ENZYMES

- Biocatalysts...
- Made up of Proteins (except ribozyme)
- Endoenzymes (intracellular) & Exoenzymes (extracellular).
- INDUCIVE ENZYMES: produced in the presence of their susbtrate; hence present only in traces (lactate dehydrogenase).
- CONSTITUTIVE ENZYMES: their concentraction is independent of the concentration of the inducer (cytochrome oxidase).
- ZYMASES/ACTIVE ENZYMES: secreted in a form which can act directly upon substrate without undergoing any prior modifications. (intracellular enzymes)
- ZYMOGENS/PROENZYMES: exist in precursor form; converted into active form by activating agents.

Based on complexity: simple & conjugated

SIMPLE ENZYMES

- Formed only of proteins.
- Hydrolysis yield only amino acids.
- Amylase, urease..

CONJUGATED ENZYMES

- Protein part (apoenzyme) + non-protein part (cofactor)
- Holoenzyme → Apoenzyme + cofactor
- Cofactors: prosthetic group & Coenzyme
- Prosthetic group: linked to apoenzyme by covalent or co-ordinate bond; usually metal ions.
- Coenzymes: not bound to apoenzyme firmly; usually organic compounds (FMN, FAD, NAD...); heat stable, low molecular weight compounds.

PROPERTIES OF ENZYMES

1. CATALYTIC EFFICIENCY

- ✓ Enzyme catalysed reactions are highly efficient.
- ✓ 10^3 to 10^8 faster than uncatalysed reactions.
- ✓ TURN OVER NUMBER : the number of molecules of substrate converted to product by 1 enzyme molecule per second (usualy 100 to 1000).

2. ACTIVE SITES

- ✓ Substrate binding occurs at a specialised portion called active site.
- ✓ Usually cleft or crevice containing special aminoacids.
- ✓ 3-D structure of active site is complementary to its substrate.
- ✓ Enzyme-substrate comlex formed soon dissociates into enzyme & product.

3. SPECIFICITY

- ✓ 2 types of specificity: substrate specificity & reaction specficity.

4. REGULATION OF ACTVITY

- ✓ Enzyme activity can be regulated by many substances naturally found in the cell.
- ✓ May be activated or inhibted by substrate utilisation or product formation.

5. SENSITIVITY TO HEAT

- ➢ Enzymes being proteins are highly sensitive to heat.
- ➢ Thermo-labile & undergo permanent denaturation above 60˚C.

6. REVERSIBILITY

- ➢ Most of the enzymatic reactions are reversible.

MECHANISM OF ENZYME ACTION

- Enzyme catalysed reactions occur within the confines of a pocket called ACTIVE SITE.
- Molecule that is bound to the active site and acted upon by the enzyme is called SUBSTRATE.
- Formation of ENZYME-SUBSTRATE COMPLEX is central to the action of enzymes.
- It is proposed that the enzymes increase the rate of the reaction by *lowering the energy of activation*.
- Substrates are raised from a low energy level (*ground state*) to a higher energy level (*transition state*) where spontaneous degradation occurs.
- All chemical reactions have an energy barrier separation the reactants from the products. This barrier is called *free energy of activation or activation energy.*
- Activation energy is a barrier to the chemical reactions.
- Speed of the chemical reactions is determined by the activation energy.

Why is activation energy required?

- Activation energy is crucial to life because it prevents the spontaneous disintegration of molecules.
- Higher activation energy is responsible for the preservation of the complex and highly ordered structures and metabolic pathways.

How do enzymes lower the activation energy?

- In enzyme-substrate complex, the substrate molecule is held at the active site of the enzyme by weak bonds like hydrogen bond, van der waals forces, ionic bonds, hydrophobic and hydrophilic interactions.
- Formation of each bond is accompanied by release of little amount of energy called binding energy.
- Binding energy is used to raise the reactant molecules to transition state.
- Binding energy and activation energy are mutually opposing, their summation results in new lower net activation energy.

