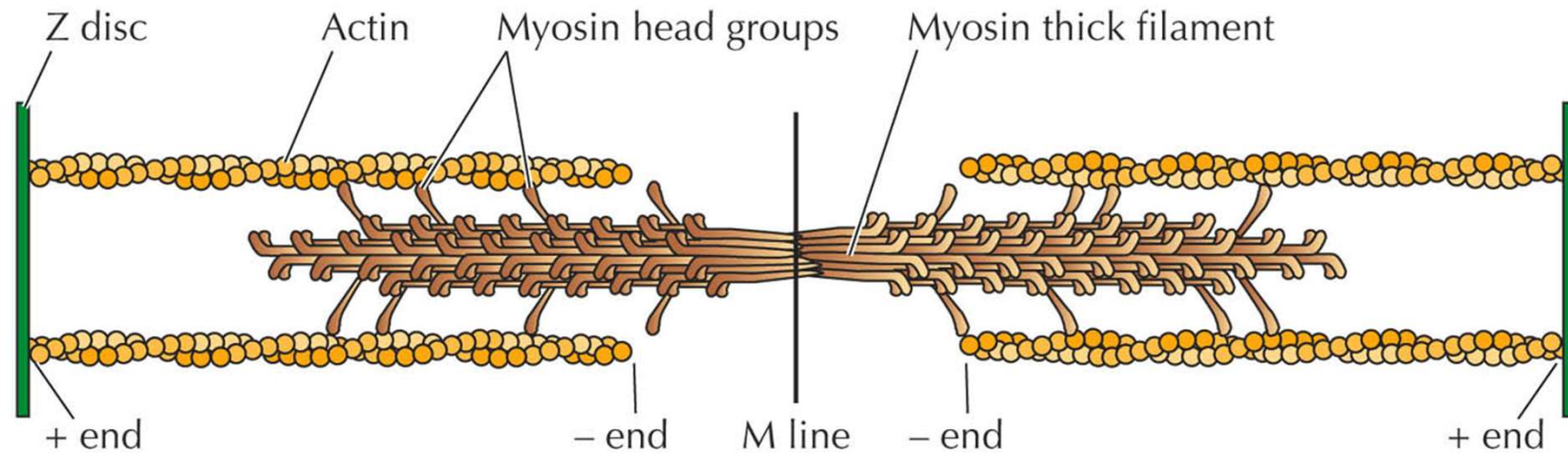
SARCOMERO



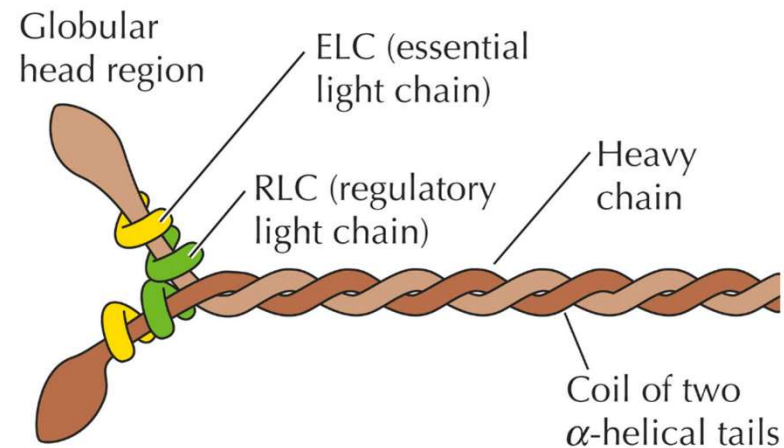
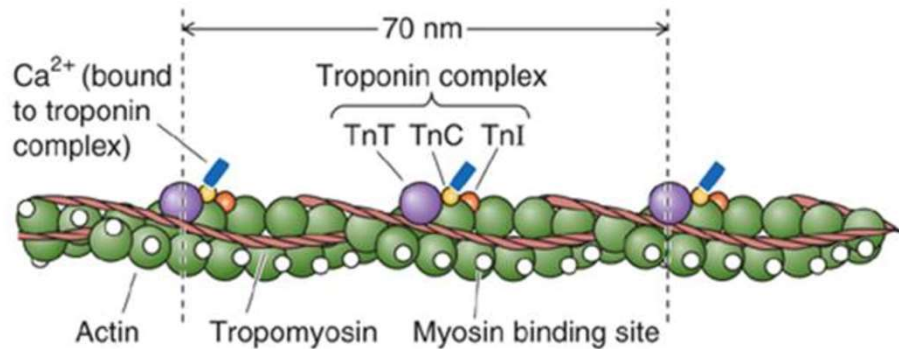
SARCOMERO - COMPLICATO



COMPLESSO ACTINA-MIOSINA

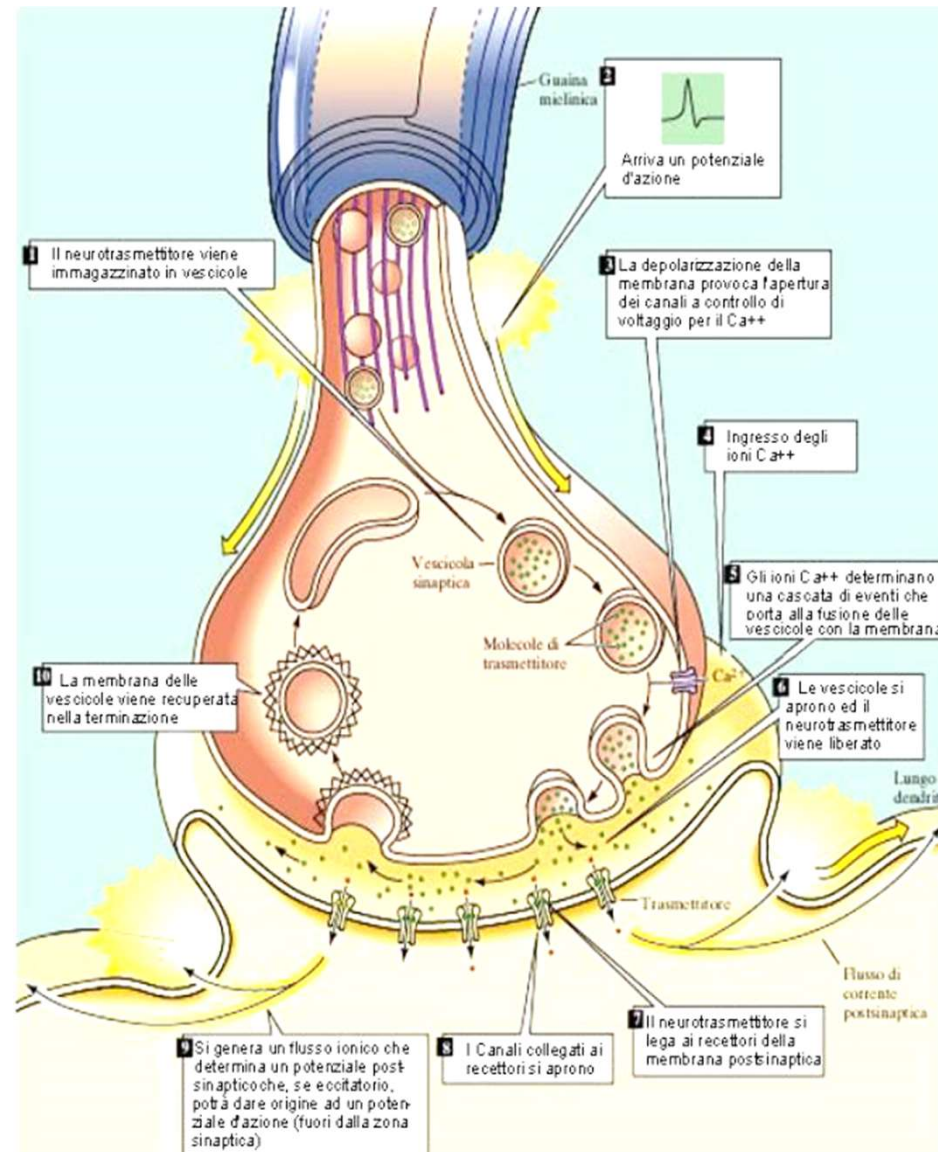


A THIN FILAMENT



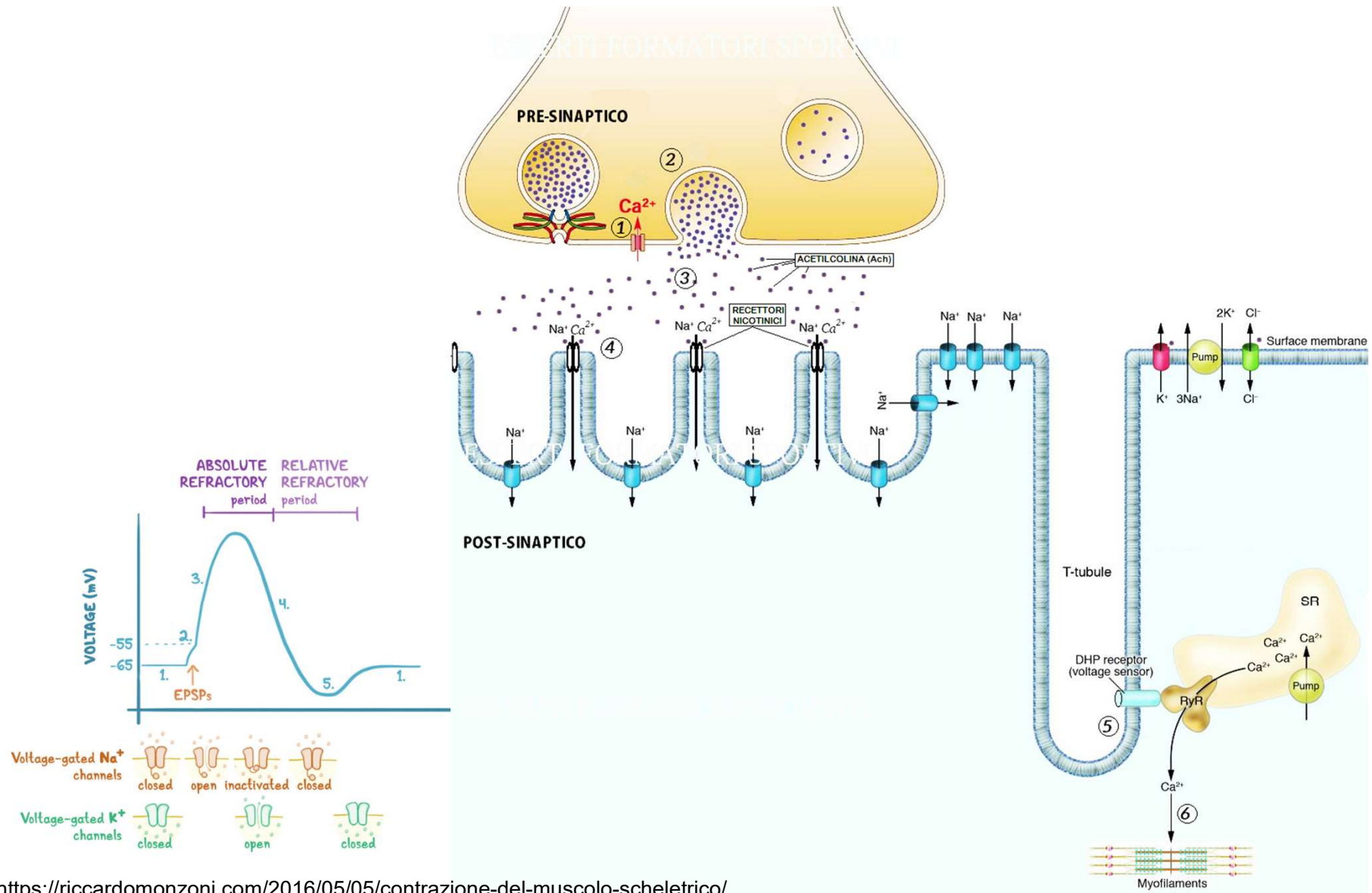
CONTRAZIONE MUSCOLARE

IMPULSO ELETTRICO



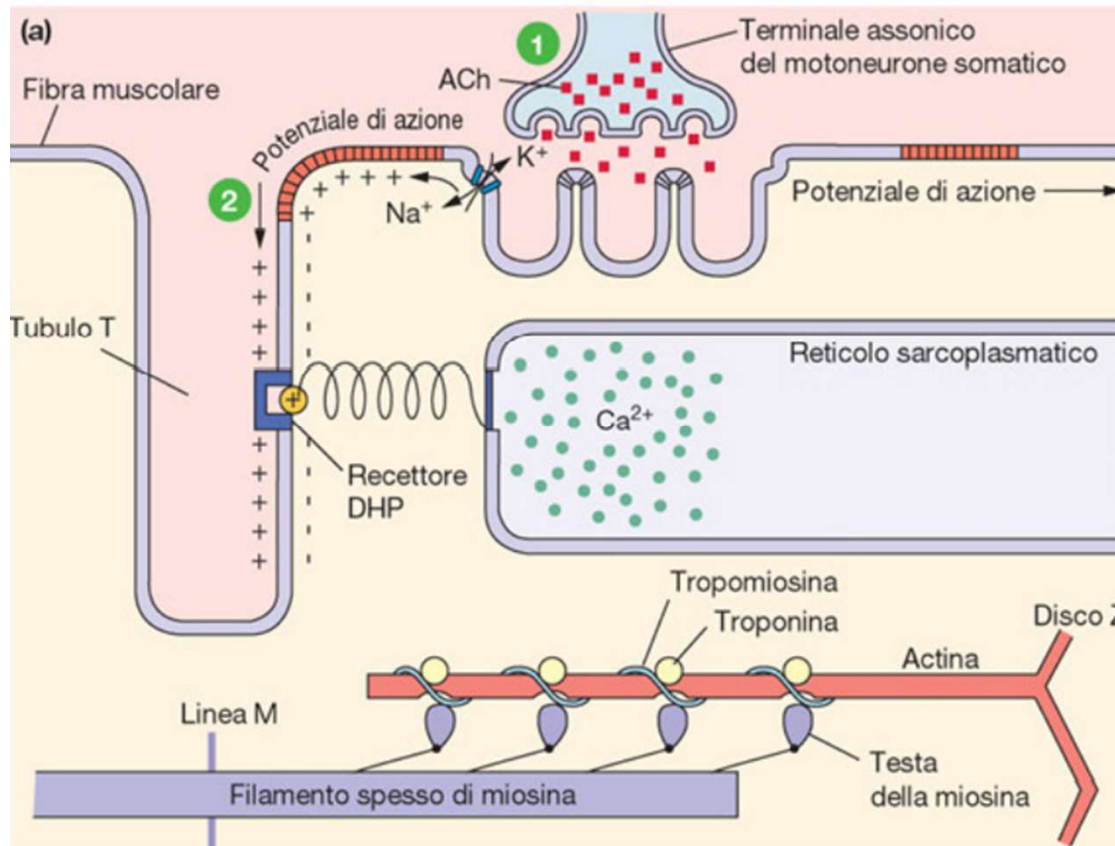
CONTRAZIONE MUSCOLARE

POTENZIALE D'AZIONE



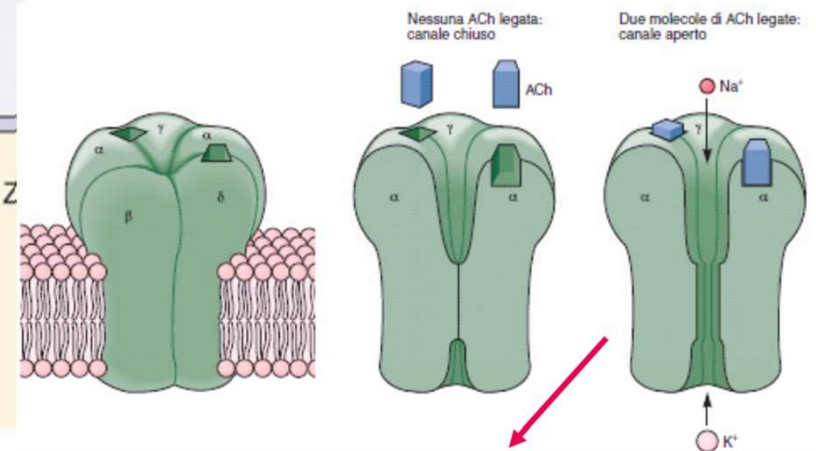
CONTRAZIONE MUSCOLARE

LIBERAZIONE ACETICOLINA



1 Il motoneurone somatico rilascia ACh a livello della giunzione neuromuscolare.

2 L'ingresso netto di Na⁺ tramite i canali controllati dai recettori per l'ACh induce un potenziale d'azione.



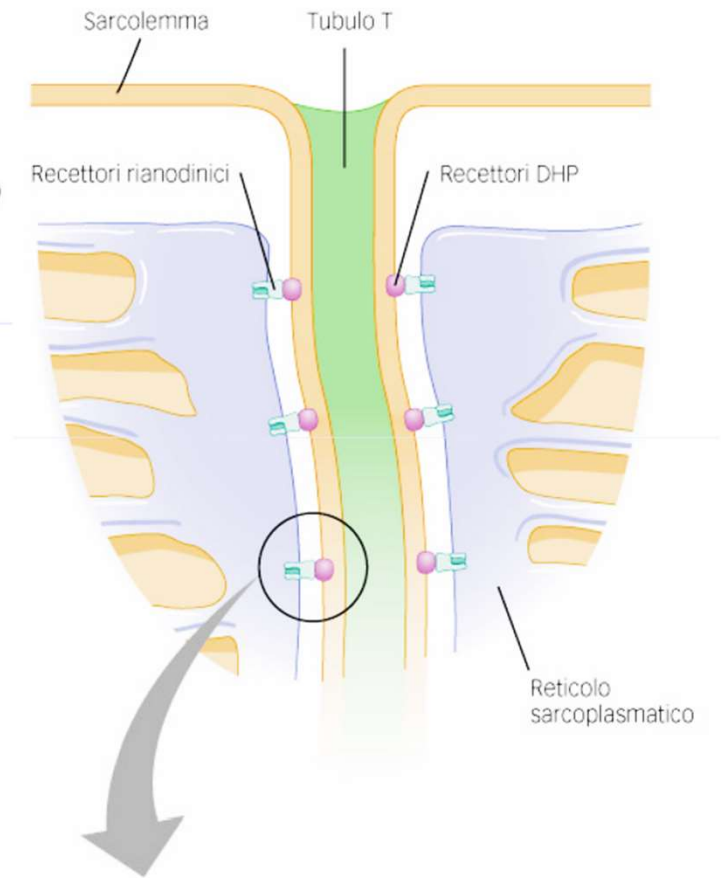
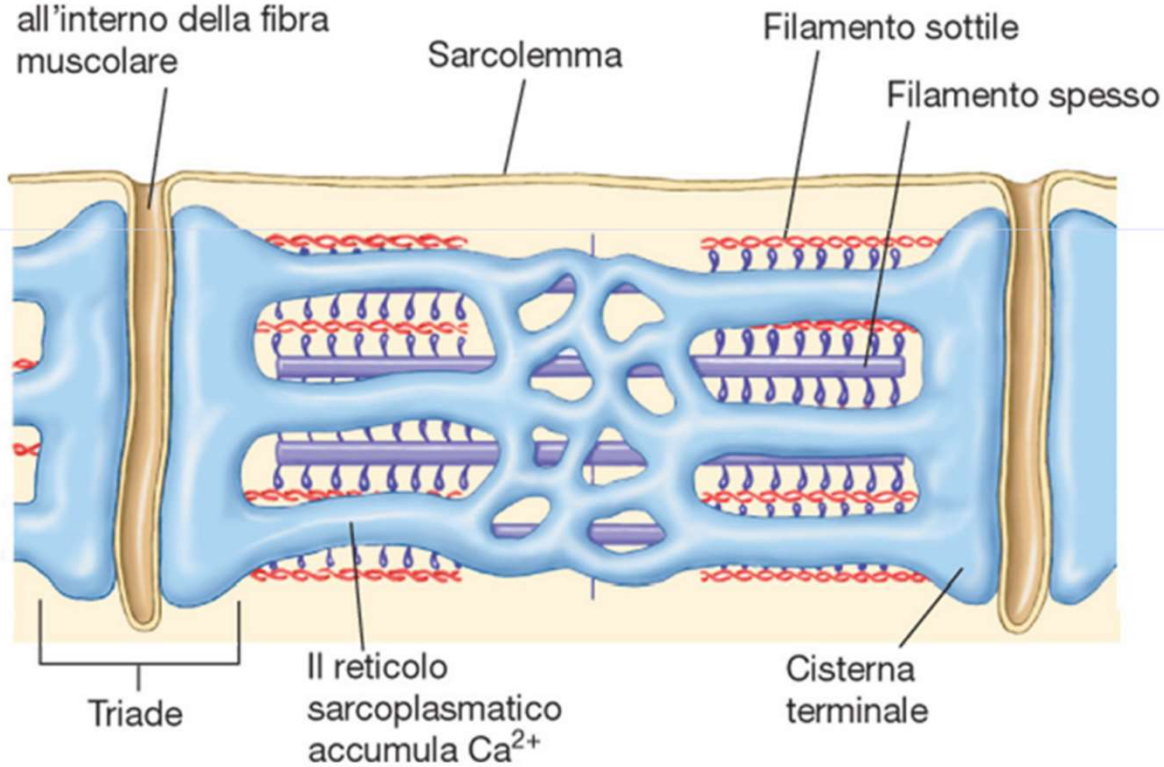
2 Ach si legano al Recettore Colinergico Nicotinico e aprono il CANALE

- INGRESSO Na supera l'uscita di K
- FIBRA DEPOLARIZZATA

CONTRAZIONE MUSCOLARE

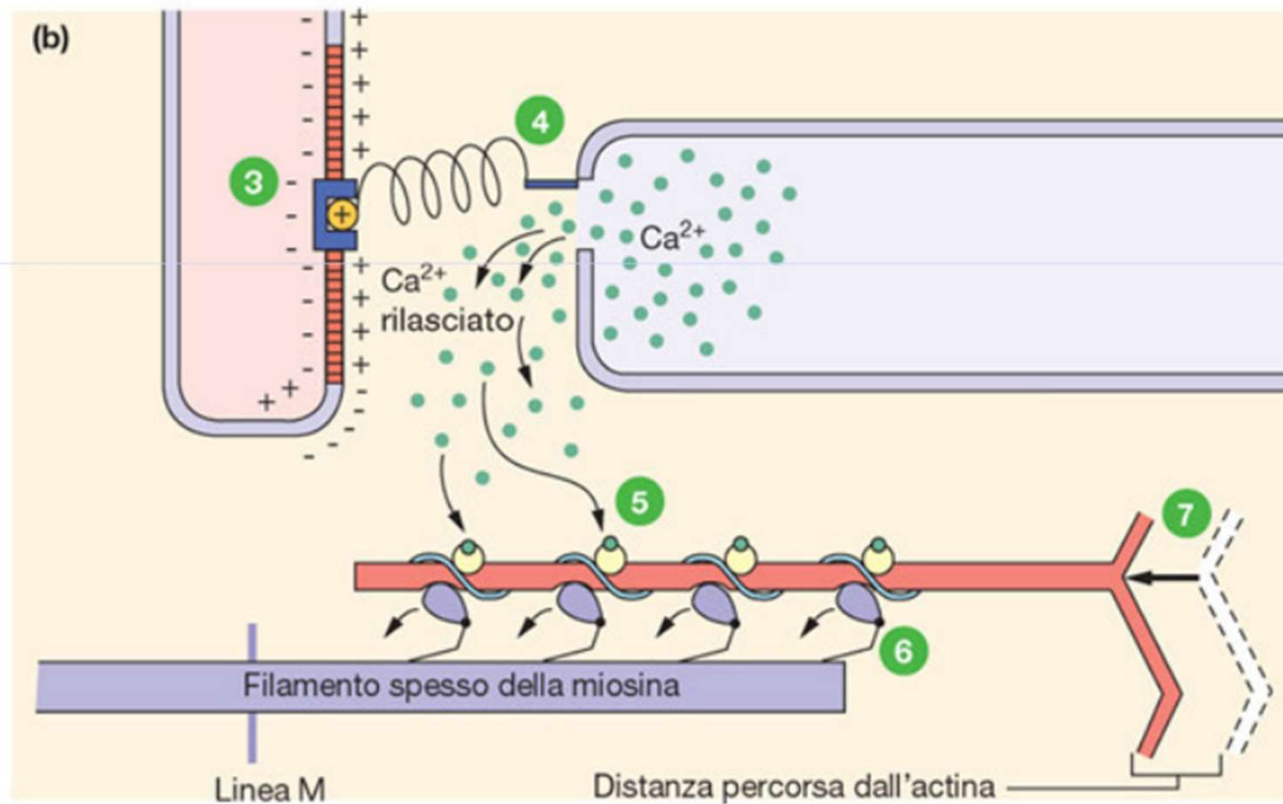
TRIADE

Il tubulo T conduce i potenziali d'azione all'interno della fibra muscolare



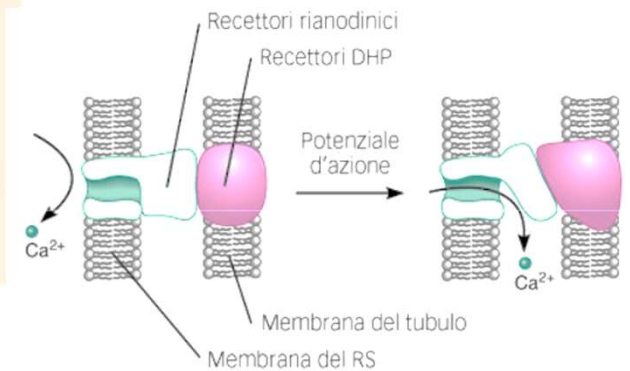
CONTRAZIONE MUSCOLARE

LIBERAZIONE DEL CALCIO



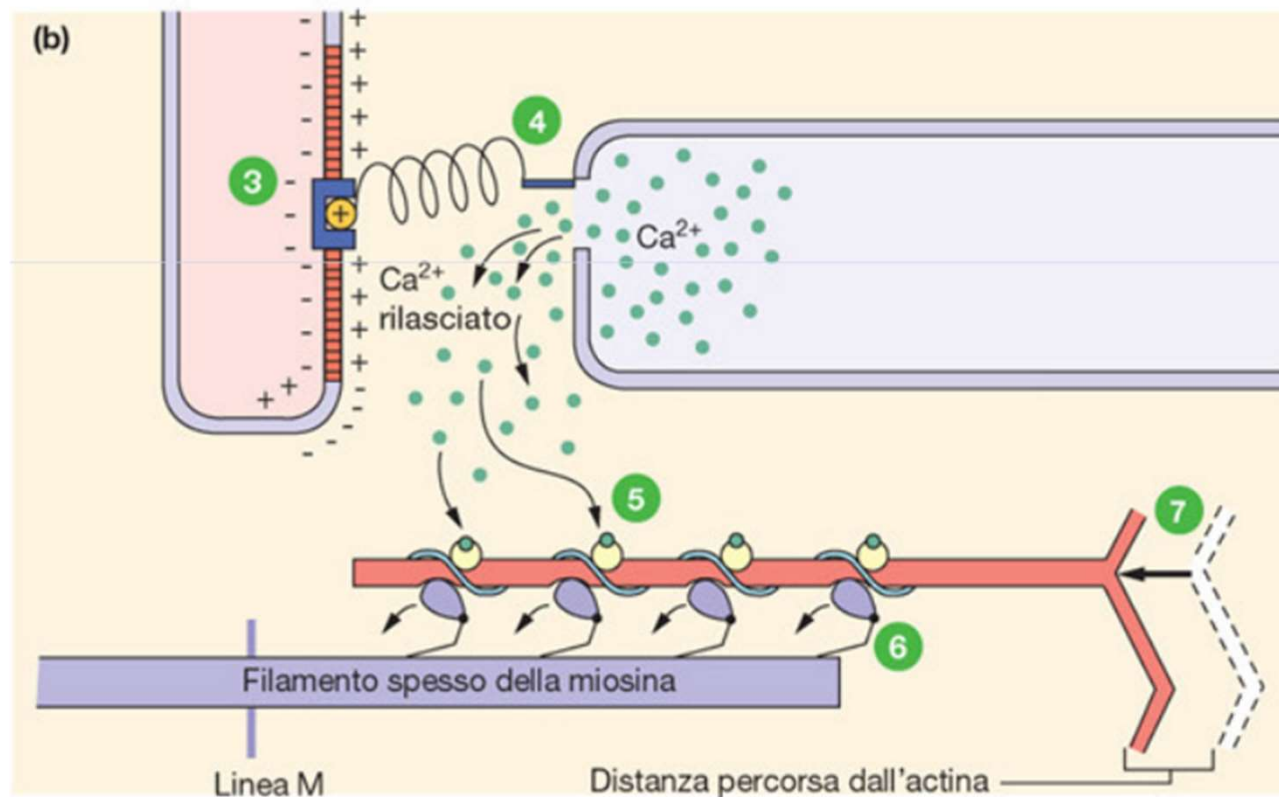
3 Il potenziale d'azione nei tubuli T altera la conformazione del recettore DHP.

