

Figure 5.1 Examples of the six major classes of the international classification of enzymes (THF is tetrahydrofolate).

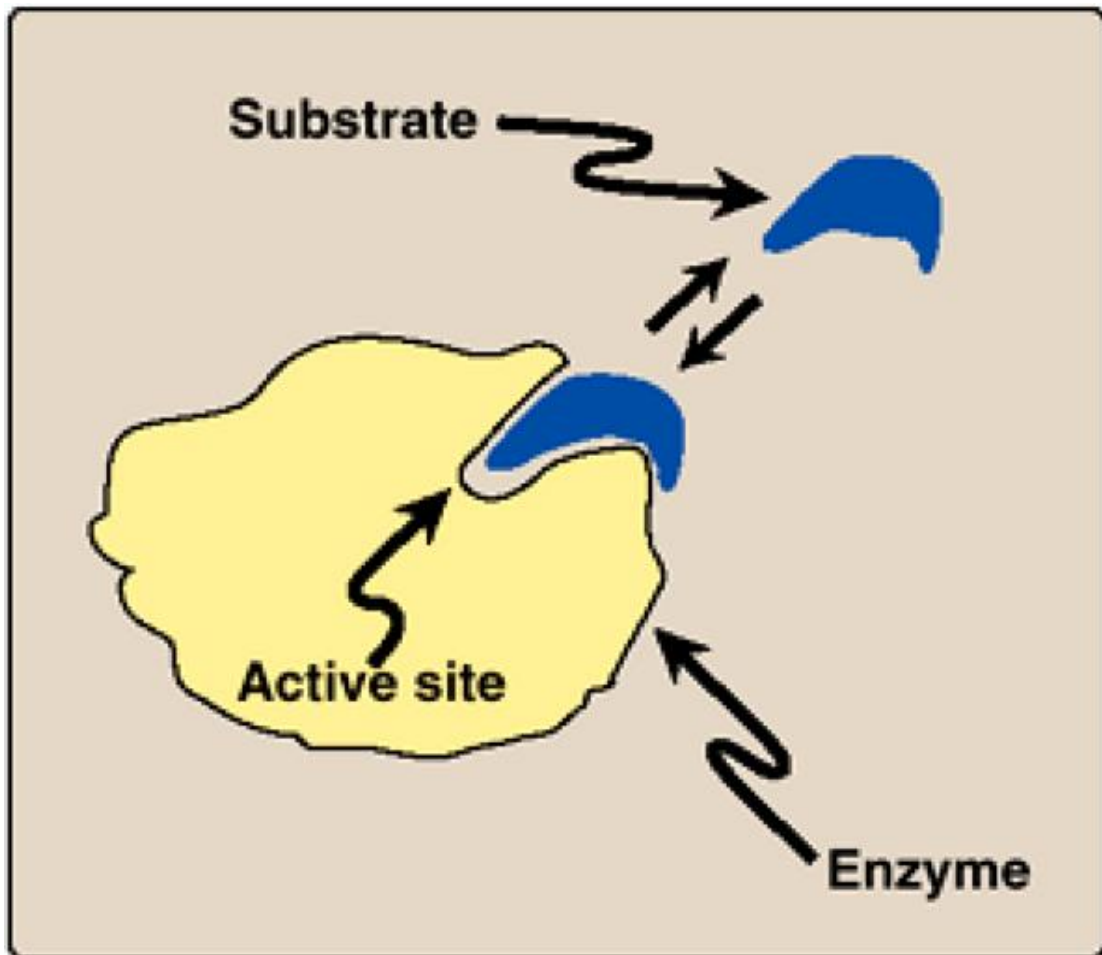


Figure 5.2 Schematic representation of an enzyme with one active site binding a substrate molecule.

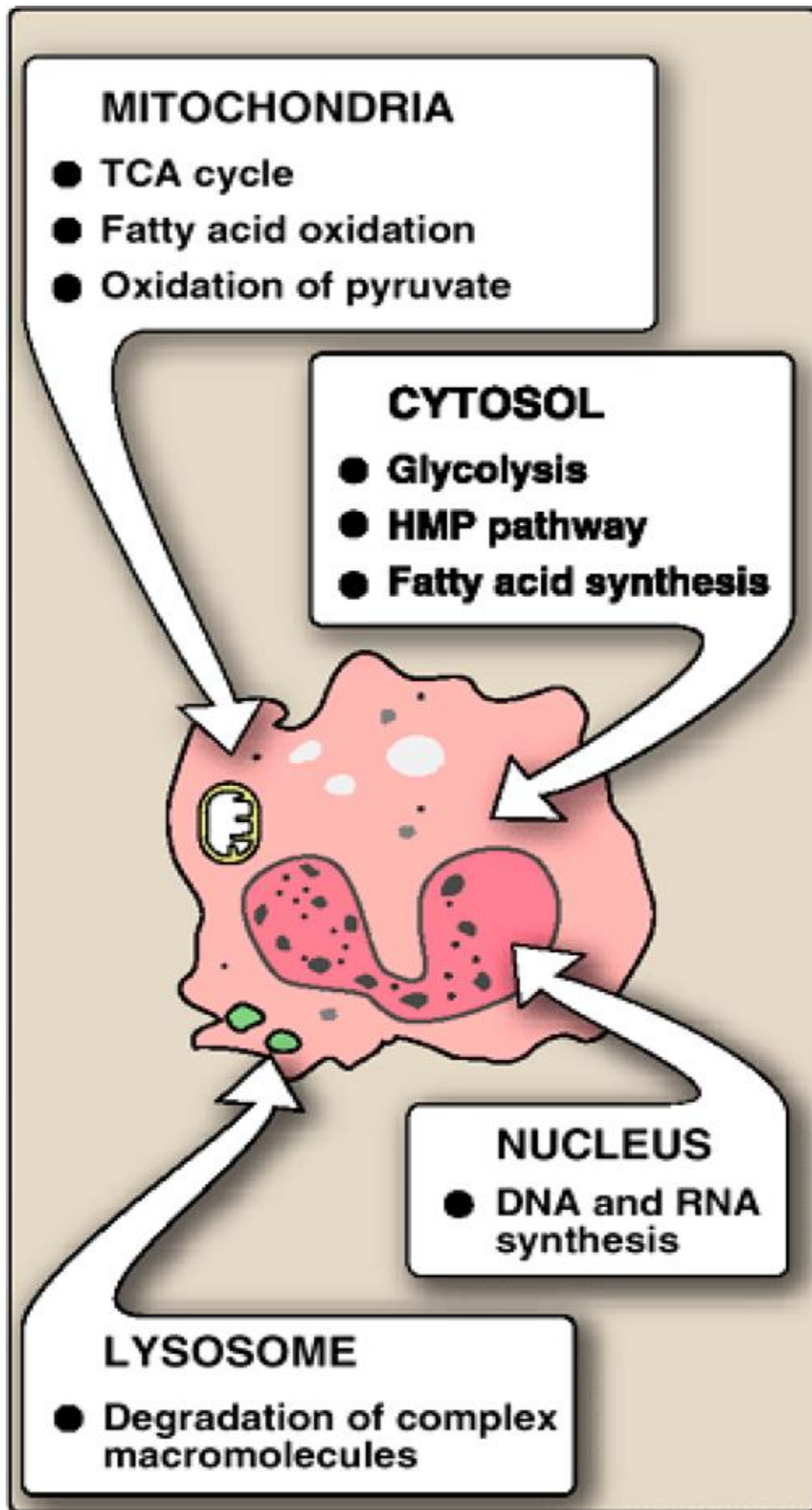


Figure 5.3 The intracellular location of some important biochemical pathways.

There is no difference in the free energy of the overall reaction (energy of reactants minus energy of products) between the catalyzed and uncatalyzed reactions.

