Sem – II (PG)
Paper ZOO-203
Group B: Parasitology

Prepared by Anindita Das

Cellular basis of Host-Parasite Interaction

Cell damage during host-parasite interaction: Histopathological studies of parasite-damaged tissues have revealed that cell damage is of three major types.

(1) *Parenchymatous or albuminous degeneration:* It occurs when the cells become swollen and packed with albuminous or fatty granules, the nuclei become indistinct, and the cytoplasm appears pale. This type of damage is characteristic of liver, cardiac muscle, and kidney cells.

(2) *Fatty degeneration:* It means that the cells become filled with an abnormal amount of fat deposits, giving them a yellowish appearance. Liver cells commonly display this type of degeneration when in contact with parasites.

(3) *Necrosis:* It occurs when any type of cell degeneration persists. The cells finally die, giving the tissue an opaque appearance. As the result of encystment of *Trichinella spiralis* in mammalian skeletal muscle cells, necrosis of the surrounding tissues is followed by calcification.

Consequences of parasitism on cells:

One of the possible consequences of parasitism associated with cell and tissue parasites is a change in the growth pattern of the affected tissue. Some of these changes may be serious, whereas others are structural and have no serious systemic importance to the whole organism. Such changes, can be divided into four main types.

Hyperplasia: Hyperplasia is an accelerated rate of cell division resulting from an increased level of cell metabolism. This leads to a greater total number of cells, but not necessarily an increase in their absolute size. Hyperplasia, as associated with parasitism, commonly follows inflammation and is the consequence of an excessive level of tissue repair. When liver flukes, *Fasciola* spp., occur in the bile duct of their host, there is a thickening of this duct. This exemplifies the

hyperplastic condition resulting from excessive division of the epithelial lining of the duct stimulated by the presence of the parasite.

Hypertrophy: Hypertrophy is an increase in cell size. This condition is commonly associated with intracellular parasites. For example, during the erythrocytic phase of *Plasmodium vivax*, the parasitized red blood cells are commonly enlarged. Another interesting example of hyperplasia lies in a parasitized invertebrate. When the spermatogonial cells of the annelid *Polymnia nebulosa* are parasitized by the protozoan *Caryotropha mesnili*, hypertophy of the host cells occurs, involving both the nucleus and cytoplasm. Some of the surrounding cells undergo similar changes and eventually fuse with the infected cell to form a giant multinucleated cell.

Metaplasia: Metaplasia describes the changing of one type of tissue into another without the intervention of embryonic tissue. When the fluke *Paragonimus westermani* occurs in human lungs, it is surrounded by a wall of host tissue composed of epithelial cells and elongate fibroblasts. Since it is known that epithelial cells and large quantities of fibroblasts do not normally occur in lungs, it can be inferred that these encapsulating cells have resulted from the transformation of certain other types of cells in the lungs, hence metaplasia.

Neoplasia: Neoplasia is the growth of cells in a tissue to form a new structure, for example, a tumor. The neoplastic tumor (1) is not inflammatory, (2) is not required for the repair of organs, and (3) does not conform to a normal growth pattern. Neoplasms may be benign, i.e., remain localized and do not invade adjacent tissues, or malignant, i.e., invade adjacent tissues or move (metastasize) to other parts of the body through the blood or lymph. Cancers are malignant neoplasms. Several species of parasites have been associated with tumors, including cancers, in mammals.

Group	Parasite	Host	Site of Tumor
Protozoa	*Eimeria stiedae*	Rabbit	Liver
Trematoda	*Schistosoma mansoni*	Human	Intestine and liver
	Schistosoma haematobium	Human	Bladder
	Schistosoma japonicum	Human	Intestine
	Paragonimus westermani	Tiger	Lung
	Clonorchis sinensis	Human	Liver
Cestoda	*Cysticercus fasciolaris**	Rats	Liver
	Echinococcus granulosus	Human	Lung
Nematoda	*Gongylonema neoplasticum*	Rat	Tongue
	*Spirocerca lupi**	Dog	Esophagus

Molecular basis of Host-Parasite Interaction

Host/Parasite	Molecular interaction	Immune defence category	Strategy
Plants/bacteria	Adhesion proteins are responsible for attachment to host tissue and are associated with host specificity.	Barrier	Pathogens mimic the target of a host receptor to gain attachment & entry.
Nematodes/bacteria	Protein mimics attach to the heparin-binding domain of the host cuticle.		
Vertebrate/*Yersinia tuberculosis* (bact.)	Invasion protein binds to a receptor on the host cell surface by mimicking the host's fibronectin molecule.		
Plant/bacteria	Host FLS2 recognition molecule responds to flg22 and flagellin proteins in bacteria (PTI).	Innate	Recognition of patterns: host PRRs recognize PAMPs and signal downstream effectors.
Plant/fungi	Host R-encoded NB-LRR proteins bind directly to the Avr effector proteins (ETI).		
Snail/trematode	Host FREPs receptors recognize mucin molecules on the surface of their trematode parasite.		
Many invertebrates, some vertebrates/bacteria or fungi	PGRP receptors recognize conserved pathogen molecular pattern and modulate innate immune defence pathways.		
Vertebrate/virus	Complement proteins recognize and bind with conserved patterns on pathogens and mark them for destruction by immune effectors.		
Plant/bacteria: *Arabidopsis thaliana/Pseudomonas syringae*	Host molecule RIN4 damaged by pathogen effectors is recognized by host NB-LRR receptors RPM1, RPS2.	Innate	Recognition of modified self: host receptors recognize modified host molecules that indicate pathogen attack and signal downstream effectors.
Vertebrates/microbes	Complement proteins recognize modified or damaged self and translate danger into immune response.		
Jawed vertebrates/virus	Pathogens that match the markers of self-identity and express them on the cell surface are not attacked by NK cells.	Innate	Recognition of missing self: pathogen mimics host self-recognition molecules to avoid attack.