4 Il recettore DHP apre canali per il rilascio del Ca²⁺ nel reticolo sarcoplasmatico e il Ca²⁺ entra nel citoplasma.



CONTRAZIONE MUSCOLARE

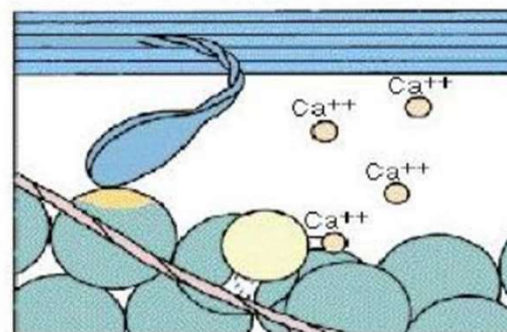
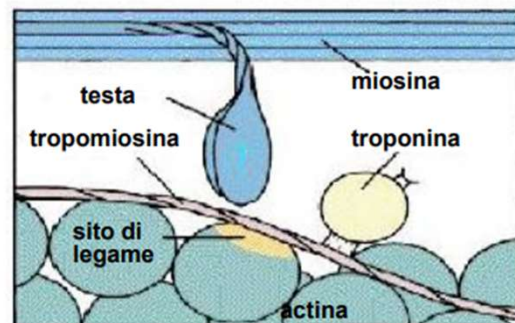
LEGAME CALCIO - TROPONINA



5 Il Ca^{2+} si lega alla troponina, consentendo il legame forte fra actina e miosina.

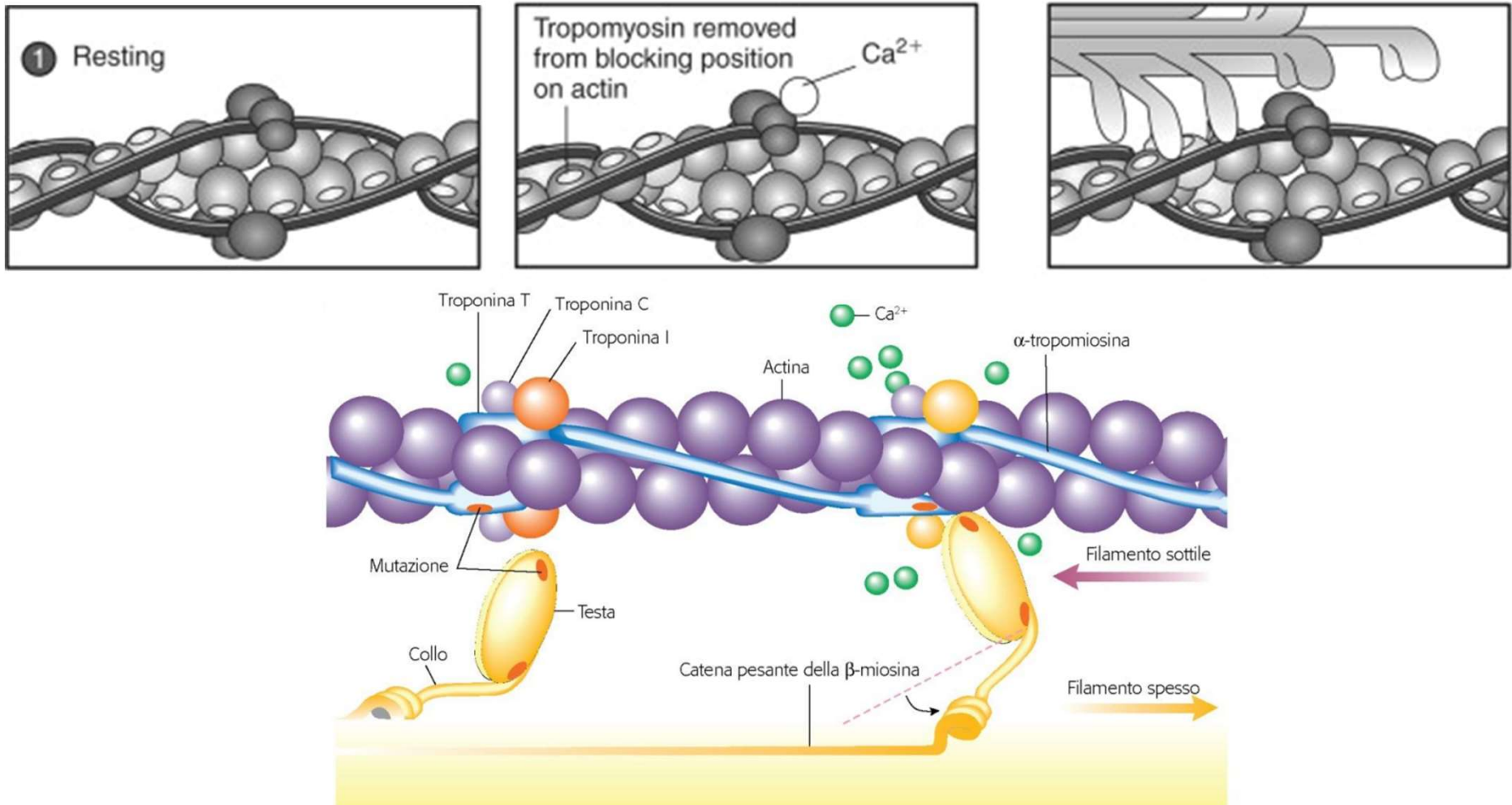
6 Le teste della miosina generano il colpo di forza.

7 I filamenti di actina scorrono verso il centro del sarcomero.



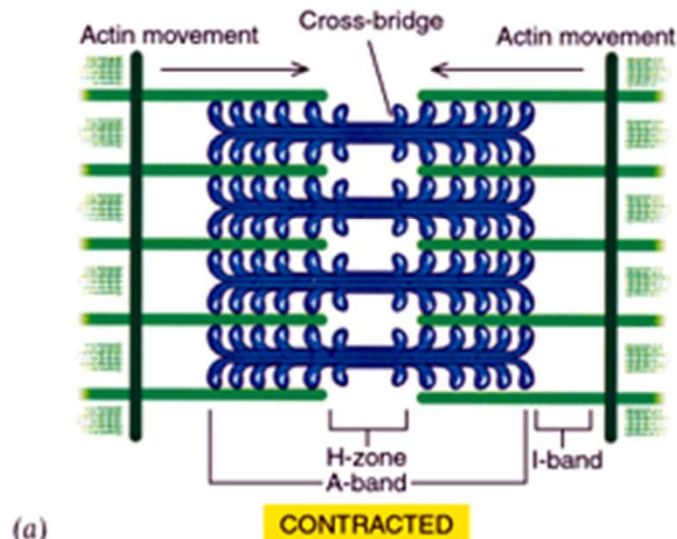
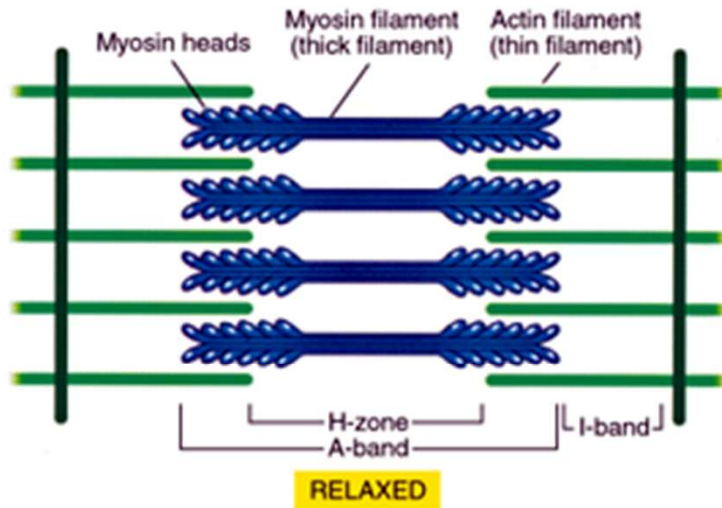
CONTRAZIONE MUSCOLARE

SCIVOLAMENTO ACTINA - MIOSINA



CONTRAZIONE MUSCOLARE

SCIVOLAMENTO ACTINA - MIOSINA



(a)

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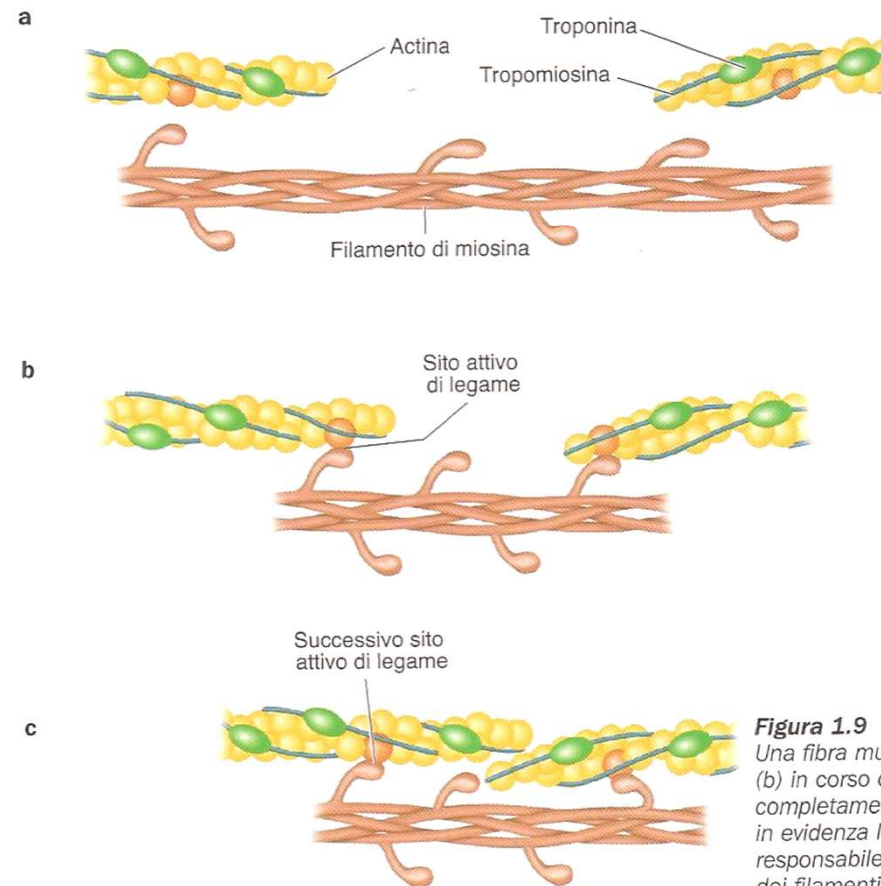


Figura 1.9
Una fibra muscolare (a) decontratta, (b) in corso di contrazione e (c) completamente contratta, che mette in evidenza l'azione agganciante responsabile dello scorrimento dei filamenti di actina e miosina.

CONTRAZIONE MUSCOLARE: ATP

FUNZIONI DELL'ATP

- ENZIMI PER ECCITABILITÀ DI MEMBRANA (Na/K ATPasi)
- GESTIONE DEL CALCIO NEL RETICOLO SARCOPLASMATICO (Ca ATPasi)
- MIOFILAMENTI (Miosin ATPasi)

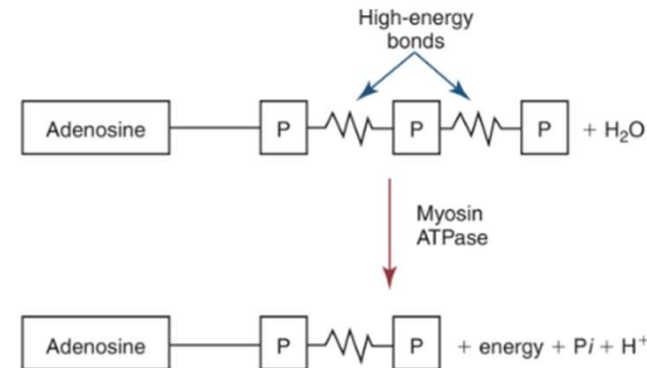
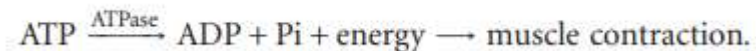


FIGURE 3-1 Hydrolysis of adenosine triphosphate (ATP) to adenosine diphosphate (ADP) by the enzyme myosin adenosine triphosphatase (ATPase), with the release of energy for use by working muscle. P, Phosphate; P_i, orthophosphate.

RISERVE ATP INTRAMUSCOLARE

- 5/8 mmol/kg di massa muscolare
- ESAURIMENTO RISERVE → 3.7 mmol ATP/kg_{xs} IN ESERCIZIO MASSIMALE
→ 0.4 mmol ATP/kg_{xs} IN ESERCIZIO SUBMASSIMALE

CONTRAZIONE MUSCOLARE: ATP

TRE SISTEMI ENERGETICI

1. SISTEMA FOSFAGENE o ANAEROBICO ALATTACIDO
2. SISTEMA GLICOLITICO o ANAEROBICO LATTACIDO
3. SISTEMA OSSIDATIVO o AEROBICO

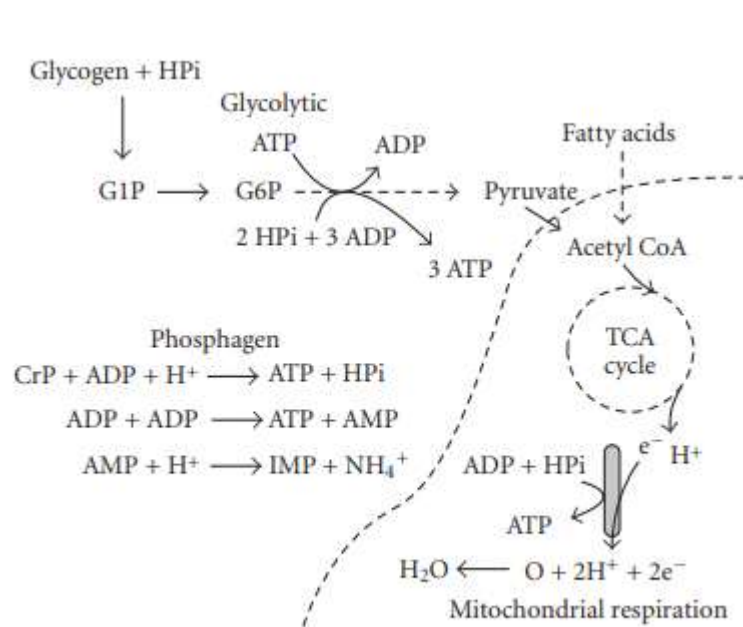


FIGURE 2: The three energy systems of muscle ATP regeneration.

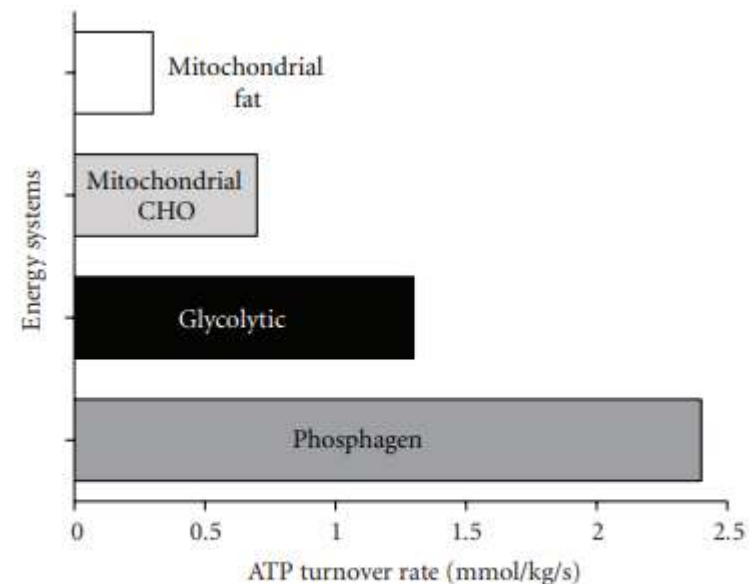
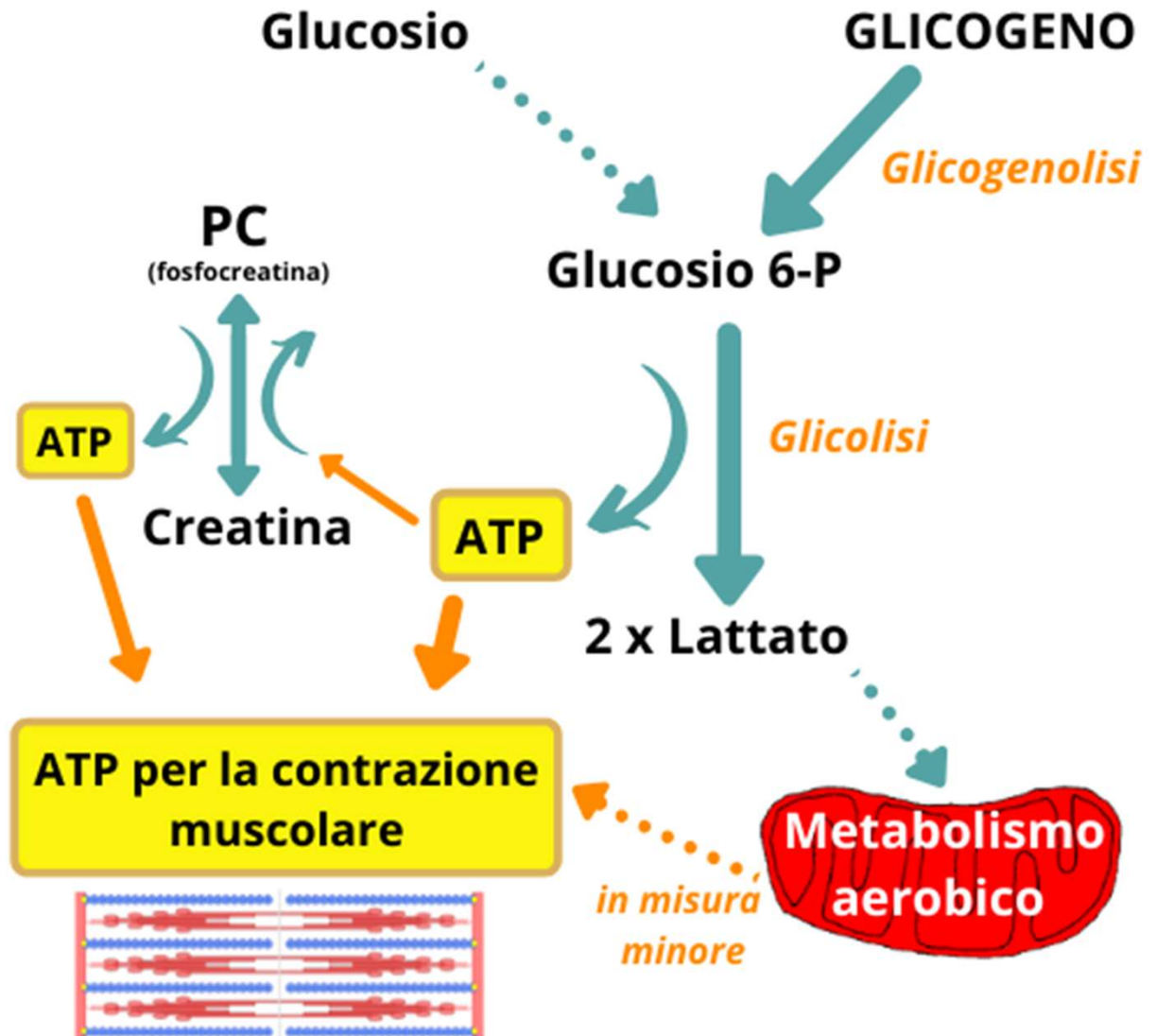
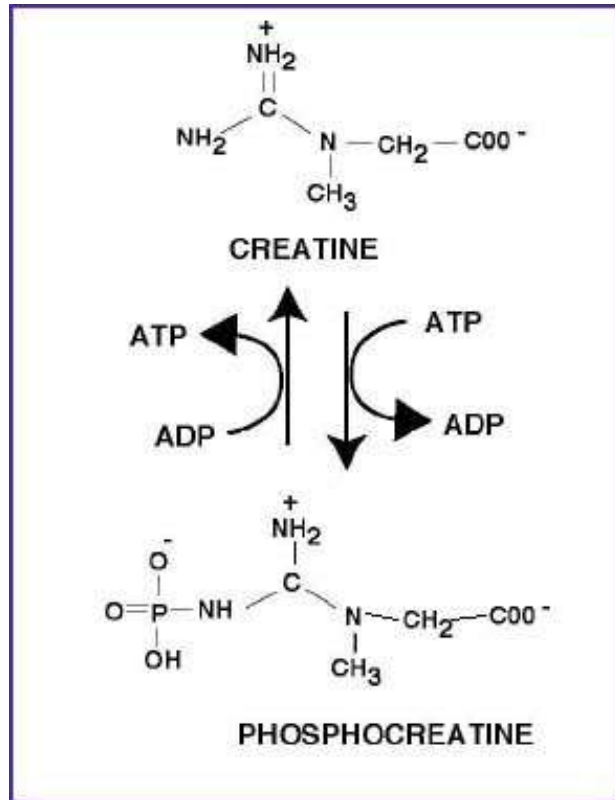


FIGURE 5: Maximal rates of ATP regeneration from the energy systems of skeletal muscle. Adapted from Sahlin et al. [25].

SISTEMA ANAEROBICO ALATTACIDO (FOSFAGENE)

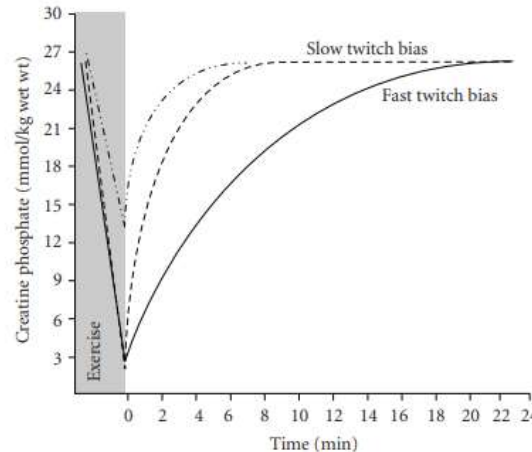
SISTEMA FOSFAGENE



SISTEMA FOSFAGENE

SPORT IN CUI SI UTILIZZA IL SISTEMA ANAEROBICO ALATTACIDO

- SPORT CON LIMITATO NUMERO DI CONTRAZIONI MUSCOLARI MA INTENSE
- DURATA DI ESERCIZIO NON SUPERIORE AI 10-15 SECONDI
- IDROLISI DELLA FOSFOCREATINA NON DIPENDE DALLA DISPONIBILITÀ DI O₂

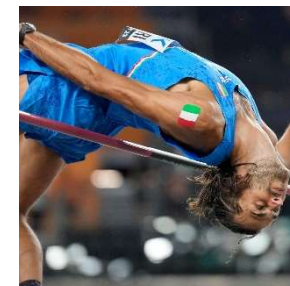
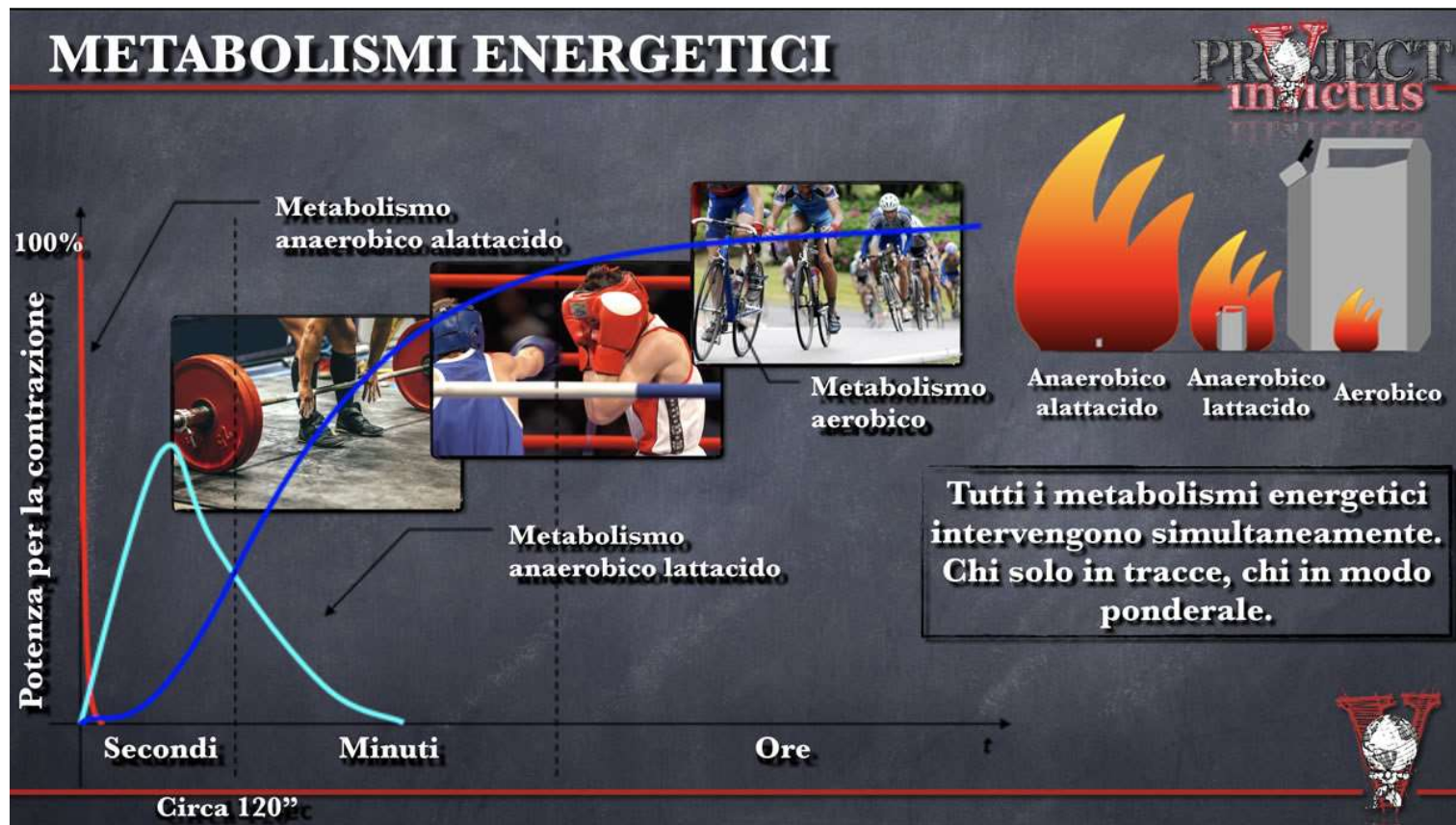


DOPO 1.5' → 65±2.8%
DOPO 4.5' → 85.5±3.5%
DOPO 13.6' → 95%

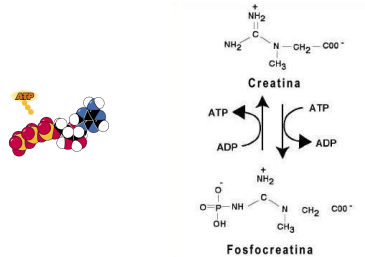
FIGURE 6: Representative kinetics of creatine phosphate (CrP) recovery in subjects with different end exercise CrP concentrations and different proportions (bias) of slow or fast twitch muscle. Note the more rapid recovery of CrP when there is less exercise-induced depletion (-----) versus near complete depletion (----). Based on unpublished research observations of the authors.

