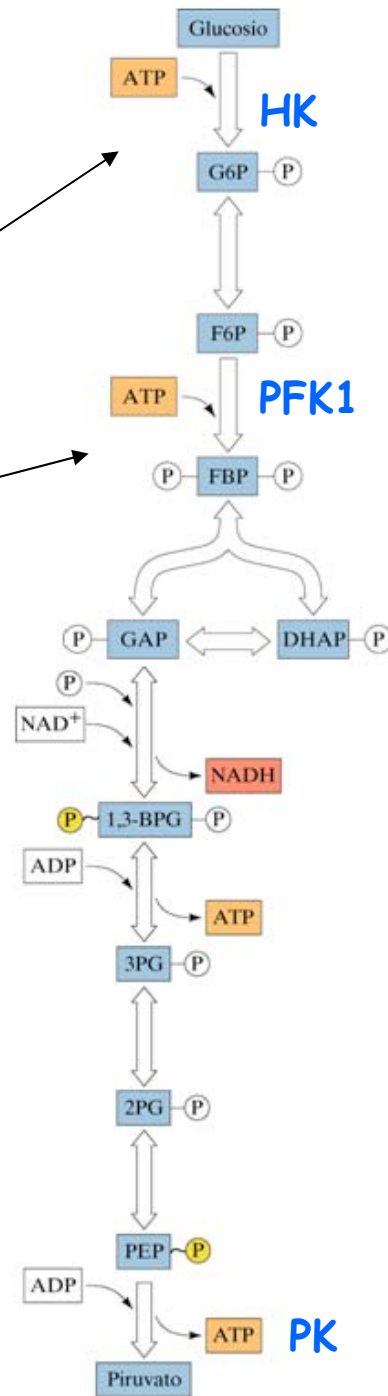


SITI DI REGOLAZIONE



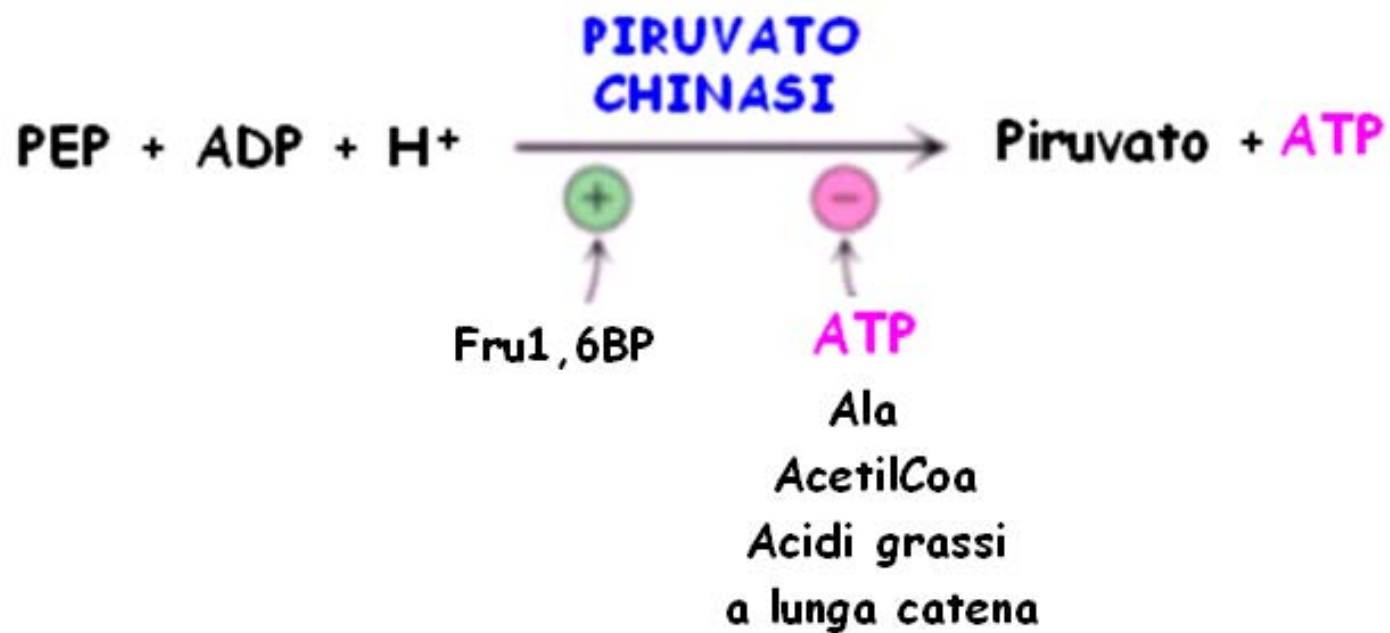
Regolazione della PK

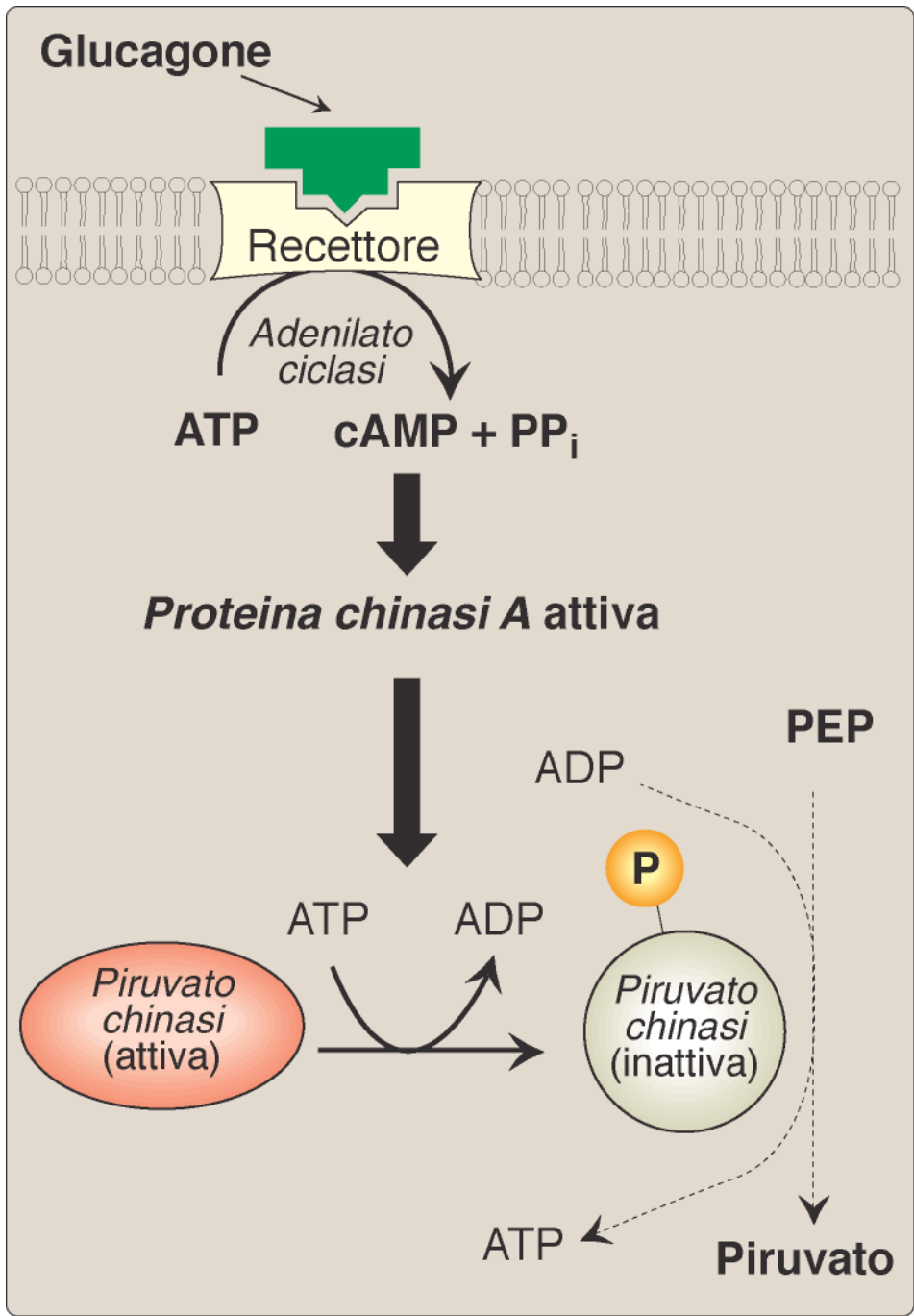
Nel **fegato**

- allosterica
- covalente (mediata dal glucagone)