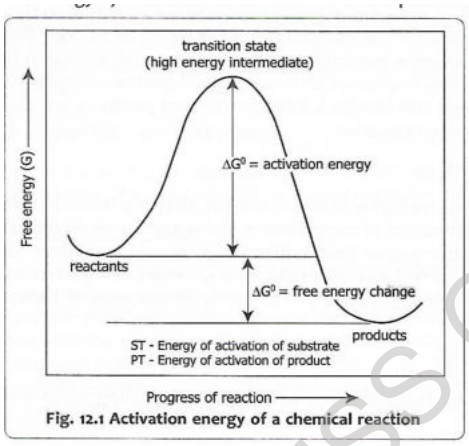

Fig. 12.1 Activation energy of a chemical reaction

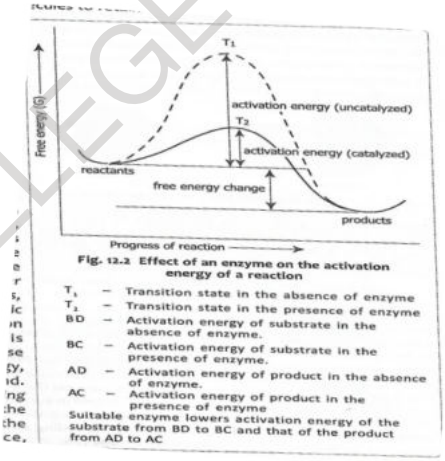

Fig. 12.2 Effect of an enzyme on the activation energy of a reaction

T_1 – Transition state in the absence of enzyme
T_2 – Transition state in the presence of enzyme
BD – Activation energy of substrate in the absence of enzyme.
BC – Activation energy of substrate in the presence of enzyme.
AD – Activation energy of product in the absence of enzyme.
AC – Activation energy of product in the presence of enzyme
Suitable enzyme lowers activation energy of the substrate from BD to BC and that of the product from AD to AC

ENZYME-SUBSTRATE COMPLEX THEORY

- Proposed by Leonor Michaelis & Maud Menten (1913).
- Enzyme (E) reversibly combines with substrate (S) to form a temporary and highly unstable enzyme-substrate complex (ES) which subsequently breakdown to products (P) and the enzyme (E) is regenerated. The free enzyme can again combine with fresh substrate molecules to continue reaction

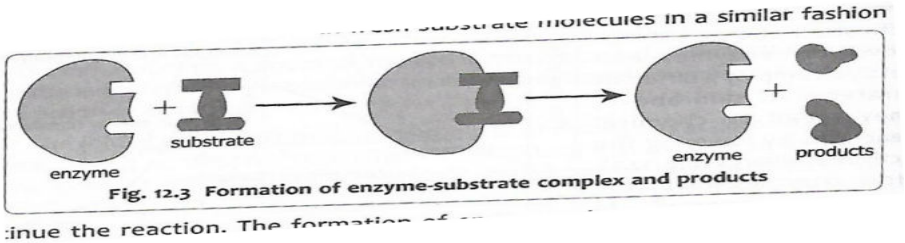

Fig. 12.3 Formation of enzyme-substrate complex and products

inue the reaction. The formation of

- ACTIVE SITE/CATALYTIC SITE/ SUBSTRATE BINDING SITE
  - Extremely specific points on the enzyme where substrate joins.
  - Area of the enzyme where catalysis occurs.
  - Occupies only small portion of an enzyme located in a small crevice or cleft.
  - Many amino acids are arranged in such a way to enable them to bind to the substrate.
  - Amino acids lying widely separated are brought to the active site by coiling or folding of polypeptide chain.
  - Active site possess a complex 3-D shape, correct molecular dimension, optimal alignment of counter ionic groups and hydrophobic regions.
  - Specificity of enzymes depends on the binding energy and functional groups of amino acids found in the active site.

MODELS FOR ENZYME SUBSTRATE COMPLEX THEORY

LOCK AND KEY MODEL

- Emil Fischer (1894)
- 3-dimensional structure of the active site of the enzyme is complementary to that of the substrate.
- Substrate fits into the active site of the enzyme like a key fits to its lock.
- Owing to the rigid nature attributed to the active site, the model is also known as <u>template model.</u>
- The model envisages active site as an already existing , rigid, pre-shaped template perfectly fitting in the size, shape and other features of the substrate.

Limitation: absolute rigidity of the active site which permits little variation in the substrate feature.

