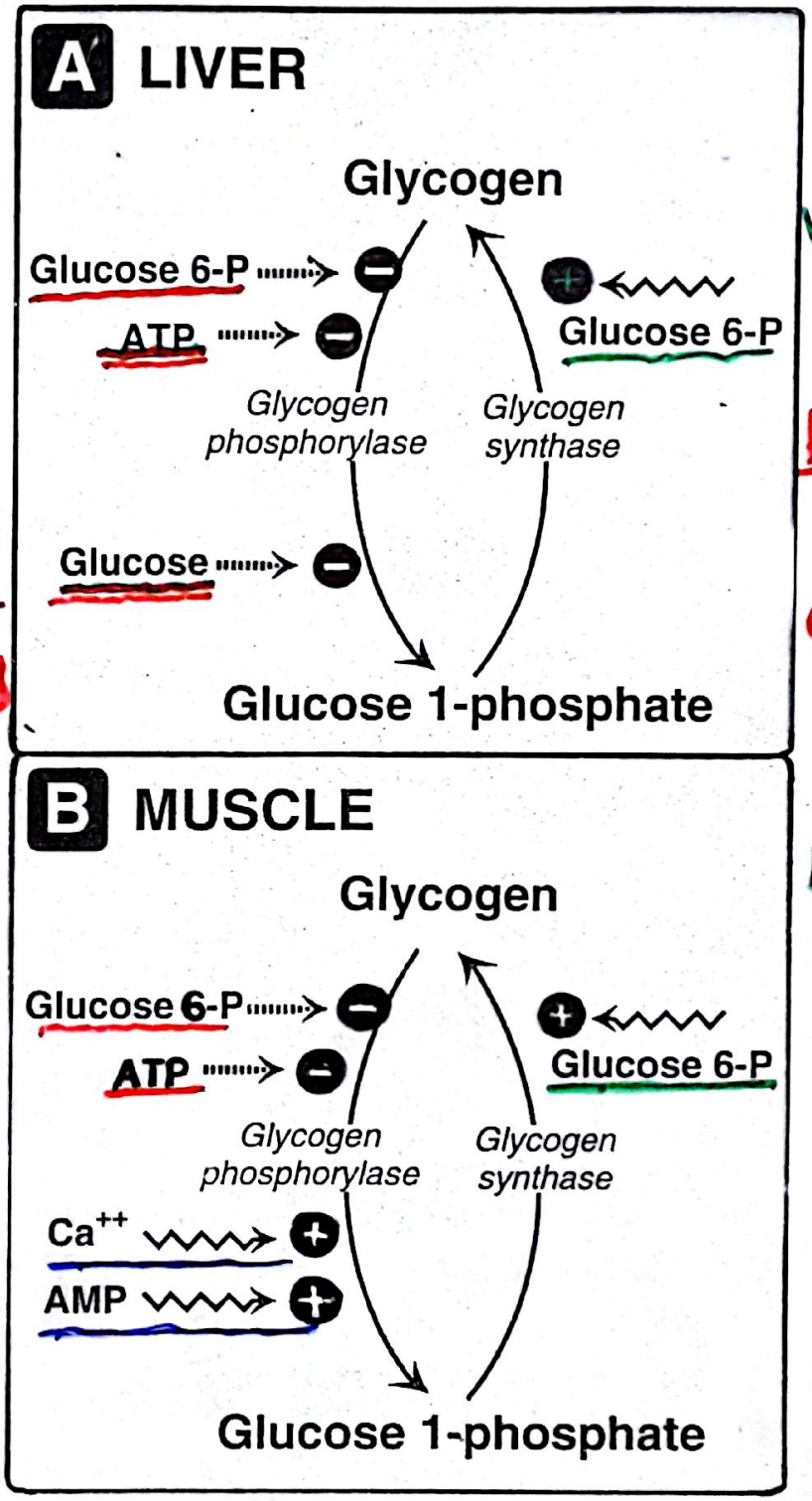
Regulation of Glycogen Synthesis and Degradation