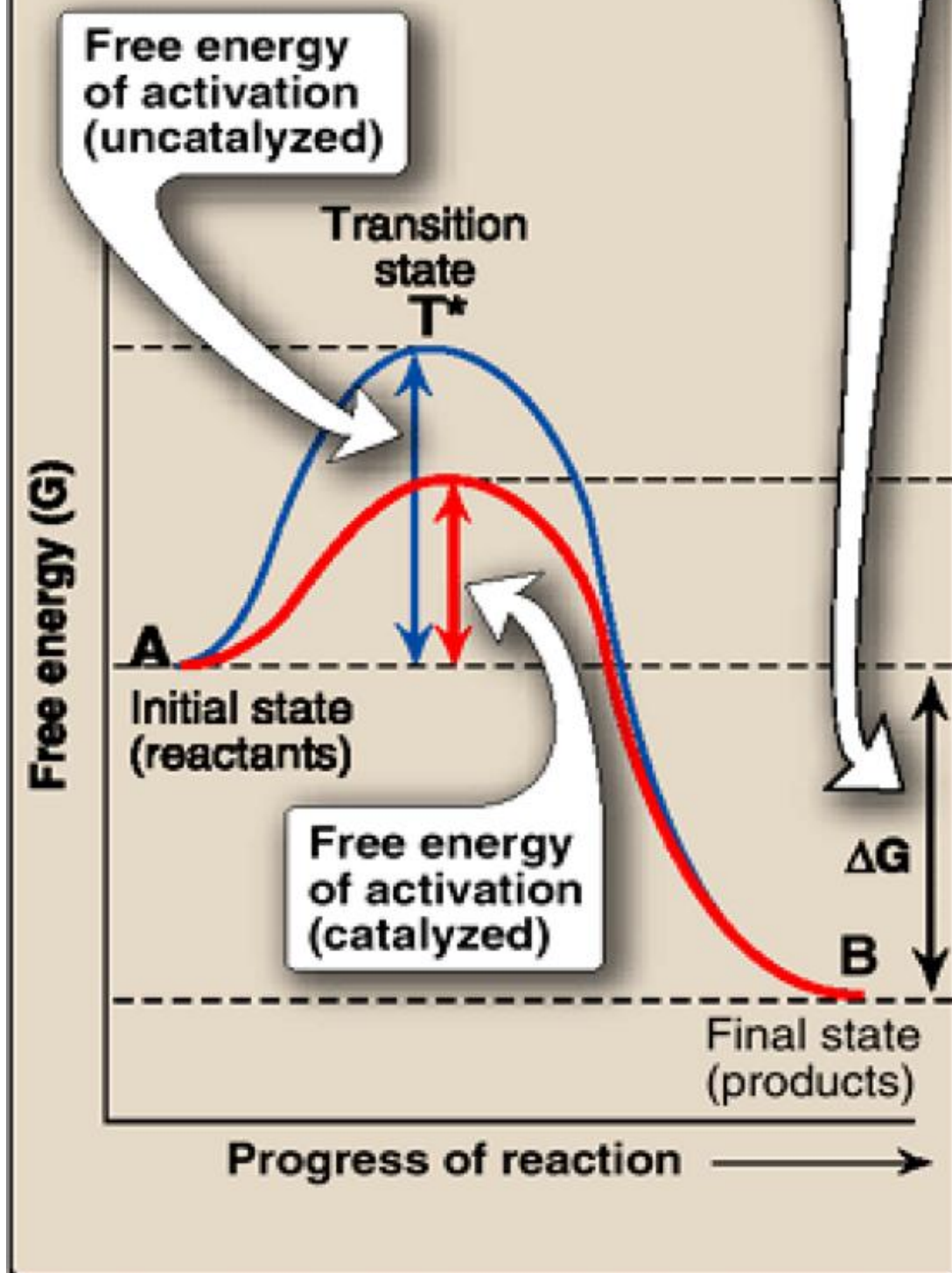


Figure 5.4 Effect of an enzyme on the activation energy of a reaction.

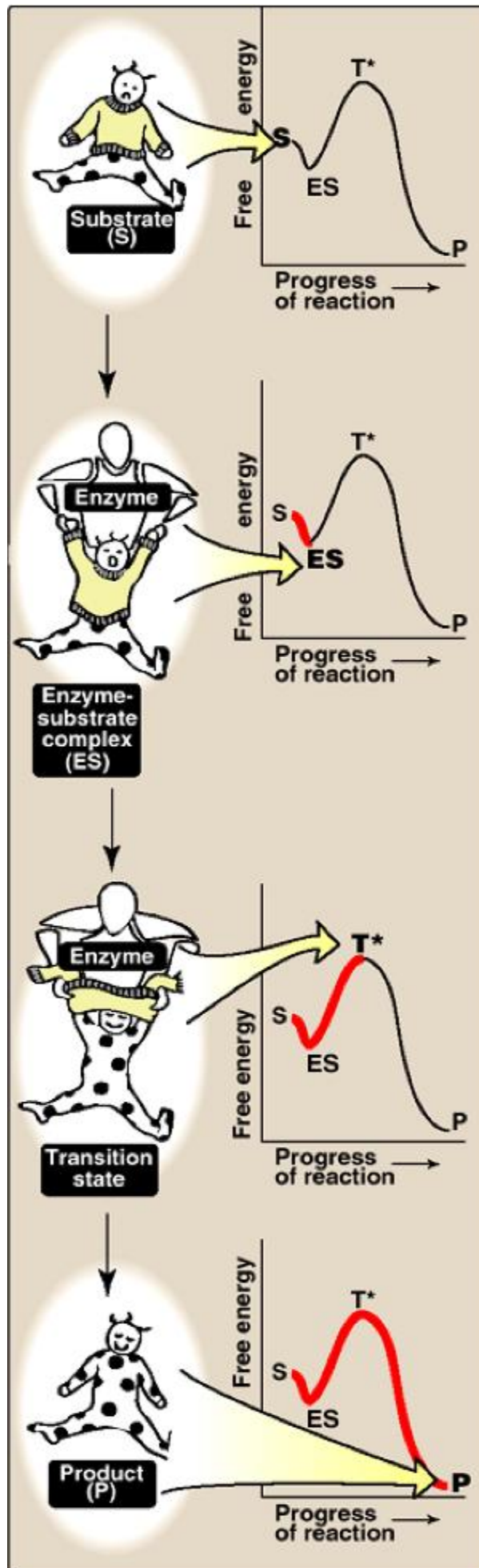


Figure 5.5 Schematic representation of energy changes accompanying formation of an enzyme-substrate complex and subsequent formation of a transition state complex.

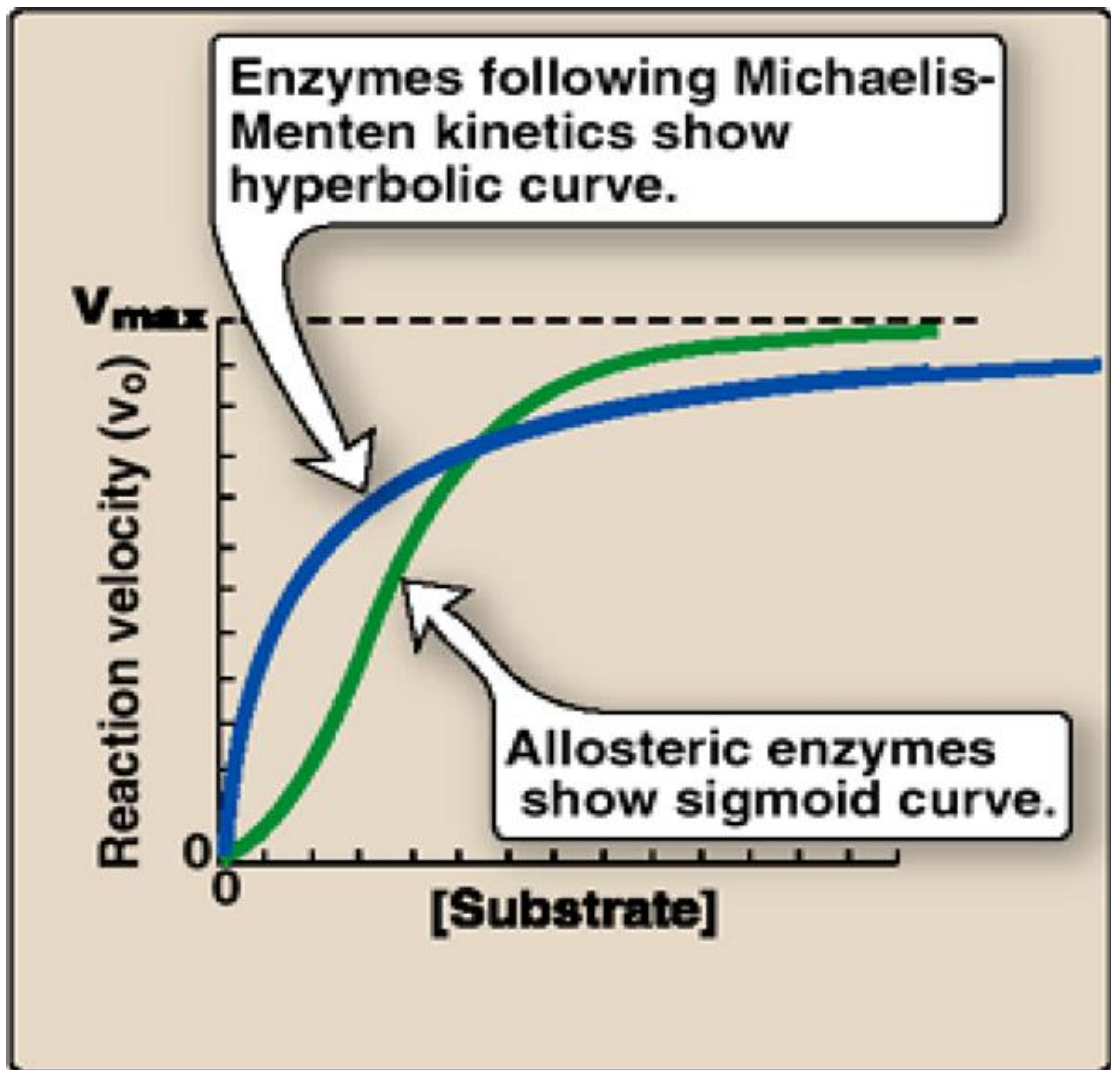


Figure 5.6 Effect of substrate concentration on reaction velocity.