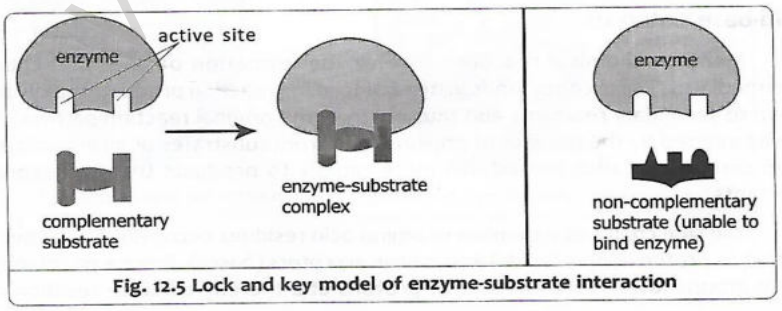

Fig. 12.5 Lock and key model of enzyme-substrate interaction

INDUCED FIT MODEL

- Koshland (1963).
- Modified version of lock & key model.
- Essential feature is the flexibility of the region of the active site.
- Active site does not possess a rigid and preformed structure to fit the substrate. Instead the enzyme undergoes conformational changes at thye active site concomitant with the substrate binding.
- Initial/Primary binding of the substrate at a specific point of the enzyme induces a conformational change in the enzyme in such a way that the catalytic groups in the active site become precisely oriented.
- This leads to the final/secondary binding of the substrate to the active site.

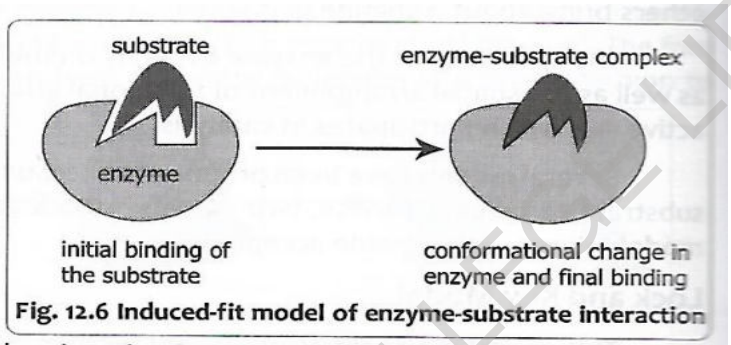

Fig. 12.6 Induced-fit model of enzyme-substrate interaction

:h as inactivation, competitive inhibition and allosteric

Once the enzyme-substrate complex is formed, further activities take place by a number of mechanisms like

1. acid-base catalysis
2. substrate strain,
3. covalent catalysis,
4. metal ion catalysis etc...

1. ACID-BASE CATALYSIS

- Many biochemical reaction form unstable charged intermediates which obstruct the original reaction pathway.
- Side chain of amino acids at active sites act as proton acceptor and proton donor. Precise positioning of these groups favour the transfer of these protons and thereby enhance the reaction rate.
- Amino acids like glutamic acid, arginine, lysine, serine etc can act as both acid and base.
- *Eg:action of ribonuclease*

- *Histidine residue at the 12th and 119th position act as acid and base respectively*
- *12th histidine donates a proton to RNA (substrate) to form charges intermediate, while 119th histidine accepts a proton and the product is released*

2. SUSTRATE STRAIN

- The substrate is believed to be held by the enzyme on the such a way as to cause distortion and weakening of chemical bonds holding its components.
- This increases the reactivity of the substrate.
- *Eg: Enzyme Lyzozyme*
  - *Catalyses the hydrolytic nreakdown of mucopolysaccharides found on bacterial cell wall by a combination of substrate strain and acid-base catalysis.*
  - *Binding of enzyme to the substrate creates a strian in the substrate (substrate strain)*
  - *Glutamic acid (35) and aspartic acid (52) hydrolyses the glycosidic linkage between the 4th and 5th monosaccharide units of the substrate*

3. COVALENT CATALYSIS

- Covalent bonds formed between the substrate and the enzyme activate the substrate to undergo further changes to yield the products.
- Functional groups of certain amino acids at the active site act as nucleophilic and electrophilic groups.
- Enzyme with serine residue at the active site (trypsine, chymotrypsine) employ this kind of catalysis.

4. METAL ION CATALYST

- Through ionic interactions, enzyme bound metal ions help to establish weak bonding with the substrate.
- They can also mediate oxidation-reduction reactions.
- Cytochrome oxidase , a major enzyme in ETC, undergoes reversible oxidation (Fe3+) reduction (Fe2+) canges during the process

* Most enzymes employ a combination of several catalytic activities.

ENZYME KINETICS

- The study of reaction rates and how they change in response to changes in different parameters is called ENZYME KINETICS.

Why do we study enzyme kinetics?