Nel **muscolo**

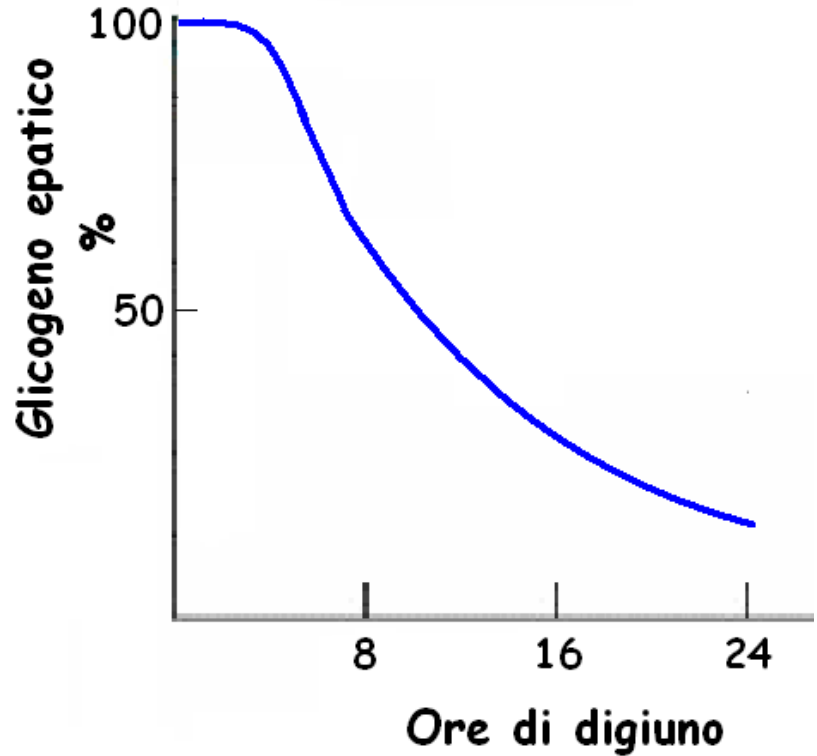
- solo allosterica





CONTENUTO DI GLUCIDI IN UN UOMO DI 70 KG

Glicogeno epatico	15-150 g
Glicogeno muscolare	150-300 g
Glucosio nel sangue e nei liquidi extracellulari	20 g



Gluconeogenesi

è la sintesi di Glc a partire da precursori non glucidici

90% fegato

10% reni



Glicogenolisi
Gluconeogenesi



Glicogenolisi
Gluconeogenesi



Insulina

Glucagone

Adrenalina

La gluconeogenesi permette il
mantenimento di
adeguati **livelli glicemici**
tra i pasti e durante gli
esercizi fisici più impegnativi

GLICOLISI

Glc → Piruvato

$$\Delta G' = -20 \text{ kcal/mole}$$

INVERSO DELLA GLICOLISI

Piruvato ~~→~~ Glc

$$\Delta G' = +20 \text{ kcal/mole}$$

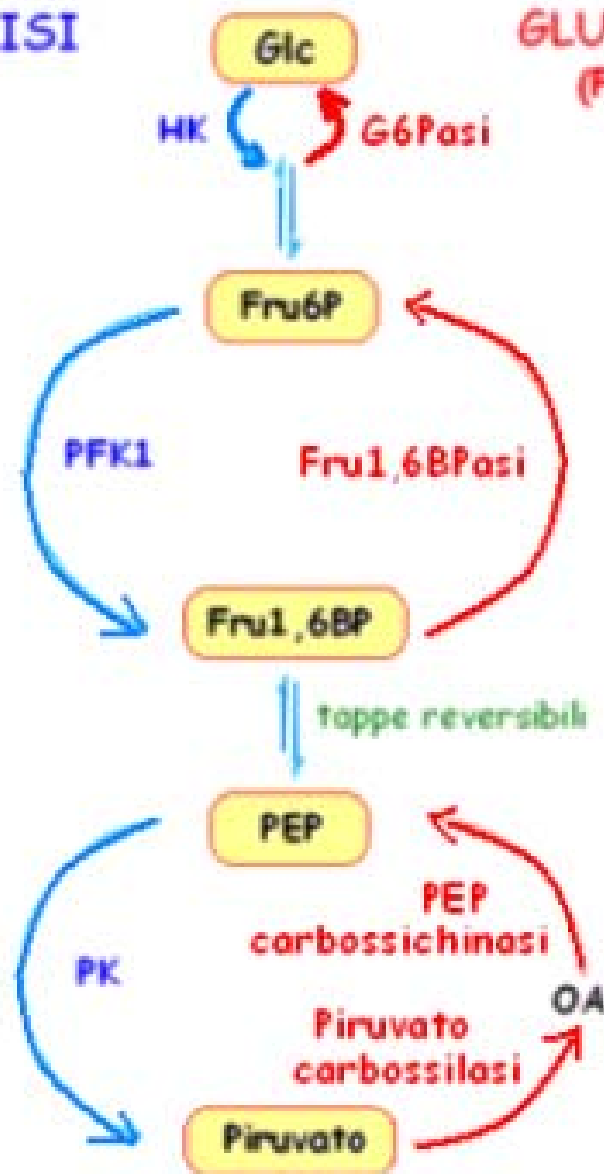
GLUCONEOGENESI

Piruvato → Glc

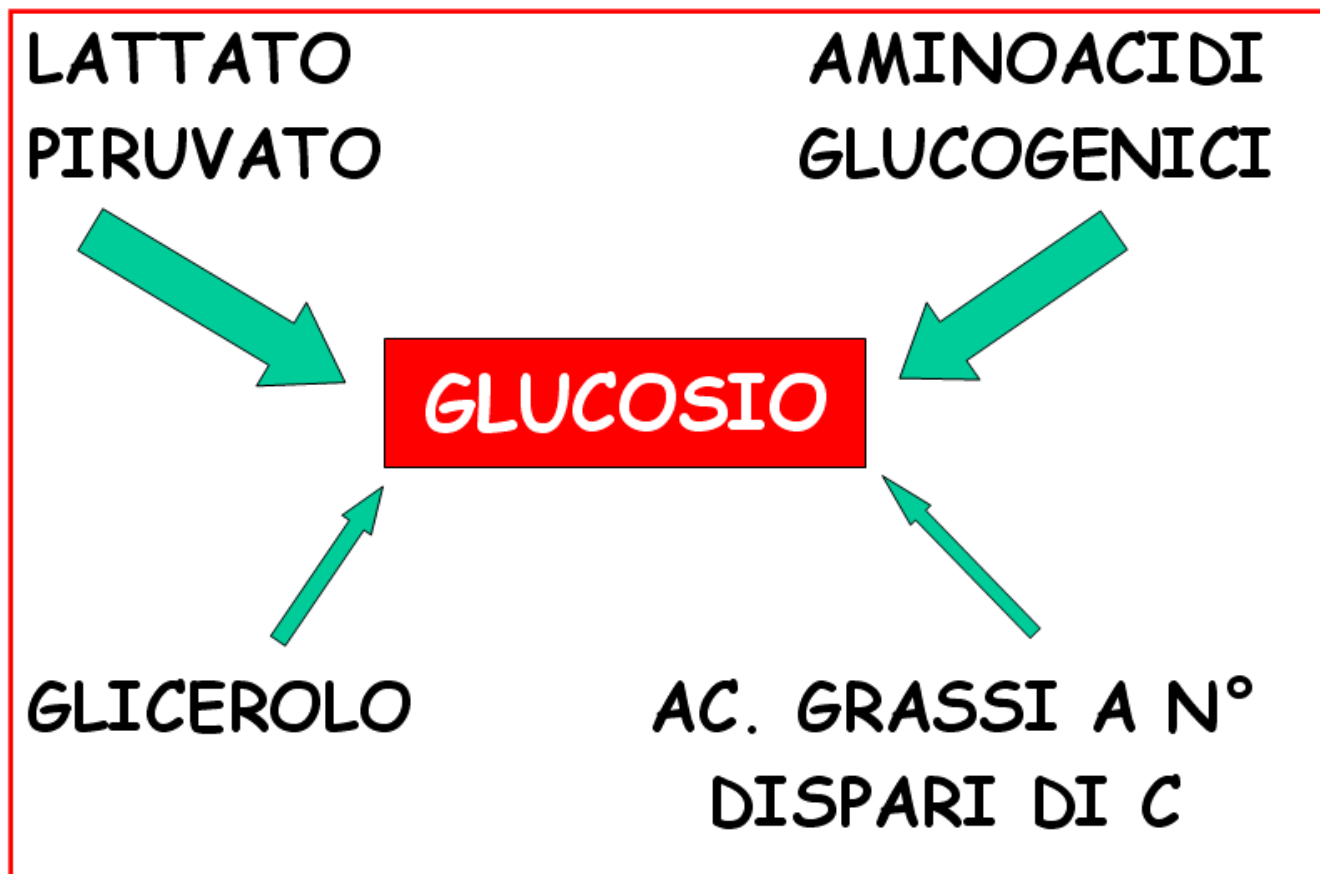
$$\Delta G' = -9 \text{ kcal/mole}$$

GLICOLISI

GLUCONEOGENESI (FEGATO E RENI)