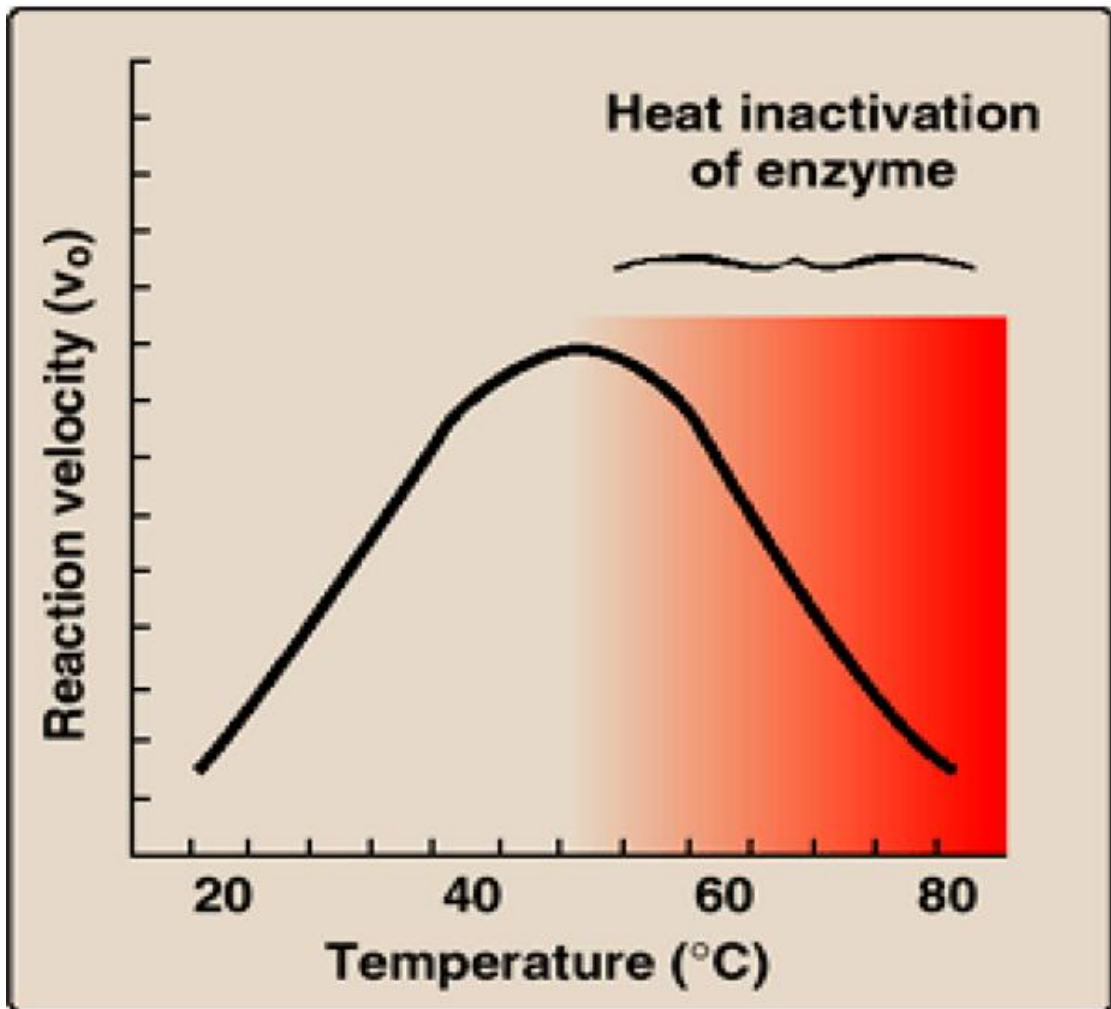


Figure 5.7 Effect of temperature on an enzyme-catalyzed reaction.

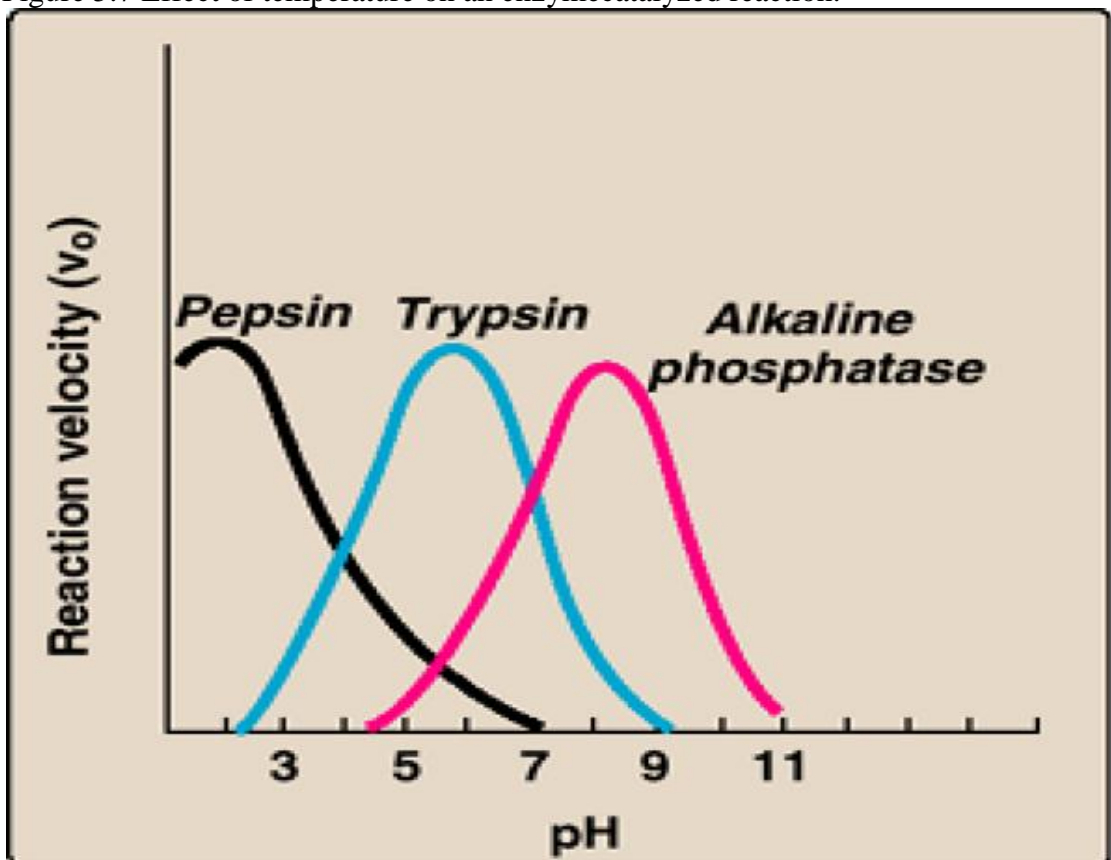


Figure 5.8 Effect of pH on enzyme-catalyzed reactions.