- It provides information on mechanism of enzyme action.
- It can give insight to the role of enzymes under conditions that exist in a cell.

VELOCITY/RATE OF REACTION: rate of change of substrate to the product per unit time.

$$A + B \leftrightarrow C = D$$

At equilibrium, rate of forward reaction = rate of backward reaction

- Rx proceeds to right if reactant concentration is increased.
- Rx proceeds to left if product concentration is increased.

VF α [A] [B] VR α [C] [D]

VF = KF [A] [B] VR = KR [C] [D]

KF & KR are proportionality constants or reaction rate constants.

At equilibrium, VF = VR

$$KF\,[A]\,[B] = KR\,[C]\,[D]$$

$$\frac{KF}{KR} = \frac{[C]\,[D]}{[A]\,[B]}$$

Equilibrium Constant $K_{eq} = \frac{[C]\,[D]}{[A]\,[B]}$

- Equilibrium constant (K_{eq})of a reaction is the ratio of the reaction rate constants of forward and backward reactions.
- At equilibrium, forward reaction is equal to backward reaction.
- Equilibrium is a dynamic state.
- At any point of time numerical value of the constant can be calculated from the concentrations of reactants and products.
- $K_{eq} > 1$, concentration of product is higher, forward reaction is favoured, spontaneous exothermic reaction.

- $K_{eq} < 1$, slow endothermic reaction.
- Concentration of enzyme does not affect the equilibrium constant, instead it increases the reaction rate and helps to reach the equilibrium quickly.

MICHAELIS-MENTEN EQUATION

- Michaelis – Menten Equation describes how reaction velocity varies with substrate concentration.

$$V_0 = \frac{V_{max}[S]}{(K_M + [S])}$$

V_0 : initial velocity

V_{max}: maximal velocity

K_m: Michaelis Constant

[S]: substrate concentration.

Assumptions made in deriving the equation

- Initial velocity is the velocity of the reaction when no product is formed yet.
- Concentration of the substrate [S] is much higher that the enzyme concentration [E].
- Concentration of enzyme substrate complex does not change.
- Michaelis Constant (Km) is a characteristic of an enzyme and its particular substrate.
- Km is defined as the substrate concentration at which the reaction velocity is equal to ½ Vmax.
- Lesser the Km value higher the affinity of the enzyme towards substrate,ie, lower concentration of substrate is needed to half saturate the enzyme.

FACTORS INFLUENCING ENZYME ACTIVITIES

1. TEMPERATURE

- ✓ Velocity of an enzyme reaction increases with temperature until a peak velocity is reached and then falls.
- ✓ Temperature at which the reaction rate is maximal is called OPTIMUM TEMPERATURE.
- ✓ Up to optimum temperature rate of enzyme action usually doubles with every 10^o rise in temp.
- ✓ The exact ratio by which the reaction velocity changes with every 10^oC rise in temp is called temperature coefficient (Q10).

- ✓ Optimum temp of most of the human enzymes is 37°C and that of some hot spring bacteria is 100°C.

. pH

- ✓ Every enzyme has a optimum pH at which it shows maximal activity.
- ✓ pH influences the enzyme activity by influencing the charge on the amino acid.
- ✓ Optimum pH of most of the enzymes falls within a range of 6 and 8.
- ✓ Extreme pH leads to denaturing of enzyles and permanent loss of catalytic activity
- ✓ Exceptions are pepsin (pH 2) and alkaline phosphatase (pH 10)

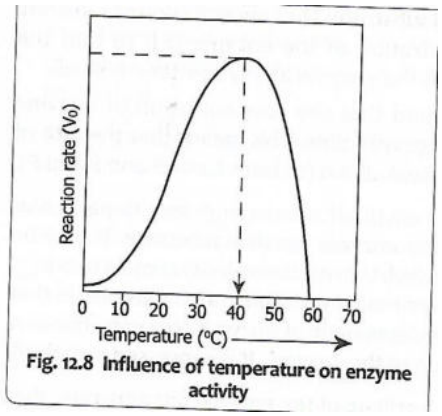

Fig. 12.8 Influence of temperature on enzyme activity

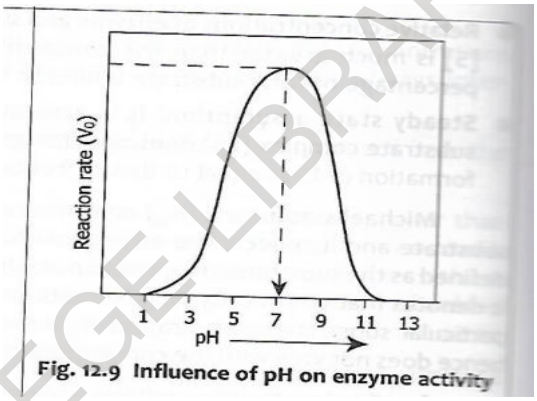

Fig. 12.9 Influence of pH on enzyme activity

3. ENZYME CONCENTRATION

Rate of reaction is directly proportional to the concentration of enzymes, provided the substrate is present in excess