- CAPACITÀ RIGENERATIVA DELLE SCORTE DAI 5 AI 15 MINUTI DIPENDE DA:
 - ACIDOSI METABOLICA
 - UNITÀ MOTORIA E CARATTERISTICHE DELLE FIBRE MUSCOLARI

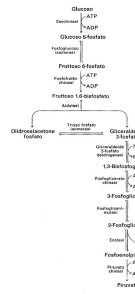
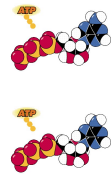
SISTEMA FOSFAGENE



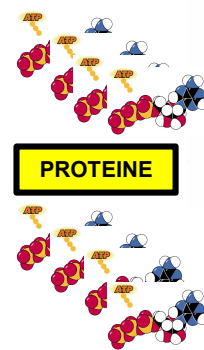
SISTEMI ENERGETICI



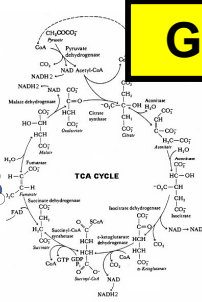
F-CREATINA



GLUCOSIO

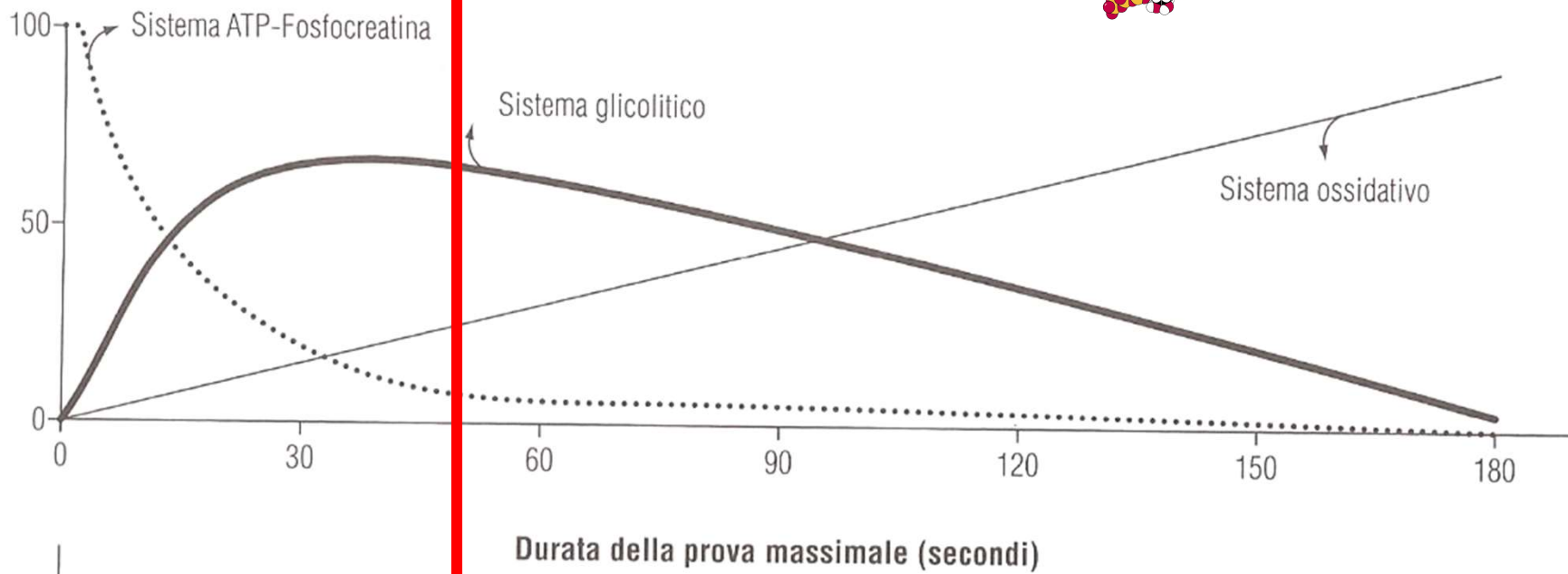


PROTEINE



GRASSI

GLUCOSIO



SISTEMI ENERGETICI

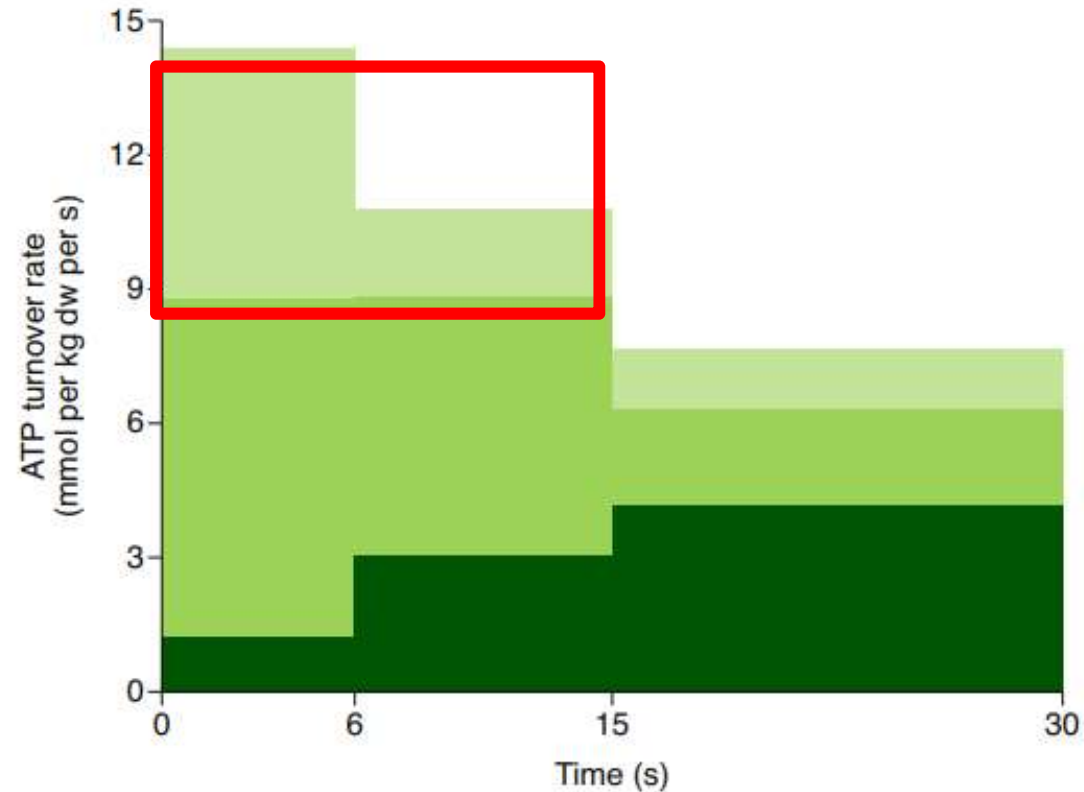
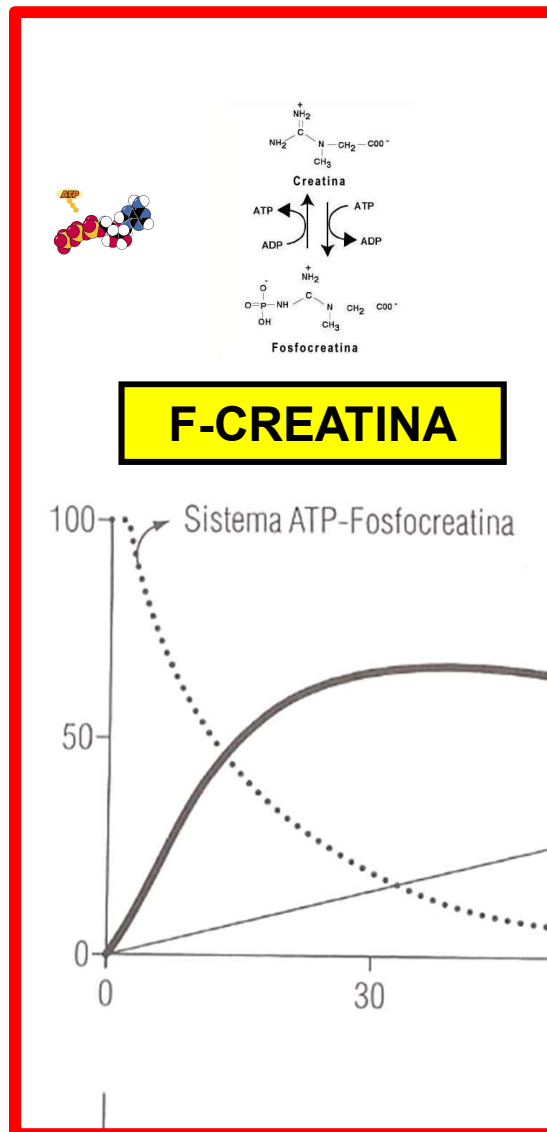
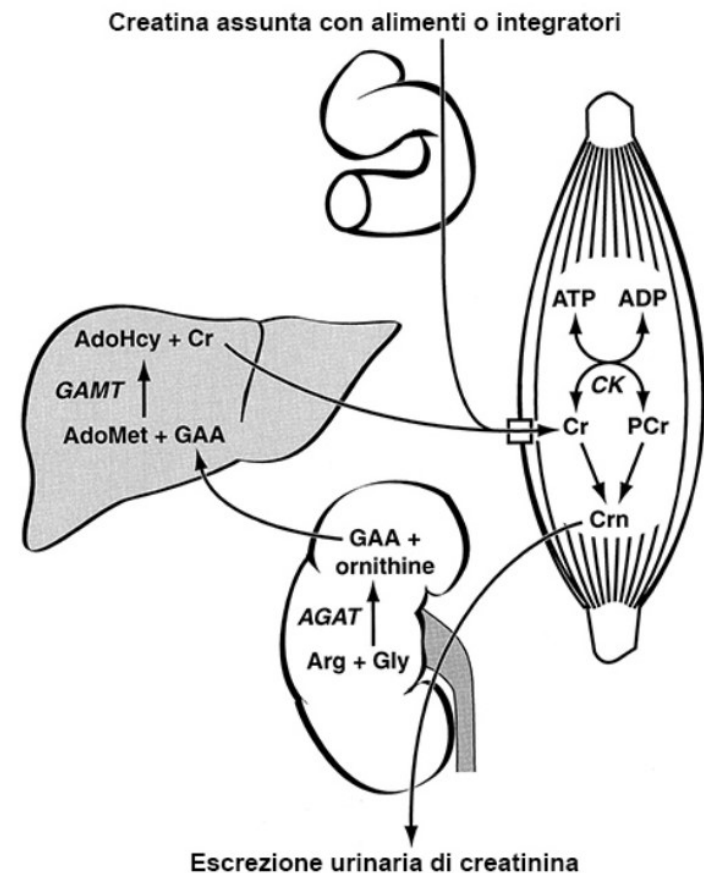


Fig. 1 | Muscle energy metabolism during intense exercise. Contributions of PCr (light green), glycolysis (medium green) and oxidative phosphorylation (dark green) to ATP turnover during maximal exercise. Muscle samples were obtained before and during 30 s of all-out cycling exercise. Dw, dry weight. Adapted with permission from ref. ⁴, American Physiological Society.

CREATINA

- AMINO ACIDO NON PROTEICO sintetizzato nel fegato, rene e pancreas a partire da ARGININA, GLICINA e METIONINA
- 50% dieta e 50 % endogeno
- Integrazione sopprime la produzione endogena
- 95% viene immagazzinato nel muscolo scheletrico ($\frac{2}{3}$ PCr e $\frac{1}{3}$ libera)
- 5% in cervello e testicoli
- 120 gr in individuo di 70 kg
- TURNOVER: 1-2,5 gr/die
- Escreta per via renale



CREATINA – FUNZIONI

FUNZIONI DELLA CREATINA:

- ↑ MASSA MUSCOLARE e CAPACITÀ ANAEROBICA
- ↑ PERFORMANCE -> POTENZA/MASSIMA FORZA MUSCOLARE (5-15%)
-> VELOCITÀ (1-5%)

EFFETTI COLLATERALI:

- NON CAUSA INSUFFICIENZA RENALE IN SOGGETTI SANI

CREATINA – ↑ IPERTROFIA

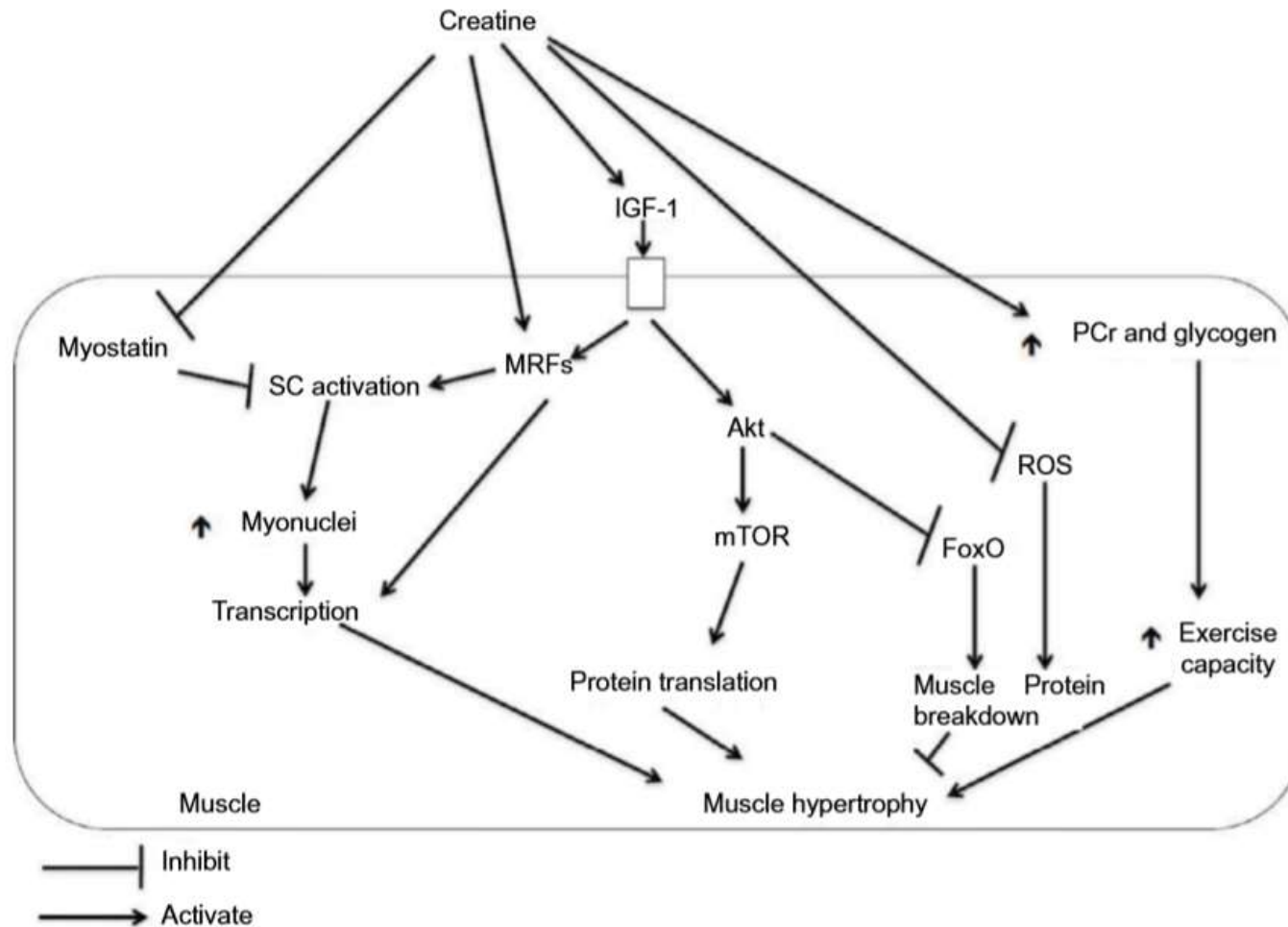
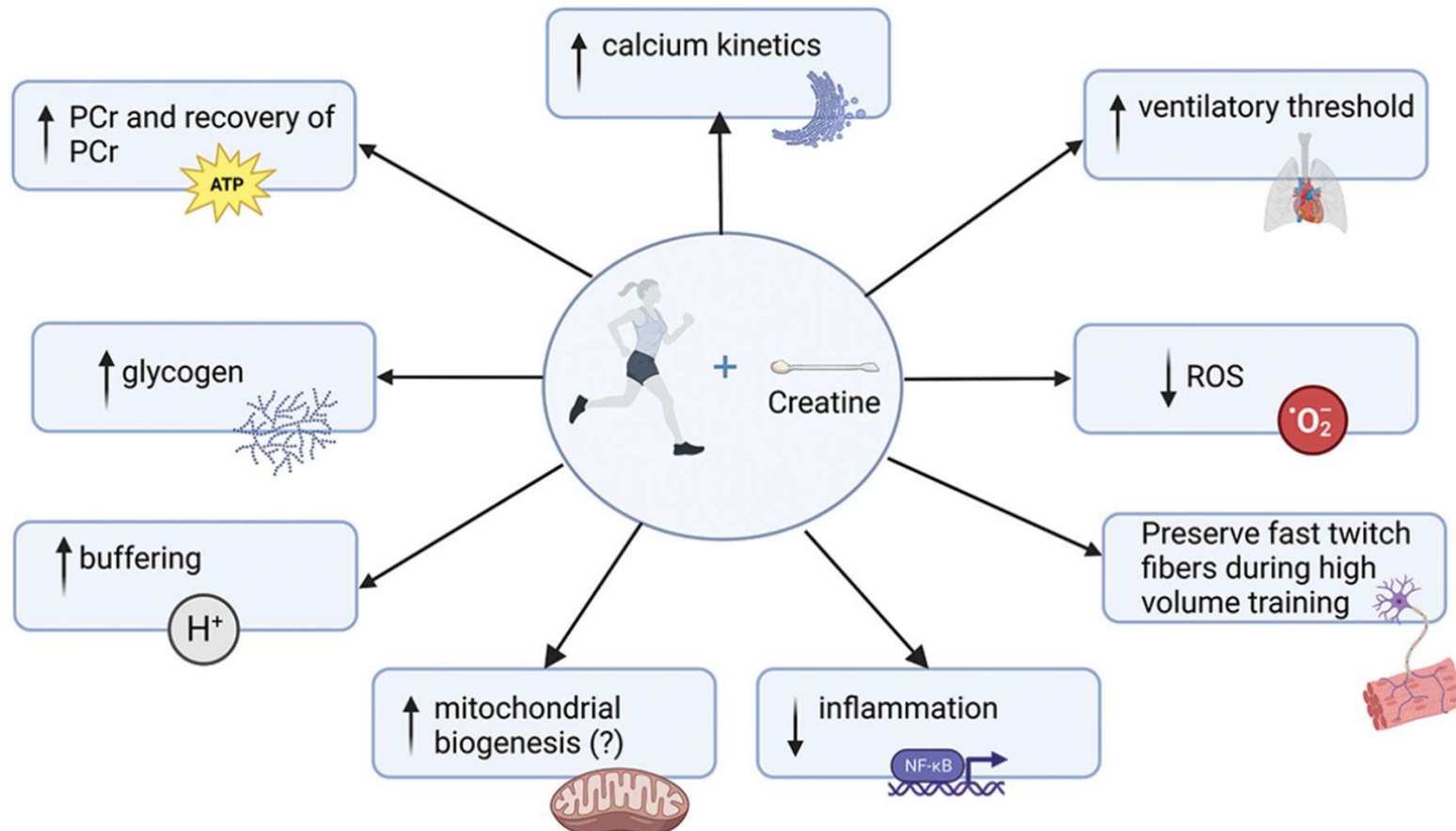


Figure 2 Potential mechanisms by which creatine supplementation leads to muscle hypertrophy.

Abbreviations: IGF-1, insulin-like growth factor 1; MRFs, myogenic regulatory factors; mTOR, mammalian target of rapamycin; PCr, phosphorylcreatine; ROS, reactive oxygen species; SC, satellite cells.

CREATINA – ↑ ENDURANCE



Review

Creatine supplementation and endurance performance: surges and sprints to win the race

Scott C. Forbes, Darren G. Candow , Joao Henrique Falk Neto, Michael D. Kennedy, Jennifer L. Forbes, Marco Machado, ...show all

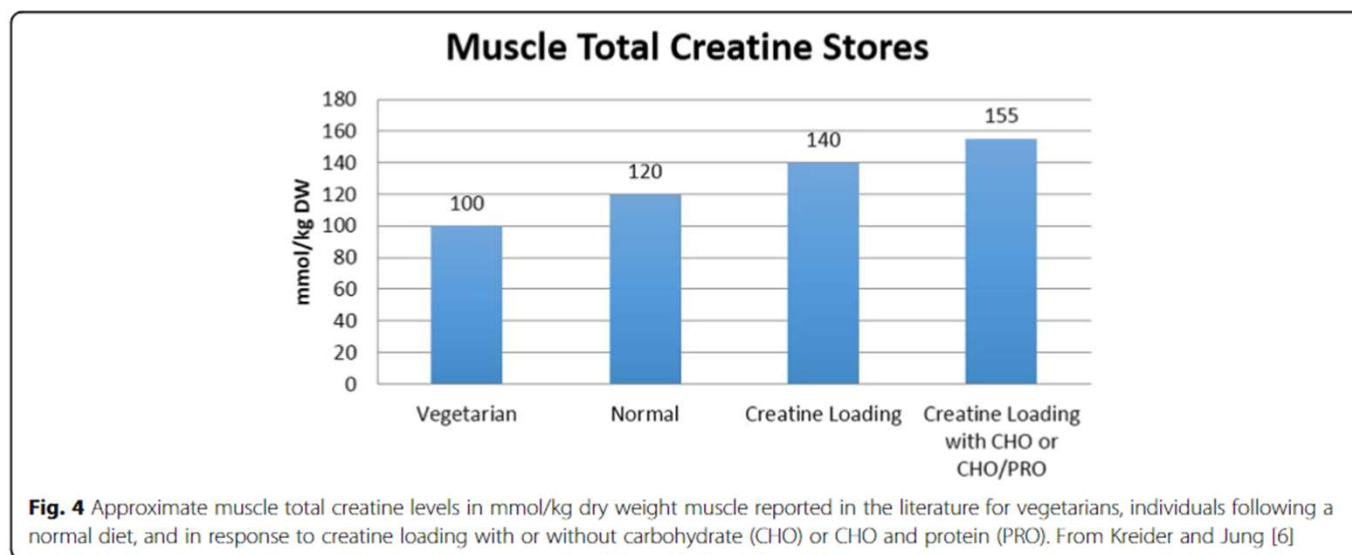
Article: 2204071 | Received 05 Jan 2023, Accepted 06 Apr 2023, Published online: 25 Apr 2023

CREATINA – FONTI e DEPOSITI

Aringa	6,5-10,0	Merluzzo	3,0
Maiale	5,0	Latte	0,1
Manzo	4,5	Mirtilli	0,02
Salmone	4,5	Gamberetti	tracce
Tonno	4,0		

Da: Incledon, 2000.

CONTENUTO DI CREATINA
(g/kg DI ALIMENTO)



In a normal diet that contains 1–2 g/day of creatine, muscle creatine stores are about 60–80% saturated. Therefore, dietary supplementation of creatine serves to increase muscle creatine and PCr by 20–40% (see Fig. 4.)

Kreider *et al.* *Journal of the International Society of Sports Nutrition* (2017) 14:18

CREATINA – INTEGRAZIONE

PROTOCOLLI DI INTEGRAZIONE:

- PROTOCOLLO DI CARICO -> AUMENTA DEPOSITI DI PCr DEL 20-40%
 - 0.3 g/kg/die x 5-7 gg
 - OVVERO per soggetto di 70 kg sono 20 gr giornalieri (5 g x 4 volte al giorno)
 - POI 3-5 g/die
- PROTOCOLLO NORMALE -> AUMENTA DEPOSITI DI PCr DEL 20%
 - x 3-4 settimane
 - 0.05->0.075 g/kg/die x 1 volta al giorno
 - OVVERO 3-5 gr/die

Creatine and Phosphocreatine: A Review of Their Use in Exercise and Sport

Joseph F. Clark, PhD, ATC

Harris et al^{21,27} have shown that feeding creatine at 2 to 5 g/day increases intracellular creatine and PCr in human muscle.³ Greenhaff and colleagues have extended these observations using 5 to 20 g/day creatine.^{9,28,29} They fed volunteers 5 g of creatine four times a day with a daily total of 20 g. This is considered the loading dose and is decreased to 2 to 5 g/day (maintenance dose) after 6 to 14 days, which is continued throughout the period of training. If athletes are given lower doses (2–3 g/day), without the loading dose, there is still increased muscle creatine but with a slower rate of accumulation. The current evidence is that the best loading of muscle creatine occurs during normal training and occurs concomitant with a low-fat, high-carbohydrate diet.²⁹

CREATINA – INTEGRAZIONE

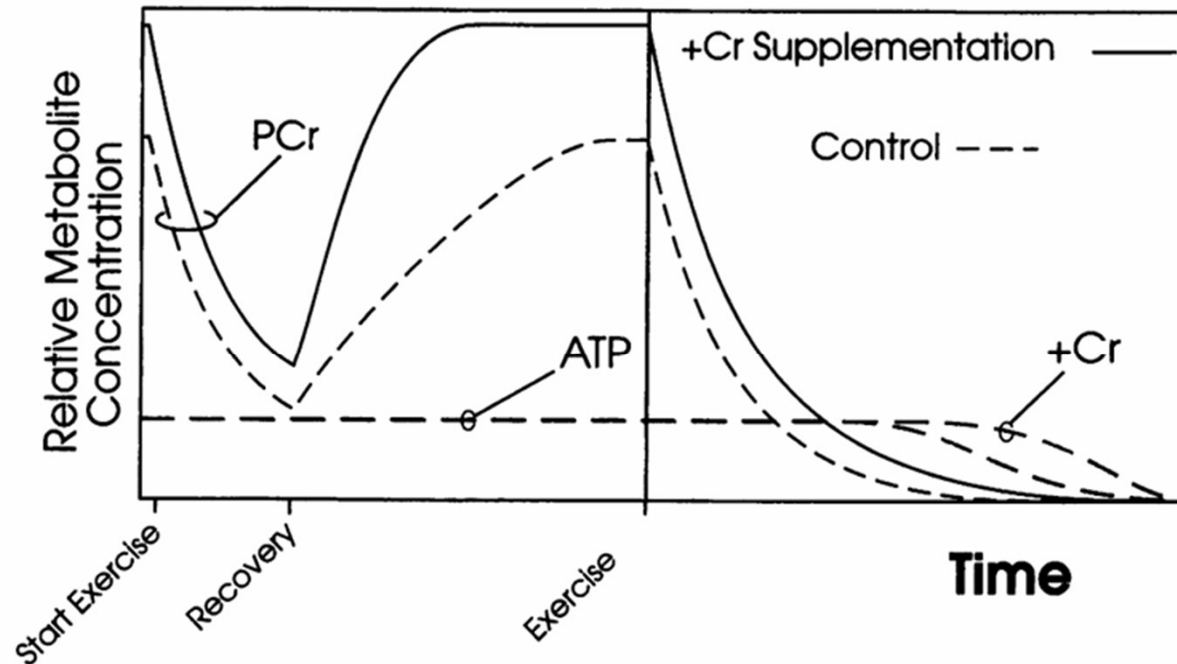


Fig 2. A schematic representation of the relative concentration changes that occur in muscle during two bouts of exercise. The PCr concentration at the start of the anaerobic exercise is greater in the muscle with creatine supplementation compared to control. The PCr concentration falls rapidly in both groups as the PCr molecule is used to buffer the ATP concentration. When exercise stops and the muscle is allowed to recover, the rate of PCr recovery is faster in the muscles with creatine supplementation (this is explained in Fig 3). Furthermore, when the anaerobic exercise is prolonged in the second bout of exercise, the ATP concentration is better maintained with creatine supplementation. The ATP buffering capacity of the muscle is enhanced due to the increased PCr concentration in the muscle. Note: the rates of concentration changes during exercise are dependent upon intensity of the exercise. For maximal anaerobic exercise, such as a sprint, the entire right hand box of this figure would take about 15 seconds.

Creatine and Phosphocreatine: A Review of Their Use in Exercise and Sport

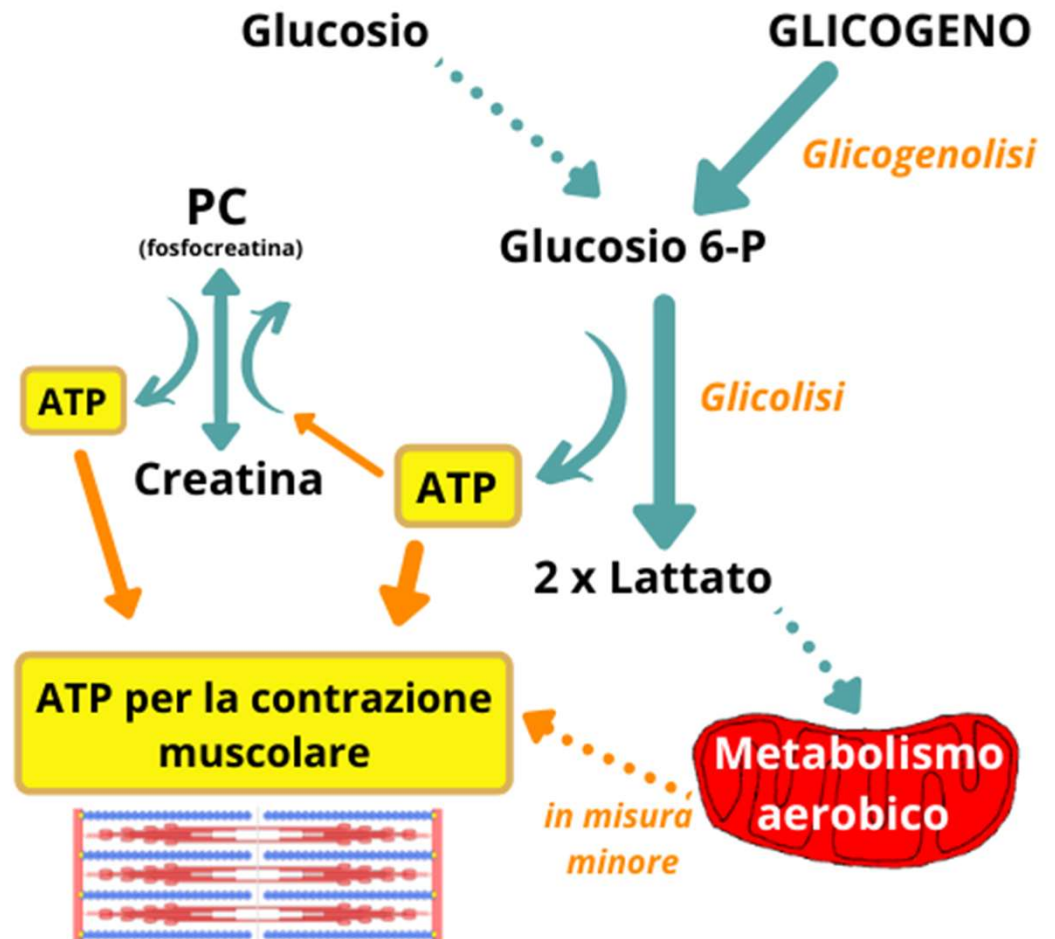
Joseph F. Clark, PhD, ATC

SISTEMA ANAEROBICO LATTACIDO (GLICOLITICO)

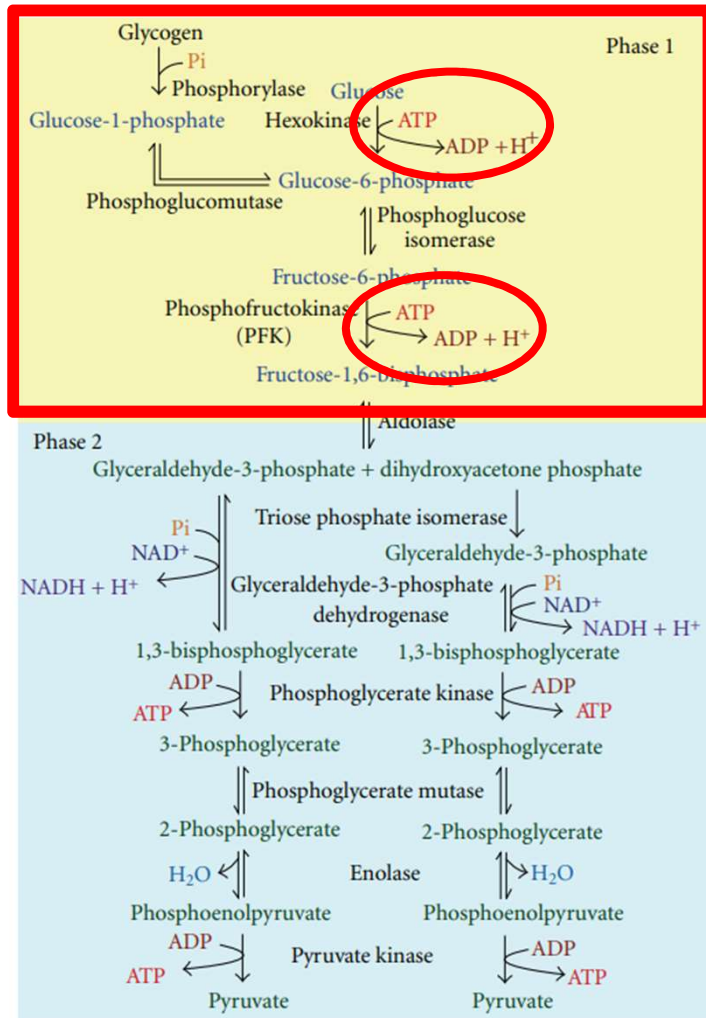
SISTEMA GLICOLITICO

ENERGIA PER ESERCIZIO DELLA DURATA DI PIÙ SECONDI

- GLUCOSIO EMATICO
- GLICOGENO MUSCOLARE



GLICOLISI – FASE 1



FASE 1

- È un costo di ATP
- Prepara la fase 2 (che rigenera ATP)
- GLUCOSIO deve essere Fosforilato (durante il riposo e nel post-prandiale) in G-6-P
- GLICOGENO già presente G-1/6-P

FIGURE 8: The glycolytic pathway. Note the duplication of phase 2 3-carbon metabolite reactions to account for the 6 carbons of each metabolite from phase 1.

