Model Answer
M.Sc IIIrd Semester
Paper Name : Developmental Biology and Immunology
Paper Code: LZT-301

Q. 1. i. a ii. d iii. a iv. b v. b vi. b

vii. a viii. d ix. a x. a

Q. 2. *Ans:* FERTILIZATION is the process whereby the sperm and the egg—collectively called the gametes fuse together to begin the creation of a new individual whose genome is derived from both parents. Although the details of fertilization vary from species to species, conception generally consists of four major events:

1. Contact and recognition between sperm and egg. In most cases, this ensures that the sperm and egg are of the same species.
2. Regulation of sperm entry into the egg. Only one sperm nucleus can ultimately unite with the egg nucleus. This is usually accomplished by allowing only one sperm to enter the egg and actively inhibiting any others from entering.
3. Fusion of the genetic material of sperm and egg.
4. Activation of egg metabolism to start development.

1. Encounter of spermatozoa and ova:

A major problem in sexual reproduction is how to bring together the spermatozoa and ova. It is important that the ripe egge and sperms be brought together in the same general locality and that individual sperm may reach the surface of the ova. Since sea urchin is an externally fertilizing animals, the process of fertilization occurs in the aquation oceanic water outside the body system of male and female parent. Since astronomical numbers of sperms as well as egges are spawned during any one spawning period, the developmental hazards are greatest in of sea urchin. In addition to the external temperature, substances liberated from the oviduct at the time of the shedding of the eggs stimulate other ripe females and also the ripe males in the vicinity to release spermatozoa. Consequently clouds of eggs and sperms are formed in the sea water at the same time and mass fertilization occurs. However, spermatozoa have to move long distance and their encounter depend on chance. The importance of life span of the gamete plays a major role in external fertilizing animals.

Sperms generally reach near the eggs and maintain their species specificity by following methods:

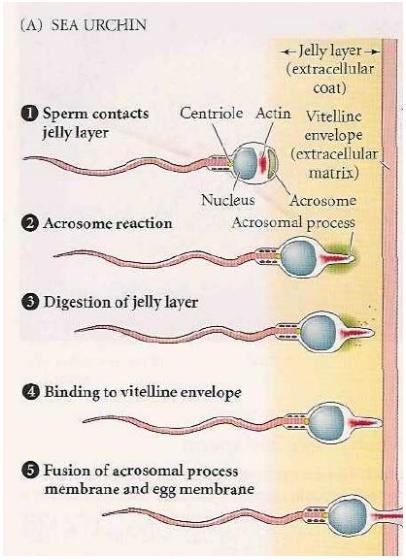
1. Chemotaxis and 2. Fertilizing and anti-fertilizin interaction

2. Acrosome Reaction and Contact of sperm and Ovum:

The first reacton of the sperm when it comes in contact with the constituents of the egg surface involve the acrosome. Onset of the acrosome reaction takes place whe there are optimal physiological conditions such as optimum pH, Ca^{2+}, Mg^{2+} ion concentration and temperature. The presence of calcium is an essential condition for acrosome reaction.

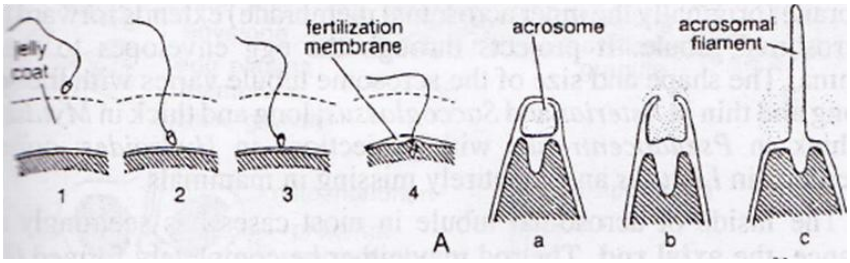
i. Trigger mechanisms for breakdown of acrosome:

The apical part of the sperm plasma membrane and underlying outer acrosomal membrane undergo dehiscence. The severed edge of the two membranes fuse to form an opening through which the contents of the acrosomal vesicles are released. Inner acrosomal membrane grows into one or many acrosomal tubules which in some cases may be almost as long as the entire spermatozoon. The membrane surrounding the acrosomal tubule is always derived from an inner membrane of the acrosomal vesicle. It is this membrane that come in direct contact with egg jelly, vitteline membrane and plasma membrane.



ii. Release of acrosomal contents:

the trigger mechanism releases lytic enzymes located in the acrosome. The lysins help the sperm penetrate the egg envelope by liquefying them locally, without affecting the egge plasma membrane. In case of sea urchin, the acrosomal reaction , the acrosomal reaction results in the liberation of a species specific egg binding protein, Bindin, and a protease or lysine called acrosomin. The bindin plays a role in causing adhesion of the sperm to egg of the same species. Acrosomin responsible for the initial digestionof the vitelline membrane that covers the unfertilized egg.



iii. Formation of Acrosomal Tubule:

The apical part of the sperm plasma membrane extends forward to form an acrosomal tubule. It projects through the egg envelopes to reach the oolemma. The shape and size of the acrosomal tubule varies with the species. Inside of the acrosomal tubule in most cases has a rigid substances, the axial rod. Discharge of the acrosomal fluid activates the actin units to form microfilaments, due to which the contractile coil extends rapidly to make contact with the sperm surface.

3. Fusion of the egg and sperm cell membranes

Once the sperm has traveled to the egg and undergone the acrosome reaction, the fusion of the sperm cell membrane with the cell membrane of the egg can begin. Sperm-egg fusion appears to cause the polymerization of actin in the egg to form a fertilization cone. Homology between the egg and the sperm is again demonstrated, since the sperm's acrosomal process also appears to be formed by the polymerization of actin. The actin from the

gametes forms a connection that widens the cytoplasmic bridge between the egg and the sperm. The sperm nucleus and tail pass through this bridge.