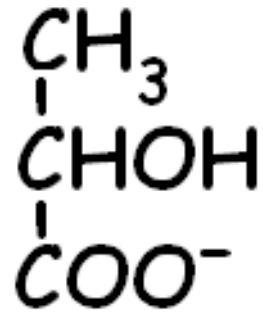
PRECURSORI NON GLUCIDICI DELLA GLUCONEOGENESI



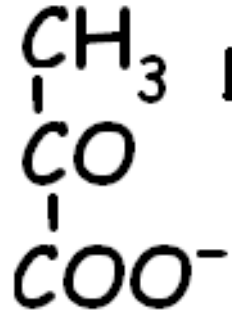
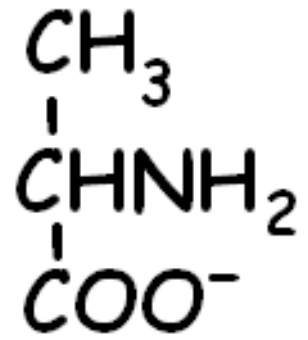
Gli aa sono quasi
tutti **glucogenici**

Ala è il più importante
aa **glucogenico**

Lattato



Ala



Piruvato

AA GLUCOGENICI



SCHELETRO CARBONIOSO



PIRUVATO



OA



α -KG



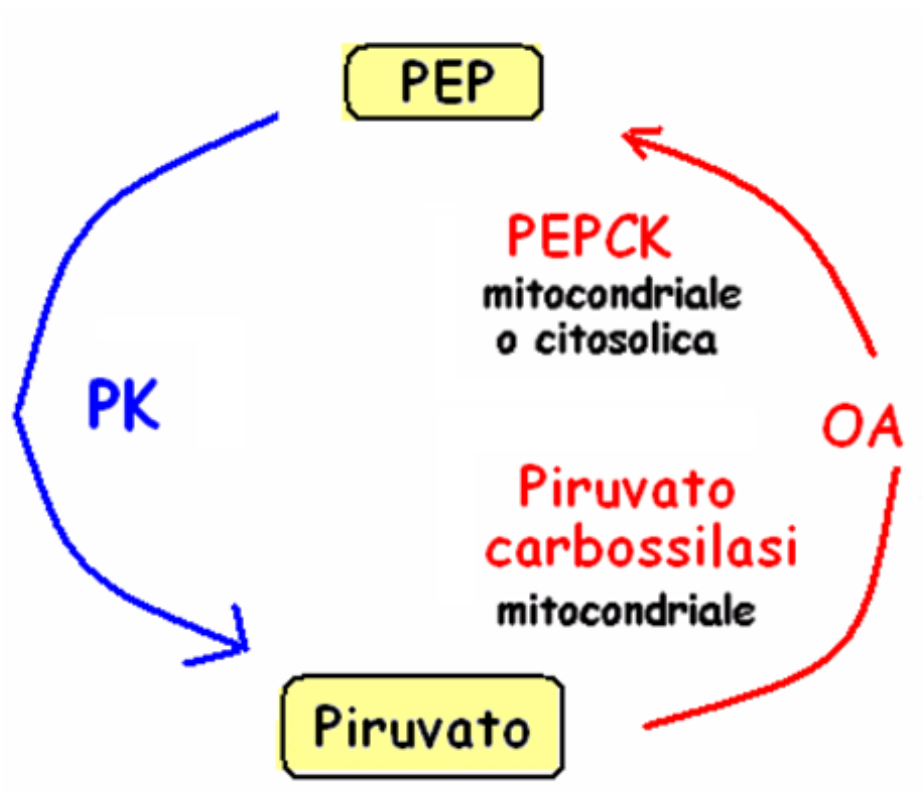
SUCCINIL CoA

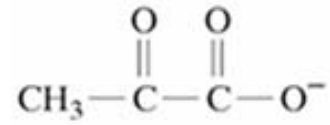


intermedi del ciclo di Krebs

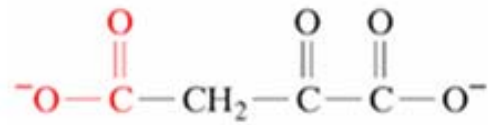
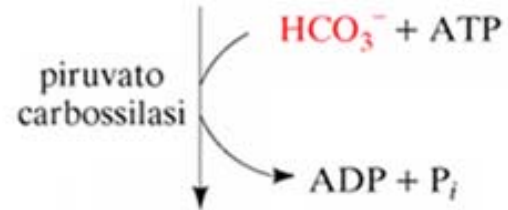
Principale rifornimento della gluconeogenesi:

- **lattato** prodotto dagli **eritrociti** e dai **muscoli** in attività (**ciclo di Cori**)
- **Ala** prodotto dai **muscoli** (**ciclo Glc-Ala**)

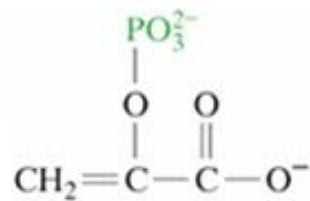
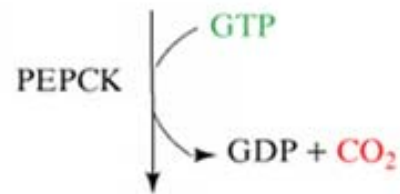