GLICOLISI – FASE 2

FASE 2

- Porta ad un guadagno di 4 ATP
- Produce ACIDO PIRUVICO

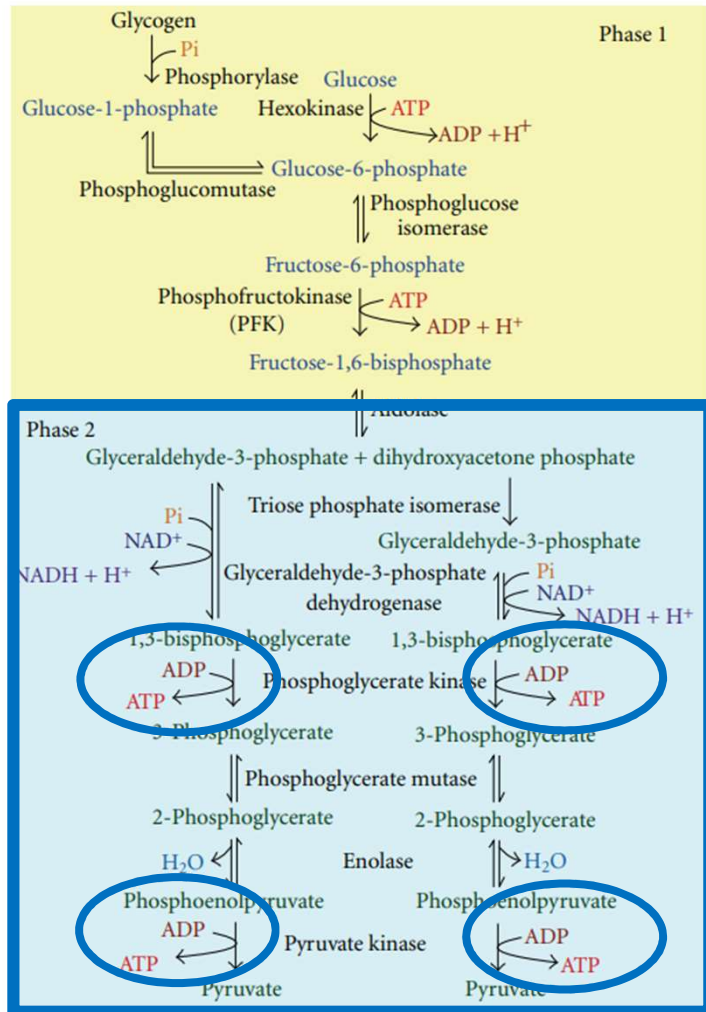


FIGURE 8: The glycolytic pathway. Note the duplication of phase 2 3-carbon metabolite reactions to account for the 6 carbons of each metabolite from phase 1.

GLICOLISI INIBITA A RIPOSO

AT REST
(glycolysis inhibited)

DIPENDE DAL RAPPORTO ATP/AMP

GLICOGENOGENESI

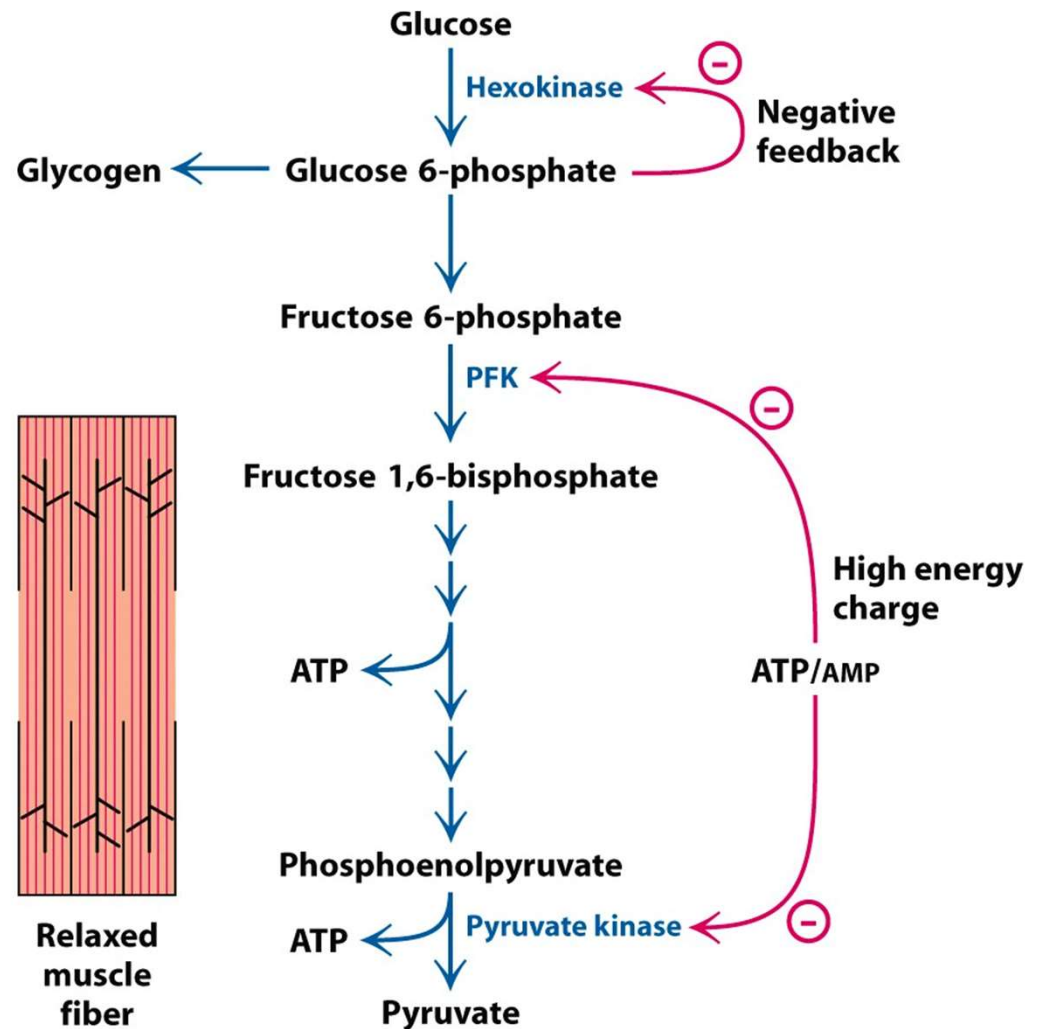


Figure 16.18 part 1

Biochemistry, Seventh Edition

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GLICOLISI STIMOLATA SOTTO SFORZO

DURING EXERCISE
(glycolysis stimulated)

DIPENDE DAL RAPPORTO ATP/AMP

GLICOGENOLISI

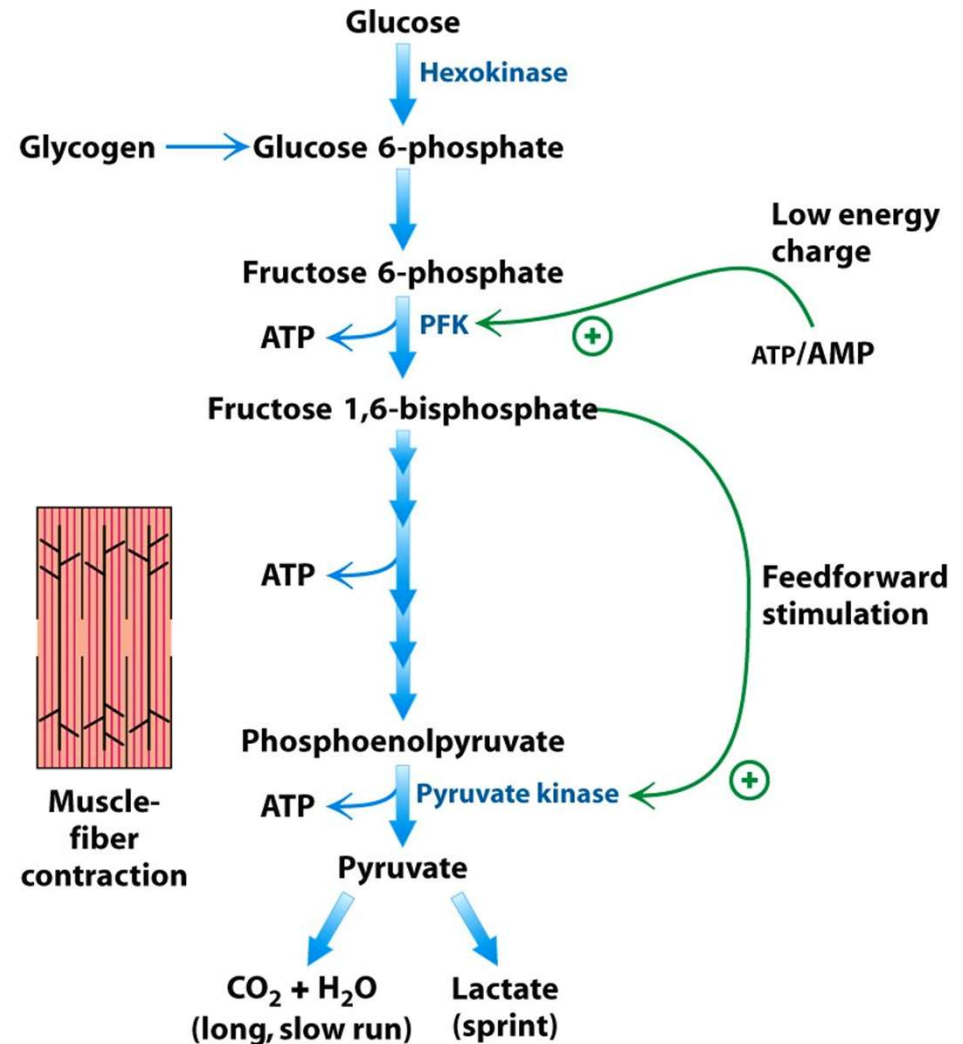


Figure 16.18 part 2
Biochemistry, Seventh Edition
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SISTEMA GLICOLITICO - GLICOGENO

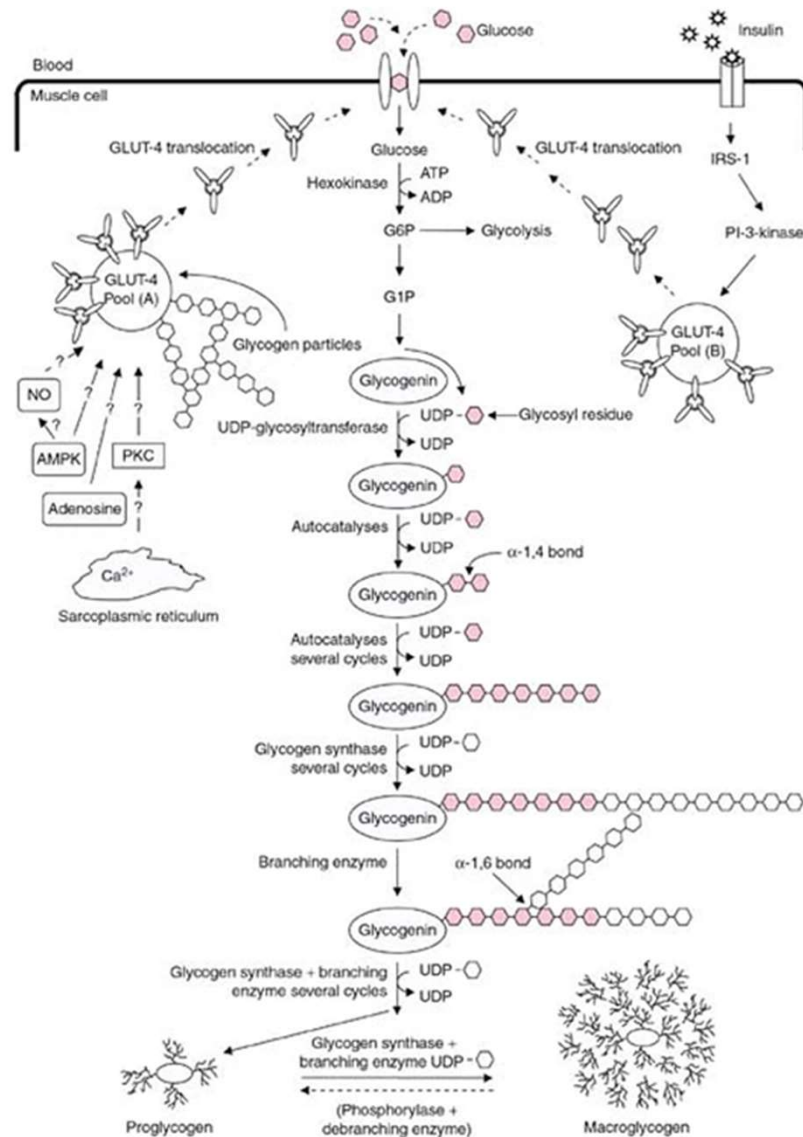
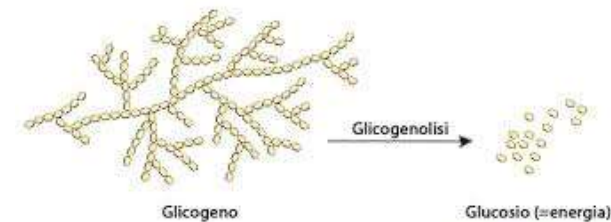


Fig. 1 (See legend on previous page.)

TABLE 2.1 Body Stores of Fuels and Associated Energy Availability

Location	g	kcal
CARBOHYDRATES		
Liver glycogen	110	451
Muscle glycogen	500	2,050
Glucose in body fluids	15	62
FAT		
Subcutaneous and visceral	7,800	73,320
Intramuscular	161	1,513
Total	7,961	74,833

Note. These estimates are based on a body weight of 65 kg (143 lb) with 12% body fat.



REVIEW ARTICLE

Open Access

Peak Week Carbohydrate Manipulation Practices in Physique Athletes: A Narrative Review

Kai A. Homer^{1*}, Matt R. Cross¹ and Eric R. Helms^{1,2}



GLICOGENO - MUSCOLARE

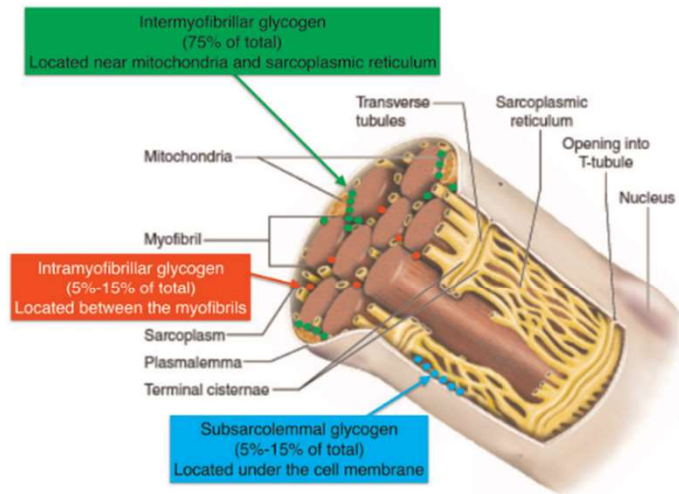


Figure 2 The intracellular locations of skeletal muscle glycogen. Image © Human Kinetics. Used with permission. Values for glycogen distribution are from Schweitzer et al (2017).⁴⁸

Table 1 The glycogen content of liver and muscle

Tissue	Average (g)	Normal range (g)
Muscle	500	300–700
Liver	80	0–160

Adapted from Hargreaves (2012).⁴⁰

**GLICOGENO OCCUPA
1-2 % VOLUME MIOCITA**

TABLE 2.1 Body Stores of Fuels and Associated Energy Availability

Location	g	kcal
CARBOHYDRATES		
Liver glycogen	110	451
Muscle glycogen	500	2,050
Glucose in body fluids	15	62
FAT		
Subcutaneous and visceral	7,800	73,320
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Total	7,961	74,833

Note. These estimates are based on a body weight of 65 kg (143 lb) with 12% body fat.

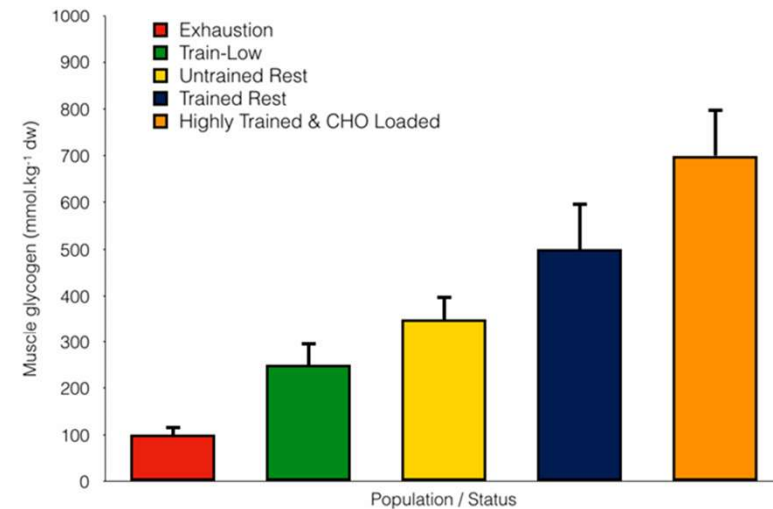


Figure 1. Variations in muscle glycogen storage according to fatigue status, training status and dietary carbohydrate (CHO) intake (data are compiled from males only and from several studies including Taylor et al. [30]; Bartlett et al. [31]; Arkinstall et al. [32]; Gollnick et al. [24]; Coyle et al. [8]).

GLICOGENO - EPATICO

Table 1 The glycogen content of liver and muscle

Tissue	Average (g)	Normal range (g)
Muscle	500	300–700
Liver	80	0–160

Adapted from Hargreaves (2012).⁴⁰

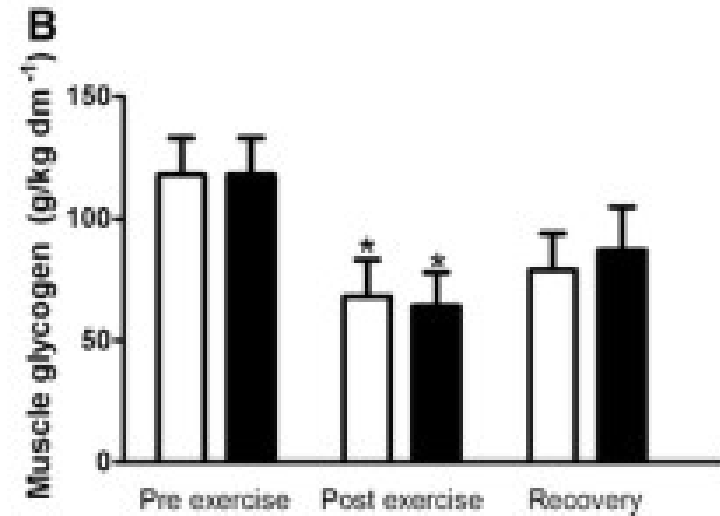
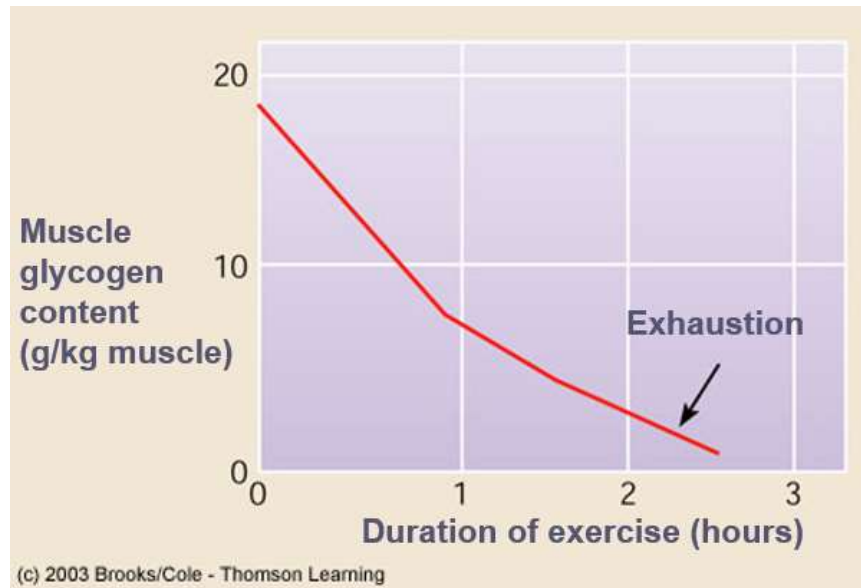
**GLICOGENO OCCUPA
5-6 % VOLUME EPATOCITA**

TABLE 2.1 Body Stores of Fuels and Associated Energy Availability

Location	g	kcal
CARBOHYDRATES		
Liver glycogen	110	451
Muscle glycogen	500	2,050
Glucose in body fluids	15	62
FAT		
Subcutaneous and visceral	7,800	73,320
Intramuscular	161	1,513
Total	7,961	74,833

Note. These estimates are based on a body weight of 65 kg (143 lb) with 12% body fat.

SISTEMA GLICOLITICO - GLICOGENO

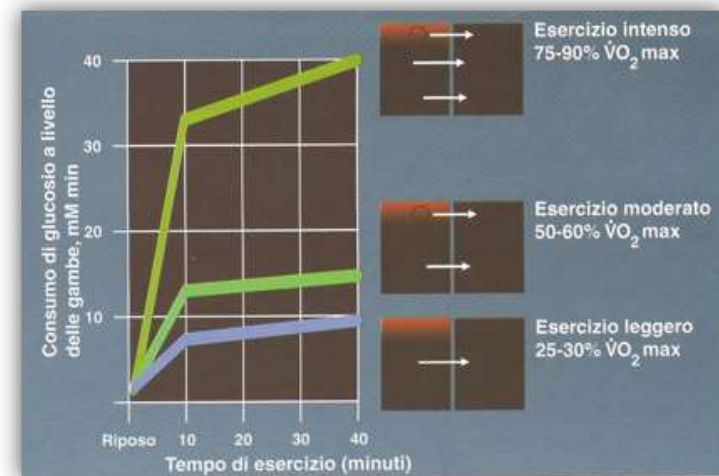


ESAURIMENTO GLICOGENO:

- Dopo 1 ora circa 55%
- Dopo 2 ore circa 100%

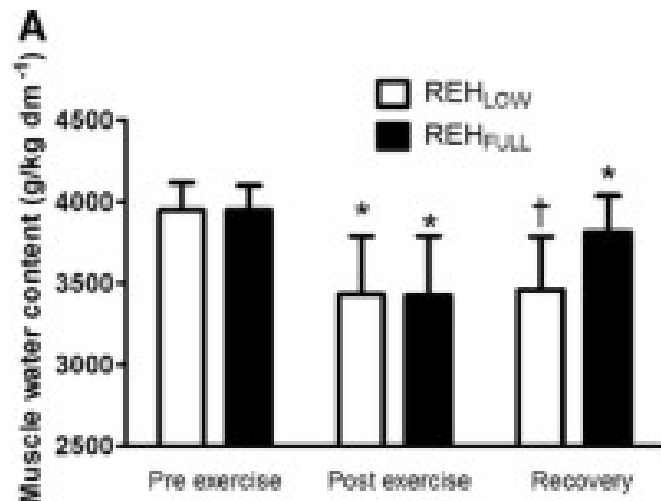
Relationship between muscle water and glycogen recovery after prolonged exercise in the heat in humans

Original Article | Published: 25 April 2015
Volume 115, pages 1919–1926, (2015) [Cite this article](#)



Alimentazione dello SPORT – [McArdle](#), [Katch](#) 2001

GLICOGENO E ACQUA



4. Muscle Glycogen and Body Hydration Status

An increase in stored glycogen is believed to be accompanied by an increase in body water content since glycogen is highly hydrophilic. Regarding the water bound to glycogen, Zants et al. initially estimated that 1 g of liver glycogen binds 3 g of water using the data provided by Pavy [34,35]. Subsequent studies showing the association between glycogen and body water are summarized in Table 1. Numerous animal studies assessing liver glycogen and water content indicated that 1 g of liver glycogen is bound to 1.6–3.8 g of water [36–39].

Concerning muscle glycogen in humans, Olsson and Saltin initially suggested that 1 g of muscle glycogen is bound to 3–4 g of water, based on the results of the tritium dilution method for detecting body water content and sampled using muscle biopsy for muscle glycogen content under glycogen-depleted and loaded conditions [10]. Furthermore, a study using magnetic resonance imaging presumed that intracellular water binding increases in glycogen-loaded muscles [47]. Our previous study using the deuterium dilution method for measuring total body water content also showed that body water content increased in glycogen-loaded conditions; the segmental BIA method indicated that the main component of increased body water was intracellular water (Figure 3) [11].

1 GR GLICOGENO RITIENE 3 GR DI ACQUA

Relationship between muscle water and glycogen recovery after prolonged exercise in the heat in humans

Original Article | Published: 25 April 2015
Volume 115, pages 1919–1926, (2015) [Cite this article](#)

Review

Muscle Glycogen Assessment and Relationship with Body Hydration Status: A Narrative Review

Keisuke Shiose ^{1,*}, Hideyuki Takahashi ² and Yosuke Yamada ³

GLICOGENO E ACQUA

5. Change in Body Composition during Carbohydrate Loading

After a few days of a high-carbohydrate diet (carbohydrate 8–12 g/kg/day), an increase in stored muscle glycogen of approximately 1.5–2.0-fold the normal level is observed.

In the classical carbohydrate loading procedure, muscle glycogen is initially depleted by exercise and a high-carbohydrate diet is consumed for 3 days following 2–3 days of a low-carbohydrate diet [51]. Interestingly, improved procedures have been described in subsequent studies [52,53]. A review article by Burke et al. provides good details [54].