Illustrative examples from a range of plants and animals are provided in the table. PRR – Pattern Recognition Receptor, PAMP – Pathogen-Associated Molecular Patterns, FLS2 – Flagellin Sensing 2, PTI – PAMP-Triggered Immunity, NB-LRR – Nucleotide-binding site Leucine-Rich Repeat, Avr – Avirulence, ETI – Effector Triggered Immunity, FREP – Fibrinogen Related Proteins, PGRP – Peptidoglycan-Recognition Proteins, NK – Natural Killer.

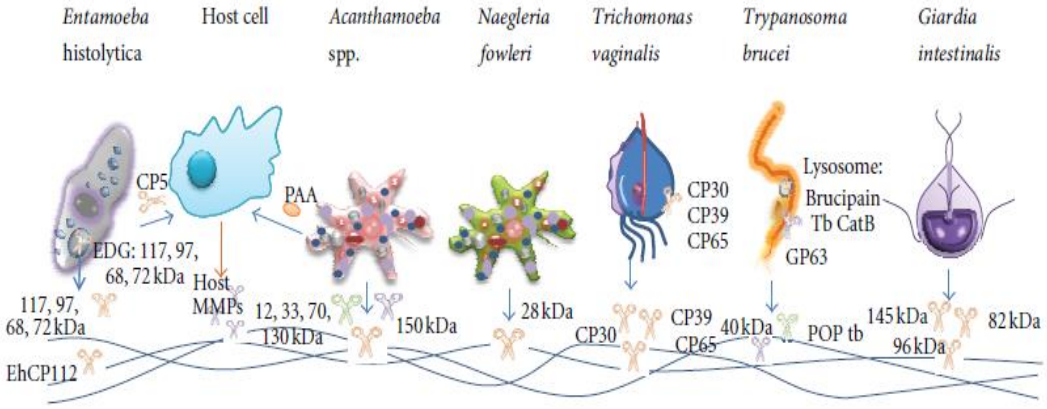

FIGURE : Extracellular parasite proteases. CPs: cysteine proteases, pink scissors; SPs: serine proteases, green scissors; MMPs: matrix metalloproteases, blue scissors; ECM extracellular matrix; EDG: electron-dense granules; POP: prolyl oligopeptidase; PAA: plasminogen activator activity; CatB: cathepsin B.

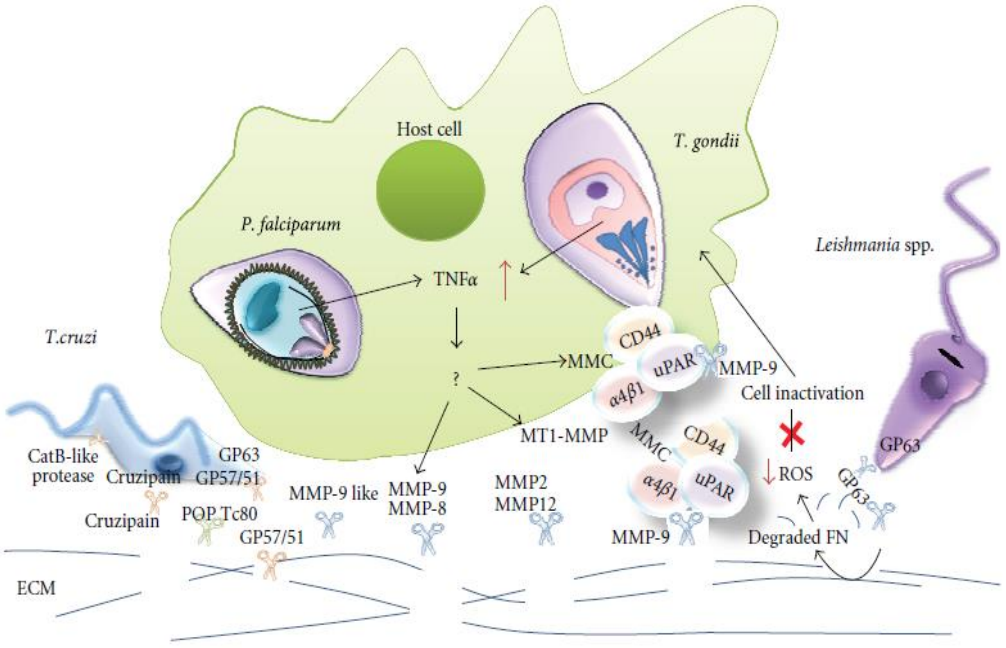

FIGURE : Intracellular parasite proteases. CPs: cysteine proteases, pink scissors; SPs: serine proteases, green scissors; MMPs: matrix metalloproteases, blue scissors; ECM extracellular matrix; POP: prolyl oligopeptidase; CatB: cathepsin B; MMC: migratory molecular complex; ROS: reactive oxygen species.

Above figures describe some parasites that secretes some proteases extracellularly and intracellularly respectively which degrade the host extracellular matrix (ECM) proteins (i.e. Collagen; Fibronectin; Laminin; Elastin) and invade into host cells and tissues.