Piruvato

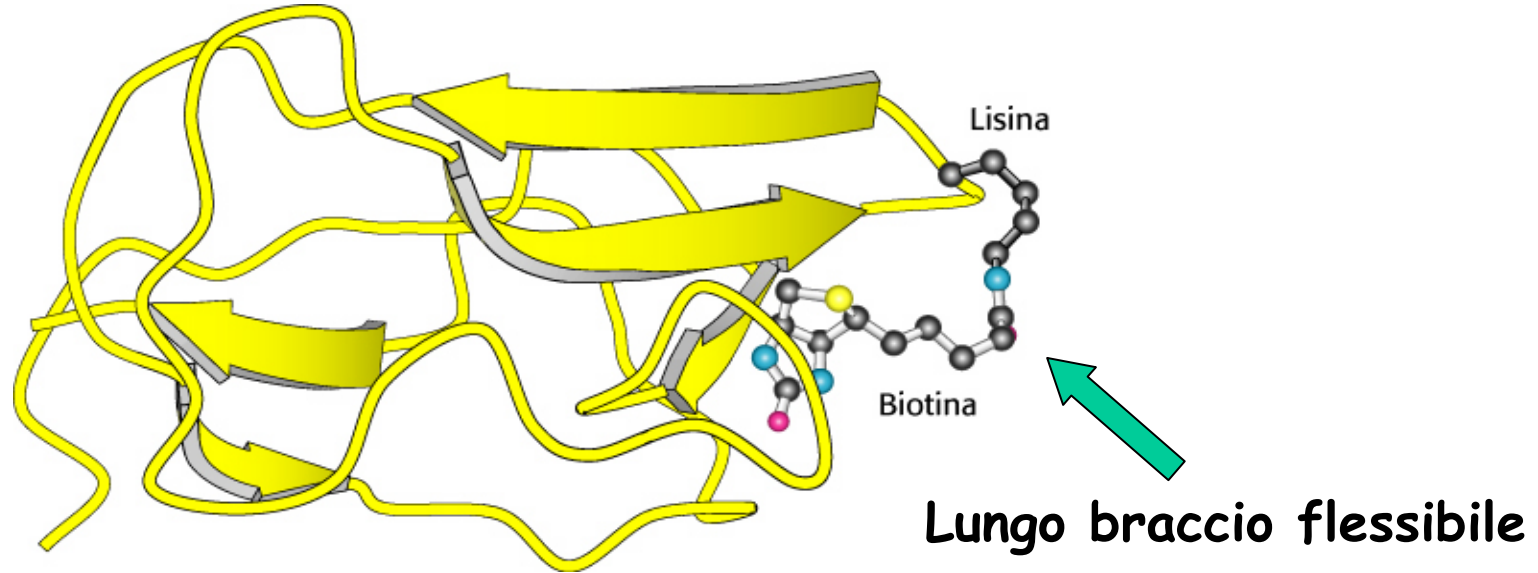


Ossalacetato



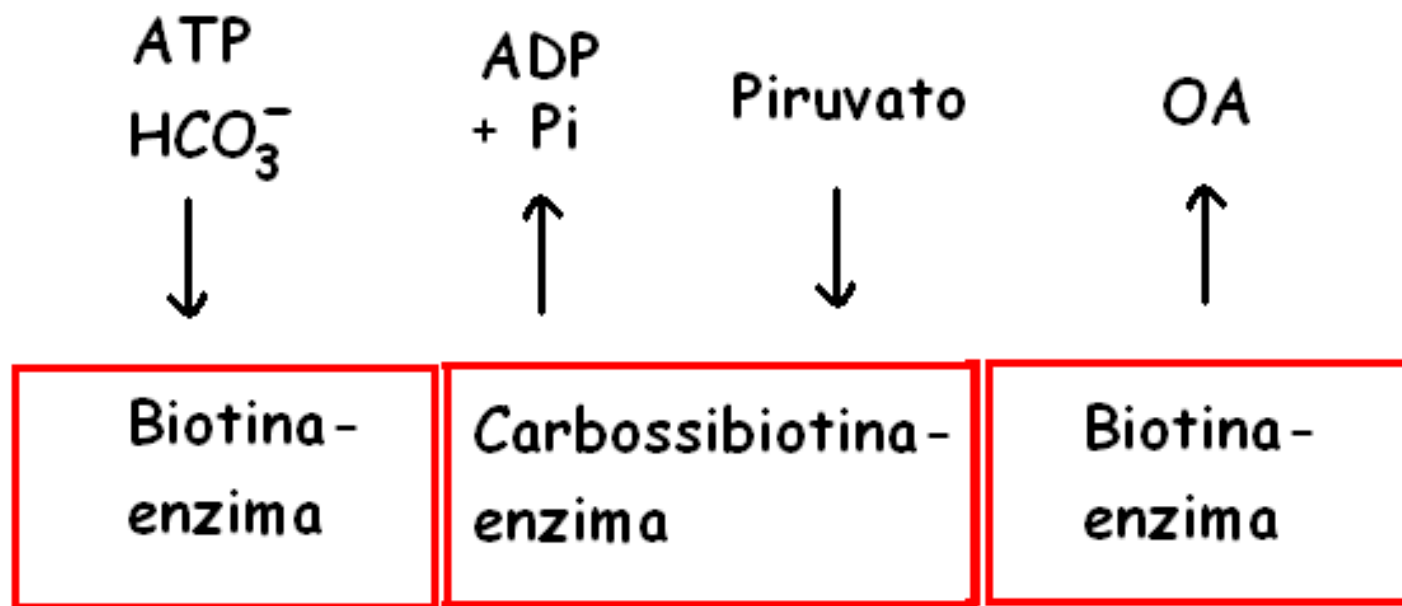
PEP

PIRUVATO CARBOSSILASI

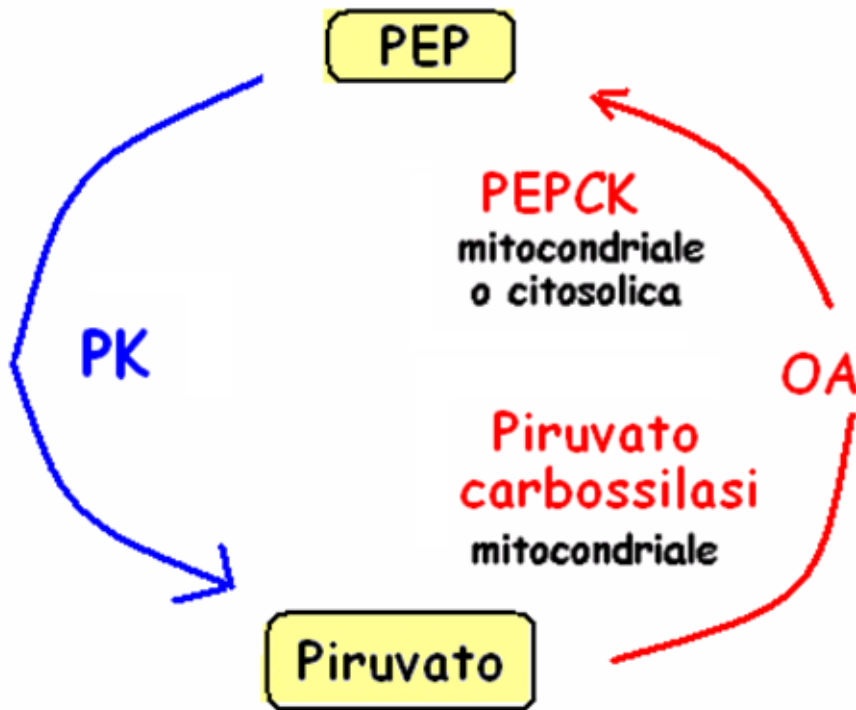


Il braccio flessibile consente alla **biotina** di **ruotare**

- dal sito dell'ATP e dell' HCO_3^-
- al sito del piruvato

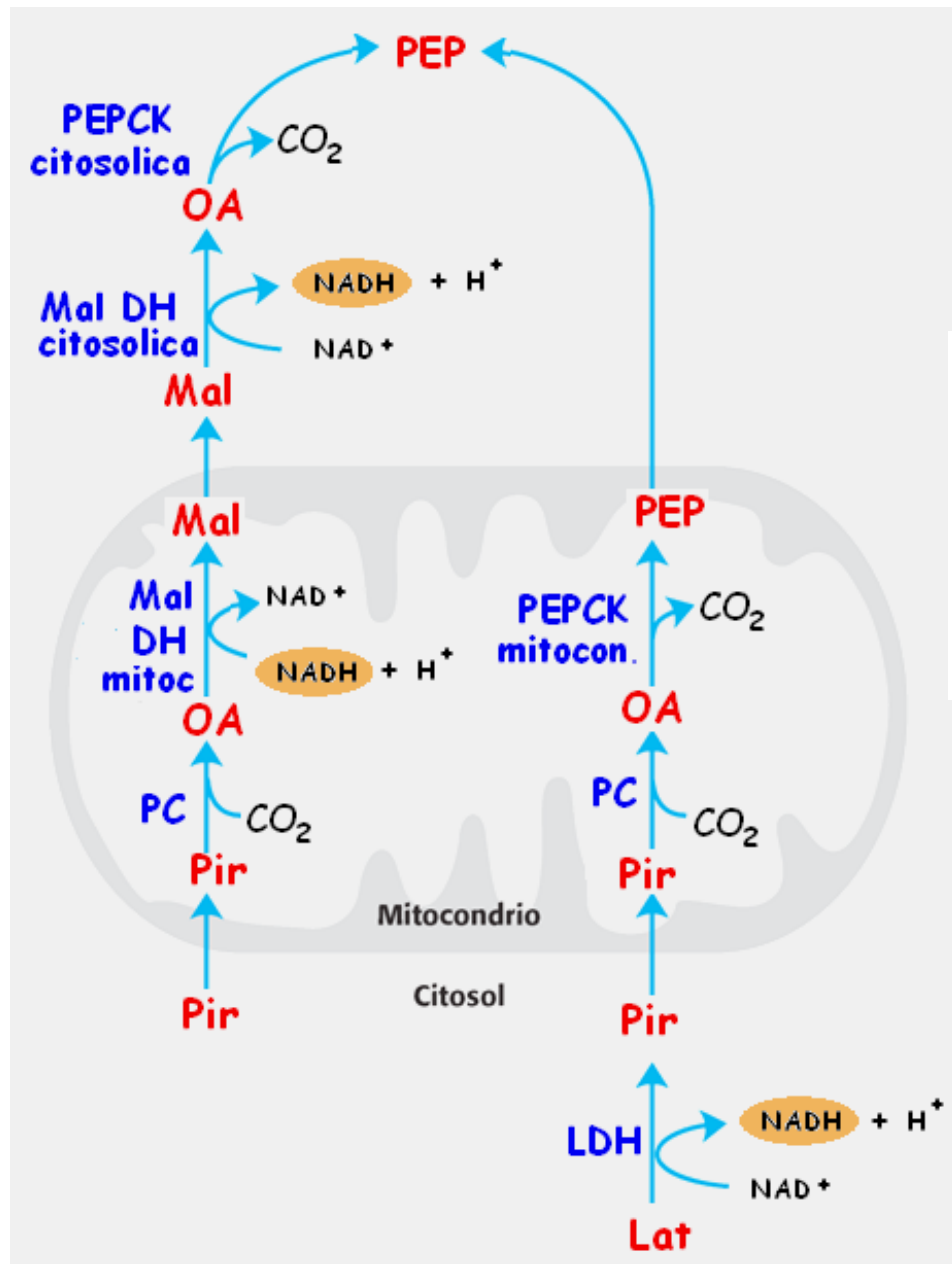


MECCANISMO A PING-PONG DELLA
PIRUVATO CARBOSSILASI



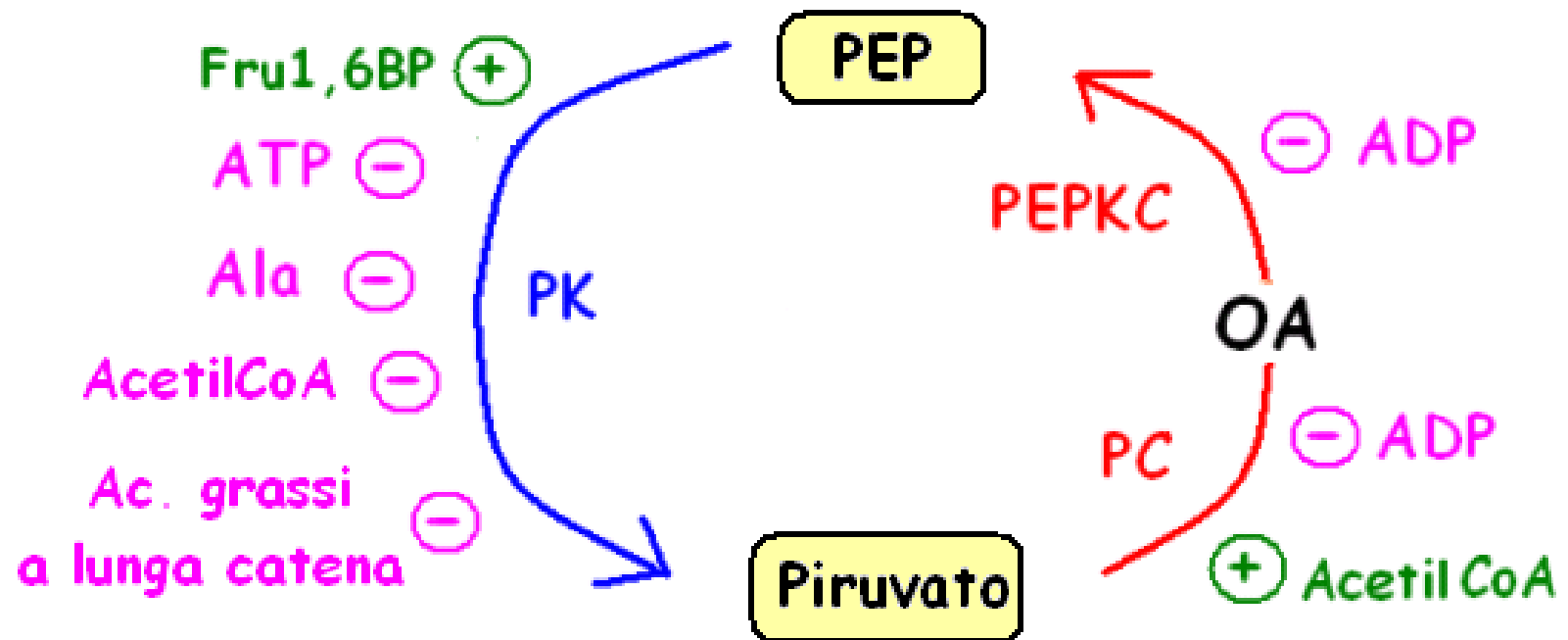