During carbohydrate loading, it is generally understood that body weight increases by approximately 1.0–1.5 kg, mainly due to an increase in body water content, as mentioned in the previous section. However, changes in other body components are uncertain,

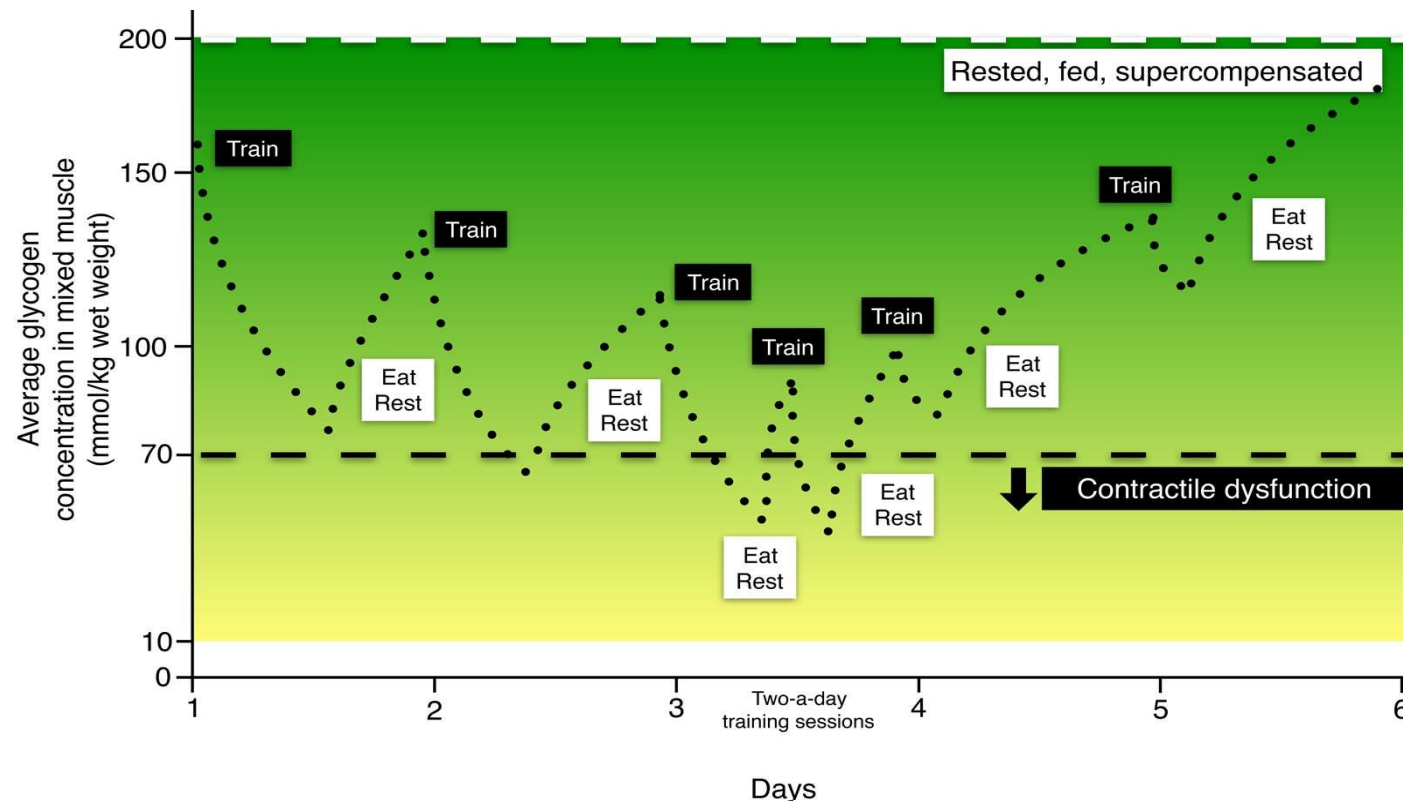
**DURANTE UN PERIODO DI CARICO GLUCIDICO
IL PESO CORPOREO PUÒ AUMENTARE ANCHE DI 1 o 2 KG**

Review

Muscle Glycogen Assessment and Relationship with Body Hydration Status: A Narrative Review

Keisuke Shiose ^{1,*}, Hideyuki Takahashi ² and Yosuke Yamada ³

GLICOGENO E SUPERCOMPENSAZIONE



Muscle glycogen levels can vary widely during training, only reaching supercompensated levels after a few days of rest and light training. In this example, muscle glycogen levels decline during training sessions and are partially restored during subsequent rest and after adequate carbohydrate intake. During hard 2-a-day training sessions (day 3), glycogen concentration can be lowered to the point at which contractile dysfunction (fatigue) occurs. Athletes typically train with muscle glycogen stores that are adequate to meet the demands of training (eg, between 75 and 150 mmol/kg wet weight) even though those stores might be considered suboptimal. Illustration based on data from Sherman and Wimer (1991).⁸⁵

GLICOGENO E SUPERCOMPENSAZIONE

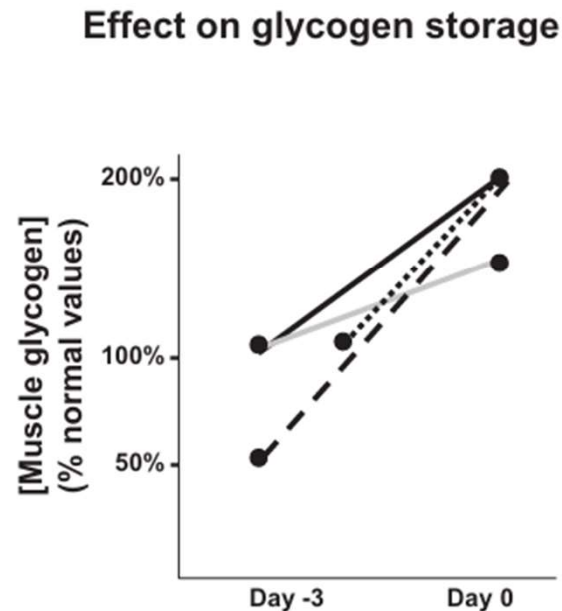
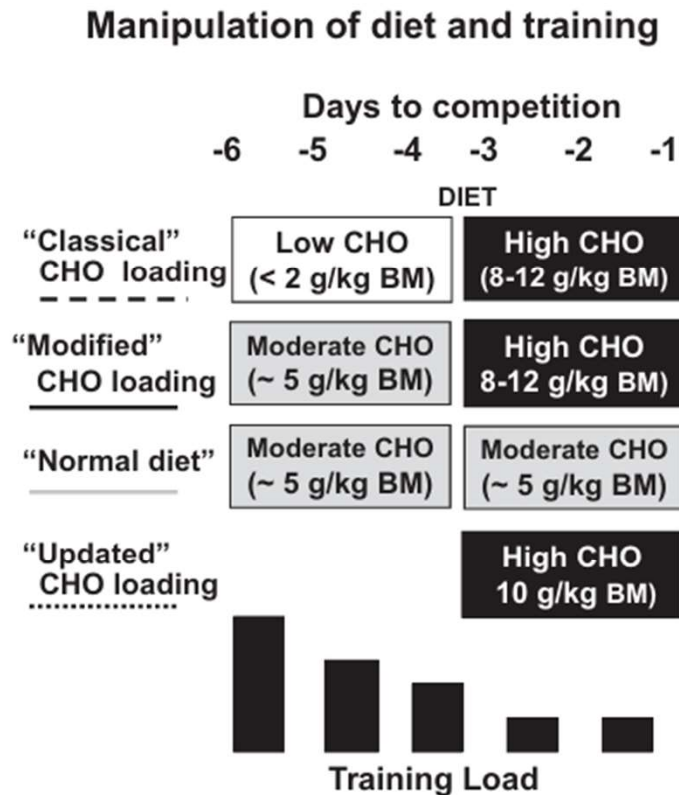


Fig. 1. Evolution of knowledge regarding protocols for carbohydrate (CHO) loading, as shown by diet and training manipulations in the 7 days before an endurance event. The "classical" loading protocol for glycogen supercompensation was developed by Bergström et al. (10) in untrained active individuals and confirmed in well-trained individuals by Sherman and colleagues (97). A "modified" protocol of high-CHO intake and exercise taper, deleting the depletion phase, was found to be similarly successful in athletes in the latter study (96). **More recent work suggests that the supercompensation occurs in 24–48 h of taper and high-CHO intake in well-trained individuals (25).** BM, body mass.

**BASTANO 2-3 GIORNI DI CARICO GLUCIDICO
PRE COMPETIZIONE**

REVIEW | *Recovery from Exercise*

Postexercise muscle glycogen resynthesis in humans

Louise M. Burke,^{1,2} Luc J. C. van Loon,^{1,3} and John A. Hawley^{1,4}

CARBOIDRATI PRE ESERCIZIO - GIORNI

- DIETA IN PERIODO DI ALLENAMENTO:
 - > HIGH-CHO 65% PER MANTENERE IL GLICOGENO MUSCOLARE
 - > ATTIVITÀ LIEVE : 3-5 GR/KG/24H
 - > ATTIVITÀ MODERATA : 5-7 GR/KG/24H
 - > ATTIVITÀ VIGOROSA : 6-10 GR/KG/24H
 - > ATTIVITÀ MOLTO VIGOROSA : 8-12 GR/KG/24H
- DIETA PRE COMPETIZIONE (3-5 GG PRIMA: FUELLING UP/CARICO GLUCIDICO):
 - > HIGH-CHO 70% PER MASSIMIZZARE IL GLICOGENO MUSCOLARE ED EPATICO E MANTENERE LA GLICEMIA DURANTE L'ESERCIZIO
 - > PER ESERCIZIO > 90 MIN : 10-12 GR/KG/24H
 - > PER ESERCIZIO < 90 MIN : 7-12 GR/KG/24H

Burke, L. M., Kiens, B., & Ivy, J. L. (2004). Carbohydrates and fat for training and recovery. *Journal of Sports Sciences*, 22, 15-30.

600 G Vs 100 GR (PER 6 GIORNI)

- PRE ES: ↑ LIVELLI DI GLICOGENO MUSCOLARE
- DURANTE ES: ↑ LIVELLI DI GLUCOSIO EMATICO
- POST ES: ↑ CONCENTRAZIONI MAGGIORI DI GLUCOSIO POST ESERCIZIO

Int.J.Sport.Nutr.Exerc.Metab, 2004 Feb;14(1):62-72.

The influence of low versus high carbohydrate diet on a 45-min strenuous cycling exercise.

Kavouras SA¹, Troup JP, Berning JR

CARBOIDRATI PRE ESERCIZIO: 3-4 h

- PERCHÉ? -> MIGLIORANO LA PERFORMANCE IN ESERCIZI DI ENDURANCE SUPERIORI A 90 MINUTI INCREMENTANDO LE SCORTE DI GLICOGENO EPATICO E MUSCOLARE
- QUANDO? -> 3-4 h prima
- QUANTO? -> 1-2 gr CHO/kg
- QUALI? -> basso IG attenuano l'iperglicemia e l'iperinsulinemia post-prandiale e riducono la soppressione dell'ossidazione degli acidi grassi liberi (FFA)

Pre-exercise carbohydrate and fat ingestion: effects on metabolism and performance

Mark Hargreaves, John A Hawley & Asker Jeukendrup
Pages 31-38 | Accepted 07 Aug 2003, Published online: 18 Feb 2007

PASTO RICCO DI CARBOIDRATI 4 H PRIMA DI UN ESERCIZIO FISICO INTENSO

- PASTO PRE ESERCIZIO: ↑ LIVELLI DI INSULINA ↑ LIVELLI DI GLUCOSIO EMATICO
- POCO PRIMA DELL'ESERCIZIO: = NORMALI LIVELLI DI INSULINA ↑ LIVELLI DI GLICOGENO MUSCOLARE
- DURANTE ESERCIZIO: ↓ CONCENTRAZIONE DI GLUCOSIO EMATICO ↓ FFA E GLIGEROLO

J Appl Physiol (1985), 1985 Aug;59(2):429-33.

Substrate usage during prolonged exercise following a preexercise meal.

Coyle EF, Coggan AR, Hemmert MK, Lowe RC, Walters TJ.

CARBOIDRATI PRE ESERCIZIO – 30/60 min

- **BASSO INDICE GLICEMICO:** LENTO E GRADUALE $\uparrow$ DELLA GLICEMIA
- **ALTO INDICE GLICEMICO:** RAPIDO $\uparrow$ DELLA GLICEMIA E RAPIDO RITORNO AI VALORI BASALI O INFERIORI (“*REBOUND HYPOGLYCEMIA*”)

TUTTAVIA, IPOGLICEMIA È TRANSITORIA (SCOMPARE NEI PRIMI 10 MINUTI DI ESERCIZIO E NON HA GROSSI EFFETTI SULLA PERFORMANCE)

CONSUMO DI CARBOIDRATI 30-60 MINUTI PRIMA DELL'ESERCIZIO COMPORTA:

- IPERGLICEMIA $\rightarrow$ IPERINSULINEMIA $\rightarrow$ IPOGLICEMIA REATTIVA (15-30' DOPO PASTO) CHE AUMENTATA LA CAPTAZIONE DI GLUCOSIO DA PARTE DEL MUSCOLO E LA RIDUZIONE DELLA PRODUZIONE DI GLUCOSIO EPATICO
- IPERISULINEMIA INIBISCE LA LIPOLISI E L'OSSIDAZIONE DEGLI ACIDI GRASSI CON CONSEGUENTE RAPIDA DEPLEZIONE DEL GLICOGENO MUSCOLARE

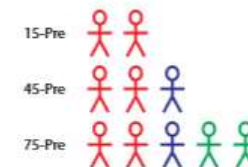
8 CICLISTI 75 GR GLUCOSIO: 15'- 45' – 75' PRE-ESERCIZIO

- CHI ASSUME 75 GR 75 MINUTI PRE-ES: 5 SU 7 SVILUPPANO IPOGLICEMIA
- CHI ASSUME 75 GR 45 MINUTI PRE-ES: 3 SU 7 SVILUPPANO IPOG
- CHI ASSUME 75 GR 15 MINUTI PRE-ES: 2 SU 7 SVILUPPANO IPOG

European Journal of Applied Physiology

January 2003, Volume 88, Issue 4-5, pp 453-458 | Cite as

Effects of timing of pre-exercise ingestion of carbohydrate on subsequent metabolism and cycling performance



CARBOIDRATI DURANTE-ESERCIZIO

- DIETA DURANTE ESERCIZIO FISICO:

-> FORNISCE UN'ADDIZIONALE FONTE DI RICARICA DI GLUCOSIO QUANDO LE FONTI DI GLICOGENO RISULTATO DEplete

-> PREVIENE LA RIDUZIONE DELLA CONCENTRAZIONE EMATICA DI GLUCOSIO

-> PREVIENE GLI EFFETTI DELLA CARENZA DI GLUCOSIO SUL SNC

- DIETA DURANTE ESERCIZIO FISICO:

-> PER ESERCIZI CHE DURANO < 60 MINUTI: CHO NON RICHIESTI

-> PER ESERCIZI CHE DURANO > 60 MINUTI (FINO A 150'): CHO 30-60 GR/H

-> PER ESERCIZI CHE DURANO > 150 MINUTI: > 90 GR/H

S94 A. E. Jeukendrup

Table 1. Recommendations for carbohydrate (CHO) intake during different endurance events.

Event	CHO required for optimal performance and minimizing negative energy balance	Recommended intake	CHO type	Single carbohydrate (e.g. glucose)	Multiple transportable carbohydrates (e.g. glucose:fructose)
< 30 min	None required	—	—	—	—
30–75 min	Very small amounts	Mouth rinse	Most forms of CHO	•	•
1–2 h	Small amounts	Up to 30 g · h ⁻¹	Most forms of CHO	•	•
2–3 h	Moderate amounts	Up to 60 g · h ⁻¹	Forms of CHO that are rapidly oxidized (glucose, maltodextrin)	o	•
> 2.5 h	Large amounts	Up to 90 g · h ⁻¹	Only multiple transportable CHO	—	•

Note: •, optimal; o, OK, but perhaps not optimal. These guidelines are intended for serious athletes, exercising at a reasonable intensity (> 4 kcal · min⁻¹). If the (absolute) exercise intensity is below this, the figures for carbohydrate intake should be adjusted downwards.

0 G Vs 37 GR Vs 74 GR Vs 111 GR (MITCHELL 1989)

- 74 gr di carboidrati per ora (in soluzione al 12%)
- AUMENTANO SIGNIFICATIVAMENTE LA PERFORMANCE IN ESERCIZIO DI 105 MINUTI

J Appl Physiol (1985), 1989 Nov;67(5):1843-9.

Influence of carbohydrate dosage on exercise performance and glycogen metabolism.

Mitchell JB¹, Costill DL, Houmard JA, Fink WJ, Pascoe DD, Pearson DR

Nutrients 2015, 7, 5733–5762; doi:10.3390/n7075249

nutrients
ISSN 2072-6643
www.mdpi.com/journal/nutrients

Review

Acute Effects of Carbohydrate Supplementation on Intermittent Sports Performance

Lindsay R. Baker ^{1,2,*}, Ian Ridd ³, Kimberly W. Stein ¹ and Aude E. Jeukendrup ¹

Journal of Sports Sciences, 2011, 29(3): 301–309

Review

Nutrition for endurance sports: Marathon, triathlon, and road cycling

ANDREW E. JEUKENDRUP

School of Sport and Exercise Sciences, University of Birmingham, Birmingham, UK

CARBOIDRATI DURANTE-ESERCIZIO

- QUALI CARBOIDRATI UTILIZZARE?

- > GLUCOSIO
- > MALTODESTRINE
- > FRUTTOSIO

Table 3. Considerations for carbohydrate ingestion during intermittent sports.

	Description	Examples
Amount:	30–60 g/h	0.5–1.0 L/h of a 6% carbohydrate solution
Type:	Rapidly digested, absorbed, and oxidized	The sugars glucose, sucrose, and maltose, as well as the glucose polymer maltodextrin and the starch amylopectin
Form:	Liquid, semisolid, or solid	Carbohydrate-electrolyte solution (<8%) or gel, chew, or bar with sufficient water to aid absorption
Other considerations:	Personal preference, experience, goals, and timing	Preference for a certain flavor and/or form of carbohydrate may promote intake and limit gastrointestinal distress

-> SINGOLA FONTE DI CARBOIDRATI: saturazione dei trasportatori specifici

-> DIVERSE FONTI DI CARBOIDRATI: possono aumentare l'assorbimento del singolo carboidrato (Shi 1995)

GLUCOSIO Vs GLUCOSIO + FRUTTOSIO (2:1)

- Glucosio (108 gr/h) da solo: non aumenta l'Indice di ossidazione
- Glucosio + Fruttosio (72+36 gr/h): aumentano l'Indice di ossidazione del 45%

Exogenous carbohydrate oxidation rates are elevated after combined ingestion of glucose and fructose during exercise in the heat

Rey L, P, G, Joutzans, Katie Underwood, Jant Adkins, Kevin Curran, Christopher H. Mann, and Asker E. Jeukendrup

CARBOIDRATI DURANTE-ESERCIZIO

- TRASPORTATORI DEL GLUCOSIO:

1) SGLT (Na⁺-dependent glucose co-transporters): nell'intestino si saturano a 60 gr/h

2) GLUT (Na⁺-independent sugar transporters)

Table 1. The glucose transporter (GLUT) family of facilitative sugar and polyol transporters (gene name SLC2A)

Isoform	Previous name	Class	Main tissue localization	Insulin sensitive?	Functional characteristics (transport)	Present in skeletal muscle?*	Present in white adipose tissue?*	Primary references
GLUT1	-	I	Erythrocytes, brain, ubiquitous	No	Glucose	Yes	Yes	Mueckler <i>et al.</i> 1985; Gould <i>et al.</i> 1991
GLUT2	-	I	Liver, pancreas, intestine, kidney	No	Glucose (low affinity); fructose	No	No	Fukumoto <i>et al.</i> 1988; Gould <i>et al.</i> 1991
GLUT3	-	I	Brain	No	Glucose (high affinity)	No	Yes (m)	Kayano <i>et al.</i> 1988; Gould <i>et al.</i> 1991
GLUT4	-	I	Heart, muscle, WAT, BAT, brain	Yes	Glucose (high affinity)	Yes	Yes	Fukumoto <i>et al.</i> 1989; James <i>et al.</i> 1989
GLUT5	-	II	Intestine, testes, kidney	No	Fructose; glucose (very low affinity)	Yes	Yes	Kayano <i>et al.</i> 1990; Davidson <i>et al.</i> 1992
GLUT6	GLUT 9	III	Brain, spleen, leucocytes	No	Glucose	No	n.d.	Doerge <i>et al.</i> 2000a; Lisinski <i>et al.</i> 2001
GLUT7	-	II	n.d.	n.d.	n.d.	n.d.	n.d.	Joost & Thorens, 2001
GLUT8	GLUT X1	III	Testes, brain and other tissues	No (yes in blastocytes)	Glucose	Yes (m)	Yes (m)	Carayannopoulos <i>et al.</i> 2000; Doerge <i>et al.</i> 2000b; Ibberson <i>et al.</i> 2000; Lisinski <i>et al.</i> 2001
GLUT9	GLUT X	II	Liver, kidney	n.d.	n.d.	No	n.d.	Phay <i>et al.</i> 2000
GLUT10	-	III	Liver, pancreas	No	Glucose	Yes (m)	n.d.	Dawson <i>et al.</i> 2001; McVie-Wylie <i>et al.</i> 2001
GLUT11†	GLUT 10	II	Heart, muscle	No	Glucose (low affinity); Fructose (long form)	Yes (m)	No	Doerge <i>et al.</i> 2001; Wu <i>et al.</i> 2002; Sasaki <i>et al.</i> 2001
GLUT12	GLUT 8	III	Heart, prostate, muscle, small intestine, WAT	Yes	n.d.	Yes	Yes	Rogers <i>et al.</i> 2002
HMIT	-	III	Brain	n.d.	H ⁺ -myo-inositol	No (m)	Yes (m)	Uldry <i>et al.</i> 2001

WAT, white adipose tissue; BAT, brown adipose tissue; m, mRNA only; n.d., not determined; HMIT, H⁺-coupled myo-inositol transporter.
 * The presence of each transporter in skeletal muscle and WAT is shown since these are the major sites of insulin-stimulated glucose uptake.
 † GLUT11 occurs in two splice variants: a short form (low-affinity glucose transport) and a long form (which may be a fructose transporter).

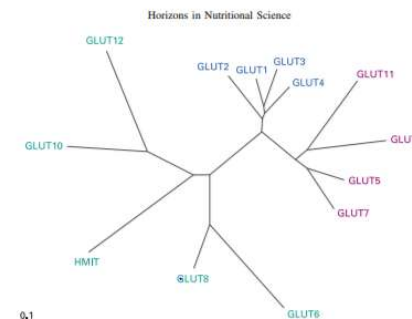


Fig. 1. Dendrogram of the glucose transporter (GLUT) family. An unrooted radial phylogram was drawn from a multiple sequence alignment of the thirteen members of the human GLUT family. The tree was constructed using neighbour-joining analysis of a distance matrix generated with PHYLIP software (Felsenstein, 1989). The three classes of GLUT proteins are colour-coded as: blue, class I; red, class II; green, class III. The scale bar represents 0.1 substitutions per amino acid position. HMIT, H⁺-coupled myo-inositol transporter.

- Usando multipli trasportatori (SGLT1 per Glucosio e GLUT5 per il fruttosio) avviene un incremento dell'Indice di assorbimento dei Carboidrati con conseguente aumento dell'Indice di Ossidazione dei Carboidrati)
- Nel caso di prolungati esercizi (< 2.5 h) è raccomandato utilizzare 90 gr/h di Carboidrati in rapporto di 2:1 in favore del Glucosio (Es. glucosio:fruttosio=2:1).
- SGLT1: nell'intestino si saturano a 60 gr/h
- Gli eccedenti 30 gr/h devono provenire da altre fonti di Carboidrati (Fruttosio, Galattosio...)

Horizons in Nutritional Science

Glucose transporters (GLUT and SGLT): expanded families of sugar transport proteins

I. Stuart Wood* and Paul Trayhurn
 Liverpool Centre for Nutritional Genomics, Neuroendocrine & Obesity Biology Unit, Department of Medicine,
 University of Liverpool, University Clinical Departments, Liverpool L69 3GA, UK

Curr Opin Clin Nutr Metab Care. 2010 Jul;13(4):452-7. doi: 10.1097/MCO.0b013e328339de9f.

Carbohydrate and exercise performance: the role of multiple transportable carbohydrates.

Jeukendrup AE¹

CARBOIDRATI DURANTE-ESERCIZIO

Carbohydrate feeding during exercise 81

- QUALI CARBOIDRATI UTILIZZARE?

- > GLUCOSIO
- > MALTODESTRINE
- > FRUTTOSIO

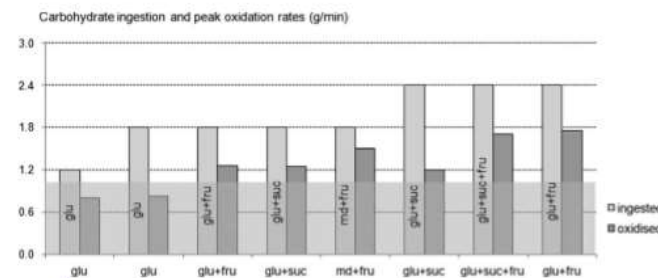


Figure 1. Oxidation of ingested carbohydrate. This figure is compiled from a number of studies in our laboratory investigating the oxidation of exogenous (ingested) carbohydrate during exercise (Jentjens & Jeukendrup, 2005; Jentjens *et al.*, 2004a, 2004b, 2005; Wallis *et al.*, 2005). The bars on the left indicate the amount of carbohydrate ingested (g min^{-1}) and those on the right the exogenous carbohydrate oxidation rate. The shaded area shows the maximum range of oxidation rates that can be expected from a single carbohydrate with a maximum oxidation rate of about 1 g min^{-1} . When multiple transportable carbohydrates are ingested at high rates, the oxidation rates can easily exceed 1 g min^{-1} and the highest oxidation rates were found with a mixture of glucose and fructose (1.75 g min^{-1}).

- > SINGOLA FONTE DI CARBOIDRATI: saturazione dei trasportatori specifici (SGLT1)
- > DIVERSE FONTI DI CARBOIDRATI: possono aumentare l'assorbimento del singolo carboidrato (Shi 1995)

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First published November 10, 2005; doi:10.1111/j.1365-2013.2005.01502.x
 Exogenous carbohydrate oxidation rates are elevated after combined ingestion of glucose and fructose during exercise in the heat

Ray L, P, G, Jentjens, Katie Underwood, Jant Adams, Kevin Cureth, Christopher H. Mann, and Asker E. Jeukendrup

CARBOIDRATI POST-ESERCIZIO

PER AVERE UNA BUONA RISINTESI DEL GLICOGENO

- 1,5 gr CHO/kg 30 minuti dopo la fine dell'esercizio
- Si possono utilizzare sia forme solide che liquide
- Carboidrati con ELEVATO INDICE GLICEMICO incrementano maggiormente il glicogeno muscolare
- Prediligere fonti miste di Carboidrati
- CHO associati a PROTEINE promuovono la ri-sintesi di glicogeno e l'attenuazione dei danni muscolari

Muscle glycogen storage after prolonged exercise: effect of the glycemic index of carbohydrate feedings

L. M. Burke, G. R. Collier, and M. Hargreaves
1 AUG 1993 / <https://doi.org/10.1152/jappl.1993.75.2.1019>

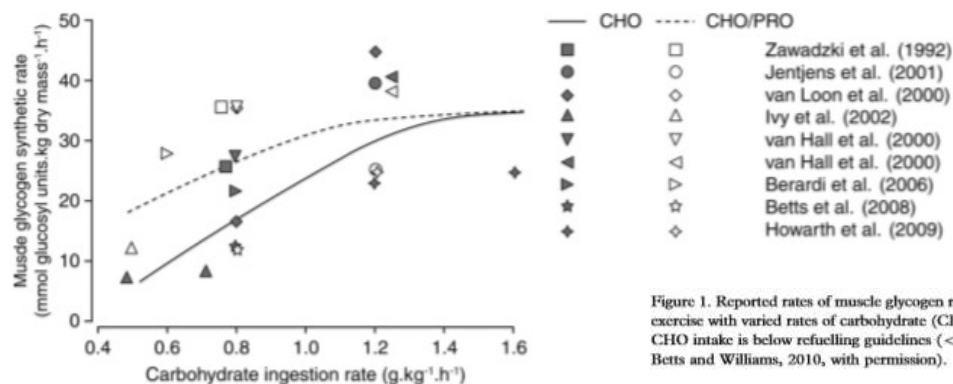


Figure 1. Reported rates of muscle glycogen resynthesis across nine studies that have compared muscle glycogen storage over >2-6 h post-exercise with varied rates of carbohydrate (CHO) intake, with or without co-ingestion of protein (PRO). This provides evidence that when CHO intake is below refuelling guidelines ($<1.2 \text{ g.kg}^{-1}.\text{h}^{-1}$), the addition of protein ($\approx 20\text{g}$) enhances glycogen synthesis. (adapted from Betts and Williams, 2010, with permission).

IN ASSENZA DI SEVERI DANNI MUSCOLARI LE RISERVE DI GLICOGENO SI NORMALIZZANO IN 24H SE AVVIENE UN ADEGUATO INTROITO DI CHO

GLUCOSIO Vs FRUTTOSIO DOPO 90 MINUTI DI ESERCIZIO

- Nel MUSCOLO: dopo 2h il GLUCOSIO migliora il ripristino di Glicogeno meglio rispetto al Fruttosio
- Nel FEGATO: dopo 2h GLUCOSIO e FRUTTOSIO promuovono lo stesso rapido accumulo di Glicogeno

Ann. Nutr. Metab. 31: 126-132 (1987)
Effects of Glucose or Fructose Feeding on Glycogen Repletion in Muscle and Liver after Exercise or Fasting
Robert K. Carter, Russell M. Lander, Patrick E. Kim
Exercise Biochemistry Laboratory, Brigham Young University, Provo, Utah, USA

CARBOIDRATI POST-ESERCIZIO

LA “SUPERCOMPENSAZIONE POST - ESERCIZIO DI GLICOGENO” È IL MECCANISMO ATTRAVERSO IL QUALE AVVIENE UN’IMPORTANTE AUMENTO DELLE CONCENTRAZIONI DI GLICOGENO MUSCOLARE IN RISPOSTA AL CARICO DI CARBOIDRATI CONSEGUENTE ALLA DEPLEZIONE DI GLICOGENO POST-ESERCIZIO FISICO.

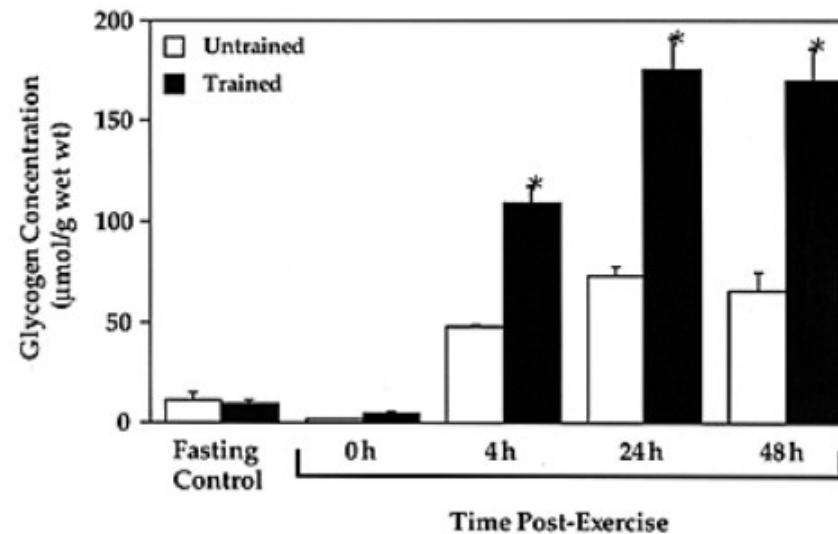


Figure 6. Time course of epitrochlearis muscle glycogen accumulation in trained (solid bars) and untrained (open bars) rats after a glycogen-depleting bout of exercise. After an overnight fast, animals performed a bout of swimming exercise, then were allowed to recover with free access to standard rodent chow and 5% sucrose in their drinking water. Muscles were harvested at the indicated times. *P<0.001 versus untrained. From reference (186).

L’ESERCIZIO INDUCE UN AUMENTO DI **GLUT4** NEL MUSCOLO SCHELETRICO CON CONSEGUENTE AUMENTO DELLA CAPACITÀ DI TRASPORTO DEL GLUCOSIO.