In the sea urchin, all regions of the egg cell membrane are capable of fusing with sperm. In several other species, certain regions of the membrane are specialized for sperm recognition and fusion. Fusion is an active process, often mediated by specific "fusogenic" proteins. Indeed, sea urchin sperm bindin plays a second role as a fusogenic protein. In addition to recognizing the egg, bindin contains a long stretch of hydrophobic amino acids near its amino terminus, and this region is able to fuse phospholipid vesicles in vitro. Under the proper ionic conditions (those in the mature unfertilized egg), bindin can cause the sperm and egg membranes to fuse.

4. Activation of Ovum and prevention of polyspermy:

The phenomenon of egg activation involves changes in ionic permeability of egg plasma membrane through activation of various pumps such proton pump, sodium pump, transient increase in intracellular calcium and pH. The process of fertilization ensure the fusion of single spermatozoa with one ova and prevents fusion of more than one sperm through cortical reaction that lead to formation of fertilization membrane and changes in membrane potential.

Significance of fertilization:

1. trigger the developmental programme in a rapid burst of metabolic activity.

2. play role in sexual recombination.

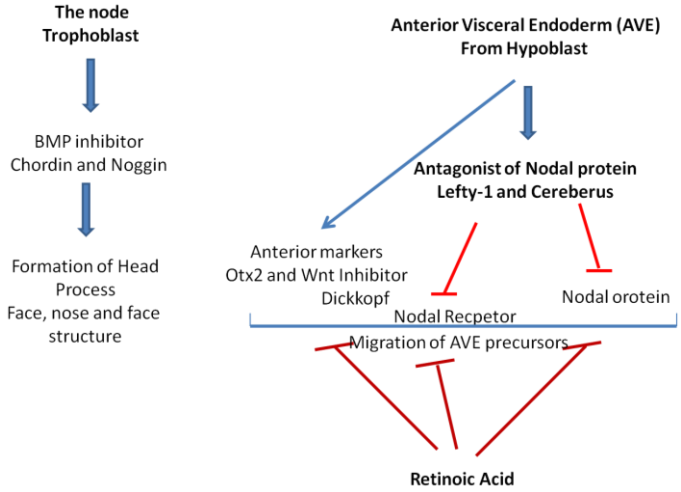
3. sustain the survivability of the egg

Q. 3. ***Ans: The anterior-posterior axis***

The mammalian embryo appears to have two signaling centers: one in the node (equivalent to Hensen's node and *the* trunk portion of the amphibian organizer) and one in the anterior visceral endoderm (AVE; equivalent to the chick hypoblast and similar to the head portion of the amphibian organizer). The node appears to be responsible for the creation of all of the body and the two signaling centers work together to form the anterior region of the embryo. The notochord forms by the dorsal infolding of the small, ciliated cells of the node. The AVE originates from the visceral endoderm hypoblast) that migrates forward. As this region migrates, it secretes two antagonists of the Nodal protein, Lefty-1 and Cerberus. While the Nodal proteins in the epiblast activate the expression of posterior genes that are required for mesoderm formation, the AVE creates an anterior region where Nodal cannot act. The AVE also begins expressing the anterior markers Otx2 and Wnt-inhibitor Dickkopf.

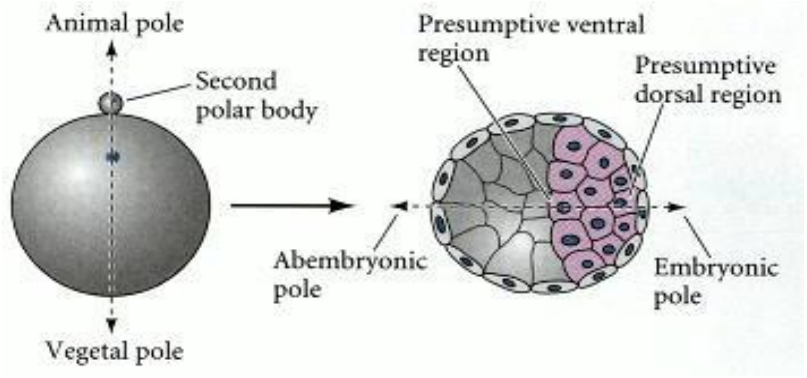
Studies of mutant mice indicate that the AVE promotes anterior specification by suppressing the formation of the primitive streak, a posterior structure, by Nodal and Wnt proteins, as in the chick. However, the AVE alone cannot induce neural tissue, as the node can. Formation of the node is dependent on the trophoblast. As in the chick embryo, the placement of the node and the primitive streak appears to be due to the blocking of Nodal signaling by the Cerberus and Lefty-1 from the AVE. Once formed, the node will secrete Chordin; the head process and notochord will later add Noggin. These two BMP antagonists are not expressed in the AVE. Dickkopf is expressed in both the AVE and in the node, but only the Dickkopf from the node is critical for head development. While knockouts of either the *chordin* or the *noggin* gene do not affect development, mice missing both genes lack a forebrain, nose, and other facial structures. It is probable that the AVE functions in the epiblast to restrict Nodal

activity, thereby cooperating with the node-produced mesendoderm to promote the head-forming genes to be expressed in the anterior portion of the epiblast.