La **PEPCK mitoc.** entra in azione quando si parte dal **lattato**

La **PEPCK citosol.** entra in azione quando si parte dal **piruvato**

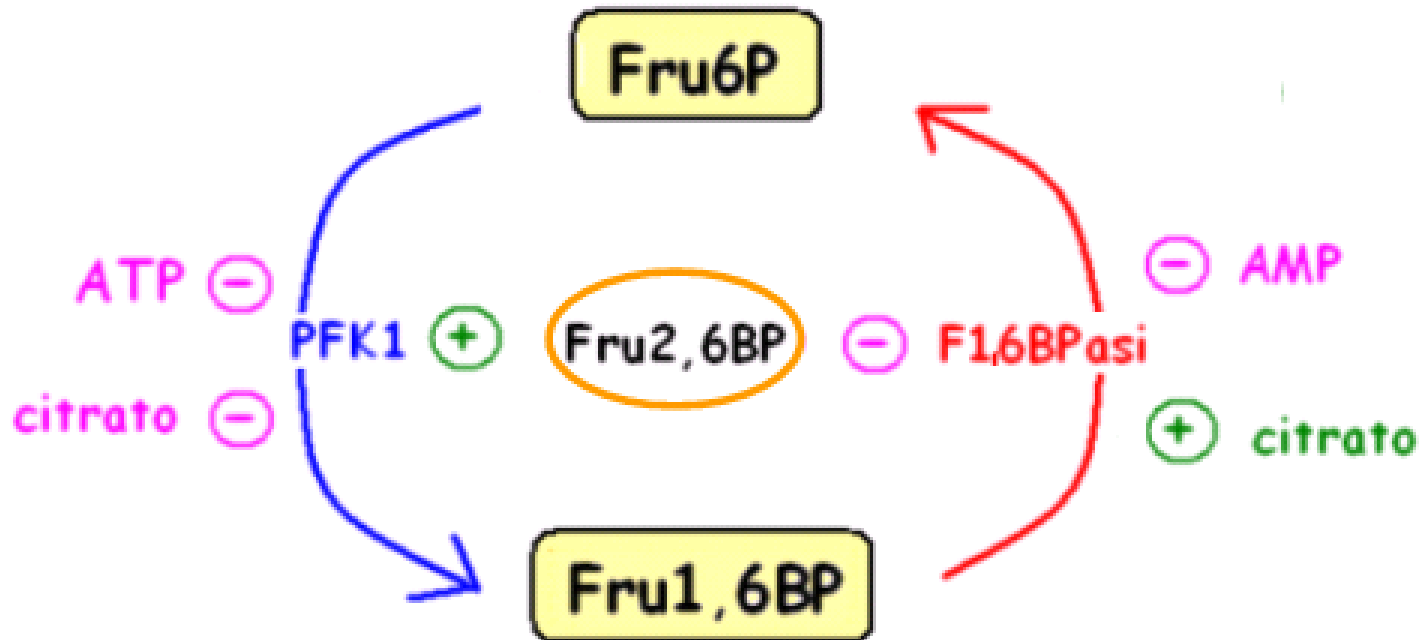


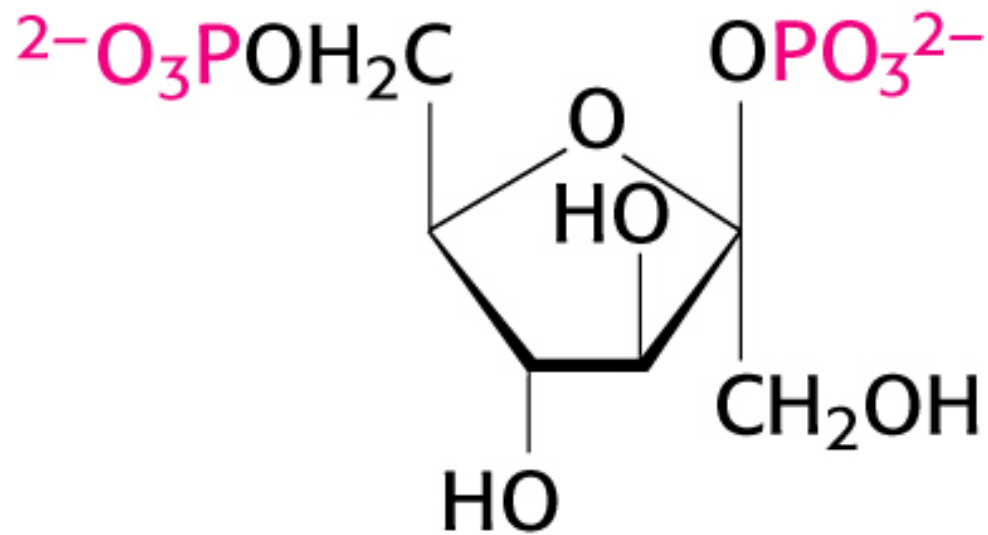
$$\frac{[\text{NADH}]}{[\text{NAD}^+]}$$

è nel citosol circa 10^5 volte più basso di quello dei mitocondri



La piruvato carbossilasi è quasi
inattiva in assenza dell'acetylCoA,
suo modulatore positivo