4. SUBSTRATE CONCENTRATION

- ✓ Impact of substrate concentration on enzyme activity is not uniform.
- ✓ If the concentration of enzyme is fixed, in the initial phase when the conc of substrate is low reaction rate is also low.
- ✓ When the conc of substrate increases, reaction rate increases.
- ✓ This trend continues until all the enzyme molecules are saturated with substrate. Then the reaction attains maximum velocity.

ENZYME ACTIVATION

- Enzyme activation is the conversion of an inactive form of an enzyme to one possessing catalytic activity.
- Enzyme activators are molecules that bind to enzymes and increase their activity.
- Enzyme activation involves
  - Activation by ions (activators)

- Activation by co-factors (coenzymes)
- Conversion of enzyme precursor (proenzyme/zymogen) to active enzyme.
- Many enzymes are secreted in inactive (precursor) called PROENZYME or ZYMOGENS.

- The activation of proenzyme to active enzyme involves limited proteolysis and removal of few amino acids
- Protein splitting enzymes of pancrease – trypsinogen, chymotrypsinogen, procarboxypeptidase and proteolase - are synthesised in inactive form
- The conversion of these proenzymes to active enzymes is initiated by enterokinase produced by mucosal cells of duodenum
- AUTOCATALYSIS: once activated trypsin (active form) enzyme can cleave and activate further trypsinogen (zymogen) molecules. This process is called autocatalysis.
- Most blood clotting enzymes are also synthesised in inactive form which are activated only at the time of blood clotting.
- Many simple enzymes are activated by inorganic ions, especially metallic ions
  - salivary amylase is activated by chloride ions
  - pancreatic lipase is activated by bile salts
  - ATPase is activated by Na^+, K^+, Ca^{2+} and Mg^{2+}

INHIBITION

- The phenomenon of suppression of the activity of enzymes is called ENZYME INHIBITION.
- Chemical agents which bring about complete or partial inhibition of enzyme activity are called ENZYME INHIBITOR.

1. Competitive inhibition
2. Non-competitive inhibition
3. Suicide inhibition
4. Allosteric inhibition
5. Substrate inhibition

1. COMPETITIVE INHIBITION

- Inhibitor is a structural analogue of the substrate.
- Inhibitor competes with the substrate for binding at the active site to form EI complex.

- The inhibitor molecule of the EI complex never undergoes degradation but can be dissociated from it to release free enzyme
- Inhibitor inhibits the enzyme-substrate complexing by lowering the affinity of enzyme towards its substrate.
- Both EI and ES complex are formed, but the relative concentration of the two complexes depend on the relative affinity of enzyme towards S or I
- The affinity of the enzyme towards its substrate progressively decreases with the corresponding increase in the concentration of inhibitor (Km decreases)
- Rate of dissociation od ES remain unaffected (Vmax is unchanged)
- Salient features
    - Lack of absolute specificity of enzyme towards substrate.
    - Close structural similarity between substrate and inhibitor.
    - Greater affinity of anzyme towards inhibitor.
    - Inhibition can be reversed by increasing the conc of substrate.

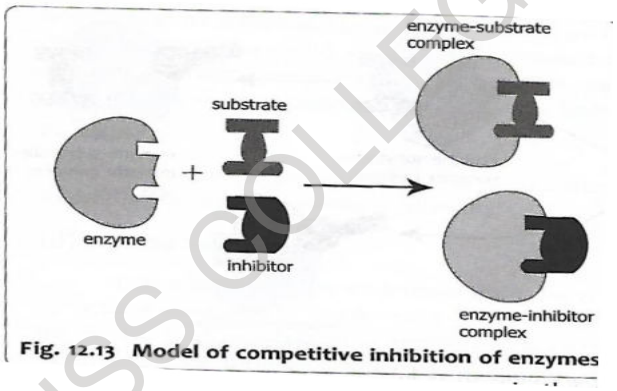

Fig. 12.13 Model of competitive inhibition of enzymes

Examples

- Inhibition of succinate dehydrogenase (succinate → fumarate) by malonate. Malonate competes for the active site of the enzyme and blocks the enzyme activity.
- Toxic Methanol is oxidised by Alcohol dehydrogenase (ADH) to formaldehyde (toxic). Antedote to methanol poisoning is ethanol which is natural substrate of ADH