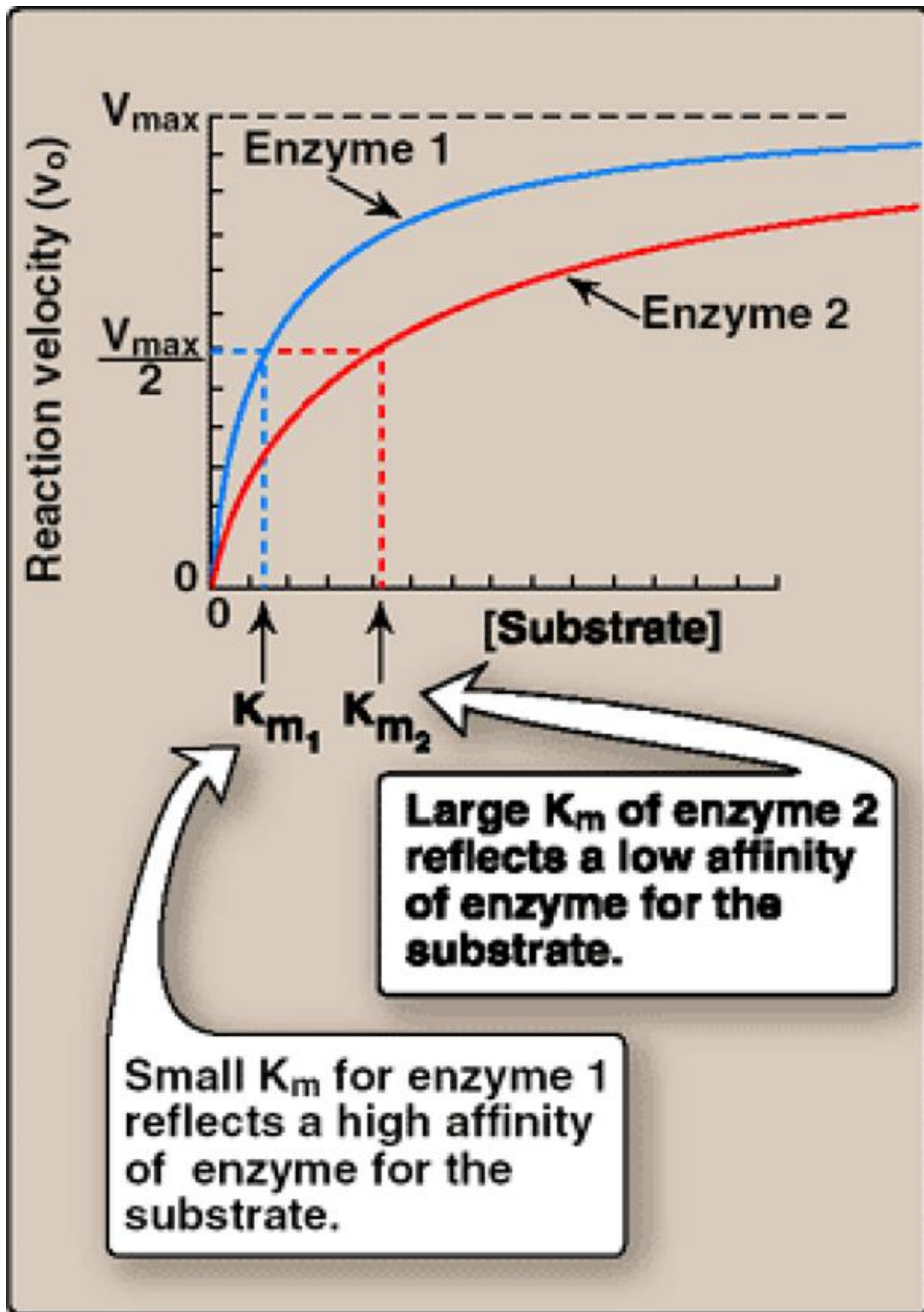


Figure 5.9 Effect of substrate concentration on reaction velocities for two enzymes: enzyme 1 with a small K_m , and enzyme 2 with a large K_m .

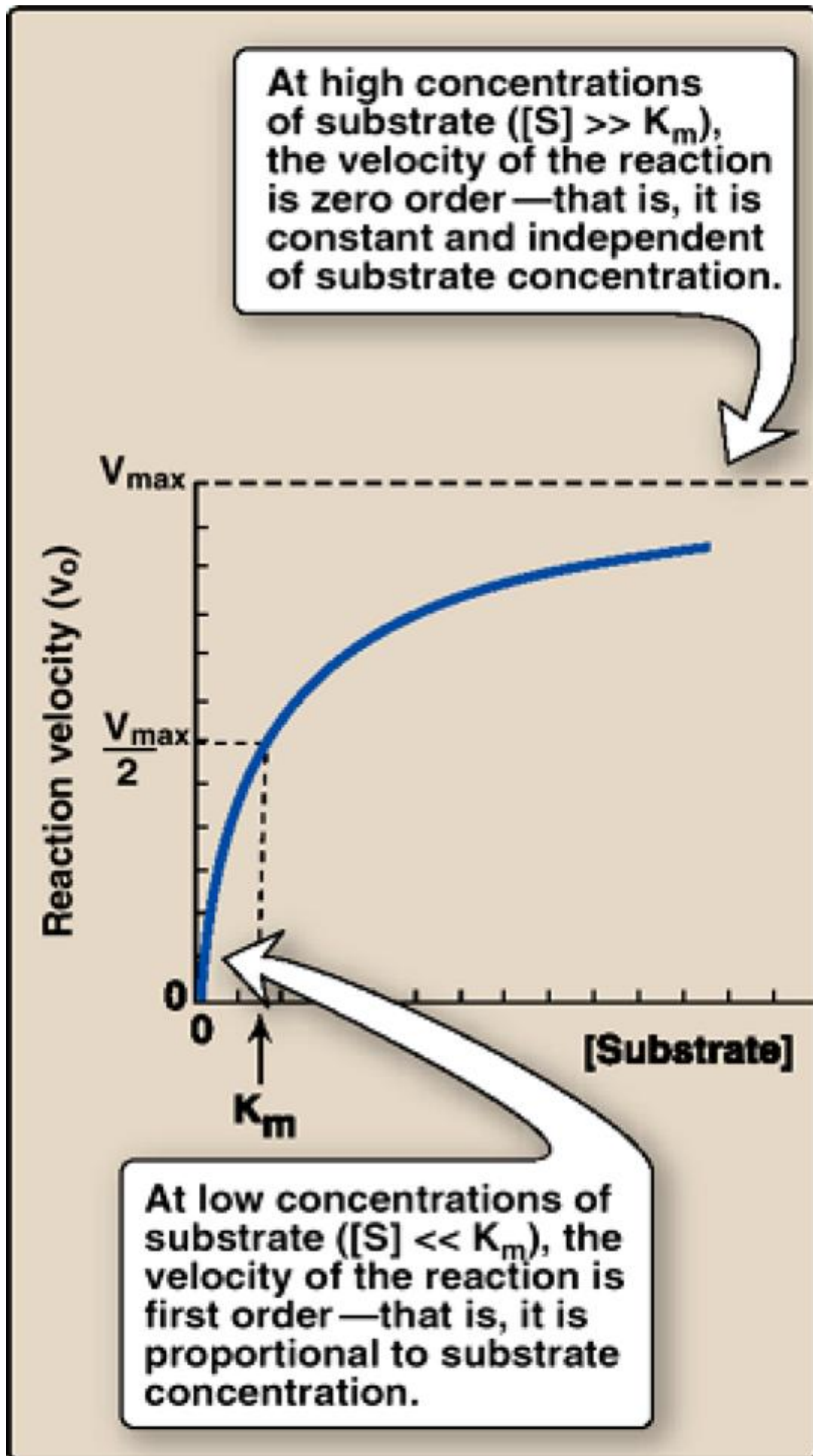


Figure 5.10 Effect of substrate concentration on reaction velocity for an enzyme-catalyzed reaction.

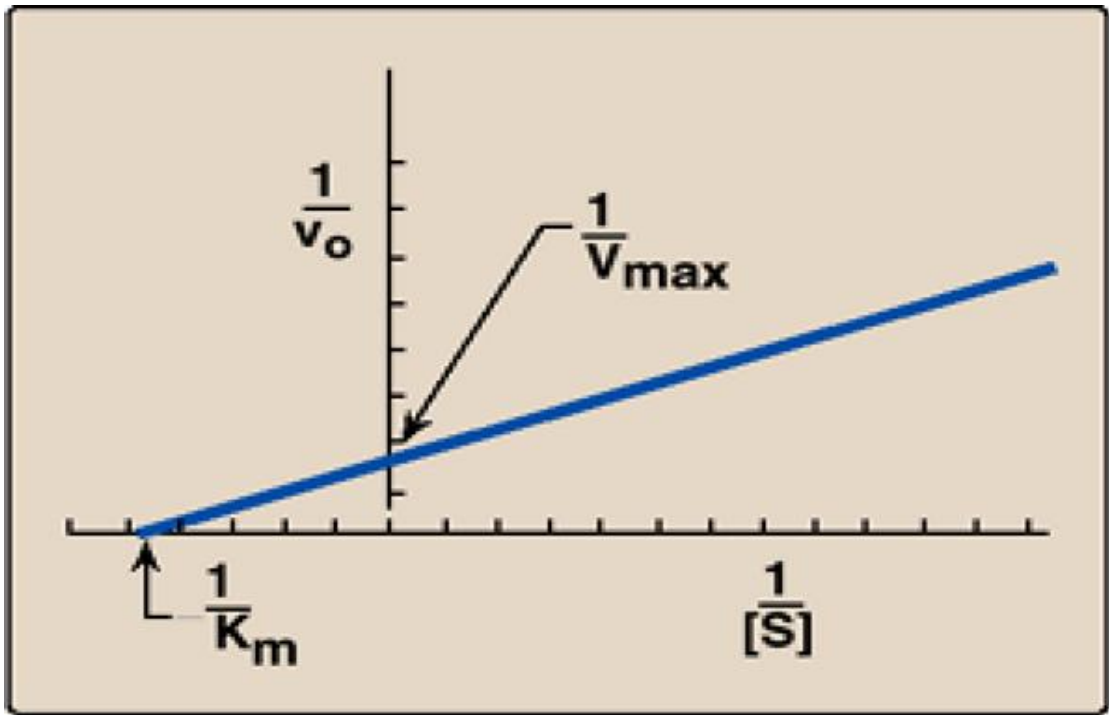


Figure 5.11 Lineweaver-Burk plot.