The dorsal-ventral axis

Very little is known about the mechanisms of dorsal-ventral axis formation in mammals. After the fifth cell division in the mouse embryo, the blastocyst cavity begins to form, and the inner cell mass resides on one side of this cavity. This axis is probably created by the ellipsoidal shape of the zona pellucida. In mice and humans, the hypoblast forms on the side of the inner cell mass that exposed to the blastocyst fluid, while the dorsal axis forms from those ICM cells that are in contact with the trophoblast and amnionic cavity. Thus, the dorsal-ventral axis of the embryo is defined, in part, by the embryonic-abembryonic axis of the blastocyst. The embryonic region contains the ICM, while the abembryonic region is that part of the blastocyst opposite the ICM. The first dorsal-ventral •clarity is seen at the blastocyst stage, and as development (proceeds, the primitive streak maintains this polarity by causing migration ventrally from the dorsal surface of the embryo.

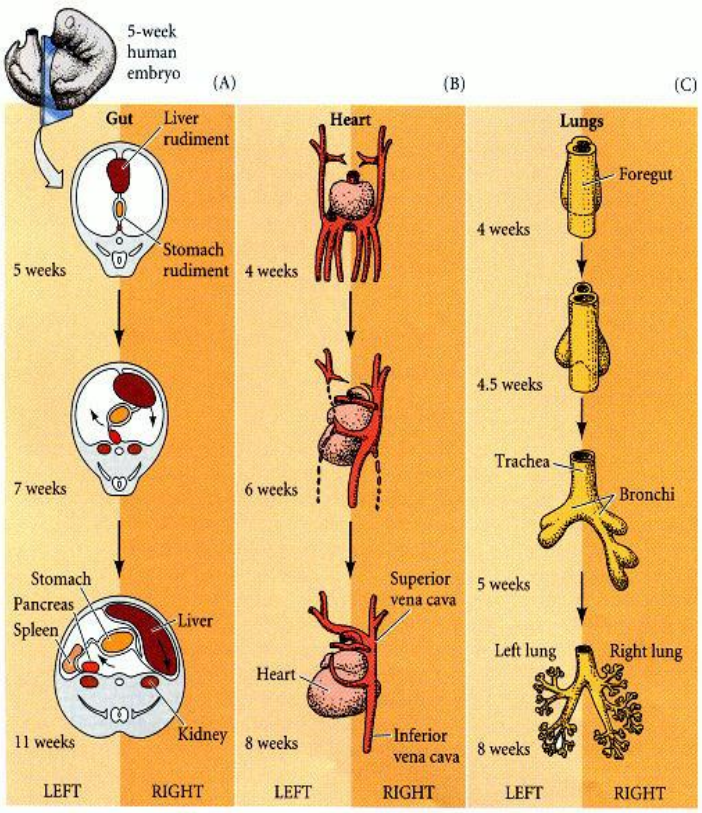


The left-right axis

The mammalian body is not symmetrical. Although the human heart begins its formation at the midline of the pnbryo, it moves to the left side of the chest cavity and loops to the right. The spleen is found solely on the left side • the abdomen, the major lobe of the liver forms on the fight side of the abdomen, the large intestine loops right *ID* left as it traverses the abdominal cavity, and the right img has one more lobe than the left lung. Mutations

in mice have shown that there are two levels • regulation of the left-right axis: a global level and an ^rgan-specific level. Mutation of the gene *situs inversus visitrum (iv)* randomizes the left-right axis for each asymmetrical organ independently. This means that the heart may loop to the left in one homozygous animal but to the right in another. Moreover, the direction of heart looping is not coordinated with the placement of the spleen or stomach. This lack of coordination can cause serious problems, even death. A second gene, *inversion of embryonic turning (inv),* causes a more global phenotype. Mice homozygous for an insertion mutation at this locus had all their asymmetrical organs on the wrong side of the body.* Since all the organs were reversed, this asymmetry did not have dire consequences for the mice.

Several additional asymmetrically expressed genes have recently been discovered, and their influence on one another has enabled scientists to arrange them into a possible pathway. The end of this pathway—the activation of Nodal proteins and the Pitx2 transcription factor on the left side of the lateral plate mesoderm—appears to be the same in mammals as in other vertebrate embryos, although the path leading to this point differs among the species.

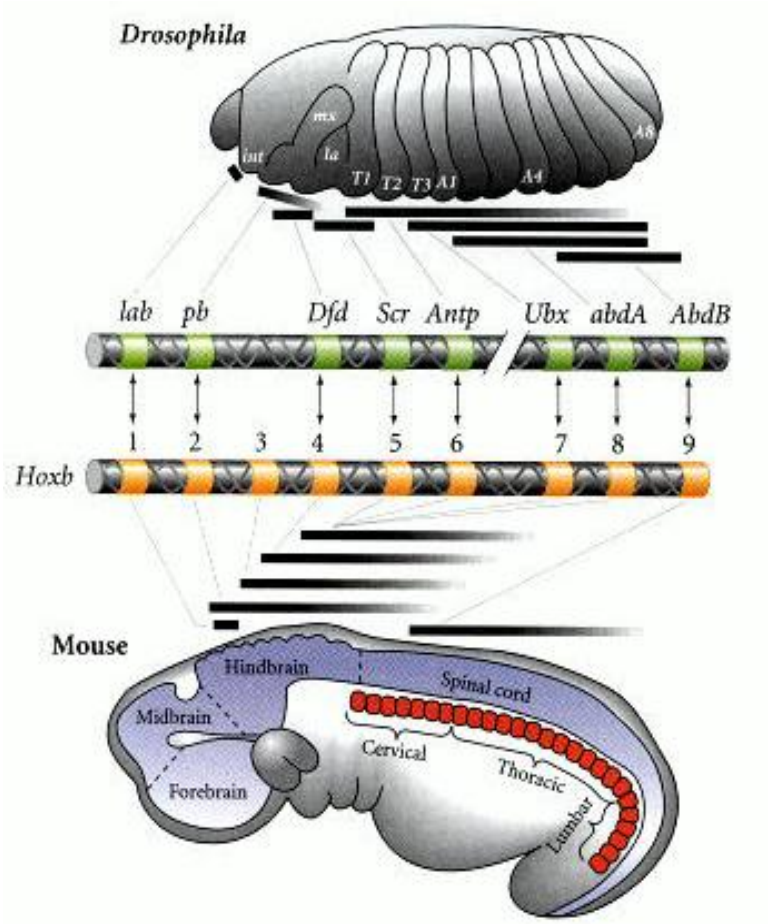


Hox gene in mammalian pattern formation:

All of the known mammalian genomes contain four copies of the Hox complex per haploid set, located on four different chromosomes *(Hoxa* through *Hoxd* in the mouse, *HOXA* through *HOXD* in humans. The order of these genes on their respective chromosomes is remarkably similar between insects and humans, as is the expression pattern of these genes. Those mammalian genes homologous to the *Drosophila labial, proboscipedia,*

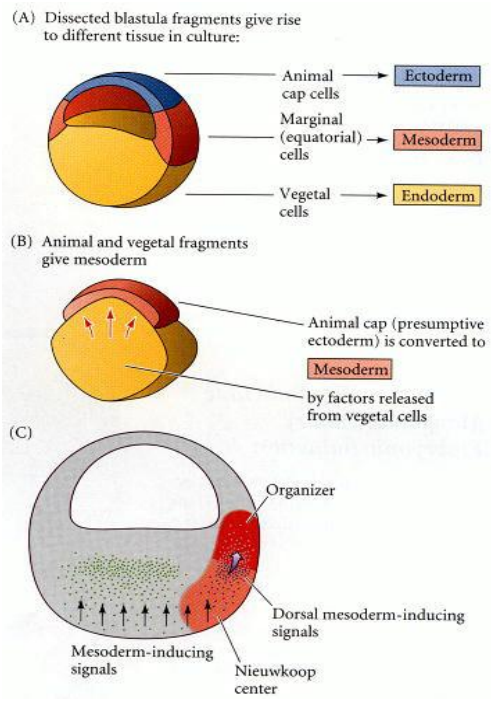
and *deformed* genes are expressed anteriorly and early, while those genes homologous to the *Drosophila AbdB* gene are expressed posteriorly and later. As in *Drosophila*, a separate set of genes in mice encodes the transcription factors that regulate head formation. In *Drosophila,* these are the *orthodenticle* and *empty spiracles* genes. In mice, the midbrain and forebrain are made through the expression of genes homologous to these—*Otx2* and *Emx.*

The mammalian Hox/HOX genes are numbered from 1 to 13, starting from that end of each complex that is expressed most anteriorly. The equivalent genes in each mouse complex (such as *Hoxal, Hoxbl,* and *Hoxdl)* are called paralogues. It is thought that the four mammalian Hox complexes were formed by chromosome duplications. Because the correspondence between the *Drosophila* Hom-C genes and mouse Hox genes is not one-to-one, it is likely that independent gene duplications and deletions have occurred since these two animal groups diverged. Indeed, the most posterior mouse Hox gene (equivalent to *Drosophila AbdB)* underwent its own set of duplications in some mammalian chromosomes.



Q. 4. i) Nieuwkoop Centre

The polarity of the induction (whether the animal cells formed dorsal mesoderm or ventral mesoderm) depends on the dorsal-ventral polarity of the endodermal (vegetal) fragment. The ventral and lateral vegetal cells (those closer to the side of sperm entry) induced ventral (mesenchyme, blood) and intermediate (muscle, kidney) mesoderm. The dorsalmost vegetal cells specified dorsal mesoderm components (somites, notochord), including those having the properties of the organizer. **The dorsalmost vegetal cells of the blastula, which are capable of inducing the organizer, have been called the Nieuwkoop center.**



The major factor that form Nieuwkoop centre : β- CATENIN which is a multifunctional protein that act as an anchor for the cell membrane cadherin and as a nuclear transcription factor induced by Wnt pathway.

Function of Nieuwkoop centre:

Nieuwkoop centre induces the dorsal blastoporal lip cells to develop specialized properties and to get differentiate into primary organizer which develop the entire body axis formation during the course of development.

ii) Segmentation Genes:

The transition from specification to determination in *Drosophila* is mediated by segmentation genes that divide the early embryo into a repeating series of segmental primordia along the anterior-posterior axis. Segmentation genes were originally defined by zygotic mutations that disrupted the body plan, and these genes were divided into three groups based on their mutant phenotypes:

1. Gap mutants lacked large regions of the body.
2. Pair-rule mutants lacked portions of every other segment.
3. Segment polarity mutants showed defects (deletions, duplications, polarity reversals) in every segment.

Gap genes

The gap genes are activated or repressed by the maternal effect genes, and are expressed in one or two broad domains along the anterior-posterior axis. These expression patterns correlate quite well with the regions of the embryo that are missing in gap mutations. The expression patterns of the gap genes are highly dynamic. Deletions caused by mutations in three gap genes— ***hunchback, Kriippel,*** **and** ***knirps***—span the entire segmented region of the *Drosophila* embryo.

Pair-rule genes

Pair rule gene indicate of segmentation in the fly embryo comes when the pair-rule genes are expressed during cell division cycle 13, as the cells begin to form at the peripheral part of the embryo. The transcription patterns of these genes divide the embryo into regions that are precursors of the developmental body plan. The primary pair-rule genes include *hairy, even-skipped,* and *runt,* each of which is expressed in seven stripes.