2. NON-COMPETITIVE INHIBITION

- Inhibitor bears no structural similarity with the substrate.
- There is no competition between the substrate and the inhibitor.

- Inhibitor usually binds to a different site on the enzyme other than the substrate binding site.
- Inhibitor binds covalently either with free enzyme or with ES complex.
- Inhibitor never blocks ES complex formation, but prevents conversion of substrate to product by bringing about some changes in the 3D structure of the active site.
- Affinity of enzyme towards substrate remains unaffected while velocity of reaction reduces.

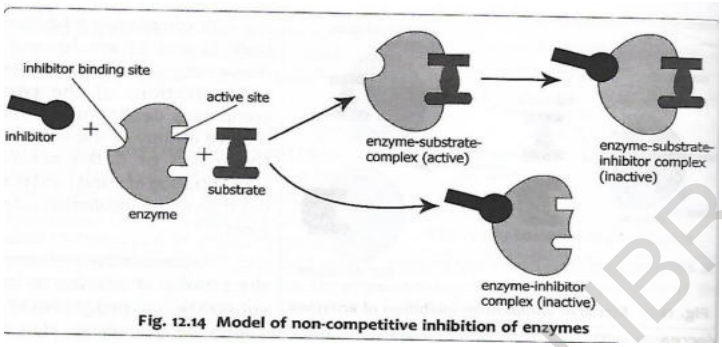

Fig. 12.14 Model of non-competitive inhibition of enzymes

- Vmax is reduced
- The affinity of enzyme towards substrate remains unaffected (Km remains unchanges)
- Drugs, poisons, pesticides, heavy metals etc act as non competetive inhibitors
- Cyanide inhibits cytochrome oxidase of ETC.

3. UNCOMPETITIVE INHIBITION

- Inhibitor shows no affinity for enzyme, but has high affinity for ES complex.
- The presence of uncompetitive inhibitor in the reaction medium enhances the affinity of the enzyme to the substrate (Km decreases)
- Inhibitor binds to ES complex and blocks conversion of substrate to product.
- Usually occurs in reactions with multiple substrate and products.
- The reaction cannot be reversed by increasing substrate concentration (Vmax decreases)

Eg: Inhibition of placental alkaline phosphatase by amino acid phenylalanine

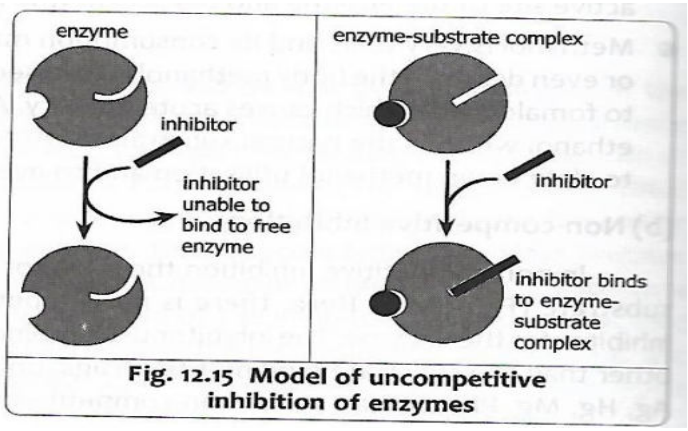

Fig. 12.15 Model of uncompetitive inhibition of enzymes

4. SUICIDE INHIBITION

- Irreversible enzyme inhibition.
- Structural analogue of substrate acts as pro-inhibitor.

1. pro-inhibitor binds to the 1st enzyme of a multienzyme reaction pathway.
2. Few steps are catalysed.
3. One of the intermediate compound formed irreversibly binds to the initial enzyme and stops the pathway.