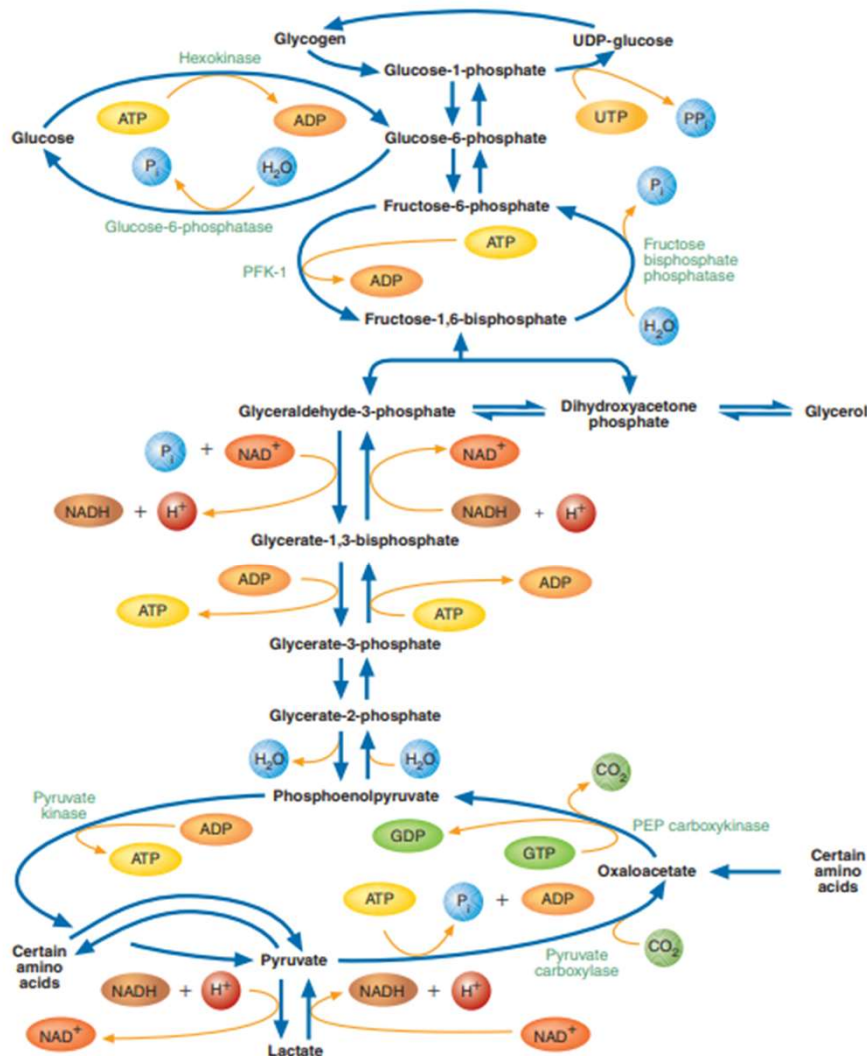
Am J Physiol Endocrinol Metab 286: E729–E736, 2003.
First published June 10, 2003; 10.1152/ajpendo.00216.2003.

Prevention of glycogen supercompensation prolongs the increase in muscle GLUT4 after exercise

Pablo M. Garcia-Roves, Dong-Ho Han, Zheng Song,
Terry E. Jones, Kathleen A. Hucker, and John O. Holloszy
Department of Internal Medicine, Washington University School of Medicine, St. Louis, Missouri 63110

GLUCONEOGENESI

È LA FORMAZIONE DI GLUCOSIO A PARTIRE DA PRECURSORI NON CARBOIDRATI

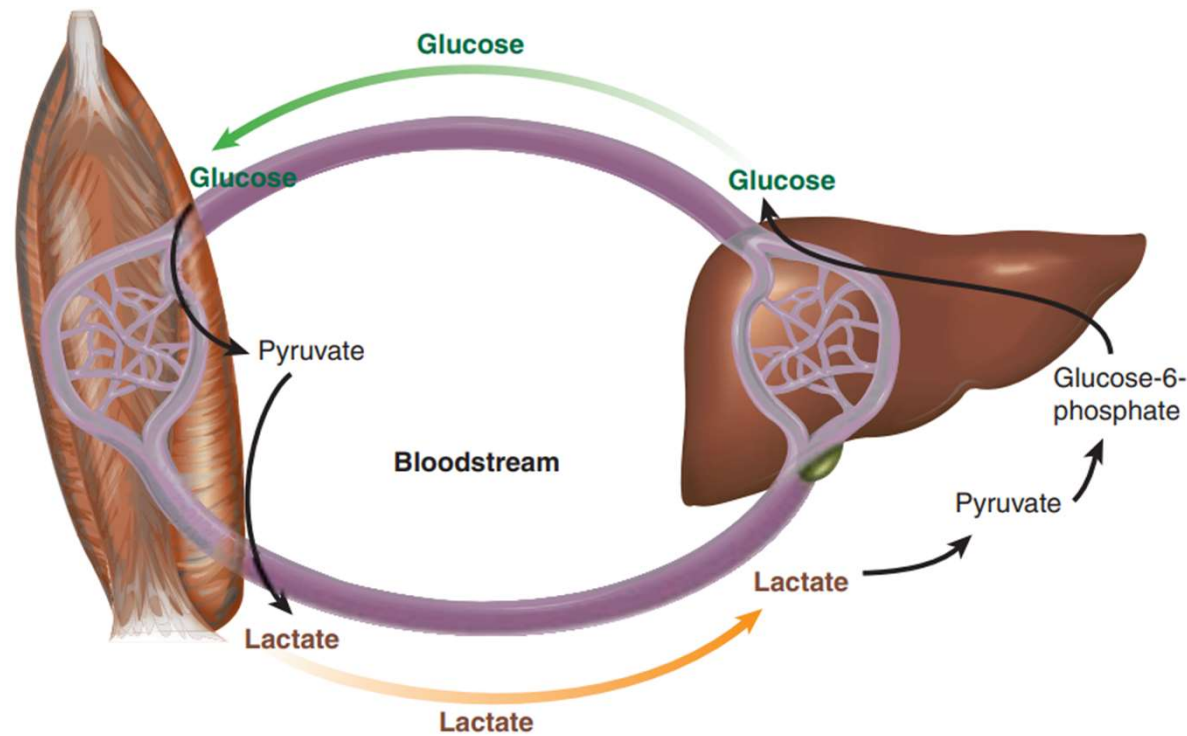


- AVVIENE QUANDO CI SONO BASSI LIVELLI DI GLICEMIA E IL GLICOGENO EPATICO È DEPLETO
- 7/10 REAZIONI DELLA GLICOLISI SONO REVERSIBILI
- 3/10 REAZIONI IRREVERSIBILI VENGONO BYPASSATE DA ALTRE REAZIONI
- SUBSTRATI DELLA GLUCONEOGENESI:
 - 1) AMINO ACIDI GLUCOGENICI
 - 2) ACIDO LATTICO (MUSCOLO E GR)
 - 3) GLICEROLO (METABOLISMO GRASSI)

FIGURE 8.10
Carbohydrate Metabolism: Gluconeogenesis and Glycolysis

GLUCONEOGENESI - CICLO DI CORI

ACIDO LATTICO TRASPORTATO NEL FEGATO DOVE VIENE CONVERTITO IN ACIDO PIRUVICO DALLA LATTATO DEIDROGENASI QUINDI IN GLUCOSIO.



GLICOLISI

PRODUZIONE DI ATP DOPO 10-15'' MA MANTENUTA PER PIÙ TEMPO

10'' ESERCIZIO MASSIMA INTENSITÀ: PCR 53% - GLICOLISI 44% - MITOCONDRI 3%

30'' ESERCIZIO MASSIMA INTENSITÀ: PCR 23% - GLICOLISI 49% - MITOCONDRI 29%

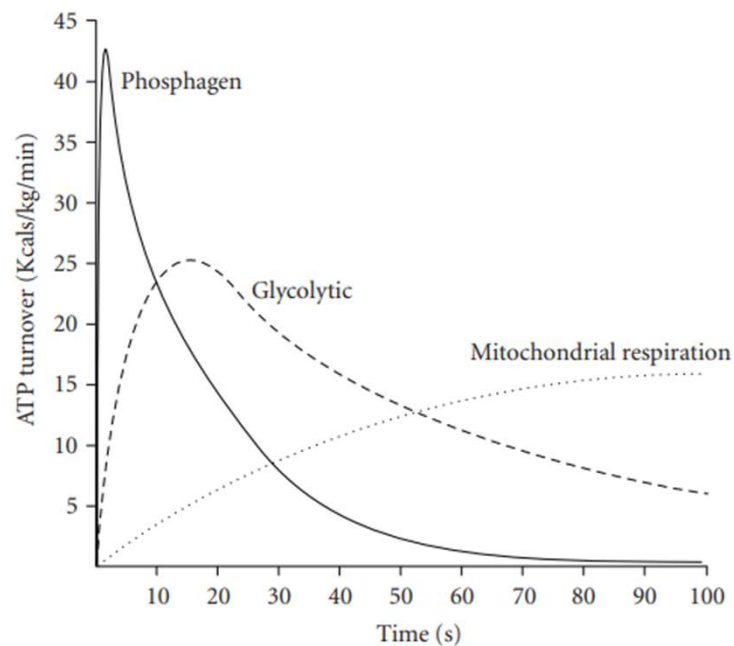
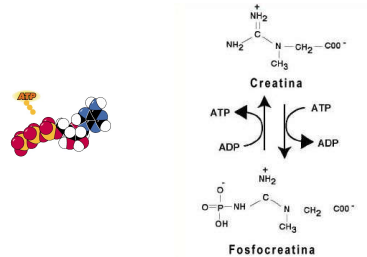
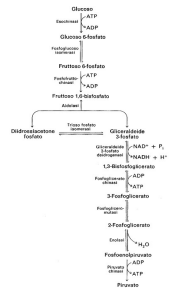
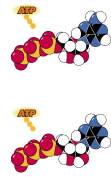


FIGURE 9: Energy system interaction and the differences in rates of ATP turnover during short term intense exercise to fatigue. The data presented is original, theoretical, and based on the authors' assessment of contemporary research evidence.

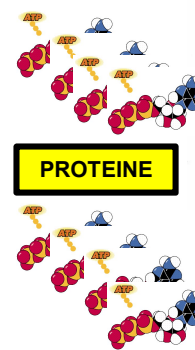
SISTEMI ENERGETICI



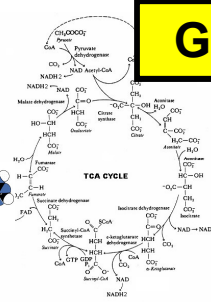
F-CREATINA



GLUCOSIO

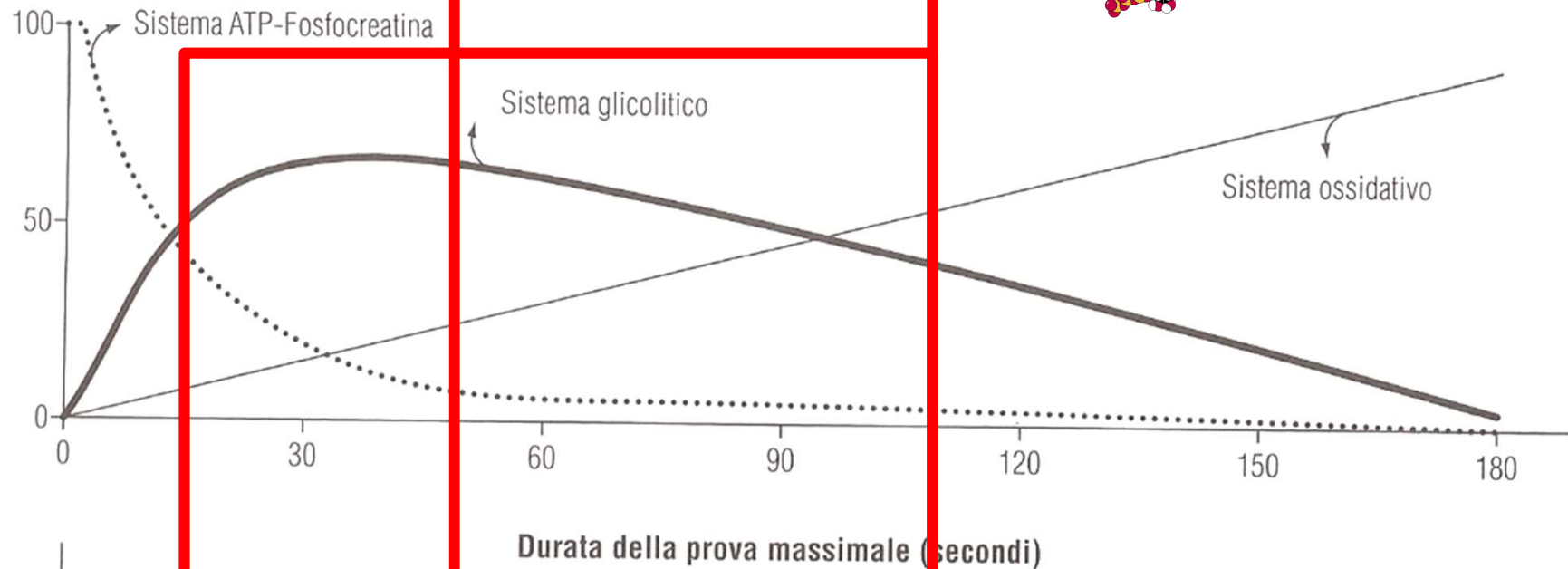


PROTEINE



GRASSI

GLUCOSIO



SISTEMI ENERGETICI

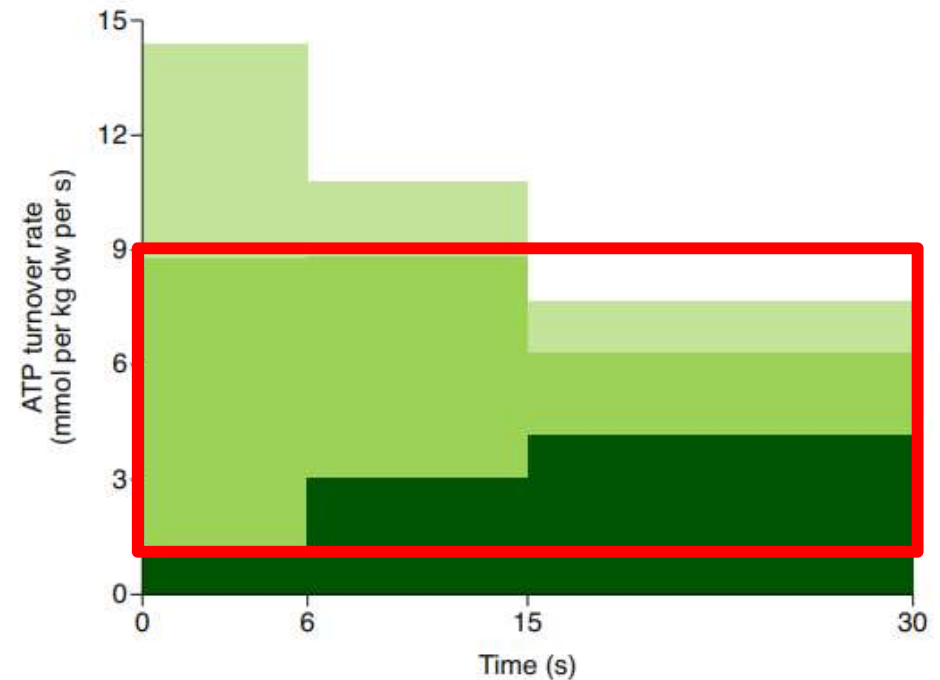
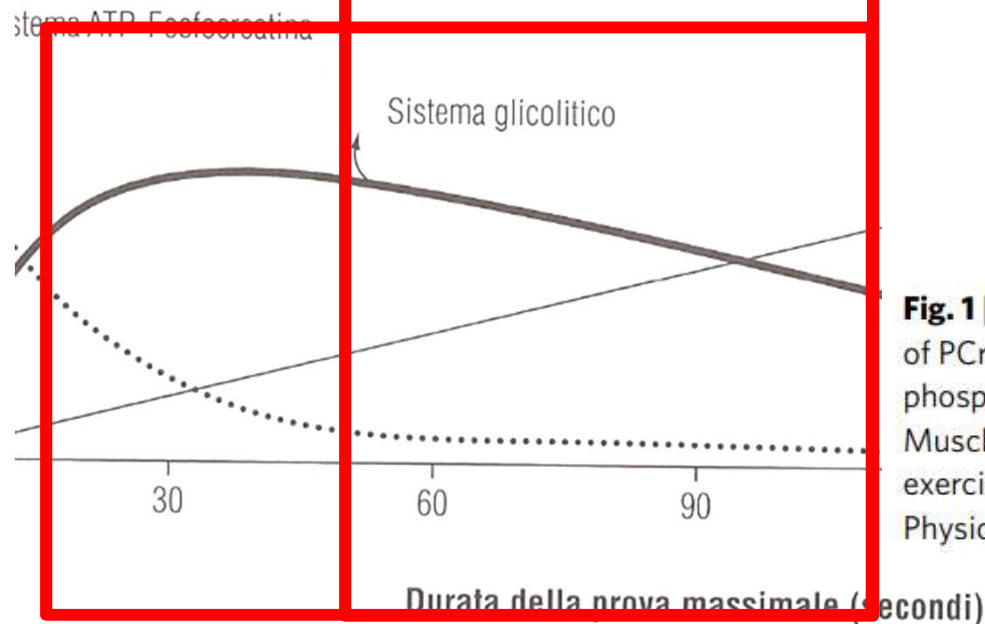
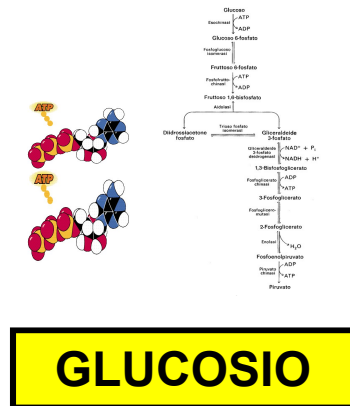
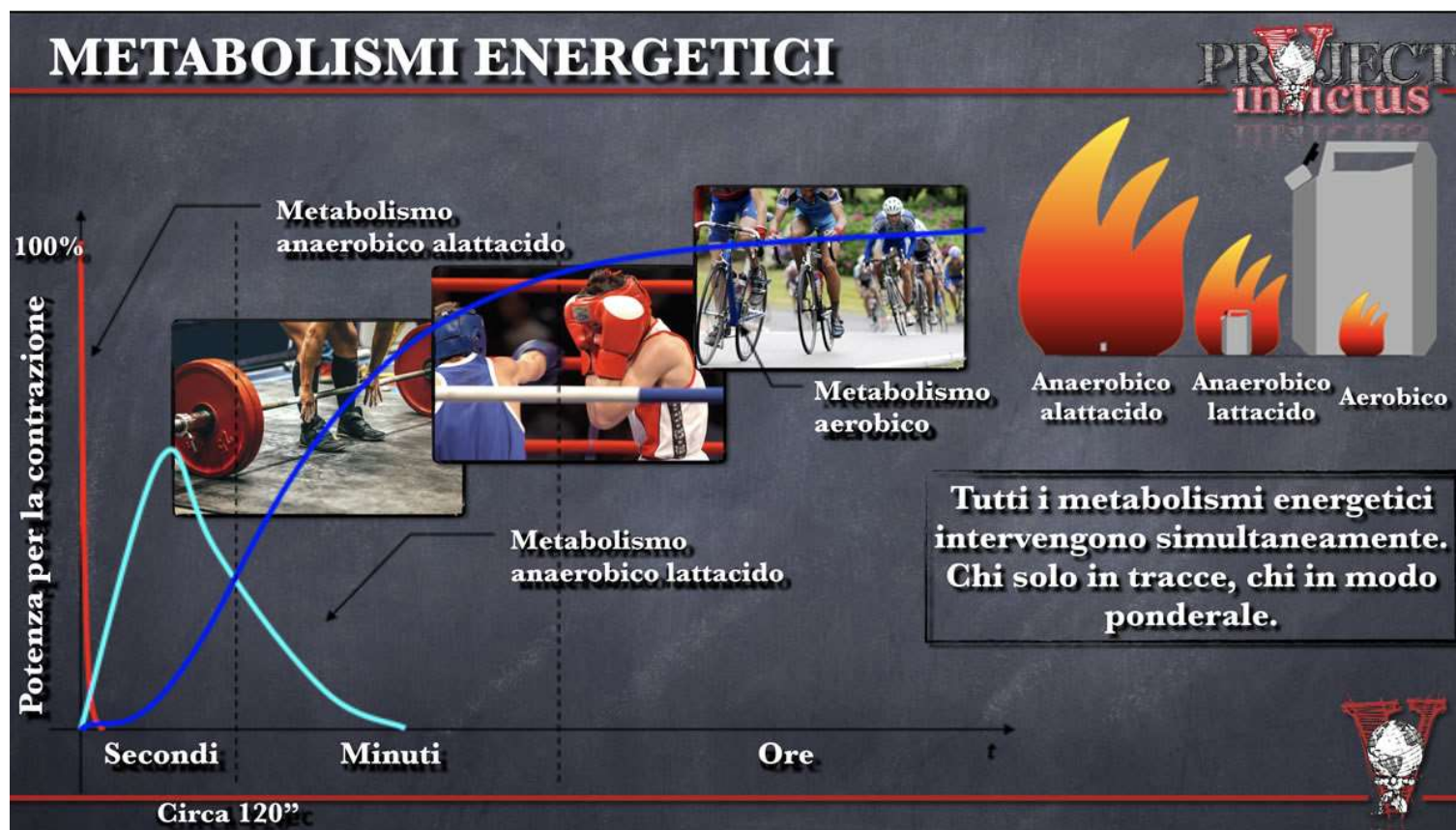


Fig. 1 | Muscle energy metabolism during intense exercise. Contribution of PCr (light green), glycolysis (medium green) and oxidative phosphorylation (dark green) to ATP turnover during maximal exercise. Muscle samples were obtained before and during 30 s of all-out cycling exercise. Dw, dry weight. Adapted with permission from ref. ⁴, American Physiological Society.

SISTEMA GLICOLITICO



SISTEMA AEROBICO (OSSIDATIVO)

RESPIRAZIONE MITOCONDRIALE

LA RESINTESI DI ATP NEI MITOCONDRI AVVIENE IN PRESENZA DI SUFFICIENTE QUANTITÀ DI OSSIGENO

LE FONTI DI ENERGIA DERIVANO DA:

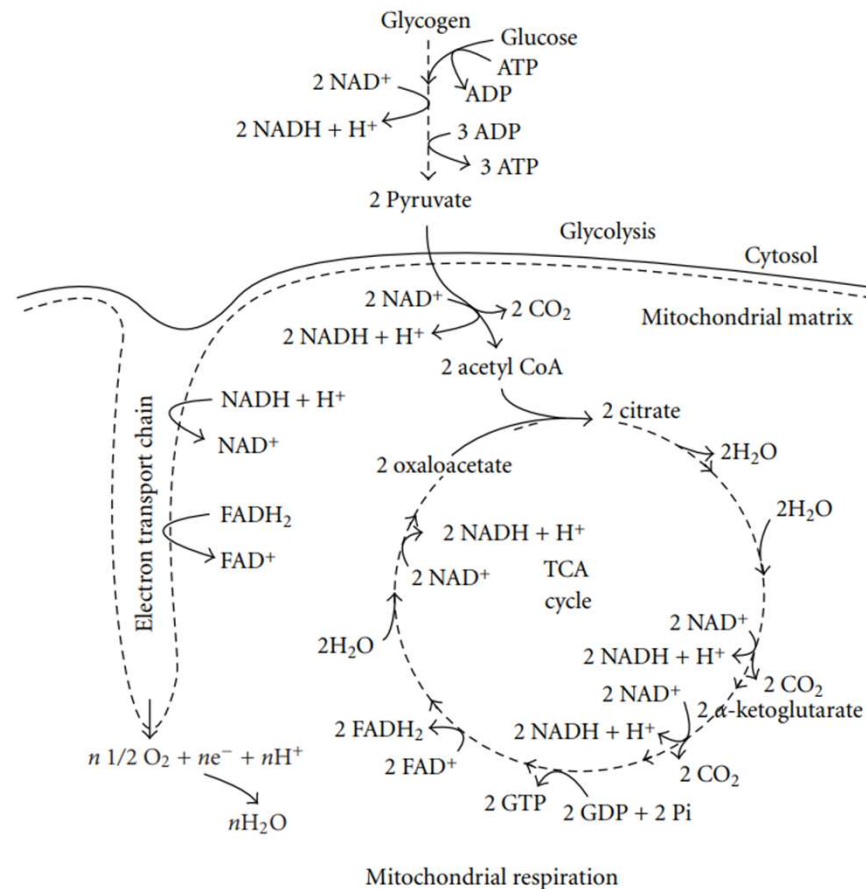
1) FONTI MUSCOLARI: GLICOGENO E ACIDI GRASSI LIBERI

2) FONTI EXTRAMUSCOLARI:

- ACIDI GRASSI LIBERI (TESSUTO ADIPOSO)
- GLUCOSIO EMATICO (DIETA E FEGATO)

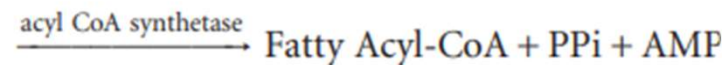
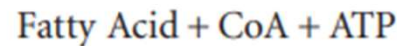
OSSIDAZIONE DI CARBOIDRATI

AVVIENE QUANDO PIRUVATO E I PRODOTTI DELLA GLICOLISI (NADH E FADH₂) VENGONO TRASFERITI ALL'INTERNO DEI MITOCONDRI (ERRONEAMENTE CHIAMATA "GLICOLISI AEROBICA")



OSSIDAZIONE DI LIPIDI

TUTTI GLI ACIDI GRASSI CON + DI 15 CARBONI RICHIEDONO ATTIVAZIONE PER ESSERE TRASPORTATI NEL MITOCONDRIO



PER PASSARE LA MEMBRANA MITOCONDRIALE LE MOLECOLE GRASSI ACYL-CoA UTILIZZANO LA NAVETTA CARNITINA

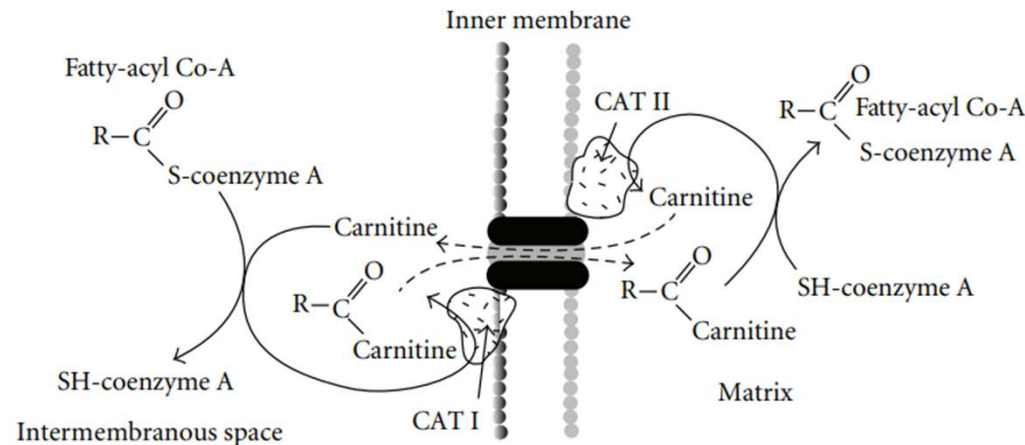
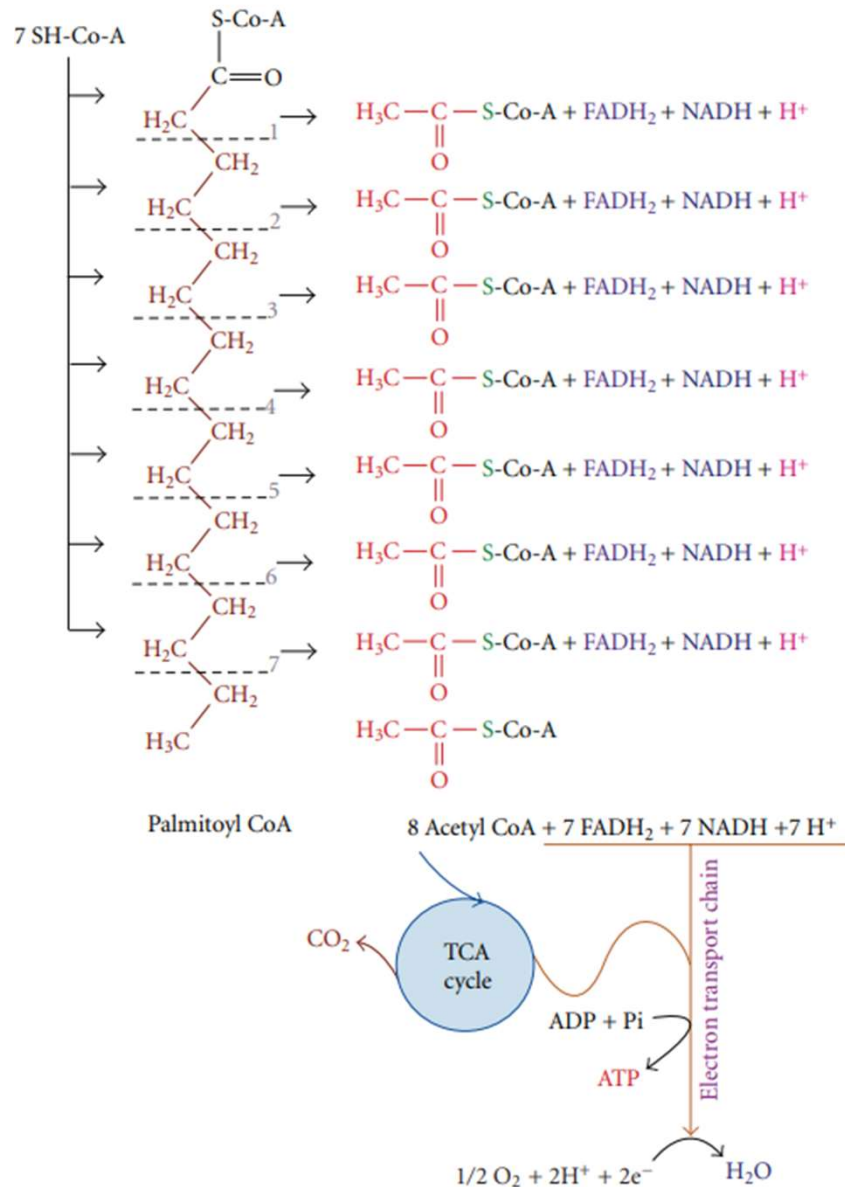


FIGURE 13: Transport of long chain (>15 carbons) fatty acyl CoA molecules into the mitochondria via the carnitine shuttle.