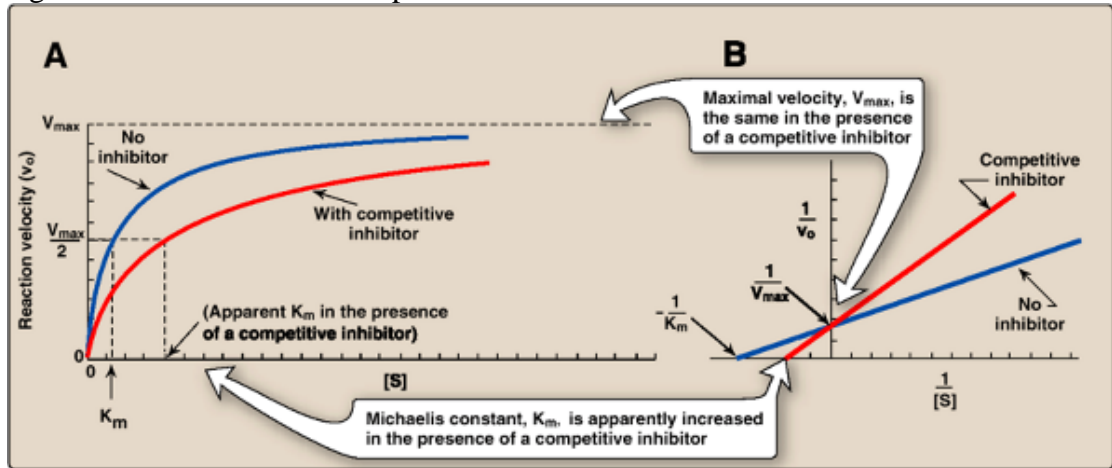


Figure 5.12 A. Effect of a competitive inhibitor on the reaction velocity (v_o) versus substrate ($[S]$) plot. B. Lineweaver-Burk plot of competitive inhibition of an enzyme.

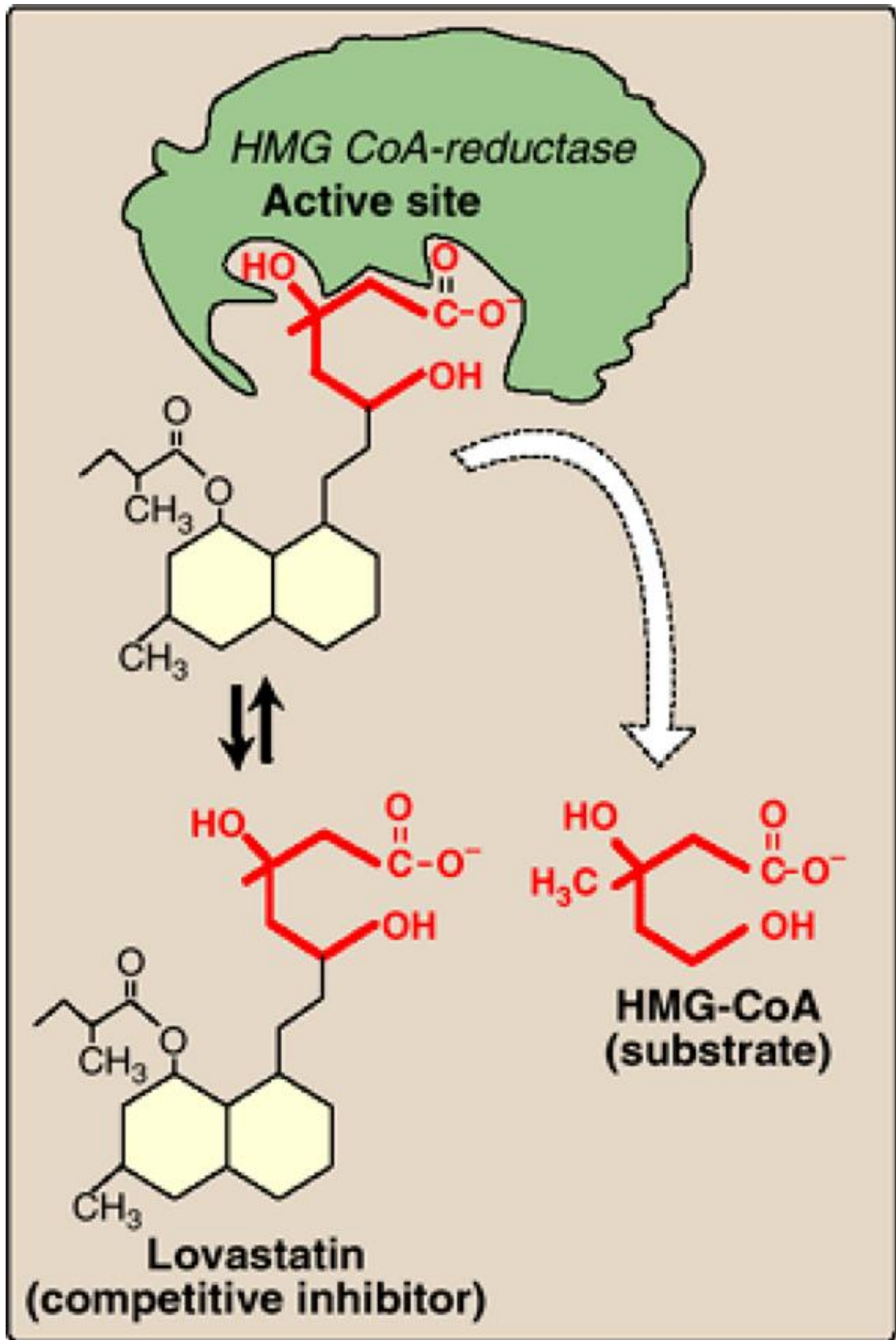


Figure 5.13 Lovastatin competes with HMG-CoA for the active site of HMG-CoA reductase.

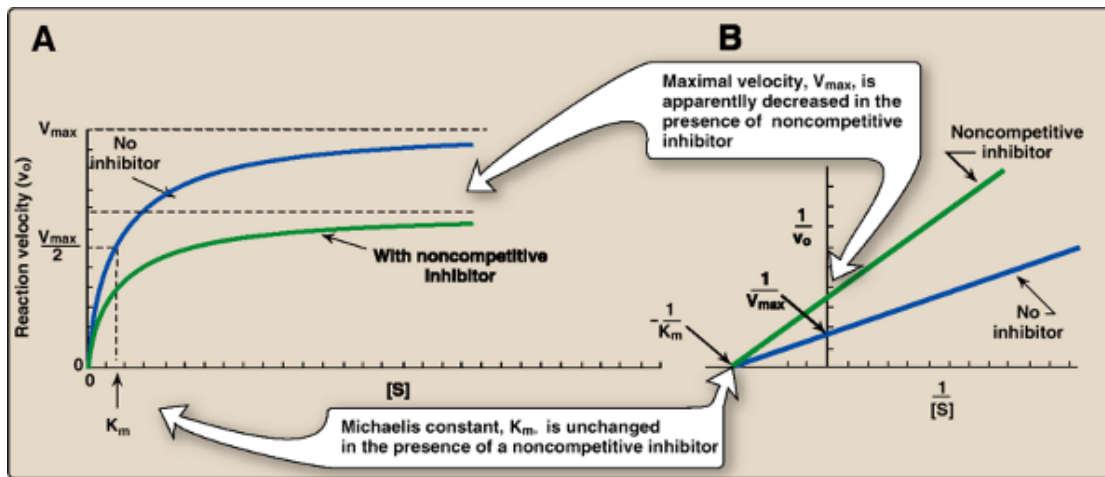


Figure 5.14 A. Effect of a noncompetitive inhibitor on the reaction velocity (v_o) versus substrate ($[S]$) plot. B. Lineweaver-Burk plot of noncompetitive inhibition of an enzyme.