Segment polarity genes

Interactions between molecules within the syncytial embryo take part in the process of development. But once cells form, interactions take place between the cells. These interactions are mediated by the segment polarity genes, and they accomplish two important tasks. First, they reinforce the parasegmental periodicity established by the earlier transcription factors. Second, through this cell-to-cell signaling, cell fates are established within each parasegment.

iii) **The *pair-rule genes***

*S*egmentation in the fly embryo comes when the pair-rule genes are expressed during cell division cycle 13 as the cells begin to form at the peripheral part of the embryo. The transcription patterns of these genes divide the embryo into regions that are precursors of the developmental body plan. one vertical band of nuclei expresses a pair-rule gene, the next band of nuclei does not express it, and then the next band expresses it again. The result is a "zebra stripe" pattern along the anterior-posterior axis, dividing the embryo into 15 subunits. Eight genes are currently known to be capable of dividing the early embryo in this fashion, and they overlap one another so as to give each cell in the parasegment a specific set of transcription factors.

The primary pair-rule genes include *hairy, even-skipped,* and *runt,* each of which is expressed in seven stripes- All three build their striped patterns using distinct enhancers and regulatory mechanisms for each stripe. These enhancers are often modular: control over expression in each stripe is located in a discrete region of the DNA, and these DNA regions often contain binding sites recognized by gap proteins. Thus it is thought that the different concentrations of gap proteins determine whether or not a pair-rule gene is transcribed. **The primary pair-rule genes** include *hairy, even-skipped,* and *runt,* each of which is expressed in seven stripes.

Once initiated by the gap gene proteins, the transcription pattern of the primary pair-rule genes becomes stabilized by interactions among their products. The primary pair-rule genes also form the context that allows or inhibits expression of the later-acting secondary pair-rule genes. One such gene *isfushi tarazu (ftz)*. Early in division cycle 14, *ftz* mRNA and its protein are seen throughout the segmented portion of the embryo. However, as the proteins from the primary pair-rule genes begin to interact with the *ftz* enhancer, the *ftz* gene is repressed in certain bands of nuclei to create interstripe regions. Meanwhile, the Ftz protein interacts with its own promoter to transcribe more ftz. The eight known pair-rule genes are all expressed in striped patterns, but the patterns are not coincident with each other. Rather, each row of nuclei within a parasegment has its own array of pair-rule products that distinguishes it from any other row. These products activate the level of segmentation genes, the segment polarity genes.

Q. 5. Ans: Metamorphosis is the process of transformation from larva stage to an adult stage. Organisms undergoing indirect development undergo metamorphosis. Insect metamorphosis primarily involves the destruction

of larval tissues and their replacement by an entirely different population of cells. Insects grow by molting—shedding their cuticle—and forming a new cuticle as their size increases.

There are three major patterns of insect development.
i. Ametabolous Metamorphosis

ii. Hemimetabolous Metamorphosis

iii. Holometabolous Metamorphosis

Hormonal control of insect metamorphosis

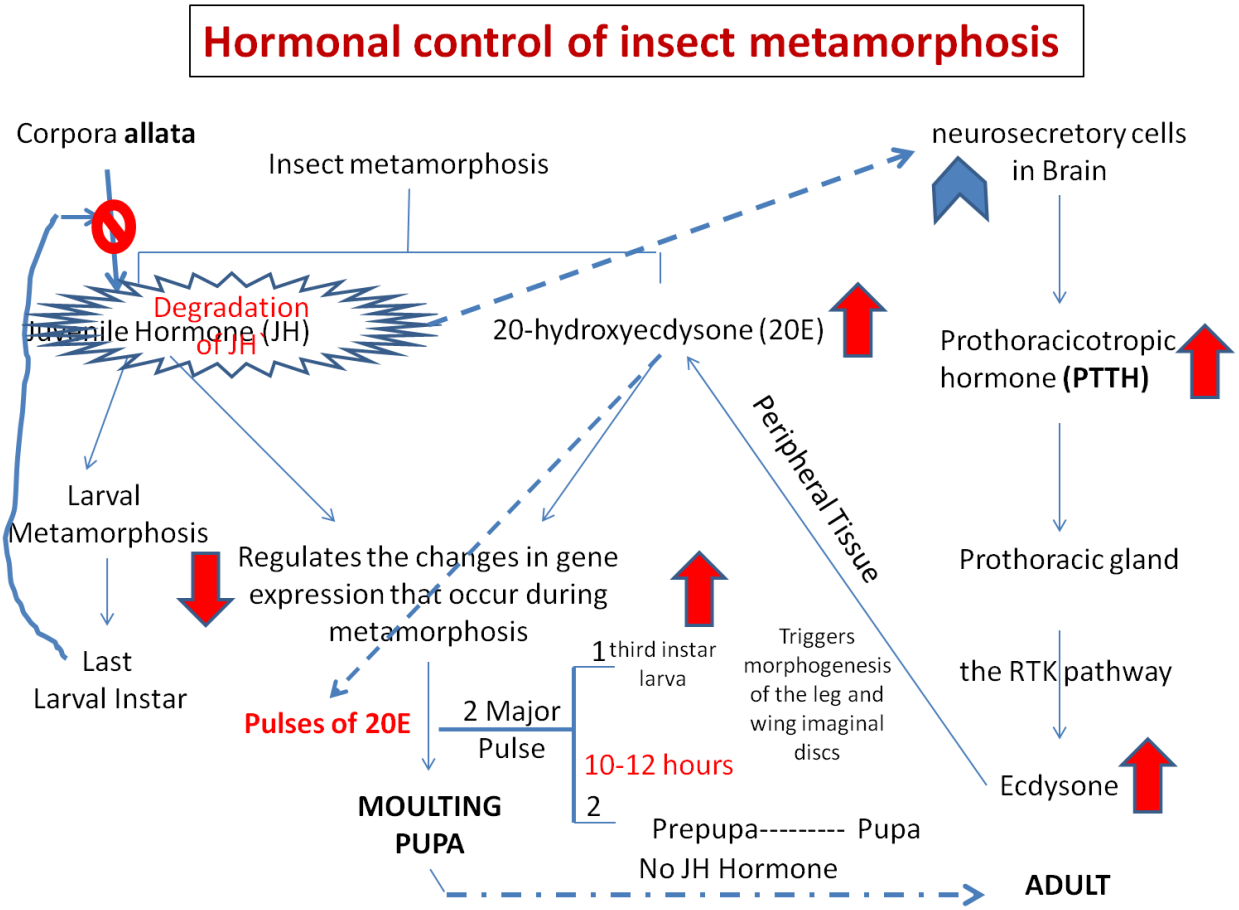
Although the details of insect metamorphosis differ among species, the general pattern of hormonal action is very similar. Like amphibian metamorphosis, the metamorphosis of insects is regulated by systemic hormonal signals, which are controlled by neurohormones from the brain. Insect molting and metamorphosis are controlled by two effector hormones: the steroid 20-hydroxyecdysone (20E) and the lipid juvenile hormone (JH). 20-Hydroxyecdysonef initiates and coordinates each molt and regulates the changes in gene expression that occur during metamorphosis. Juvenile hormone prevents the ecdysone-induced changes in gene expression that are necessary for metamorphosis. Thus, its presence during a molt ensures that the result of that molt is another larval instar, not a pupa or an adult.