- Also called mechanism based inactivation

Eg: Inhibition of xanthine oxidase by allopurinol

Xanthine is the natral sunstrate of xanthine oxidase (XO). The proinhibitor allopurinol binds with XO and gets converted into more effective inhibitor alloxanthine. This compound irreversibly inactivates the enzyme XO

5. ALLOSTERIC OR END PRODUCT OR FEEDBACK INHIBITION

- Inhibitor is usually the end product of the reaction pathway.
- Inhibitor (end product) binds to the an allosteric site of the key enzyme which is different from catalytic active site.
- Binding of inhibitor to the allosteric site brings conformational changes in the active site which makes substrate binding difficult.
- Substrate affinity of enzymes decreases (Km increases)
- Vmax remains unaffected.
- Inhibition can be reversed by partially increasing the substrate concentration as well as decrease in the product concentration.
- Allosteric inhibition does not follow Michaelis-Menten kinetics, instead it gives sigmoid kinetics.

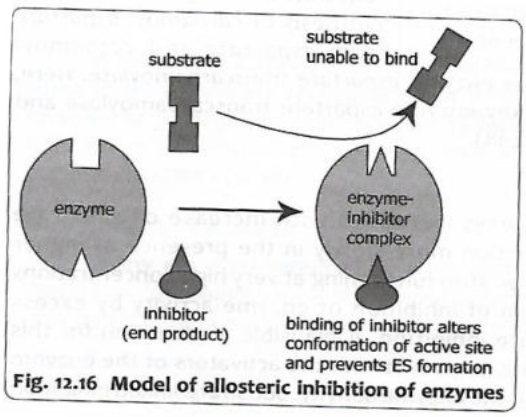

Fig. 12.16 Model of allosteric inhibition of enzymes

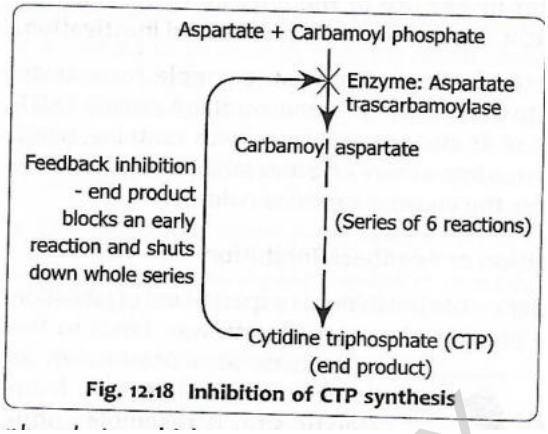

Fig. 12.18 Inhibition of CTP synthesis

- Feedback inhibition is the commonest way for the regulation of biosynthetic pathways

Eg: In the synthesis of cytidine triphosphate (CTP), the end product (CTP) inhibits the key enzyme aspartate transcarbamoylase and blocks the reaction pathway

6. SUBSTRATE LINKED INHIBITION

- Some enzymes work more slowly at higher concentrations or may even stop working. This phenomenon of inhibition of enzyme activity by excess substrate is referred to as substrate inhibition
- Inhibition disappears when substrate concentration comes to normal

Eg: Hexokinase, lipase, enolase, LDH etc...

ISOZYMES

- Multiple forms of the same enzyme are called isozymes or isoenzymes
- They differ in their electrophoretic mobility, molecular structure, affinity towards different substrates and response to different inhibitors.
- The difference between isozymes are genetically determined
- HOMOLOGOUS: isozymes with similar structural and catalytic properties. They are produced by multiple allels of same gene.
- ANALOGOUS: isozymes with similar catalytic properties and different molecular structure. They are produced by different genes located on different chromosomes.
- Examples of isozymes:
    - Lactate dehydrogenase (LDH) exists as 5 different isozymes (LD1 to LD5). LD1 is abundant in heart muscles while LD5 is predominant in skeletal muscles
    - Alkaline phosphatase exsits in 2 forms
    - Creatin phosphokinase exist in 3 forms

COFACTORS

- A cofactor is a non-protein chemical compound that is required for the activity of some enzymes and are regenerated for further reactions.
- There are 2 types of cofactors – inorganic and organic

INORGANIC COFACTORS

They require metal ions for maintenance of protein conformation and catalysis. Metal ions participate in enzyme reactions in 3 ways

1. Metallo Enzymes: metals are integral part of the enzyme and bound by coordinate bond.

Eg: Iron is required for cytochrome oxidase, catalase, succinate dehydrogenase etc

Copper is required for cytochrome oxidase, ceruplasmin etc

Zinc is required in carbonic anhydrase, carboxypeptidase etc

2. Metal-dependent enzymes: metal is loosely associated with enzyme molecule

Eg: Mg^{2+} : Mg-ATP complex is essential for enzymes using ATP (hexokinase, galctokinase, pyruvate kinase)

Ca^{2+}: required for the activity of caplin, a Ca-dependent protease

3. Metal activated enzymes: enzymes are activated in the presence of metals.

Eg: Chloride activates amylase and angiotensin converting enzyme

Calcium activates trypsin

ORGANIC COFACTORS

The organic cofactors are classified depending on how tightly they bind to an enzyme.