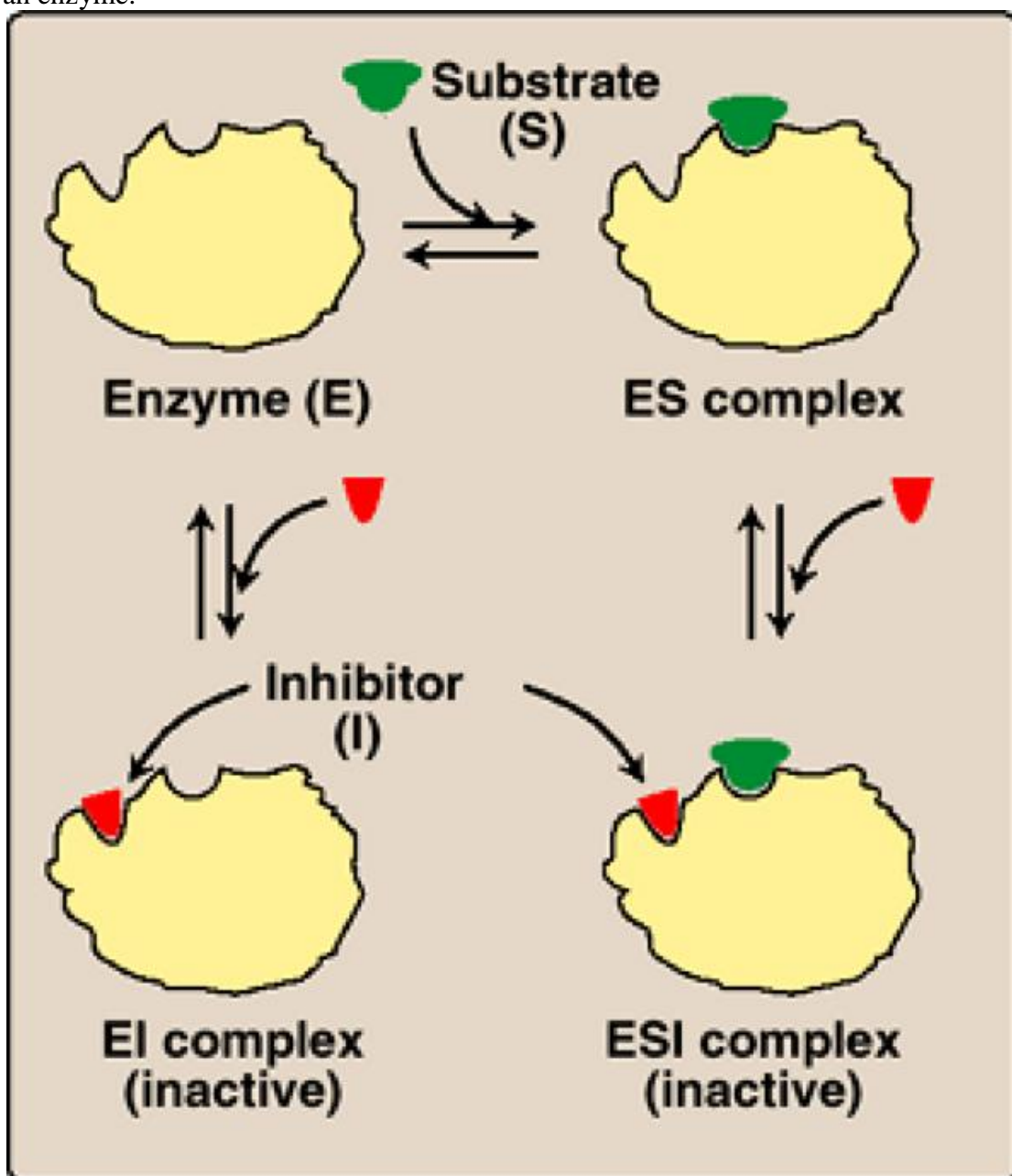


Figure 5.15 A noncompetitive inhibitor binding to both free enzyme and enzyme-substrate (ES) complex.

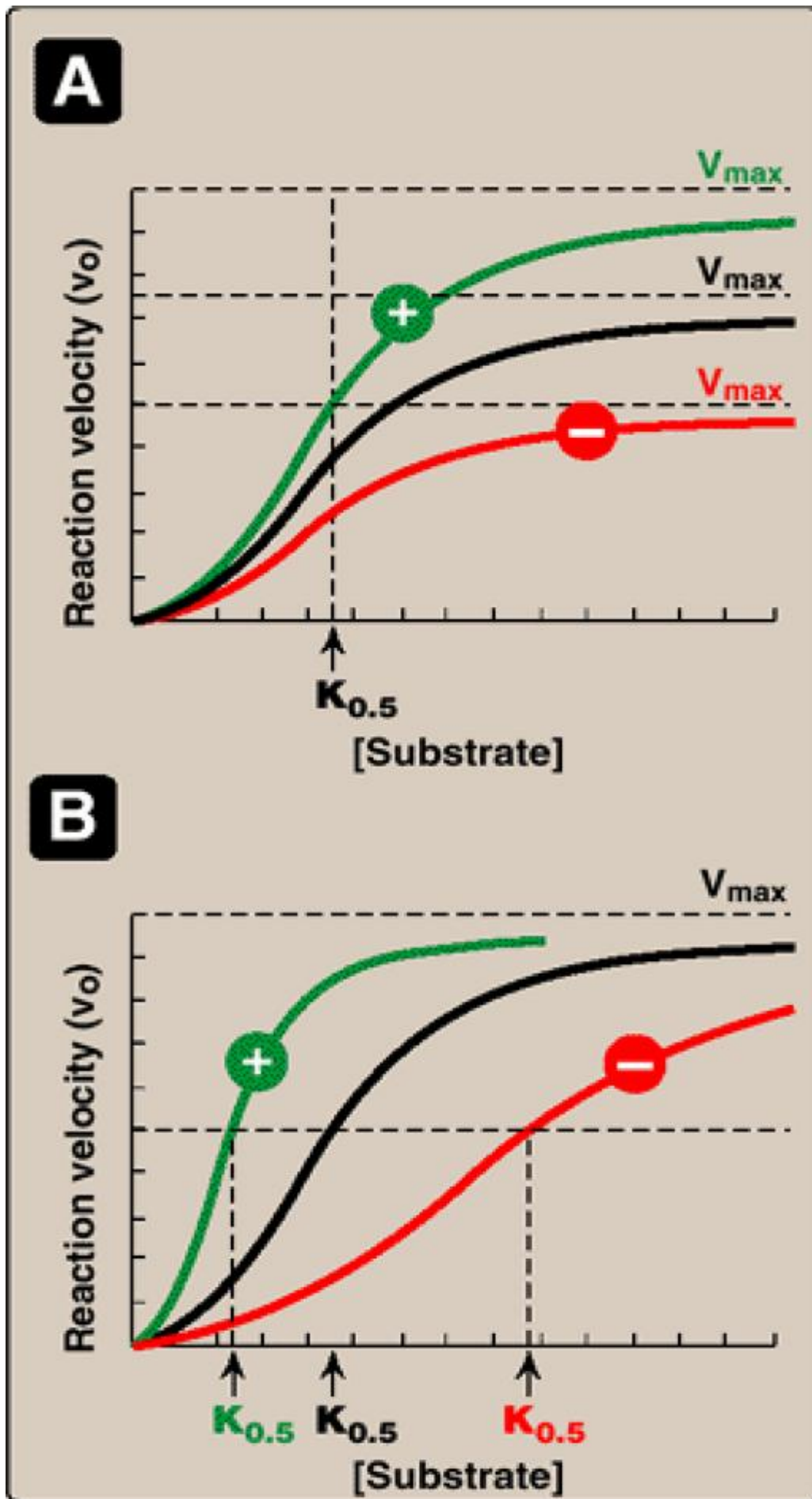


Figure 5.16 Effects of negative - or positive + effectors on an allosteric enzyme. A. V_{max} is altered. B. The substrate concentration that gives half-maximal velocity ($K_{0.5}$) is altered.

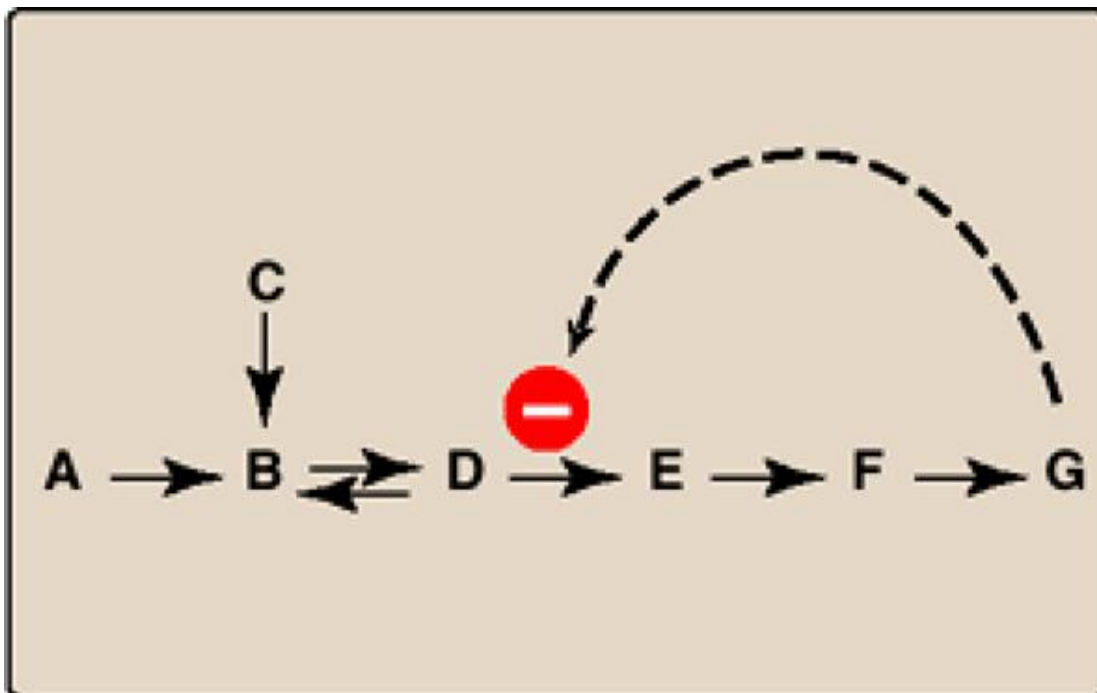


Figure 5.17 Feedback inhibition of a metabolic pathway.