The molting process is initiated in the brain, where neurosecretory cells release prothoracicotropic hormone **(PTTH)** in response to neural, hormonal, or environmental signals. PTTH is a peptide hormone with a molecular weight of approximately 40,000, and it stimulates the production of ecdysone by the prothoracic gland by activating the RTK pathway in those cells . Ecdysone is modified in peripheral tissues to become the active molting hormone 20E. Each molt is initiated by one or more pulses of 20E. For a larval molt, the first pulse produces a small rise in the 20E concentration in the larval hemolymph (blood) and elicits a change in cellular commitment in the epidermis.

A second, larger pulse of 20E initiates the differentiation events associated with molting. These pulses of 20E commit and stimulate the epidermal cells to synthesize enzymes that digest the old cuticle and synthesize a new one. Juvenile hormone is secreted by the corpora **allata.** The secretory cells of the corpora allata are active during larval molts but inactive during the metamorphic molt and the imaginal molt. As long as JH is present, the 20E-stimulated molts result in a new larval instar. In the last larval instar, however, the medial nerve from the brain to the corpora allata inhibits these glands from producing JH, and there is a simultaneous increase in the body's ability to
degrade existing JH. Both these mechanisms cause JH levels to drop below a critical threshold value, triggering the release of PTTH from the brain. PTTH, in turn, stimulates the prothoracic gland to secrete a small amount of ecdysone. The resulting pulse of 20E, in the absence of high levels of JH, commits the epidermal cells to pupal development. Larva-specific mRNAs are not replaced, and new mRNAs are synthesized whose protein products inhibit the transcription of the larval messages.

There are two major pulses of 20E during *Drosophila* metamorphosis. The first pulse occurs in the third instar larva and triggers the initiation of ("prepupal") morphogenesis of the leg and wing imaginal discs. The larva stops eating and migrates to find a site to begin pupation. The second 20E pulse occurs 10-12 hours later and tells the "prepupa" to become a pupa. The head inverts and the salivary glands degenerate. It appears, then, that the first ecdysone pulse during the last larval instar triggers the processes that inactivate the larva-specific genes and

initiates the morphogenesis of imaginal disc structures. The second pulse transcribes pupa-specific genes and initiates the molt. At the imaginal molt, when 20E acts in the absence of juvenile hormone, the imaginal discs fully differentiate and the molt gives rise to an adult.

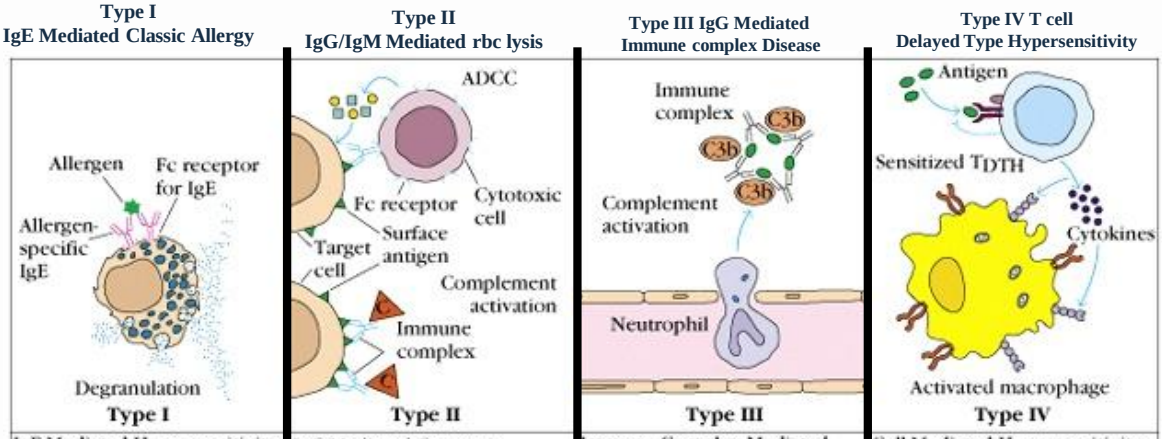


Q.6.

Ans 6 : Hypersensitivity: Hypersensitivity reactions are harmful antigen-specific immune responses , occur when an individual who has been primed by an \innocuous antigen subsequently encounters the same antigen , produce tissue injury and dysfuntion.