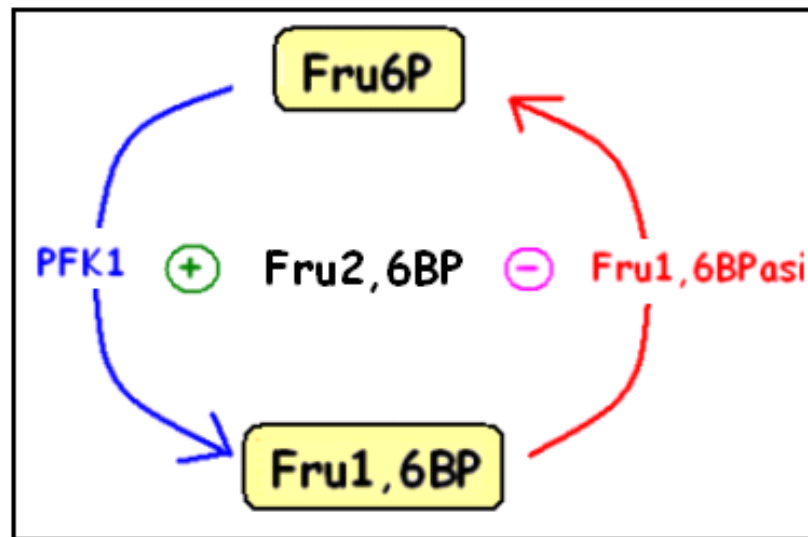
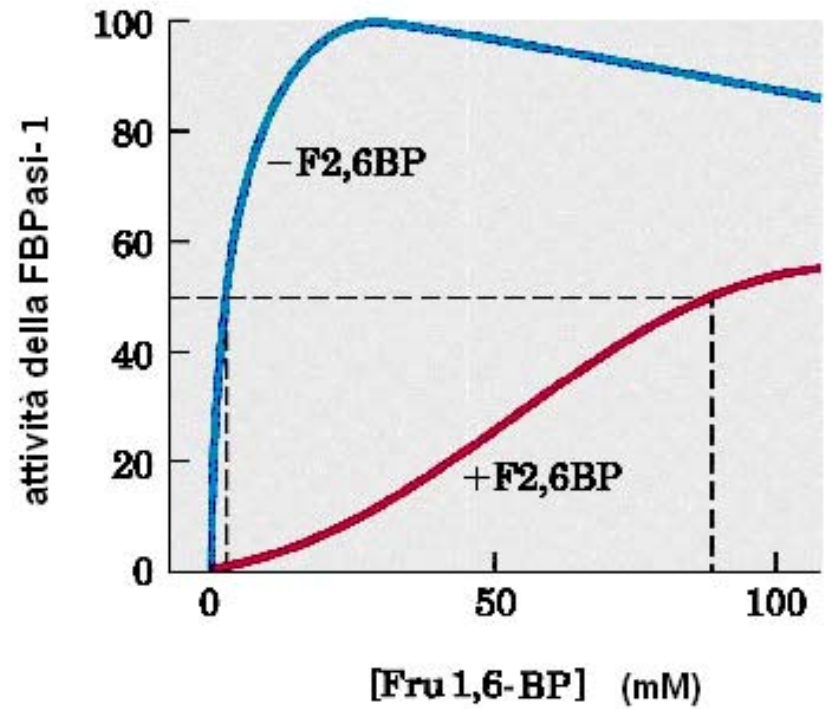
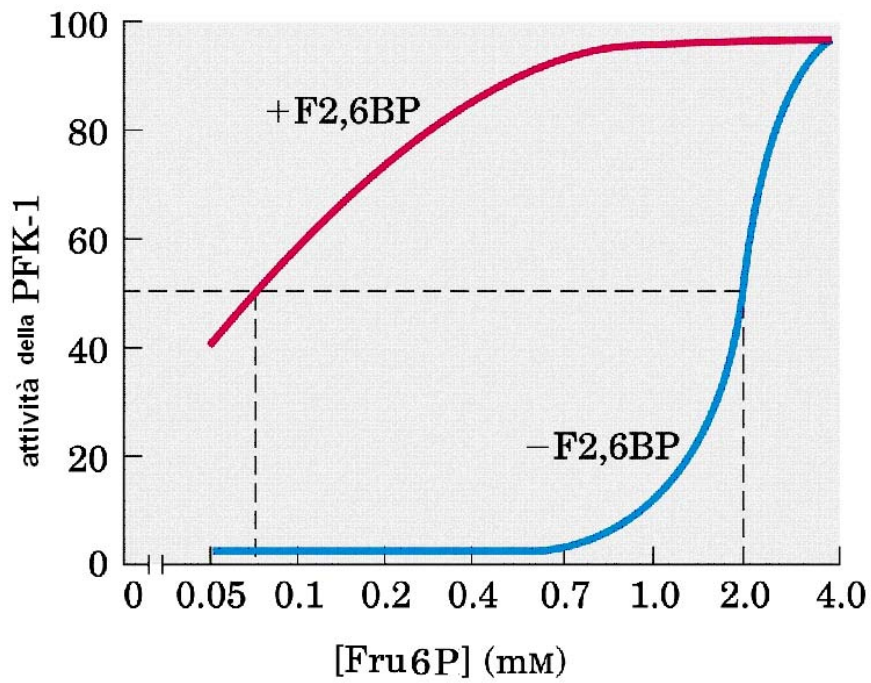