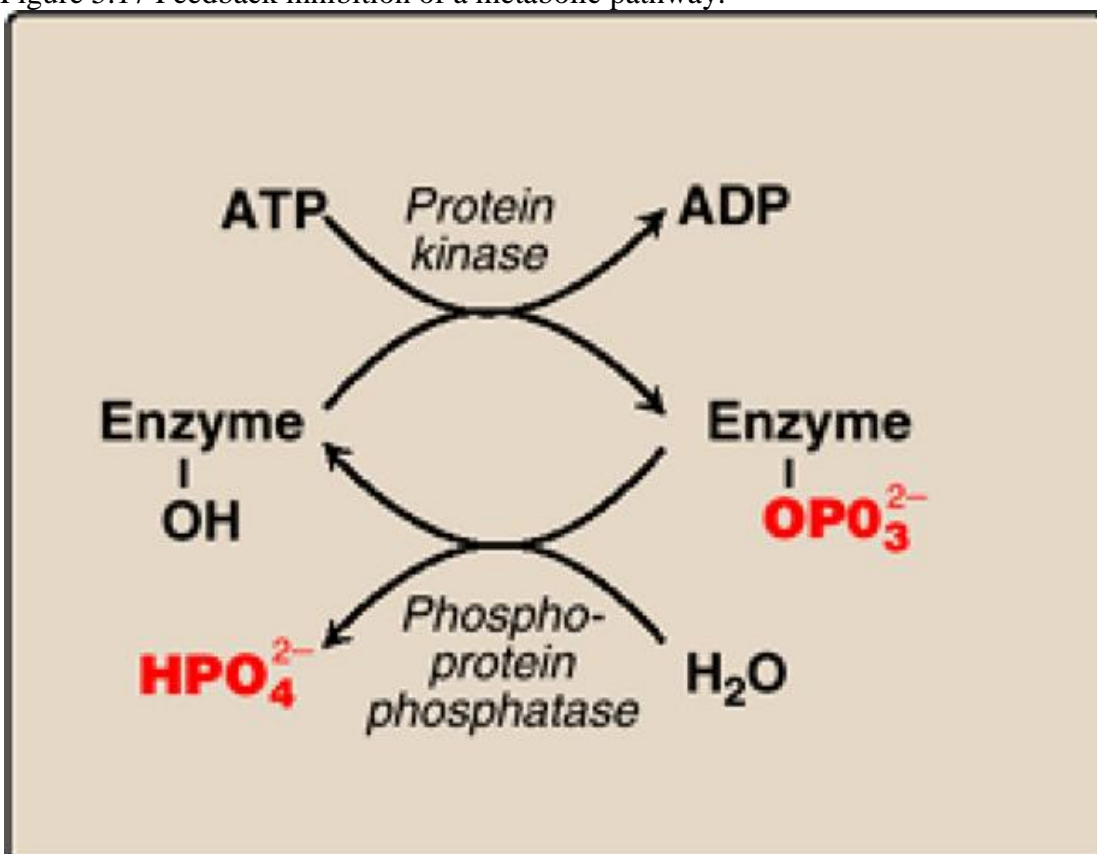


Figure 5.18 Covalent modification by the addition and removal of phosphate groups.

REGULATOR EVENT	TYPICAL EFFECTOR	RESULTS	TIME REQUIRED FOR CHANGE
Substrate inhibition	Substrate	Change in velocity (v_o)	Immediate
Product inhibition	Product	Change in V_m and/or K_m	Immediate
Allosteric control	End product	Change in V_m and/or K_m	Immediate
Covalent modification	Another enzyme	Change in V_m and/or K_m	Immediate to minutes
Synthesis or degradation of enzyme	Hormone or metabolite	Change in the amount of enzyme	Hours to days

Figure 5.19 Mechanisms for regulating enzyme activity.

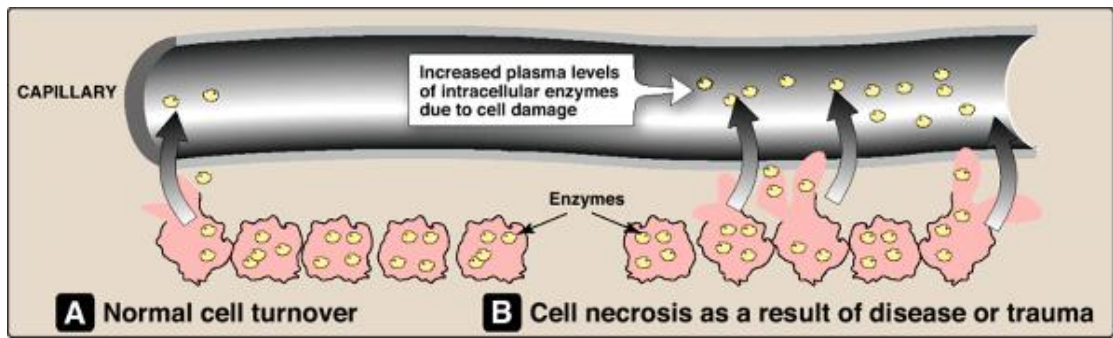


Figure 5.20 Release of enzymes from normal and diseased or traumatized cells.

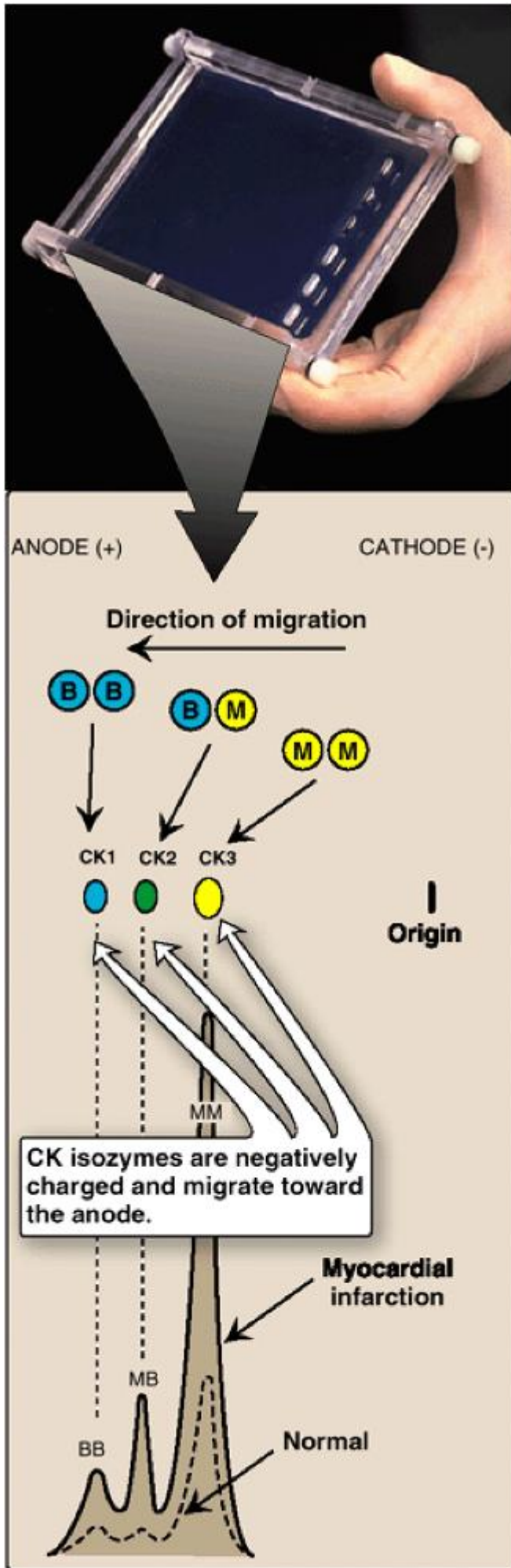


Figure 5.21 Subunit structure and electrophoretic mobility and enzyme activity of creatine kinase (CK) isoenzymes.