A. Allosteric Regulation

- During Well-fed state
  Glu↑, G6P↑
  ATP↑

- Activation of glycogen degradation in muscle by Ca^{2+}

- Activation of Glycogen degradation in muscle by 5-AMP

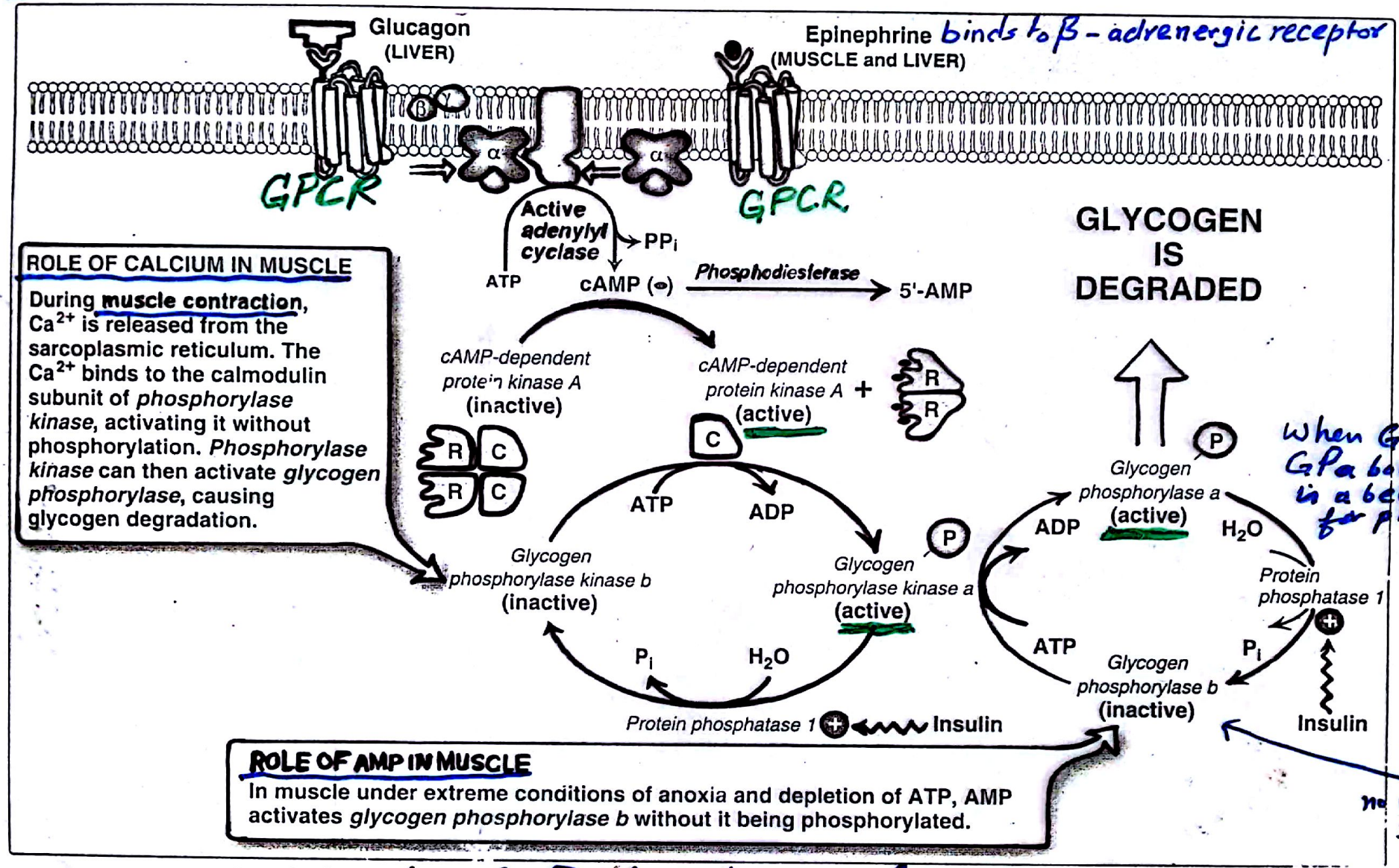


Well fed:-
synthesis ↑
Degradation ↓
High Energy
High [G] = Glu

Fasting:-
Synthesis ↓
Degradation ↑
(low energy, low glu)