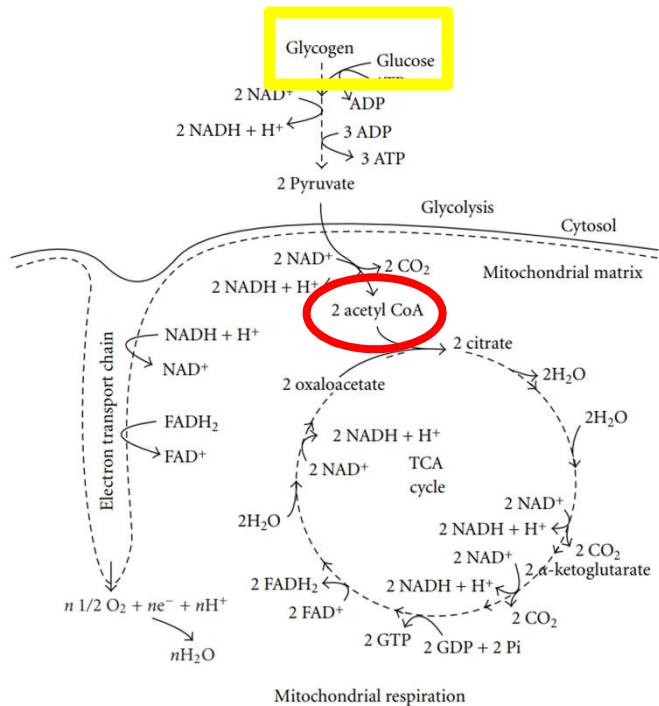
OSSIDAZIONE DI LIPIDI



UNA VOLTA ARRIVATO ALL'INTERNO DEL MITOCONDRIO GLI ACIDI GRASSI VENGONO DEGRADATI 2 CARBONI ALLA VOLTA (β OSSIDAZIONE) RILASCIANDO:

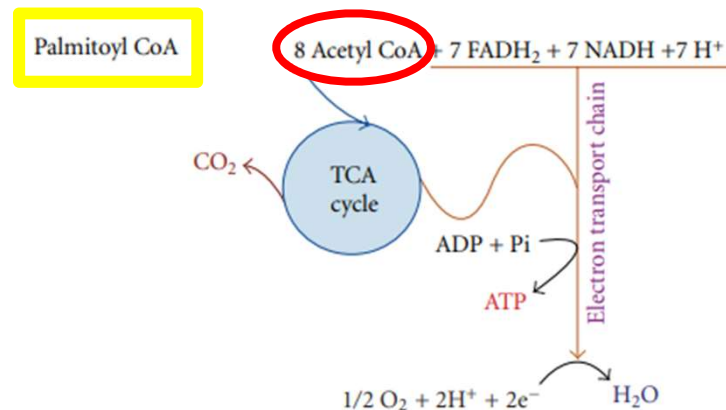
- **Acetil CoA**
- **NADH**
- **FADH**

LIPIDI Vs CARBOIDRATI



UNA VOLTA ARRIVATO ALL'INTERNO DEL MITOCONDRIO GLI ACIDI GRASSI VENGONO DEGRADATI 2 CARBONI ALLA VOLTA (β OSSIDAZIONE) RILASCIANDO:

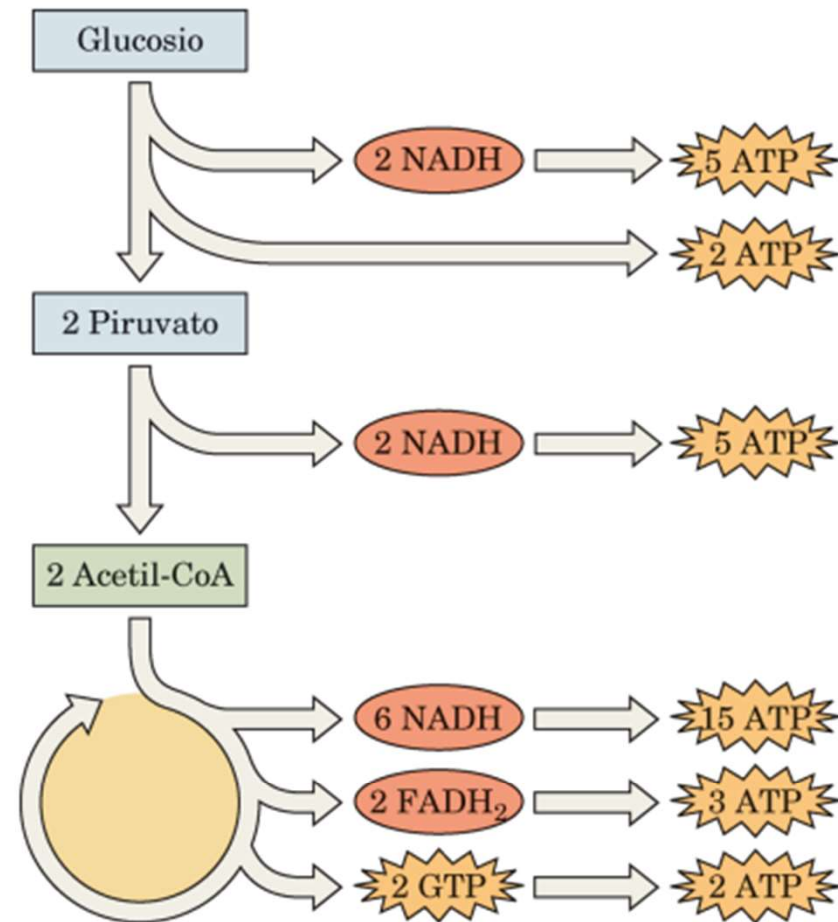
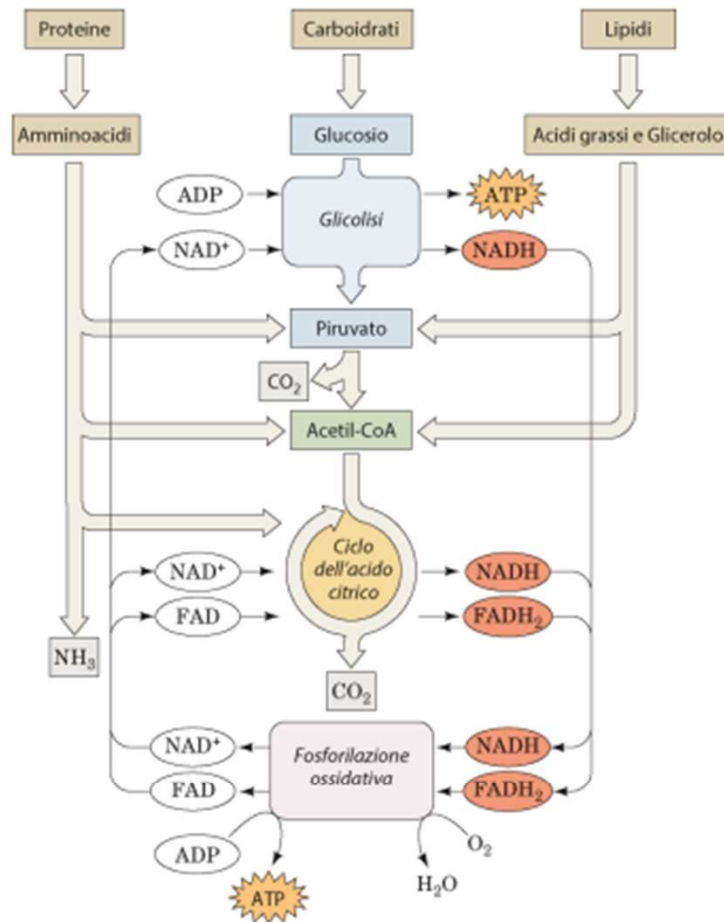
- **Acetil CoA**
- **NADH**
- **FADH**



OSSIDAZIONE DI AMINOACIDI

IN CASO DI ESERCIZIO INTENSO E PROLUNGATO ASSOCIATO
A INADEGUATE FONTI DI CARBOIDRATI AVVIENE UNA
MAGGIORE DEGRADAZIONE PROTEICA E OSSIDAZIONE DEGLI
AMINOACIDI.

FOSFORILAZIONE OSSIDATIVA

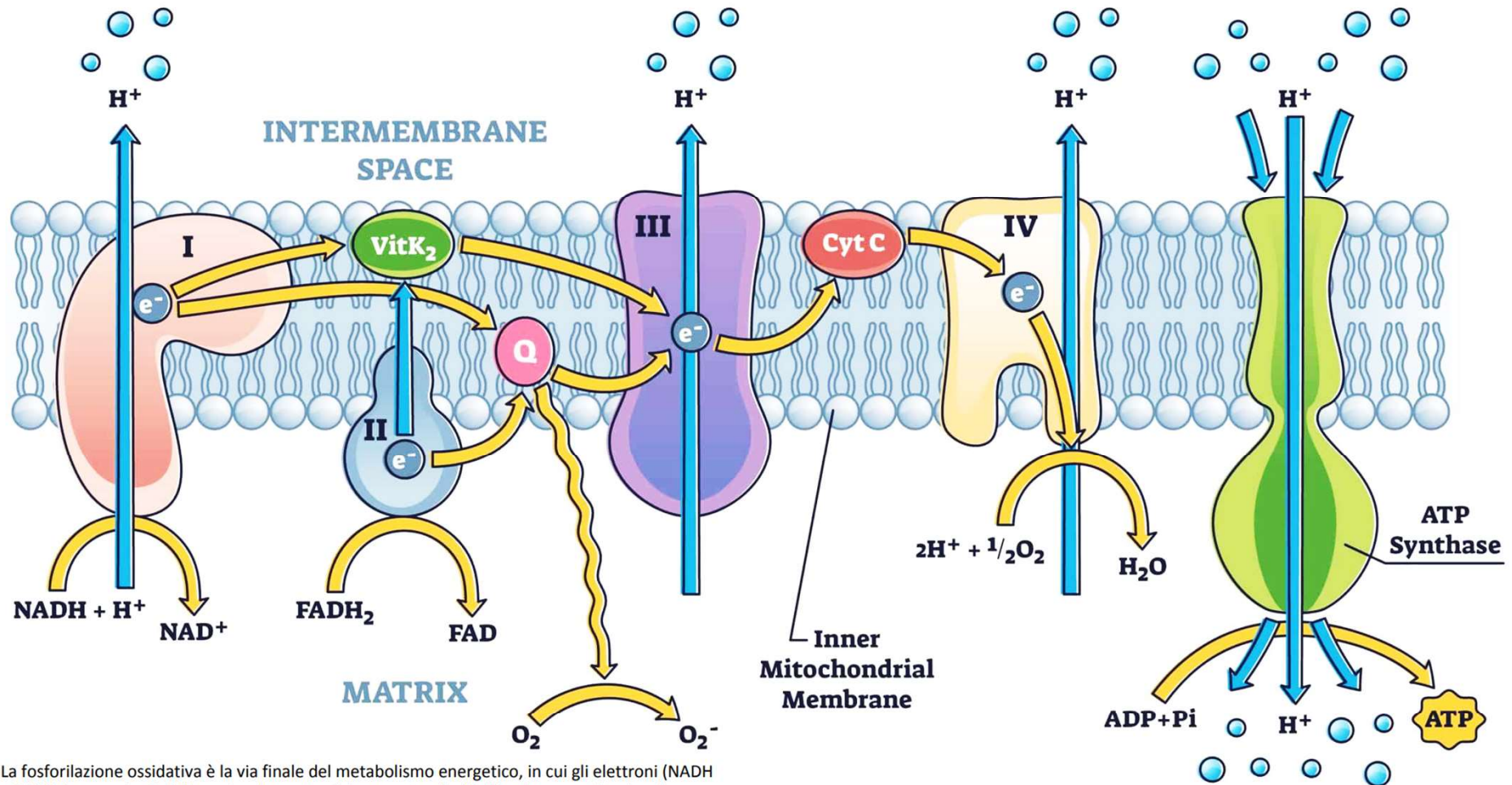


La fosforilazione ossidativa è la via finale del metabolismo energetico, in cui gli elettroni (NADH e FADH₂) provenienti dalle molecole energetiche sono trasferiti all'ossigeno molecolare in modo da alimentare la sintesi di ATP.

È la fonte principale di ATP negli animali (p.e., essa produce 26 delle 30 molecole di ATP generate dalla ossidazione completa del glucosio).

La **fosforilazione ossidativa** è un processo che genera ATP tramite il trasferimento di elettroni dai coenzimi redox ridotti fino all'ossigeno, mediante una serie di **trasportatori**.

FOSFORILAZIONE OSSIDATIVA

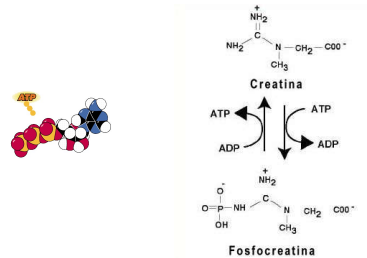


La fosforilazione ossidativa è la via finale del metabolismo energetico, in cui gli elettroni (NADH e FADH_2) provenienti dalle molecole energetiche sono trasferiti all'ossigeno molecolare in modo da alimentare la sintesi di ATP.

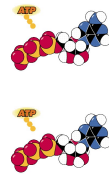
È la fonte principale di ATP negli animali (p.e., essa produce 26 delle 30 molecole di ATP generate dalla ossidazione completa del glucosio).

La **fosforilazione ossidativa** è un processo che genera ATP tramite il trasferimento di elettroni dai coenzimi redox ridotti fino all'ossigeno, mediante una serie di **trasportatori**.

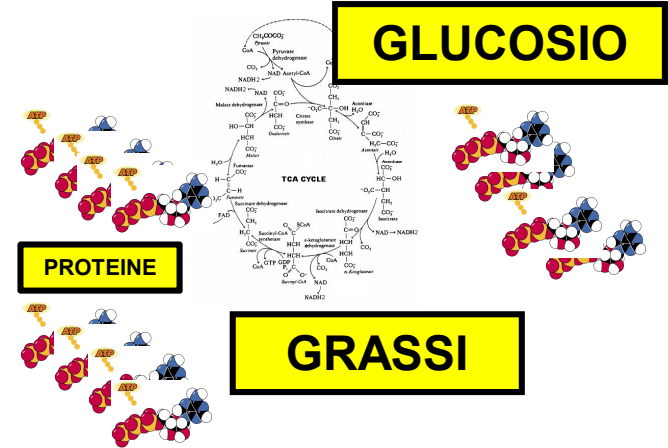
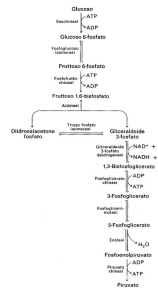
SISTEMI ENERGETICI



F-CREATINA

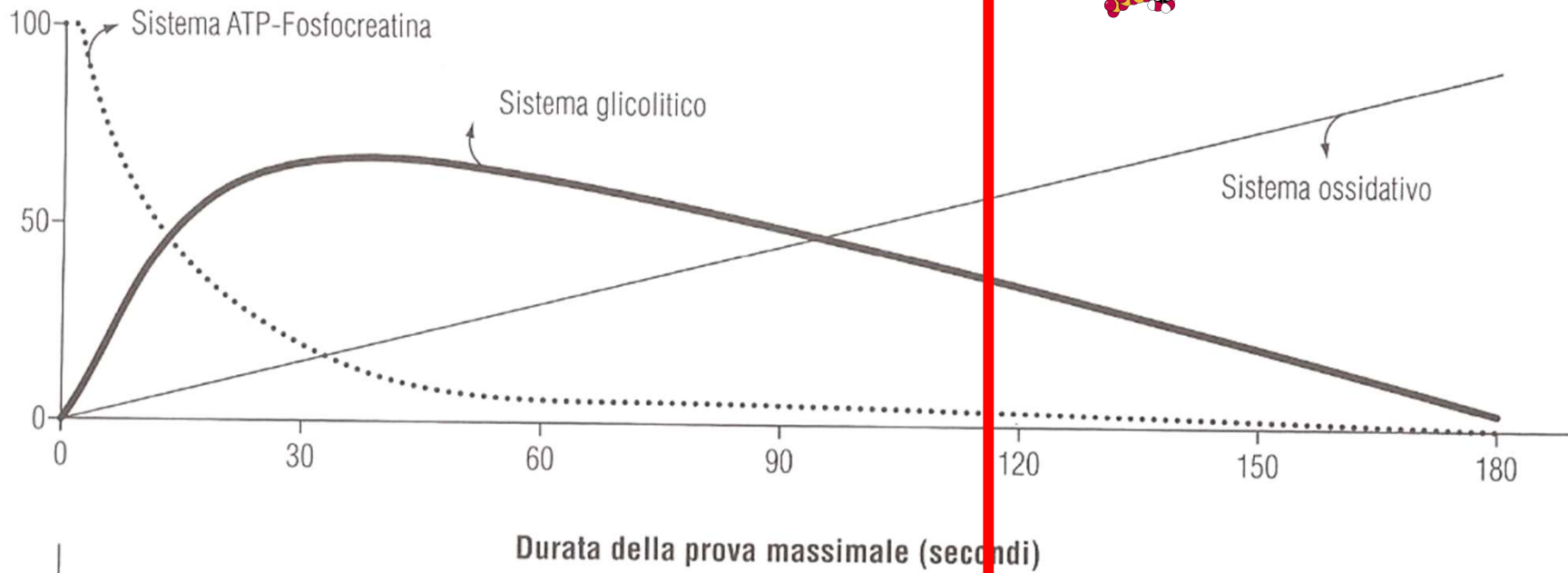


GLUCOSIO



PROTEINE

GRASSI



SISTEMI ENERGETICI

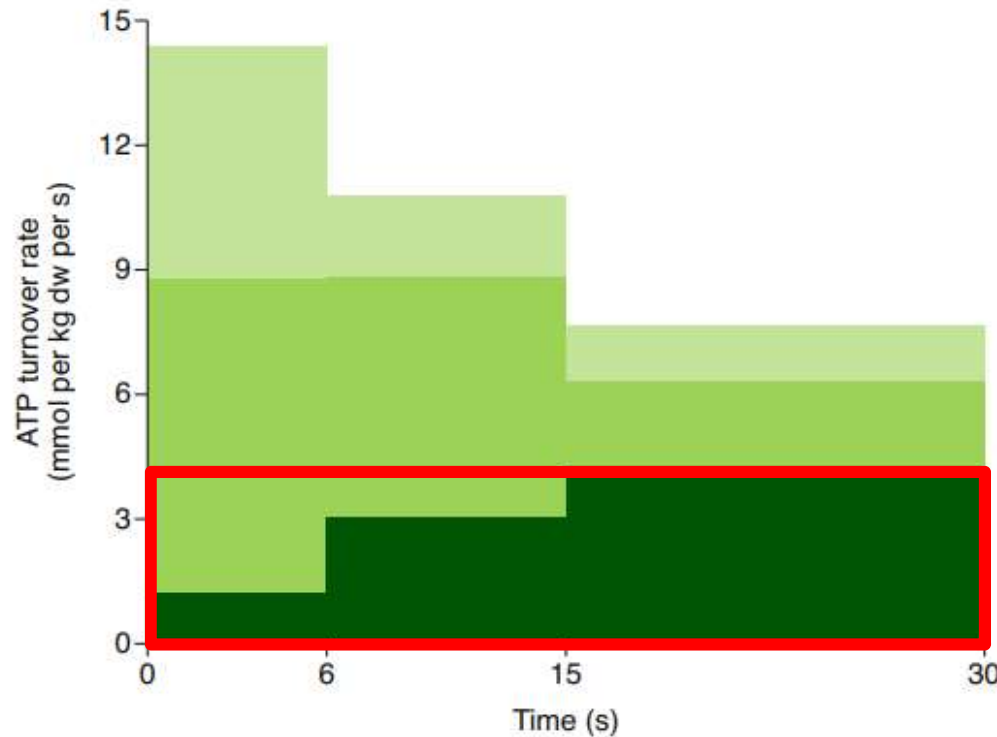
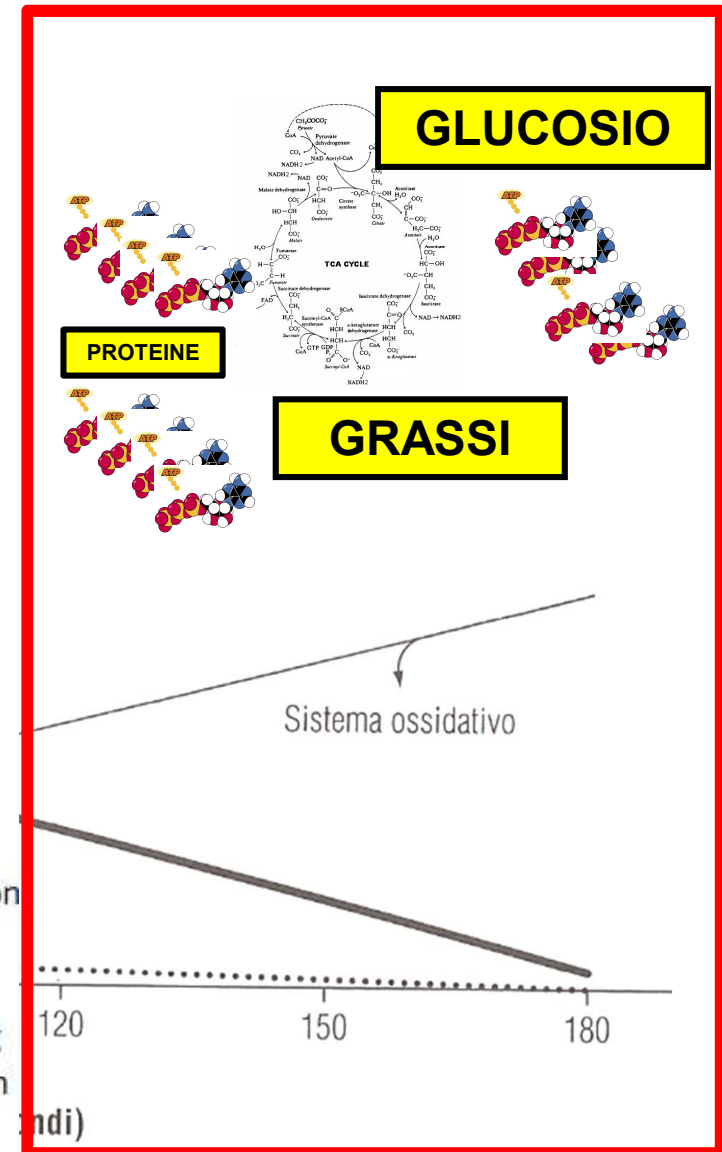
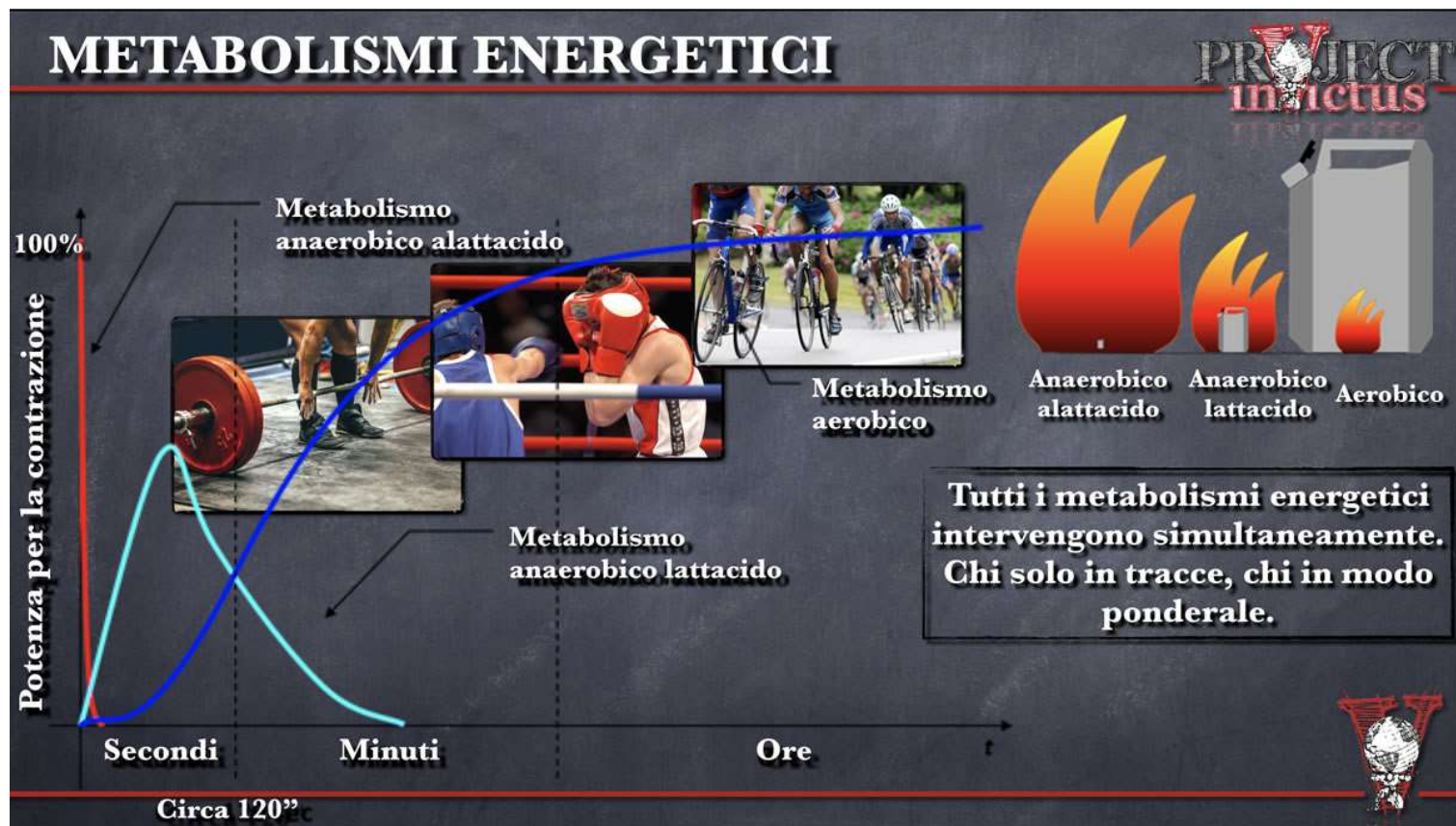


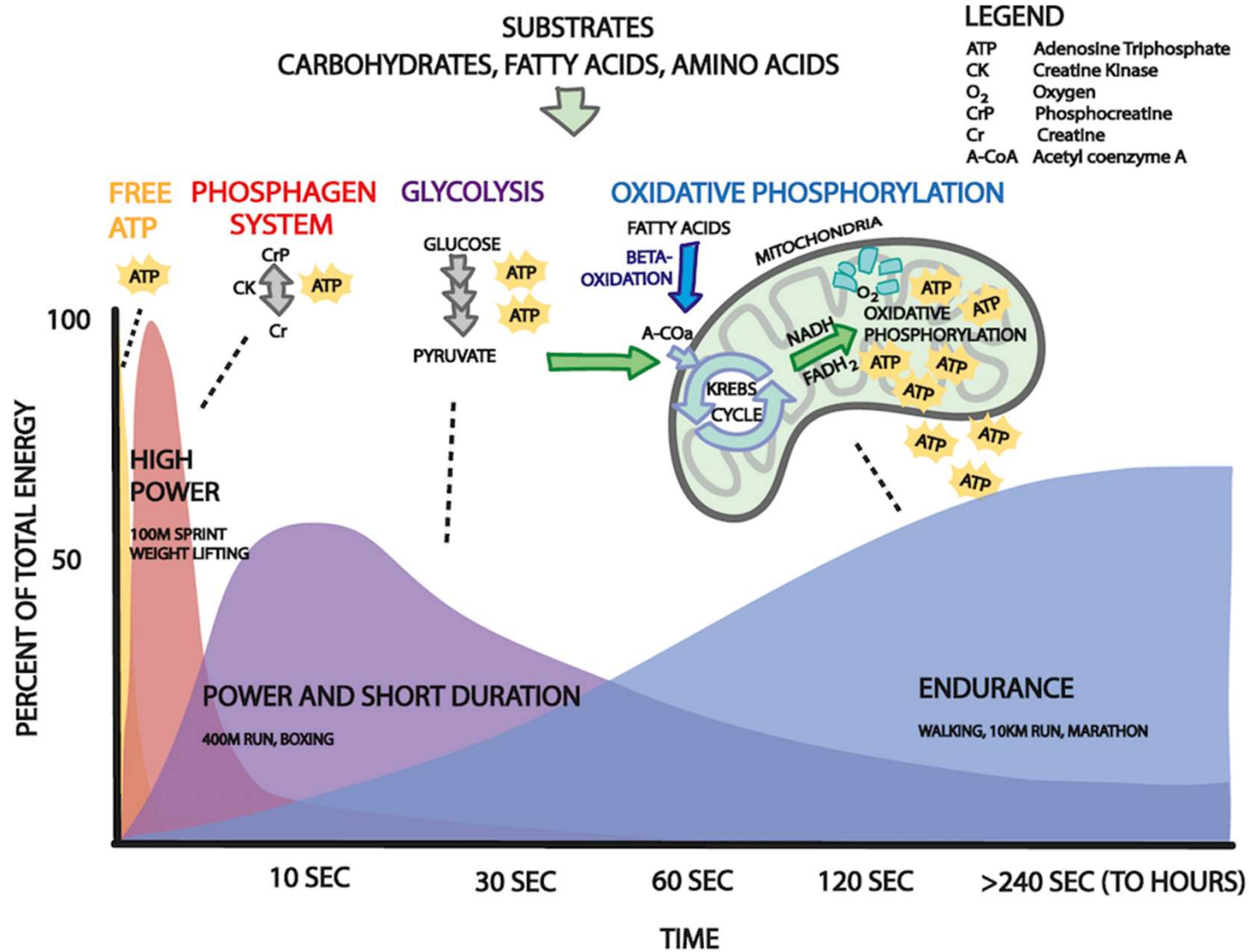
Fig. 1 | Muscle energy metabolism during intense exercise. Contribution of PCr (light green), glycolysis (medium green) and oxidative phosphorylation (dark green) to ATP turnover during maximal exercise. Muscle samples were obtained before and during 30 s of all-out cycling exercise. Dw, dry weight. Adapted with permission from ref. ⁴, American Physiological Society.