1. Prostheic group:
    - bind very tightly to the enzyme
    - covalently attached to the enzyme;
    - heme in cytochrome oxidase enzyme.
2. Co-enzymes:
    - loosely (non-covalent) attached to the enzyme.
    - They undergo change during reaction along with the substrate (co-substrate)

COENZYMES

- Complex molecules associated with enzymes and are essential for the catalytic activity

- These are heat stable low molecular weight organic molecules generally derived frpm B complex vitamins
- Loosely bound to apoenzyme and can be separated easily by dialysis

Apoenzyme + Co-enzyme Holoenzyme

(Protein) (Non-protein) (Active Enzyme)

How do co-enzymes work?

- Substrate binding site may be formed by apoenzyme and co-enzyme jointly
- Few co-enzymes serve as intermediate carriers helping to transfer electrons, atoms and special chemical groups.
- Also act as donors or acceptors of specific atoms or chemical groups.
- Some co-enzymes counterbalance the chemical changes taking place in the substrate

MAJOR COENZYMES

Coenzymes may be divided into 2 classes on the basis of groups they transfer

1. Coenzymes involved in oxidation-reduction reaction. They serve as acceptors or donors of hydrogen or electron

   NAD, NADP, FMN, FAD, Co-enzyme Q etc..

2. Coenzymes which transfer groups other than hydrogen and electrons

   ATP, ADP etc..

1. NAD and NADP

- Nicotinamide Adenine Dinucleotide (NAD)
- Nicotinamide Adenine Dinucleotide Phosphate (NADP).
- Formed from B complex vitamin NICOTINIC ACID
- NAD is coenzyme1 and NADP is coenzyme2 and perform similar functions.
- Serve as carrier of hydrogen atom and are involved in a variety of oxidation-reduction reactions.
- Oxidised state (NAD+ and NADP+).
- Reduced state (NADH and NADPH).
- Oxidation : $SH + NAD^+ \rightarrow P + NADH$
- Reduction : $S + NADH \rightarrow PH + NAD^+$

2. FLAVIN NUCLEOTIDE COENZYMES

- Derived from vitamin B2 or RIBOFLAVIN.
- FMN (Flavin mononucleotide) & FAD (Flavin Adenine Dinucleotide)
- FMN has phosphate group attached to the riboflavin
- FAD is formed by linkage of AMP to FMN.
- Both FMN and FAD participate in oxidation-reduction reaction by accepting and donating hydrogen or electrons
- Oxidised state : FMN+ and FAD+
- Reduced state : FMNH2 and FADH2

$$FMN + + 2H \rightarrow FMNH2$$

$$FAD+ + 2H \rightarrow FADH2$$

3. COENZYME Q

- Ubiquinone is a lipid material found abundantly in the mitochondria.
- Plays important role in the oxidation-reduction reactions of the ETC.
- It can exist in oxidised and reduced state

4. ADENOSINE NUCLEOTIDE

- 3 coenzymes: AMP, ADP, ATP.
- They are synthesised in all mammalian cells by phosphorylation of adenosine
- ATP is known as *energy currency of the cell* as the terminal phosphate bond stores enormous energy obtained from oxidation of nutrients
- 5. PYRIDOXAL PHOSPHATE
- Derived from vtamin B6 (pyridoxine). It serves as coenzyme for transamination and decarboxylation reactions
- 6. THYMINE PYRO-PHOSPHATE
- Derived from Vitamin B1 (thiamine).
- It is also called *Cocarboxylase* and is involved in the removal of CO2 from α ketoacids (decarboxylation)
- 7. COENYME A: Complex molecule containing free sulflhyryl groups. It is involved in acetylation reactions. CoA accepts acetyle group from one metabolite and transfers them to another in the presence of specific enzymes.