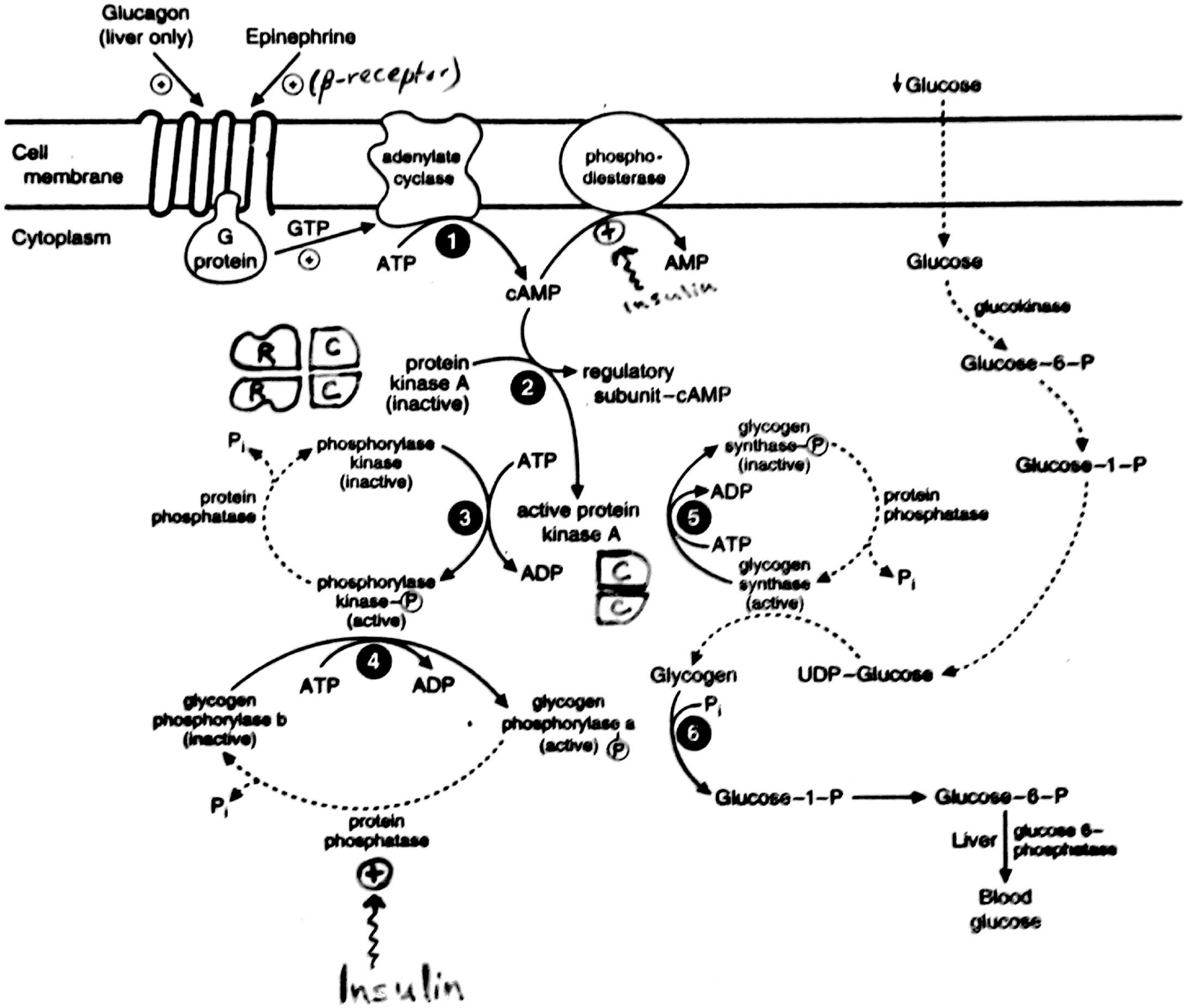
Exercise:-
Degradation ↑

Activation of Glycogen Degradation by cAMP-directed Pathway



1- Activation of Protein kinase A
2- Activation of phosphorylase Kinase
3- Activation of Glycogen phosphorylase