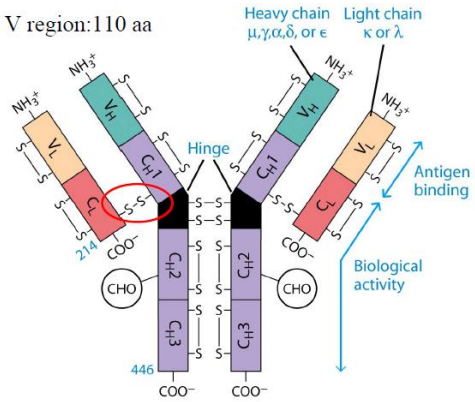
Allergen：the antigens that give rise to immediate hypersensitivity; **Atopy**：the genetic predisposition to synthesize inappropriate levels of IgE specific for external allergens

Classification of hypersensitivities: Types of hypersensitivity：I、II、III、IV

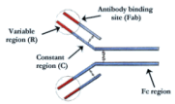
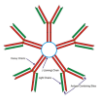
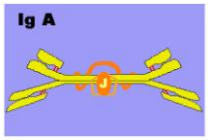
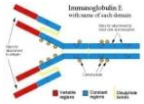


Type I	Type II	Type III	Type IV
IgE-Mediated Hypersensitivity	IgG-Mediated Cytotoxic Hypersensitivity	Immune Complex-Mediated Hypersensitivity	Cell-Mediated Hypersensitivity
Ag induces crosslinking of IgE bound to mast cells and basophils with release of vasoactive mediators	Ab directed against cell surface antigens meditates cell destruction via complement activation or ADCC	Ag-Ab complexes deposited in various tissues induce complement activation and an ensuing inflammatory response mediated by massive infiltration of neutrophils	Sensitized T_{DTH} cells release cytokines that activate macrophages or T_C cells which mediate direct cellular damage
Typical manifestations include systemic anaphylaxis and localized anaphylaxis such as hay fever, asthma, hives, food allergies, and eczema	Typical manifestations include blood transfusion reactions, erythroblastosis fetalis, and autoimmune hemolytic anemia	Typical manifestations include localized Arthus reaction and generalized reactions such as serum sickness, necrotizing vasculitis, glomerulnephritis, rheumatoid arthritis, and systemic lupus erythematosus	Typical manifestations include contact dermatitis, tubercular lesions and graft rejection

Answer 7: Immunoglobulin structure and function and types



IgG: γ
IgM: μ
IgA: α
IgD: δ
IgE: ε

Type	Number of ag binding sites	Site of action	Functions
IgG 	2	•Blood •Tissue fluid •CAN CROSS PLACENTA	•Increase macrophage activity •Antitoxins •Agglutination
IgM 	10	•Blood •Tissue fluid	Agglutination
IgA 	2 or 4	•Secretions (saliva, tears, small intestine, vaginal, prostate, nasal, breast milk)	•Stop bacteria adhering to host cells •Prevents bacteria forming colonies on mucous membranes
IgE 	2	Tissues	•Activate mast cells → HISTAMINE •Worm response

- **Answer 8 a: MHC: Major Histocompatibility Complex is -** Cluster of genes found in all mammals,Its products play role in discriminating self/non-self,Participant in both humoral and cell-mediated immunity
- MHC Act As Antigen Presenting Structures
- In Human MHC Is Found On Chromosome 6 - Referred to as HLA complex
- In Mice MHC Is Found On Chromosome 17 - Referred to as H-2 complex
- **Genes Of MHC Organized In 3 Classes**

  -Class I MHC genes :Glycoproteins expressed on all nucleated cells- Major function to present processed Ags to T_C

  -Class II MHC genes: Glycoproteins expressed on MΦ, B-cells, DCs- Major function to present processed Ags to T_H

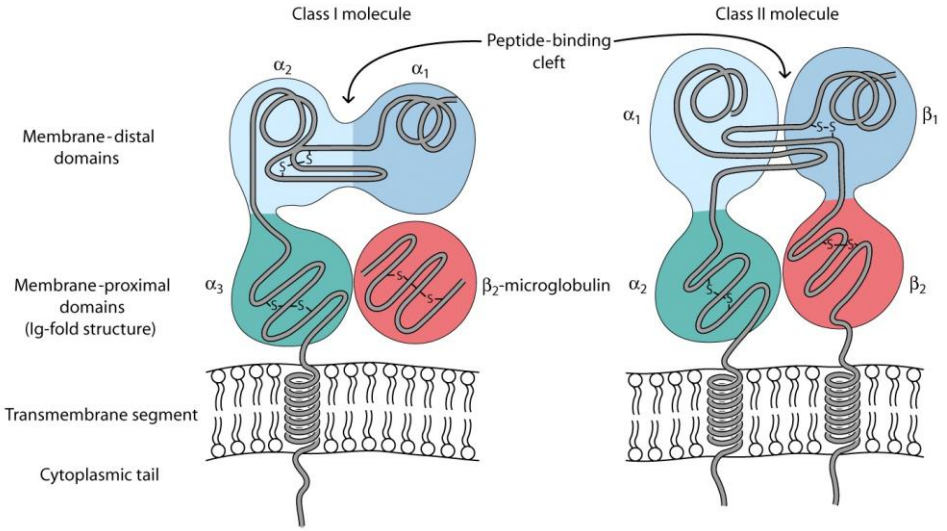
  -Class III MHC genes: Products that include secreted proteins that have immune functions. Ex. Complement system, inflammatory molecules

Mouse H-2 complex

Complex	H–2						
MHC class	I	II		III		I	
Region	K	IA	IE	S		D	
Gene products	H–2K	IA αβ	IE αβ	C′ proteins	TNF-α TNF-β	H–2D	H–2L