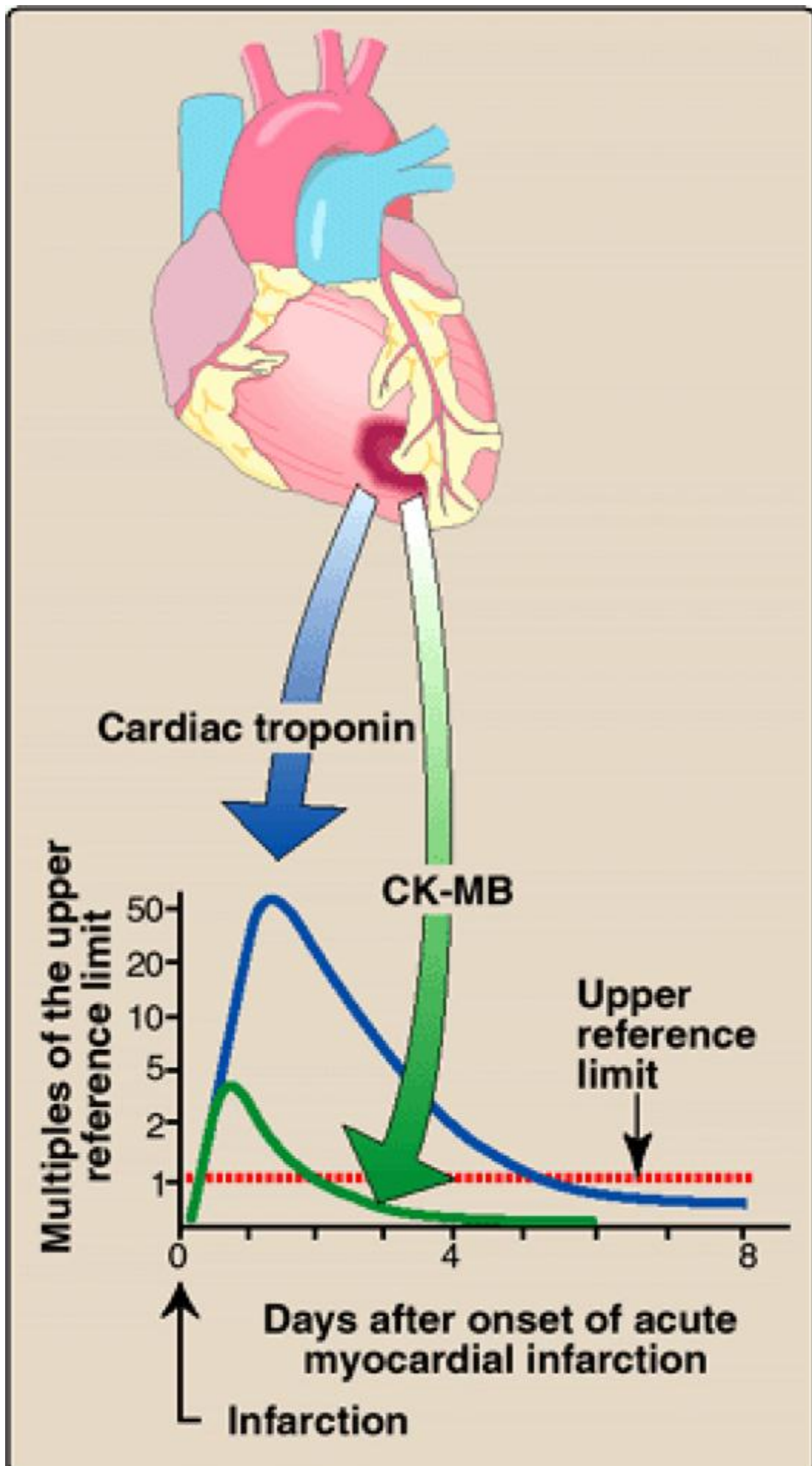


Figure 5.22 Appearance of creatine kinase (CK) and cardiac troponin in plasma after a myocardial infarction.

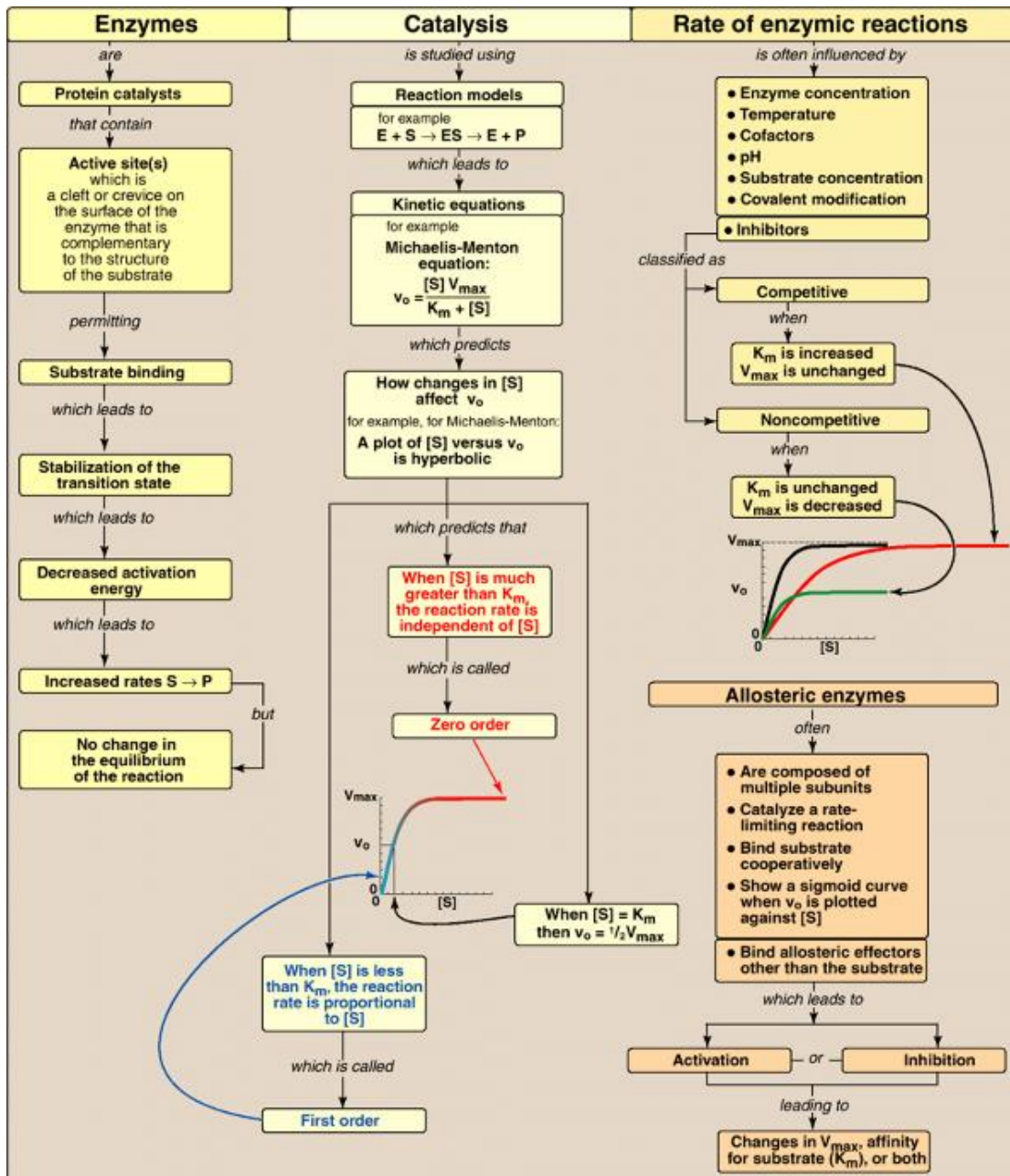


Figure 5.23 Key concept map for the enzymes. S = substrate, [S] = substrate concentration, P = product, E = enzyme, v_o = initial velocity, V_{max} = maximal velocity, K_m = Michaelis constant.

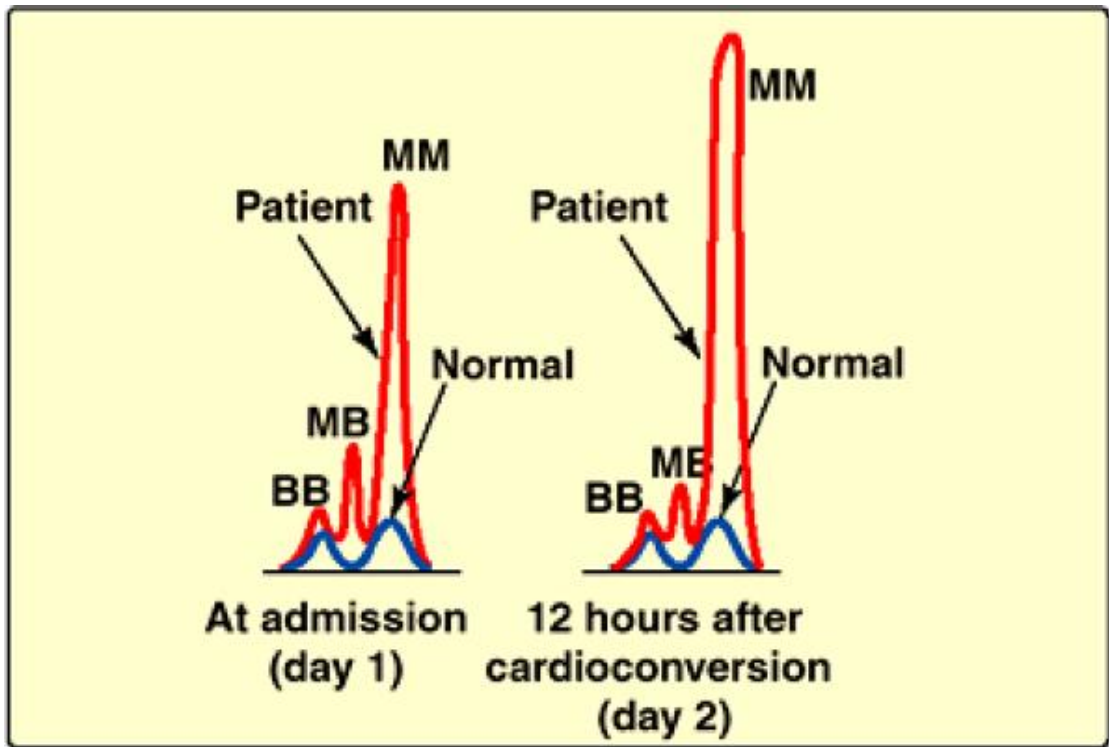


Figure 5.24 Serum creatine kinase levels.