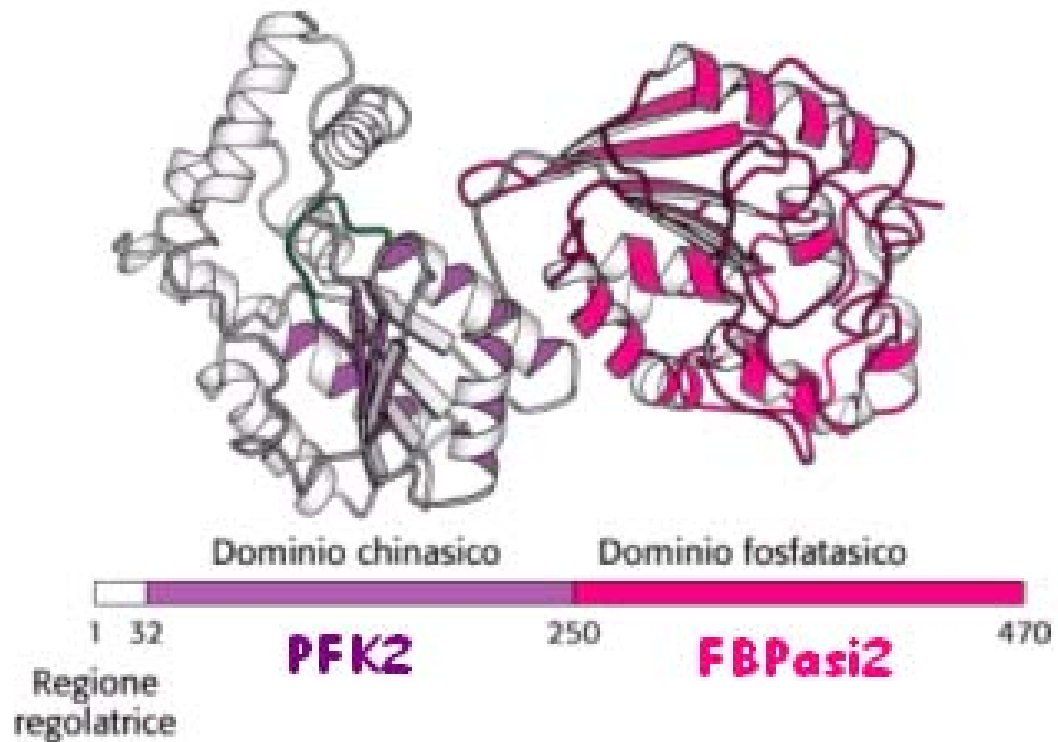
**Fruttosio 2,6-bisfosfato
(F-2,6-BP)**

**Il Fru2,6BP consente una
reciproca regolazione
di glicolisi e gluconeogenesi**

Il Fru2,6BP è un potente

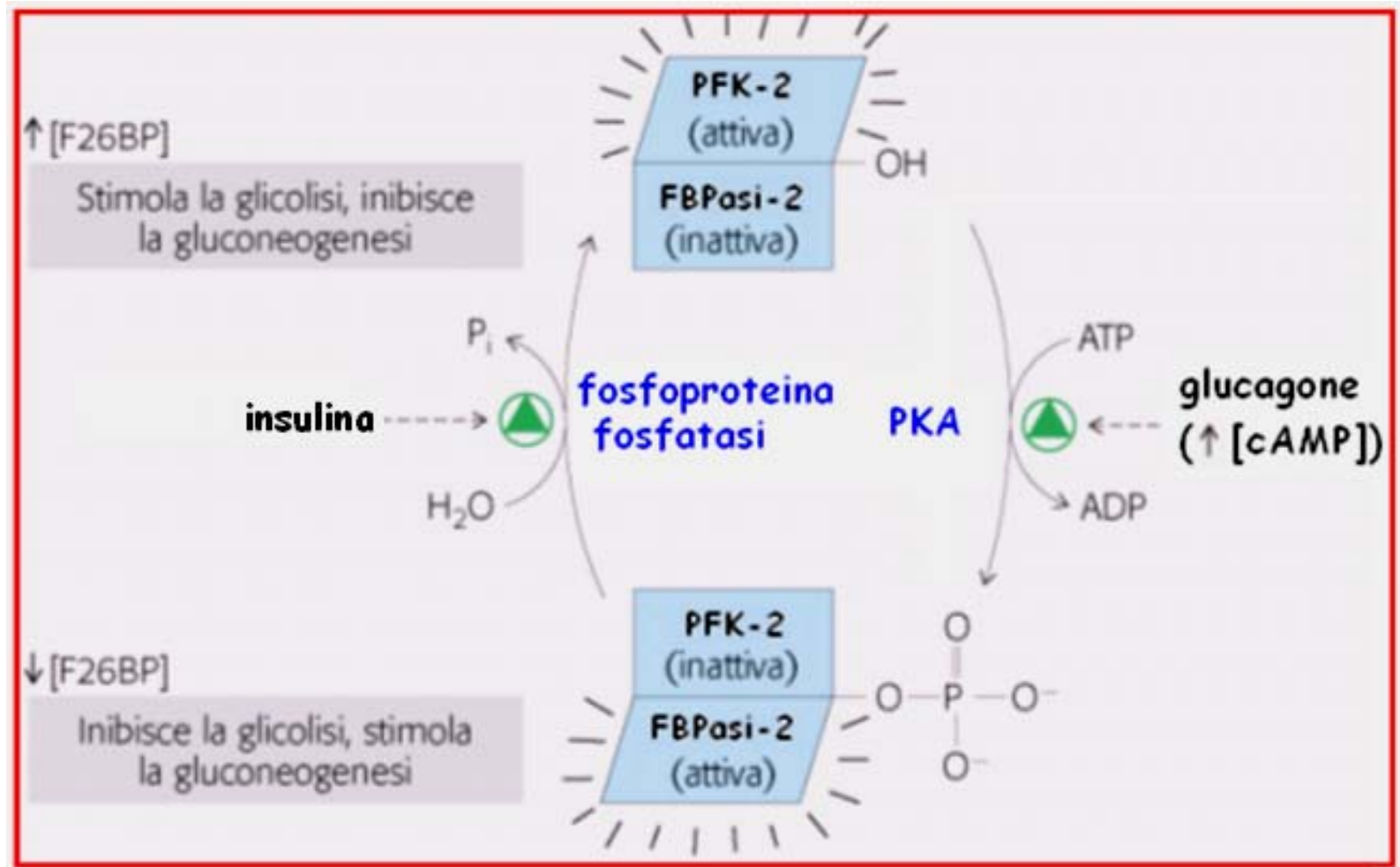
- **attivatore** della **PFK1**
e quindi della glicolisi
- **inibitore** della **Fru1,6BPasi**
e quindi della gluconeogenesi