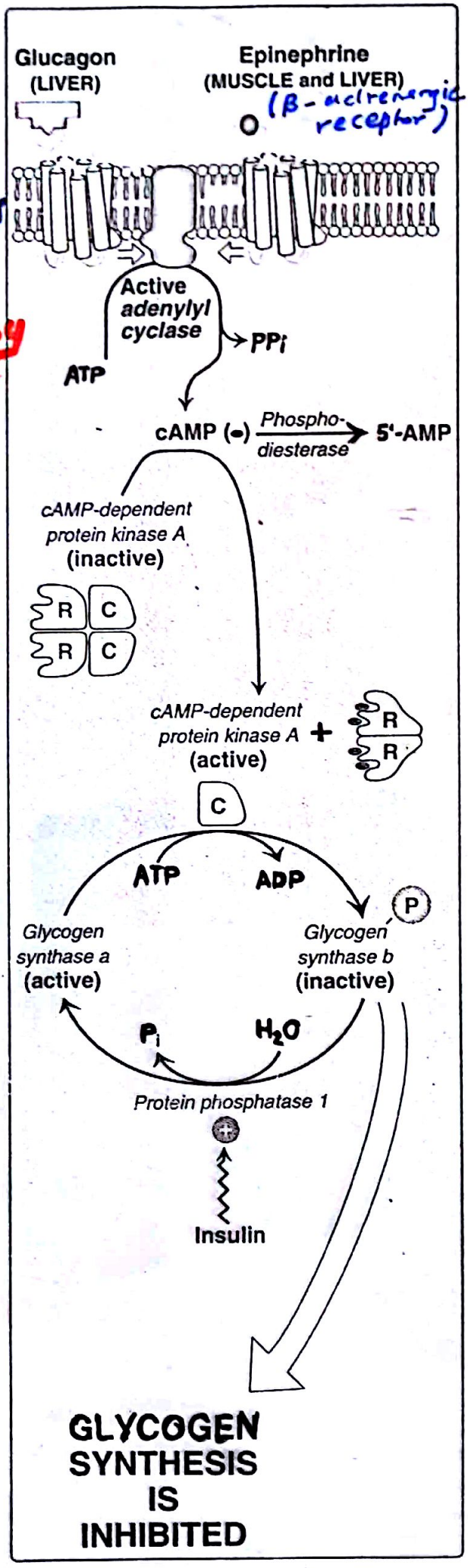
Regulation of Glycogen Synthesis and Degradation in the Liver :-



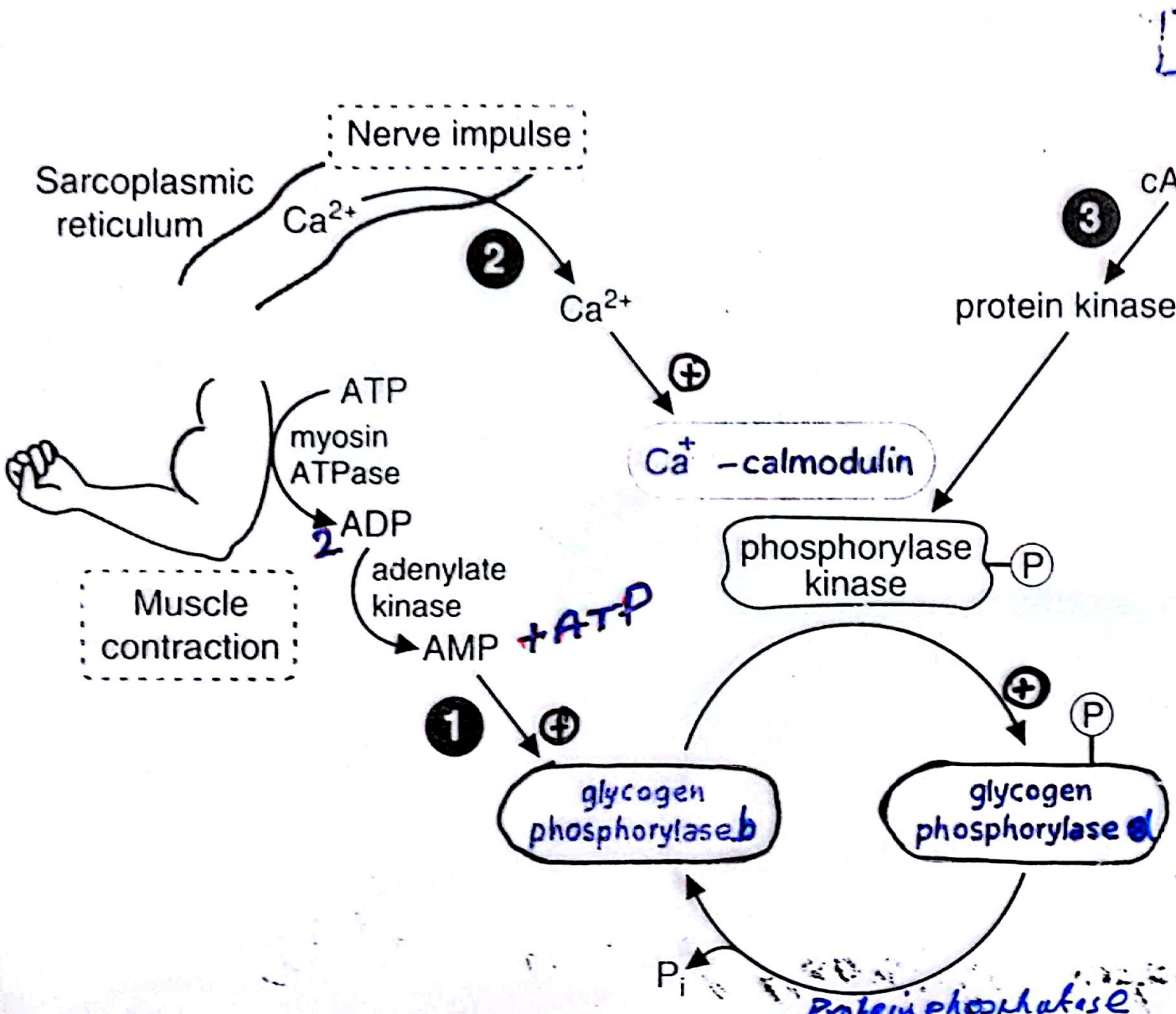
Hormonal Regulation of Glycogen Synthesis