FOSFORILAZIONE OSSIDATIVA



VISIONE D'INSIEME



VISIONE D'INSIEME

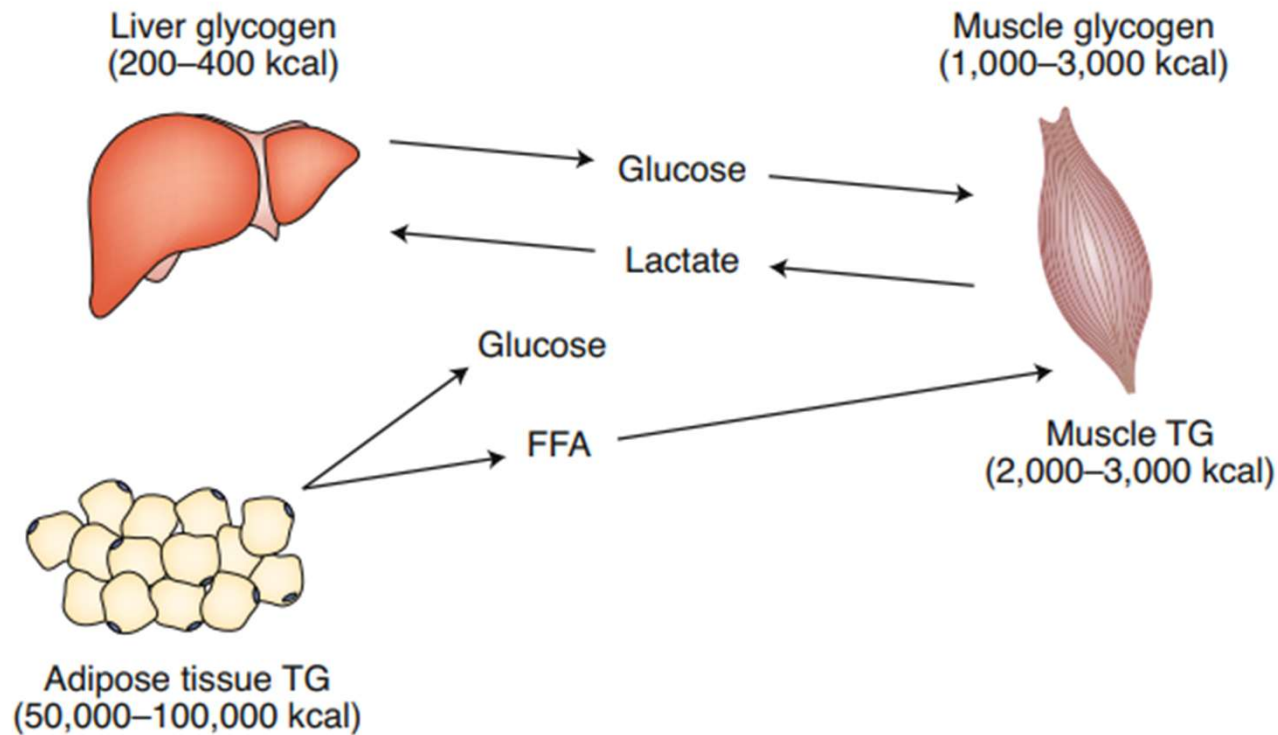


Fig. 2 | Intramuscular and extramuscular fuel sources for exercise metabolism. Major sources of carbohydrate in the muscle and liver and of fat in the muscle and adipose tissue during exercise. The estimated potential energy available from each fuel source is also provided. TG, triglyceride; FFA, free fatty acids.

DISPENDIO ENERGETICO

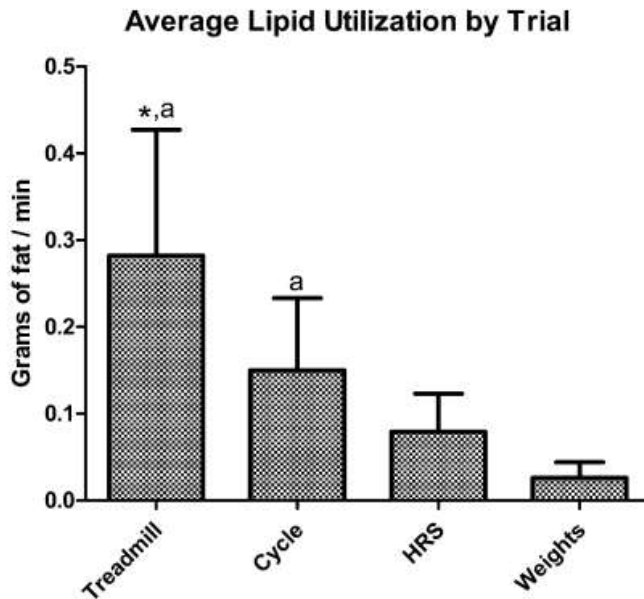


Figure 2. Average lipid use by trial. Data represent mean $\pm$ SD. Statistical significance set at $p \leq 0.05$. Significant differences between groups are represented as *, significantly different from HRS; a, significantly different from weights.

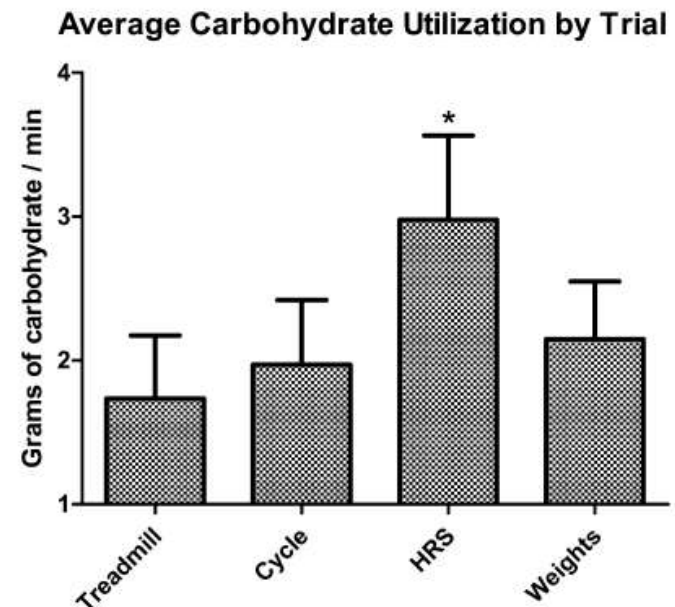


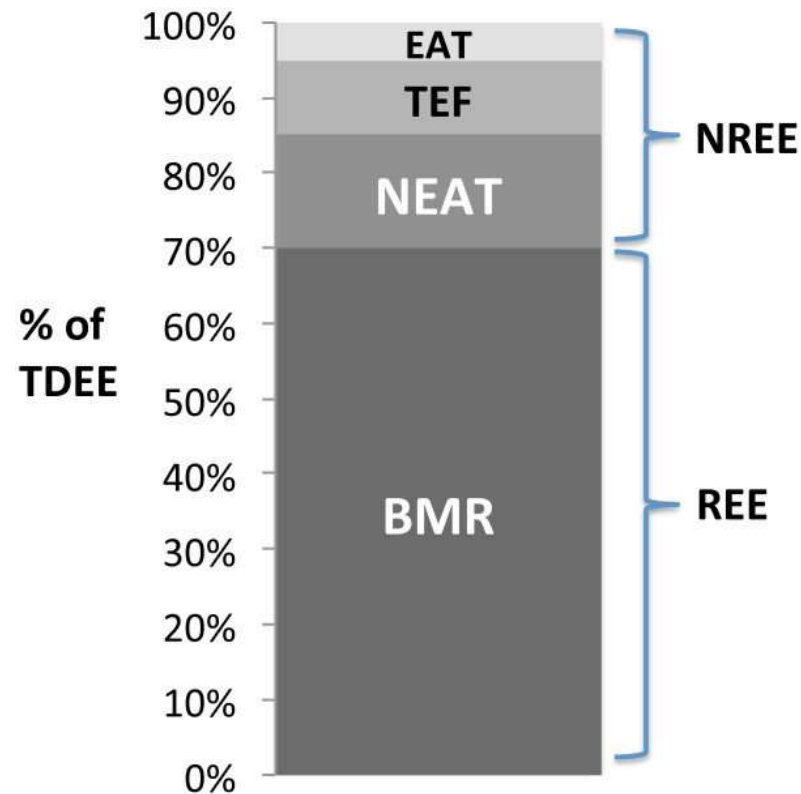
Figure 3. Average carbohydrate use by trial. Data represent mean $\pm$ SD. Statistical significance set at $p \leq 0.05$. Significant differences between groups are represented as *, significantly different from treadmill, cycle, and weights.

CALORIC EXPENDITURE OF AEROBIC, RESISTANCE, OR COMBINED HIGH-INTENSITY INTERVAL TRAINING USING A HYDRAULIC RESISTANCE SYSTEM IN HEALTHY MEN

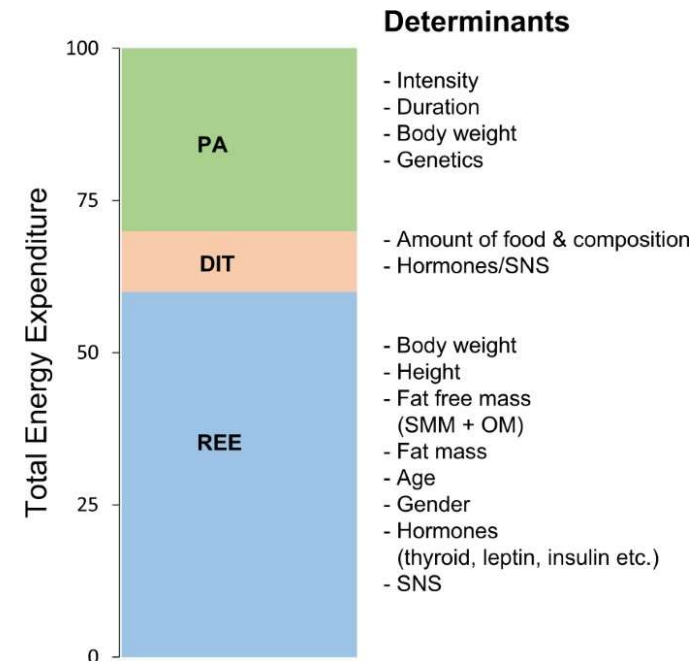
PAUL H. FALCONE,¹ CHIH-YIN TAI,¹ LAURA R. CARSON,¹ JORDAN M. JOY,² MATT M. MOSSMAN,¹ TYLER R. MCCANN,² KEVIN P. CHONA,² MICHAEL P. KIM,¹ AND JORDAN R. MOON^{1,4}

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DISPENDIO ENERGETICO



Components of total daily energy expenditure (TDEE). BMR = basal metabolic rate; NEAT = non-exercise activity thermogenesis; TEF = thermic effect of food; EAT = exercise activity thermogenesis; REE = resting energy expenditure; NREE = non-resting energy expenditure. Adapted from Maclean et al., 2011.



Total daily energy expenditure (TDEE)

AKA How many calories do you **need to maintain body weight**

Basal Metabolic Rate:

Men: $BMR = 66 + (13.7 \times \text{weight (kg)}) + (5 \times \text{height (cm)}) - (6.8 \times \text{age (years)})$

Women: $BMR = 655 + (9.6 \times \text{weight (kg)}) + (1.8 \times \text{height (cm)}) - (4.7 \times \text{age (years)})$

Multiply by **Activity level**

1.2 (sedentary) 1.4 (lightly active) 1.7 (moderately active) 2.0 (active all day)

Add on Estimated **Exercise Energy Expenditure**

= **Total Daily Energy Expenditure**

Ben Turner

— High Performance, Nutrition and Fitness Consultant —

DISPENDIO ENERGETICO

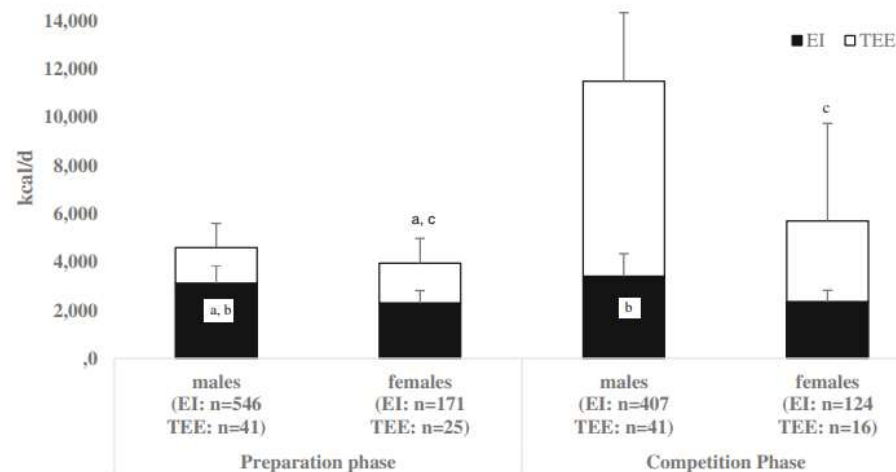


Fig. 3 Energy intake (EI) and total energy expenditure (TEE) in kcal/day of endurance athletes. Data are shown in weighted mean and standard deviation of the weighted mean ($\bar{X}_w \pm SD_w$). n = number of cumulative subjects

Study or subgroup	Energy intake (kcal/d)			Total energy expenditure (kcal/d)			Weight	Mean difference IV, Fixed, 95% CI	Mean difference IV, Fixed, 95% CI
	Mean	SD	Total	Mean	SD	Total			
1.1.1 Preparation phase									
Sjodin et al. 1994	7,218	1,099	4	7,218	1,004	4	2.4%	0 [-1,459, 1,459]	
Boulay et al. 1994	3,872	382	7	4,063	956	7	8.9%	-191 [-954, 572]	
Fudge et al. 2006	3,165	318	9	3,492	249	9	74.1%	-327 [-591, -63]	
Subtotal (95% CI)			20			20	85.4%	-304 [-549, -58]	
Heterogeneity: Chi ² = 0.28, df= 2 (p = 0.87); I ² = 0% Test for overall effect: Z = 2.42 (p = 0.02)									
1.1.2 Competition phase									
Bescós et al. 2012	5,549	2,127	8	10,253	1,625	8	1.5%	-4,704 [-6,559, -2,849]	
Costa et al. 2014	5,497	2,868	19	13,862	2,390	19	1.8%	-8,365 [-10,044, -6,686]	
Rehrer et al. 2010	6,525	908	4	6,549	478	4	5.1%	-24 [-1,030, 982]	
Hulton et al. 2010	4,918	810	4	6,420	470	4	6.1%	-1,502 [-2,420, 584]	
Subtotal (95% CI)			35			35	14.6%	-2,177 [-2,772, -1,582]	
Heterogeneity: Chi ² = 79.02, df= 3 (p < 0.00001); I ² = 96% Test for overall effect: Z = 7.17 (p < 0.00001)									
Total (95% CI)			55			55	100%	-577 [-804, -349]	
Heterogeneity: Chi ² = 111.80, df= 6 (p < 0.00001); I ² = 95% Test for overall effect: Z = 4.97 (p < 0.00001) Test for subgroup differences: Chi ² = 32.50, df= 1 (p < 0.00001); I ² = 96.9%									

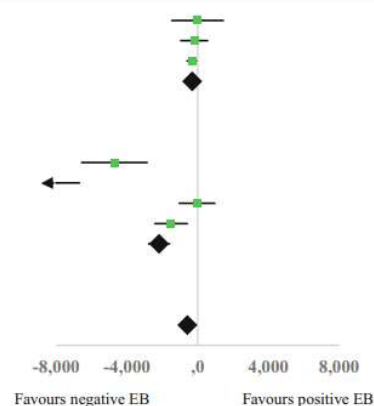


Fig. 5 Energy balance (EB) of male endurance athletes during preparation and competition phase

DISPENDIO ENERGETICO

Table 4 Energy intake in kcal/day and kcal/kg/day of endurance athletes in preparation and competition phase

Endurance discipline	Preparation			Competition		
	n	Energy intake [kcal/day]	Energy intake [kcal/kg-day]	n	Energy intake [kcal/day]	Energy intake [kcal/kg-day]
Cyclists						
Total	46	3789 ± 764 ^{d,e,f}	52.3 ± 13.3 ^{d,e}	133	3600 ± 1102 ^d	46.9 ± 17.7 ^{d,f}
Male	46	3789 ± 764 ^{d,e}	52.3 ± 13.3 ^{d,e}	125	3603 ± 1137	45.9 ± 18.0
Female	–	–	–	–	–	–
Runners						
Total	278	2489 ± 425 ^a	38.2 ± 7.8 ^a	272	3042 ± 788	42.7 ± 4.7
Male	207	2640 ± 366 ^{a,b,f}	38.3 ± 8.6 ^a	203	3298 ± 713 ^b	43.8 ± 3.2 ^b
Female	71	2046 ± 230 ^a	38.0 ± 4.6 ^c	69	2291 ± 443	39.4 ± 6.4
Swimmers						
Total	73	3366 ± 902 ^{a,d,e,g}	48.7 ± 9.6 ^{a,d,e}	55	2769 ± 681 ^{g,h}	40.1 ± 7.7 ^g
Male	39	3963 ± 762 ^{a,b}	53.2 ± 9.5 ^{a,b,d,e}	24	3462 ± 341 ^b	46.2 ± 6.5 ^b
Female	34	2683 ± 450 ^{a,d,e}	43.6 ± 6.9 ^{a,e}	31	2234 ± 256	35.4 ± 4.7
Rowers						
Total	70	2426 ± 448 ^a	33.9 ± 4.5 ^a	15	3633 ± 1097	46.8 ± 10.9
Male	24	2921 ± 326 ^{b,f}	36.0 ± 0.1 ^b	–	–	–
Female	46	2168 ± 330	32.8 ± 5.2 ^c	–	–	–
Cross-country skiers						
Total	138	3224 ± 917 ^{a,d,e,g}	48.3 ± 12.7 ^{a,d,e}	33	2091 ± 53.2 ^{d,e,f,g}	32.7 ± 2.9 ^c
Male	124	3287 ± 876 ^{d,f,g}	48.3 ± 11.6 ^{d,e}	–	–	–
Female	14	2663 ± 1107 ^{d,e}	49.1 ± 20.3	–	–	–
Triathletes						
Total	16	3162 ± 159 ^{d,e}	45.7 ± 2.6 ^e	–	–	–
Male	16	3162 ± 159 ^{f,g}	45.7 ± 2.6	–	–	–
Female	–	–	–	–	–	–
Other endurance athletes						
Total	96	3261 ± 282 ^{a,d,e,g}	46.5 ± 5.1 ^{a,d,e}	14	4656 ± 1070	–
Male	90	3274 ± 286 ^{a,d,f,g}	46.3 ± 5.2 ^{a,d,e,f}	14	d,f,g,h	–
Female	–	–	–	–	4656 ± 1070 ^c	–
Total						
Total	717	2915 ± 761 ^a	42.8 ± 10.5	531	3156 ± 967	43.5 ± 11.3
Male	546	3111 ± 717 ^{a,b}	44.0 ± 10.6 ^b	407	3405 ± 940 ^b	44.8 ± 11.9 ^b
Female	171	2291 ± 525	39.0 ± 9.1	124	2337 ± 483	39.3 ± 7.9

DISPENDIO CALORICO

Tab. I - Fabbisogno energetico in Kcal/minuto per alcuni tipi di attività fisica o sport.

Camminare lentamente	2,6	Ginnastica	5,9
Camminare 5 km/h	3,7	Basket	14,3
Corsa campestre ricreativa	10,4	Pallavolo	8,5
Corsa 100 m gara	280,0	Tennis singolo	11,1
Ballo ritmo lento	4,3	Tennis doppio	9,1
Ballo ritmo veloce	11,3	Calcio	11,7
Ciclismo ricreativo	5,9	Sci di fondo ricreativo	12,0
Ciclismo gara	26,0	Sci di fondo gara	21,5
Canottaggio ricreativo	9,1	Sci alpino ricreativo	12,0
Canottaggio gara	25,5	Sci alpino gara	21,5
Nuoto ricreativo	9,1	Judo	22,8
Nuoto gara	25,0	Rugby	10,0

Sport	CE (kcal/kg/h)	50 kg		70 kg	
Pallavolo	3-5	150	250	210	350
Ginnastica	4-6	200	300	280	420
Equitazione	3-6	150	300	210	420
Aerobica	3-7	150	350	210	490
Golf	4-7	200	350	280	490
Walking	5-8	250	400	350	560
Tennis tavolo	5-8	250	400	350	560
Trekking	5-10	250	500	350	700
Basket	5-10	250	500	350	700
Nuoto	7-10	350	500	490	700
Calcio	6-10	300	500	420	700
Ciclismo	5-12	250	600	350	840
Spinning	10-12	500	600	700	840
Judo	10-12	500	600	700	840
Corsa	6-12	300	600	420	840
Sci di fondo	8-12	400	600	560	840
Squash	8-12	400	600	560	840

Average Caloric Expenditure by Trial

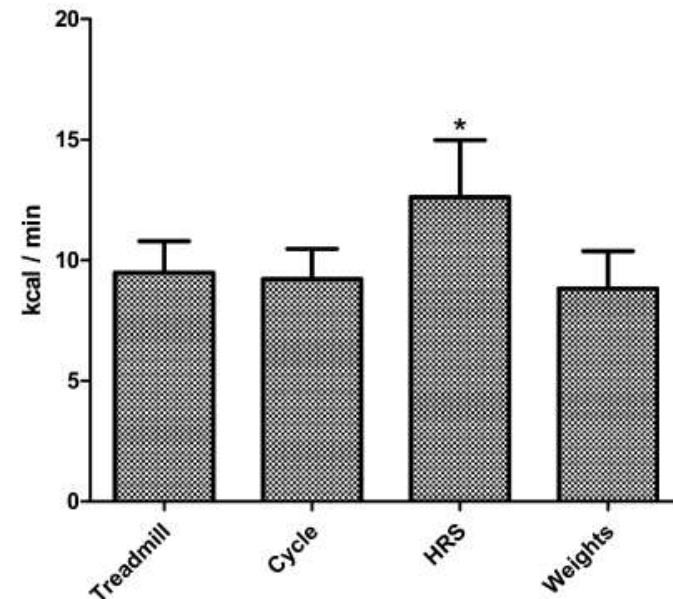


Figure 1. Average caloric expenditure by trial. Data represent mean $\pm$ SD. Statistical significance set at $p \leq 0.05$. Significant differences between groups are represented as *, significantly different from treadmill, cycle, and weights.

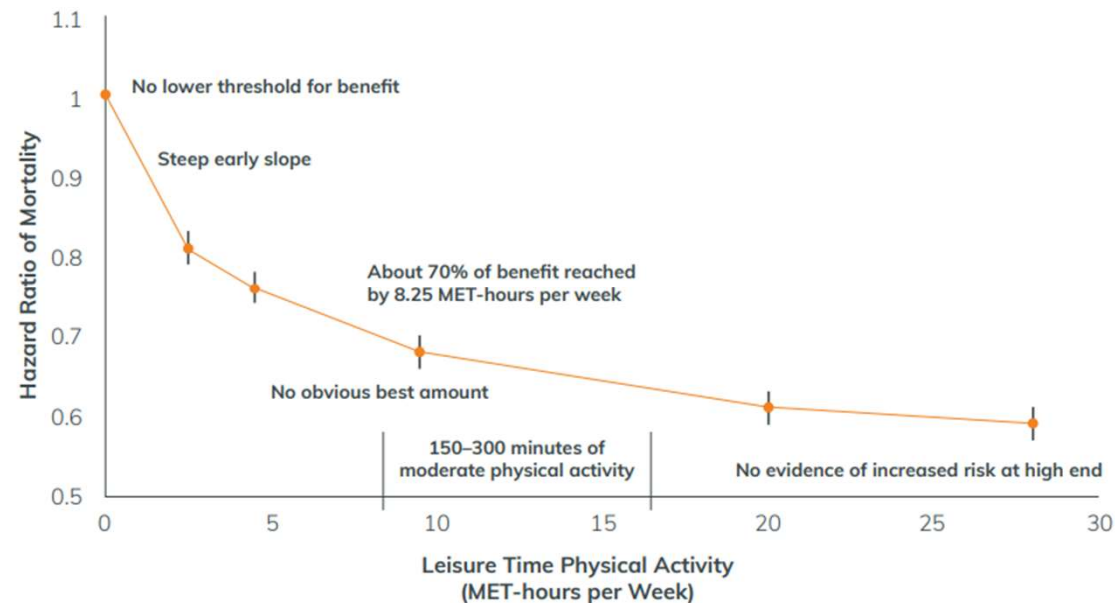
CALORIC EXPENDITURE OF AEROBIC, RESISTANCE, OR COMBINED HIGH-INTENSITY INTERVAL TRAINING USING A HYDRAULIC RESISTANCE SYSTEM IN HEALTHY MEN

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¹MusclePharm Sports Science Institute, Denver, Colorado; ²University of Colorado, Boulder, Colorado; ³University of Colorado, Denver, Colorado; and ⁴Department of Sports Exercise Science, United States Sports Academy, Daphne, Alabama

FINE.

RELAZIONE ATTIVITÀ FISICA - MORTALITÀ

Figure 2-1. Relationship of Moderate-to-Vigorous Physical Activity to All-Cause Mortality



Source: Adapted from data found in Moore SC, Patel AV, Matthews CE. Leisure time physical activity of moderate to vigorous intensity and mortality: a large pooled cohort analysis. PLoS Med. 2012;9(11):e1001335. doi:10.1371/journal.pmed.1001335.

- First, only a few lifestyle choices have as large an effect on mortality as physical activity. It has been estimated that people who are physically active for approximately 150 minutes a week have a 33 percent lower risk of all-cause mortality than those who are not physically active.
- Second, it is not necessary to do large amounts of activity or vigorous-intensity activity to reduce the risk of all-cause mortality. Benefits start to accumulate with any amount of moderate- or vigorous-intensity physical activity.

Il consumo energetico in equivalenti metabolici

Nella tabella sono illustrati alcuni esempi di consumi energetici espressi in equivalenti metabolici (MET).

Attività	MET
Camminare a ritmo lento in pianura	2,0
Camminare a ritmo veloce in pianura	4,0
Camminare a ritmo veloce in salita	6,0
Correre a ritmo lento (jogging)	7,0
Correre a ritmo veloce (running)	14,0 - 15,0
Correre a ritmo molto veloce	18,0
Corsa campestre	9,0
Salire le scale di corsa	15,0
Andare in bicicletta in pianura	4,0
Ciclismo in pianura, veloce	10,0 - 12,0
Danza, aerobica	6,0
Nuotare a ritmo lento	4,0
Nuotare a ritmo veloce a stile libero	10,0
Attività di palestra con pesi e macchine	8,0
Pattinare sul ghiaccio	7,0
Pattinare sul ghiaccio, veloce (gara)	15,0
Sciare	6,0 - 7,0
Sciare su sci di fondo, velocemente (gara)	14,0
Sciare, discesa, velocemente	8,0
Andare sullo skateboard	5,0
Praticare escursionismo	6,0
Giocare a tennis	7,0

1 MET = 3,5 ml O₂/(kg × min)
= 1 kcal/(kg × ora) circa



HARVARD ALUMNI HEALTH STUDY

HARVARD ALUMNI HEALTH STUDY suggerisce che gli uomini che praticano regolarmente ESERCIZIO FISICO guadagnano circa 2 ORE di aspettativa di vita ogni ORA di allenamento.

Se una persona si allena 4 ore alla settimana per 50 anni, ovvero, 10.400 ore.

10.000 ore di allenamento equivalgono a 20.000 ore di vita guadagnate.

20.000 ore significano quasi 2 anni e mezzo di aspettativa di vita in più.



Exercise Intensity and Longevity in Men
The Harvard Alumni Health Study

ESERCIZIO FISICO

RESISTENZA MUSCOLARE

- ALMENO **30 MINUTI** AL GIORNO PER **ALMENO 5 GIORNI** ALLA SETTIMANA DI ATTIVITÀ **DI RESISTENZA** AD INTENSITÀ **MODERATA (60-70% Fc MAX)**
- ATTIVITÀ **DI RESISTENZA** AD INTENSITÀ **VIGOROSA** PER ALMENO **20 MINUTI** AL GIORNO PER **ALMENO 3 GIORNI** ALLA SETTIMANA (**> 80% Fc MAX**)

adulti raccomandazione settimanale	150 minuti a media intensità		2 volte a settimana esercizi di rafforzamento muscolare
	oppure 75 minuti a intensità elevata		
bambini e ragazzi raccomandazione giornaliera	60 minuti al giorno con un'intensità media ed esercizi di coordinazione		3 volte a settimana esercizi di rafforzamento muscolare e delle ossa
			

N.B. IN AGGIUNTA
ALL'ATTIVITÀ FISICA QUOTIDIANA DI
ROUTINE !!!

FORZA MUSCOLARE

- ALMENO **2 GIORNI** ALLA SETTIMANA NON CONSECUTIVI
- **8-10 ESERCIZI** USANDO I MAGGIORI GRUPPI MUSCOLARI (**8-12 RIPETIZIONI** PER OGNI ESERCIZIO AL **60-70% DEL 1 RM**)

FLESSIBILITÀ ARTICOLARE

- **OGNI GIORNO** ALLUNGAMENTI STATICI E DINAMICI DI TUTTI I GRUPPI MUSCOLARI
- ESEGUITO DOPO RISCALDAMENTO CV OPPURE AL TERMINE DI UN ALLENAMENTO