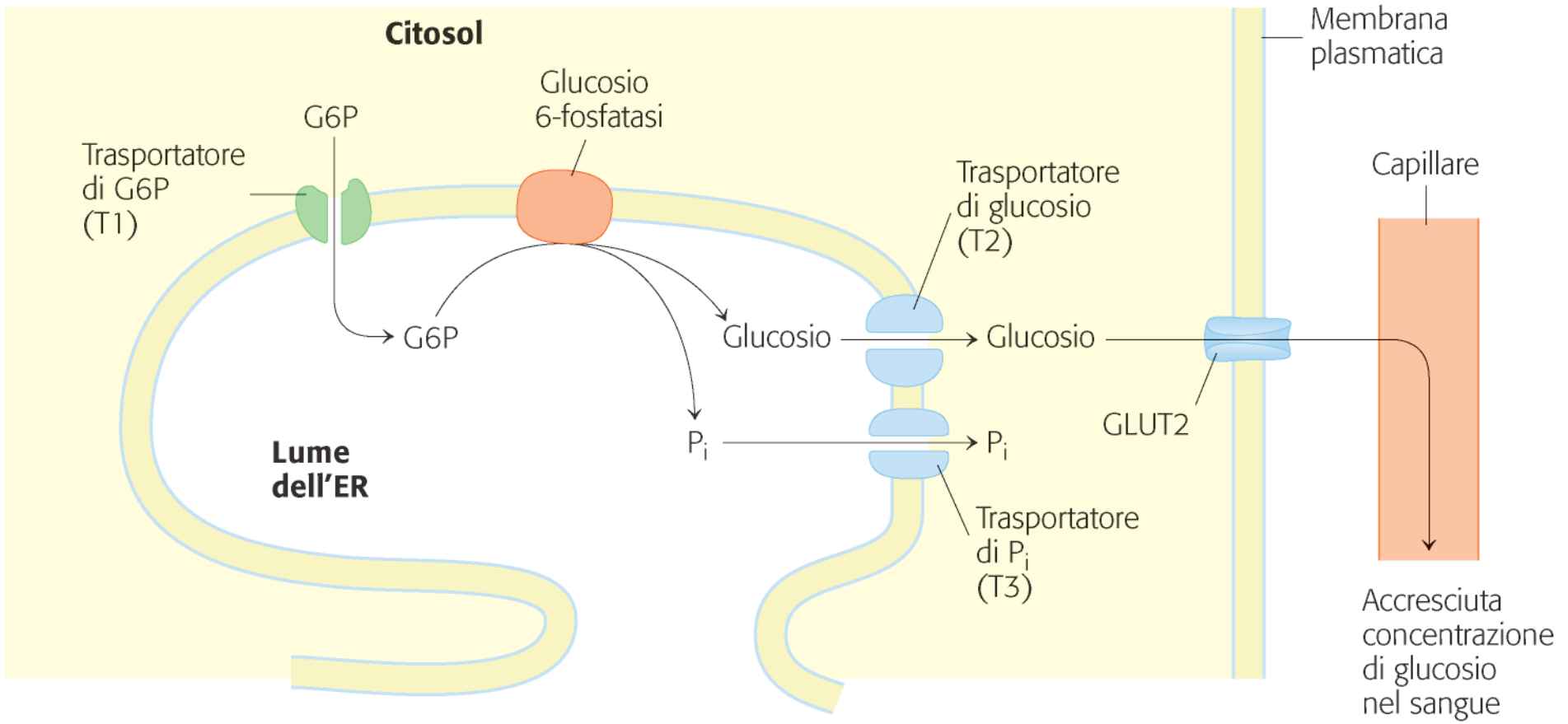


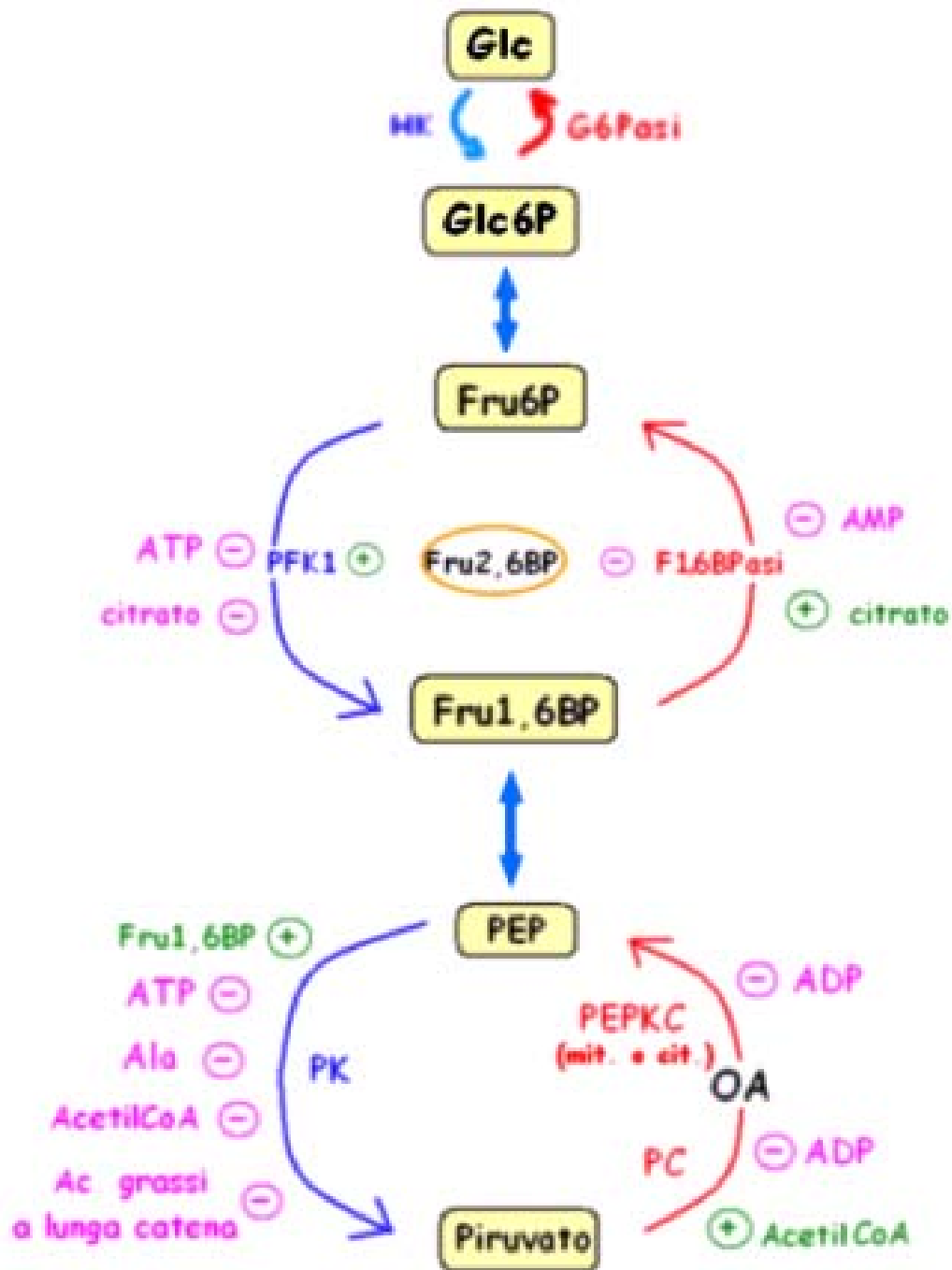


ENZIMA TANDEM

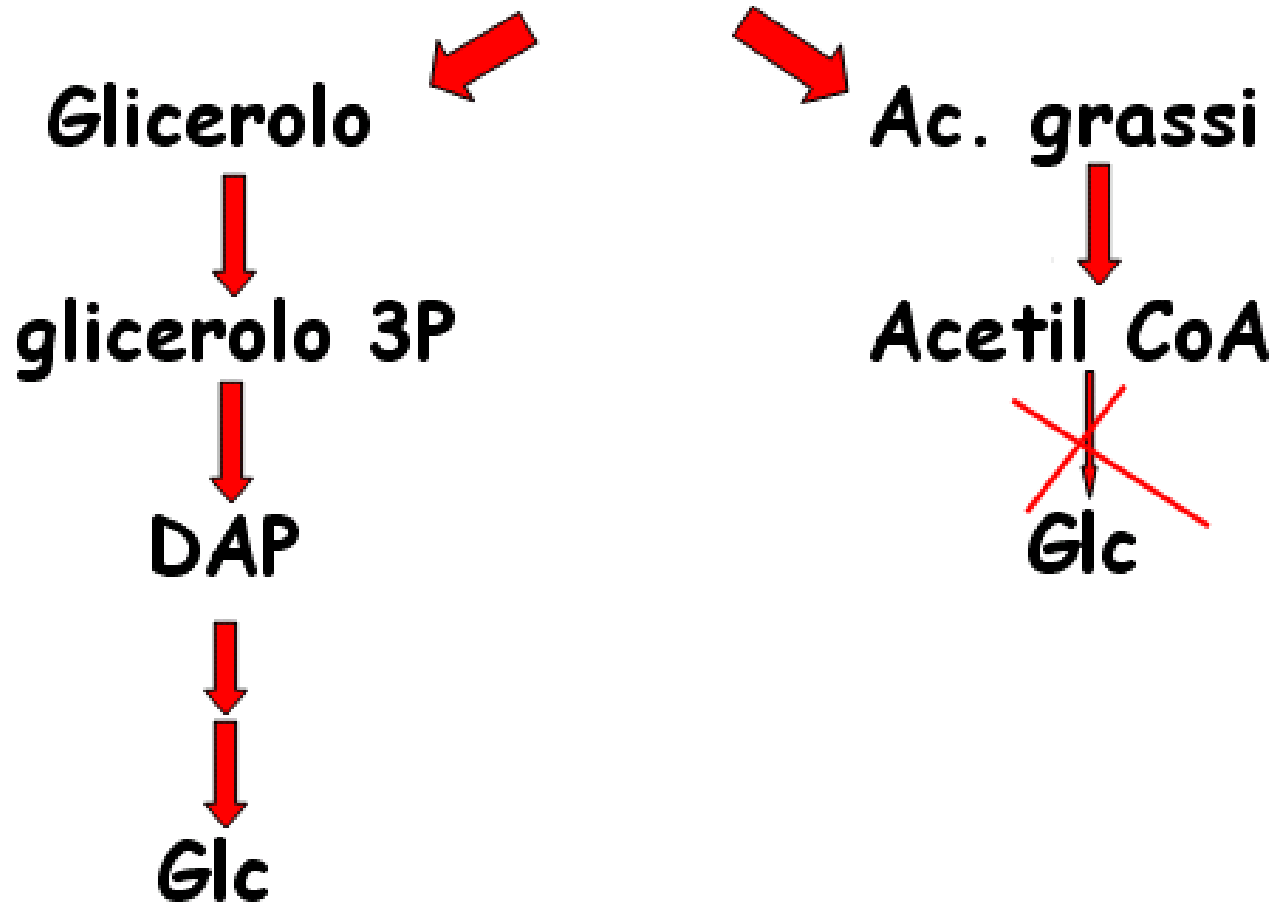
I livelli di F26BP sono controllati dal glucagone e dall'insulina







TRIGLICERIDI



IN CONDIZIONI FISIOLOGICHE
LA REAZIONE È **IRREVERSIBILE**

Piruvato  Acetil CoA

GLI ACIDI GRASSI **NON** SI POSSONO
TRASFORMARE IN CARBOIDRATI

C19 -> 8 Acetil CoA (C2)

+

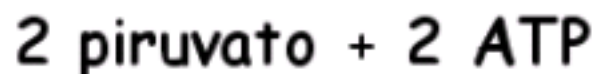
1 Propionil CoA (C3)



Succinil CoA

(intermedio del ciclo di Krebs)

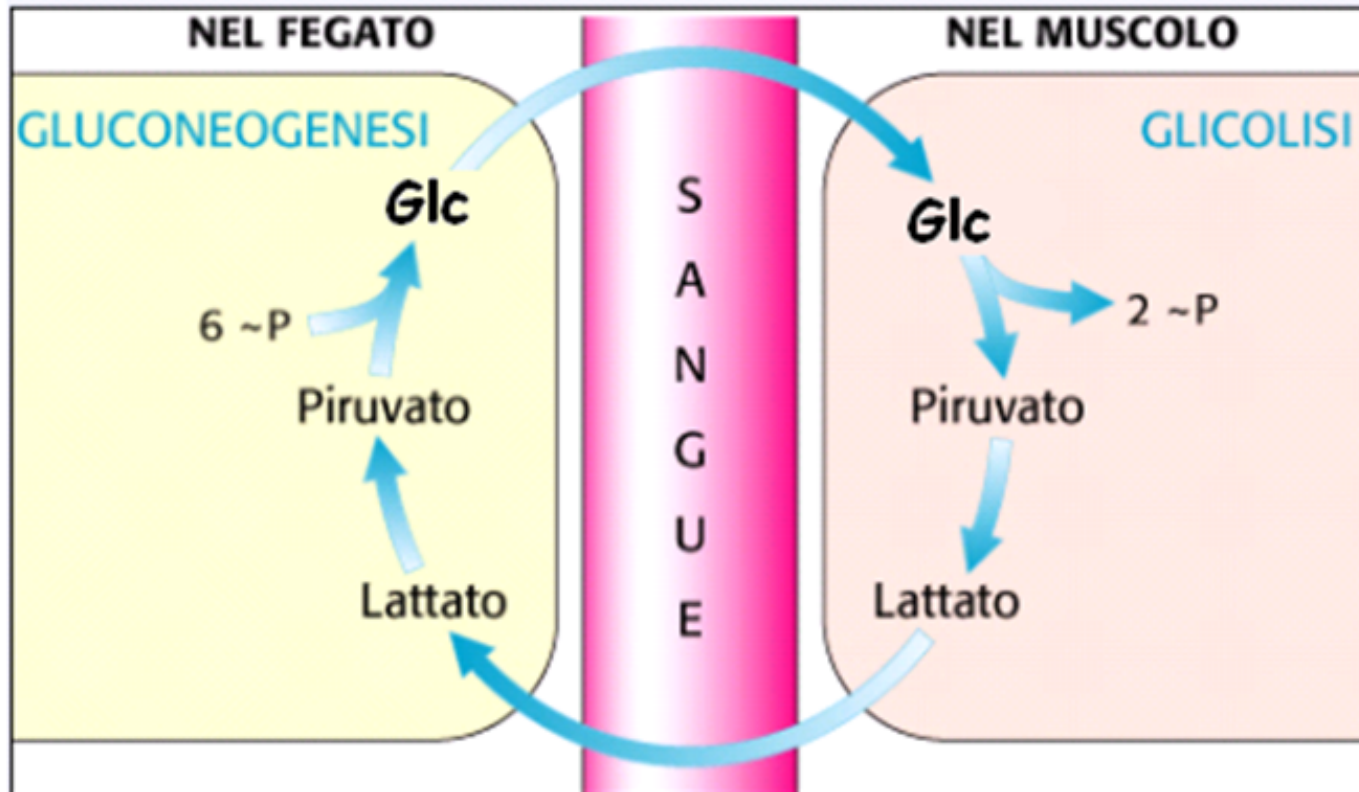
REAZIONE NETTA DELLA GLICOLISI



REAZIONE NETTA DELLA GLUCONEOGENESI



ciclo di Cori



CICLO Glc-Ala