- Inhibition by C-AMP mediated Process

+ - Inhibition by other Protein kinases (cAMP indep. endent)



Activation of Muscle Glycogen Phosphorylase During Excercise



Differences in Regulation of Glycogen Deg. & Syn. between Liver & muscle:-

- Glucagon has no effect on muscle
- AMP is an allosteric effector of muscle GP only (not liver)
- neural stimulation ↑ Ca^+
- Glu is not a physiological stimulator of GS in muscle
- Glycogen is stronger inhibitor of GS in muscle than in liver
- Effect of epinephrine-stimulated Protein Kinase A is similar in liver & muscle.

Calmodulin mediates many effects of intracellular Ca^{2+}

Epinephrine → α-agonist receptor
→ ↑ phospholipase C

PIP_2 —(phospholipase C)→ DAG + IP_3

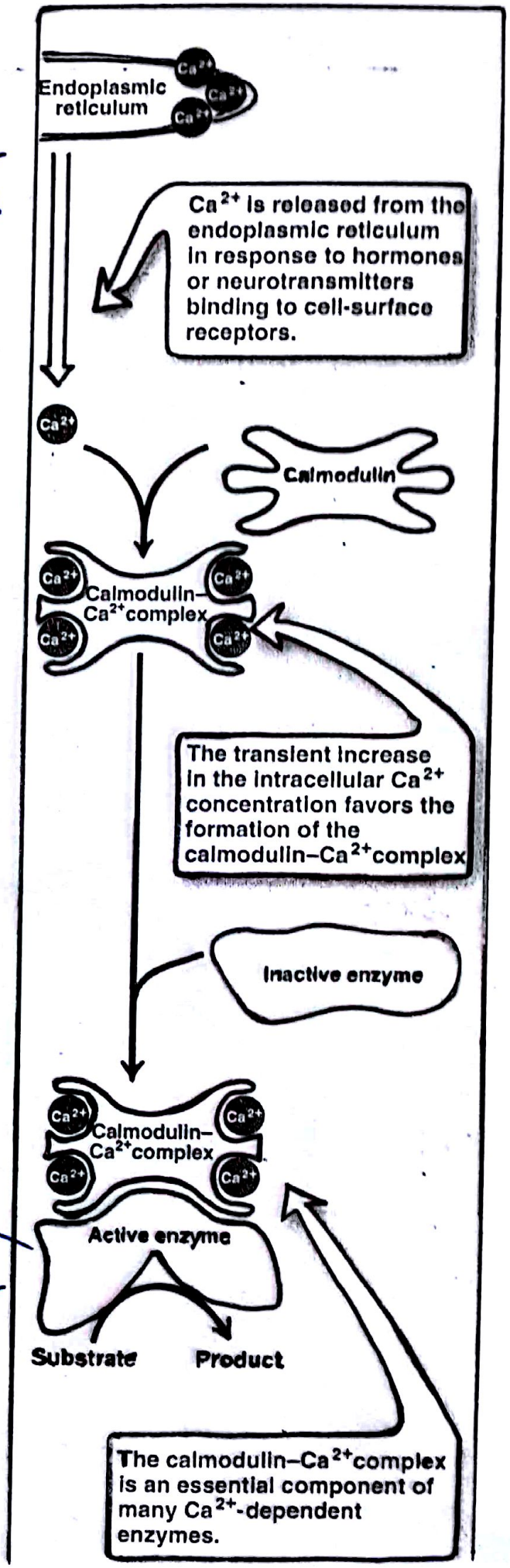
IP_3 → E.R. ⇉ Ca^{2+}

Ca^{2+} also activate protein kinase C

e.g. :-

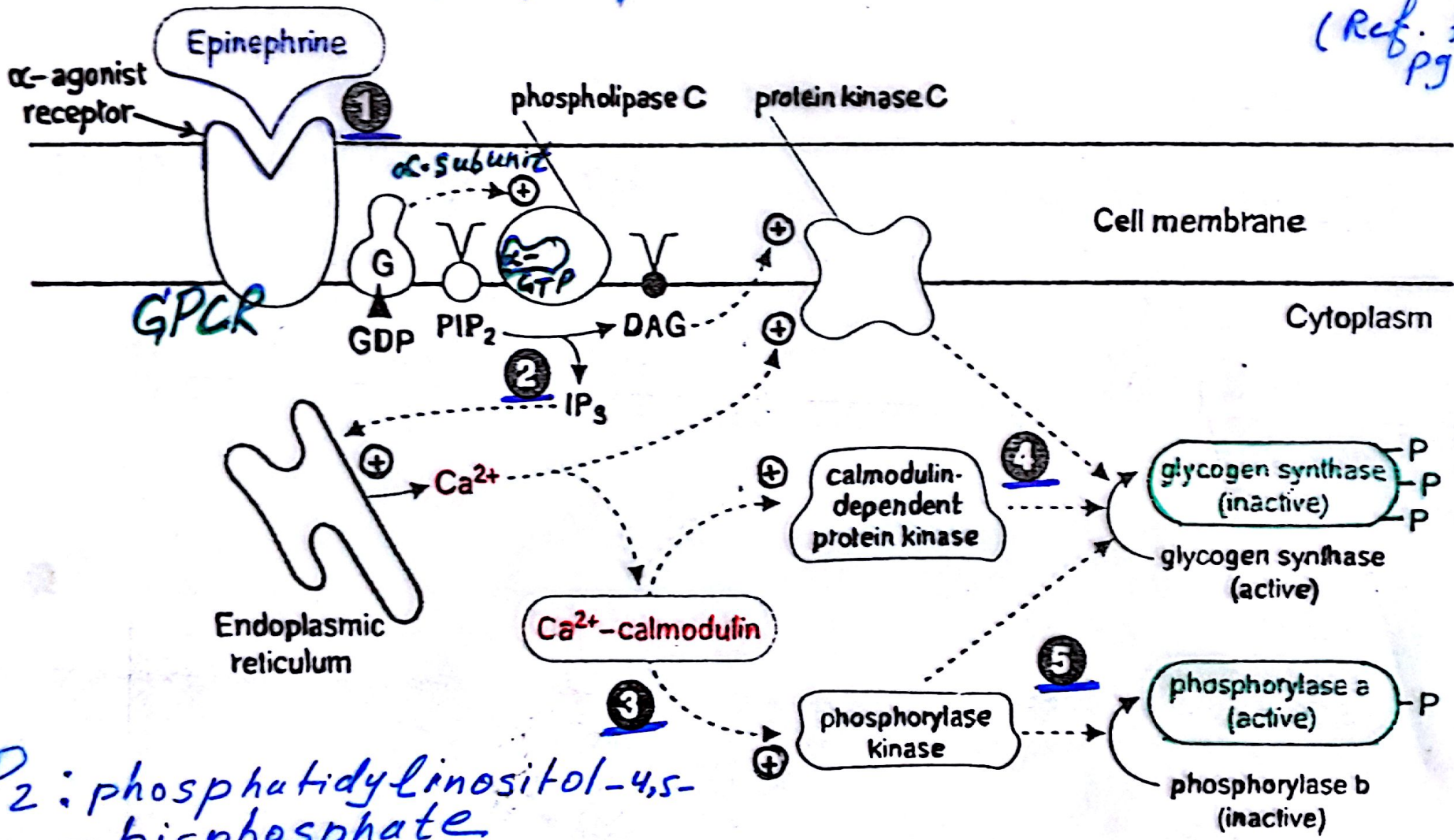
(1) Calmodulin-dependant protein kinase

(2) phosphorylase kinase



Regulation of Glycogen Synthesis and Degrdation by Epinephrine and Ca^{2+} in the Liver

(Ref. fig 17.8, Pg 205)



PIP_2 : phosphatidylinositol-4,5-bisphosphate

IP_3 : Inositol 1,4,5-trisphosphate

DAG : Diacylglycerol