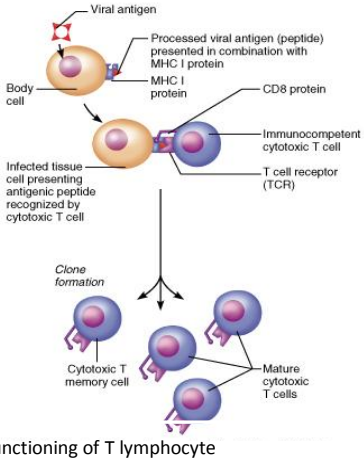
Human HLA complex

Complex	HLA							
MHC class	II			III		I		
Region	DP	DQ	DR	C4, C2, BF		B	C	A
Gene products	DP αβ	DQ αβ	DR αβ	C′ proteins	TNF-α TNF-β	HLA-B	HLA-C	HLA-A



Ans 8 b: T-Lymphocyte

- **Mature T-cells have T cell receptors which have a very similar structure to antibodies and are specific to 1 antigen.**
- **They are activated when the receptor comes into contact with the Ag with another host cell (e.g. on a macrophage membrane or an invaded body cell)**
- **After activation the cell divides to form:**
- **T-helper cells – secrete CYTOKINES**
  → help B cells divide
  → stimulate macrophages
- **Cytotoxic T cells (killer T cells)**
  → Kill body cells displaying antigen
- **Memory T cells**
  → remain in body



Functioning of T lymphocyte

Ans 8c:

An **antigen** is the substance that binds specifically to the respective antibody. Each antibody from the diverse repertoire binds a specific antigenic structure by means of its variable region interaction (CDR loops.
The antigen may originate from within the body or from the external environment. "**Self**" antigens are usually well tolerated by the immune system, which has been educated to non-reactivity against the structures present inside the body under the physiological conditions**. "Non-self"** antigens can be identified as invaders from the outside world or modified/harmful substances present under the distressed conditions in the body and only these are supposed to be attacked by the immune system. **In other words, the immune system will try to destroy or neutralize any antigen that has been recognized as a foreign substance and /or signal of harmed tissues.**

Cells present their antigenic structures to the immune system via a histocompatibility molecule. Depending on the antigen presented and the type of the histocompatibility molecule, several types of immune cells can become activated. Antigen was originally a structural molecule that binds specifically to the antibody, but the term now also refers to any molecule or molecular fragment that can be recognized by highly variable antigen receptors (B-cell receptor or T-cell receptor) of the adaptive immune system. **For T-Cell Receptor (TCR) recognition, it must be processed into small fragments inside the cell and presented to a T-cell receptor by major histocompatibility complex (MHC). Antigen by itself is not capable to elicit the immune response without the help of an Immunologic adjuvant**

CONT…..8C:

An immunogen is in analogy to the antigen a substance (or a mixture of substances) that is able to provoke an immune response if injected to the body. An immunogen is able to **initiate** an indispensable innate immune response first, later leading to the activation of the adaptive immune response, whereas an antigen is able to **bind** the highly variable immunoreceptor products (B-cell receptor or T-cell receptor) once these have been produced.

At the molecular level, an antigen can be characterized by its ability to be bound by the variable Fab region of an antibody. Note also that different antibodies have the potential to discriminate between specific epitopes present on the surface of the antigen. Hapten is a small molecule that changes the structure of an antigenic epitope. In order to induce an immune response, it has to be attached to a large carrier molecule such as protein. Antigens are usually proteins and polysaccharides, less frequently also lipids. This includes parts (coats, capsules, cell walls, flagella, fimbrae, and toxins) of bacteria, viruses, and other microorganisms. Lipids and nucleic acids are antigenic only when combined with proteins and polysaccharides Non-microbial exogenous (non-self) antigens can include pollen, egg white, and proteins from transplanted tissues and organs or on the surface of transfused blood cells. Vaccines are examples of antigens in an immunogenic form, which are to be intentionally administered to induce the memory function of adaptive immune system toward the antigens of the pathogen invading the recipient.

Epitope - The distinct molecular surface features of an antigen capable of being bound by an antibody (a.k.a. *antigenic determinant*). Antigenic molecules, normally being "large" biological polymers, usually present several surface features that can act as points of interaction for specific antibodies. Any such distinct molecular feature constitutes an epitope. Therefore, most antigens have the potential to be bound by several distinct antibodies, each of which specific to a particular epitope. Using the "lock and key" metaphor, the antigen itself can be seen as a string of keys - any epitope being a "key" - each of which matching a different lock. Different antibody **idiotypes**, each having distinctly formed complementarity determining regions, correspond to the various "locks" that can match "the keys" (epitopes) presented on the antigen molecule.

Allergen - A substance capable of causing an allergic reaction. The (detrimental) reaction may result after exposure via ingestion, inhalation, injection, or contact with skin.

Superantigen - A class of antigens that cause non-specific activation of T-cells, resulting in polyclonal T cell activation and massive cytokine release.

Tolerogen - A substance that invokes a specific immune non-responsiveness due to its molecular form. If its molecular form is changed, a tolerogen can become an immunogen.

Immunoglobulin-binding protein - These proteins are capable of binding to antibodies at positions outside of the antigen-binding site. That is, whereas antigens are the "target" of antibodies, immunoglobulin-binding proteins "attack" antibodies. Protein A, protein G, and protein L are examples of proteins that strongly bind to various antibody isotypes.

T-dependent antigen - T-dependent antigens are usually proteins. They require an assistance of T cells to induce the formation of specific antibodies.

T-independent antigen - T-independent antigens are usually polysaccharides stimulating B cells directly.

Immunodominant antigens are the ones that dominate (over all others from a pathogen) in their ability to produce an immune response. It is commonly assumed that T cell responses are directed against a relatively few immunodominant epitopes, although at least in some cases (e.g., infection with the malaria pathogen it is dispersed over a relatively large number of parasite antigens