Repair and Regeneration of oral tissues:

The response of the body to tissue destroyed can lead to complete restoration of tissue architecture and function (regeneration) or to restoration of function and of tissue continuity this called (repair).

Wound healing in oral mucosa:

Skin and oral mucosa have the primary functions of protection the underlining tissues and limiting entry of microorganisms and toxins. The damage to the oral m. result from physical, radiation and chemical irritation.

Initial response to wounding (hemostasis):

Damage to mucosal surf. Cause vascular damage, hemorrhaging into tissue defect, deposition of fibrin, aggregation of platelets and coagulation to form a clot. This clot forms a haemostatic barrier and protects the exposed tissues. Because of the moist environment of the oral cavity and salivary flow rate, the clot does not resemble the hard clots in skin that is easily lost.

Inflammatory cell activaion, migration and function:

Inflame. Cells in wound derive from 3 sources: cells normally present in tissues, cells extravasated when bl.vs. are damaged, cells carried in intact bl.vs. adjacent to wound, platelate-derived cytokines recruit leukocyte to site of tissue damage called chemotaxis process.
Neutrophils (1st inflame. Cells) invade the wound and activated in response to phagocytic stimuli and this cell contain enzymes that kill engulfed bacteria (short life span 24 hours),
Macrophages and other leuk. Cell enter the wound after 24 h. and remain in damaged tissue at 5 days. This function are phagocytosis of damaged t. and foreign material, release potent growth factors (ex: interleukin-1) for next phase of wound repair.

Reparative phase:

Successful repair of the injured tissues requires resolution of the inflame. Reaction, regeneration occur first in epith. and then in conn. T., damage to the epith. Results in mobilization and migration of epithelial cells at wound

margin. The cells lose their close attachment to each other and to the underlying connective tissue within 24 hours of wounding; histologically appear as a widening of the intercellular spaces.

After 24-48 hours cell division in the basal epith. Increases and those cells adjacent to the margin to migrate beneath the clot.

Epith. Cells continue to migrate until they reach the cells from the opposing wound margin at this time an increase in cell division leads to differentiation and reestablishing a normal epith. Tissue. The fibroblasts involved in wound derive from 2 sources:

- division of un damaged fibroblast at the wound periphery.
- Un differentiated connective tissue cells.

These cells from both sources migrate into the wound defect to form collagen of scar tissue. New bl. Vs. are participating in conn. Tissue formation to provide nutrients and oxygen.., secreting bioactive substances (endothelial cells)and allowing for inflame. Cell migrate to the site of injury.

Wound contraction and scarring:

Scar formation is physiologic and inevitable outcome of wound repair, the function of which is to restore tissue integrity quickly.
In skin the 1st fibroblast that enter the wound contain actin and myosin that have contraction properties.(called myofibroblasts cell) , these cell have junctions with each other and with conn. Tissue fibrils, by contracting the myofibr. Cell draw the edges of wound together and reducing the surface area and facilitating healing. Collagen is laid down form scar tissue and lead to rigidity and immobilization of area with impairment of function. In oral mucosa the scar tissue formed is remodeled so that most surgery within the mouth can be undertaken without fear of producing disabling scar tissue.

Wound healing of the dentogingival junction:

When the gingivitis progress to periodontitis the junctional epith. Migrate apically and is responsible for the pocket formation, this process requires cell proliferation and migration of the cells. Conn. tissue is destroyed during

periodontal disease and the junctional epith. Extends until it reaches intact conn. Tissue that stop its migration.

Repair of enamel:

Enamel cannot reform after it is destroyed because the cells that formed it no longer exist, if the carious process is arrested and the enamel surface layer has not broken down, remineralization can occur in the sub surface enamel, the remineralized enamel becomes more resistant than normal enamel to further demineralization.

Repair of the dentin-pulp complex:

Dentin is a vital tissue, and its repair is complex and varied because of several factors that include the extent and duration of the stimulus, the variability of dentin structure, and age of the tooth. Formation of dentin continues throughout the life, the pulp chamber becomes increasing smaller. At the same time, the cellularity of dentine decreases, its collagen content increases, the blood supply is diminished as is nerve supply and these events influence the reparative response of dentine-pulp complex.
When dental pulp is injured the immune system triggers an inflammatory response similar to that take place in any other connective tissues in the body.

The responses of the complex can be described in two ways:

1. in response to prolonged insult of slow onset, such as attrition or dental caries, a number of different events cause occlusion of the tubule within the tubular compartement, increased collagen deposition by odontoblast process may plug the tubule or mineral deposited in the tubule. All these reactions are aimed at depositing a calcified barrier to protect the dental pulp.
2. if the insult is sever and rapid in onset (fracture of tooth or cavity preparation), the odontoblast survives this degree of trauma and capable of depositing further reactionary dentin. If the odontoblast does not survive, the wounded pulp reacts by the classic repair mechanism involving cellular proliferation and scar tissue formation to form a bridge of reparative dentin that seals off the site of exposure.

Dental caries:

Under the light microscope can distinguish three zones in the early carious lesion. At the inner advancing edge is translucent zone, which is followed by a dark zone. The third zone is the body of the lesion, which occupies the space between the dark zone and the intact enamel surface.

Repair following tooth extraction:

After the tooth extracted, the defect is filled immediately by a blood clot (hemostatic response). The epith. Cells bordering the socket rim begin to proliferate and migrate across the clot so that after about 10 days the socket is epithelial zed.

The inflammatory response take place within the clot, the neutrophil and macrophages are present the proliferative and synthesizing phase differs from that in skin because the cells invade the clot are not fibroblasts but cells from the adjacent bone marrow that have osteogenic potential.(bone formation).

Comparison of repair responses in skin and teeth:-

Repair response	In skin	In teeth
Epithelial response	Proliferation and migration of cells to cover the defect	No epith, response because ameloblasts are lost at time of tooth eruption
Connective tissue response	Polymorph response, macrophage response, fibroblast response from undifferentiated perivascular cells and undamaged fibroblasts	Polymorph response, macrophage response, fibroblast response by division of undamaged pulpal and perivascular cells.
	New fibroblasts form collagen	New fibroblasts form collagen, which mineralizes to form dentin

Alterations of the periodontal connective tissues with the development of periodontal inflammation:

As dental plaque accumulates adjacent to ging. Margins, an inflammatory response is induced with in the gingival connective tissues. With in 3to4 days, connective tissue destruction result and 70% of collagen being lost with in foci of inflammation and if left untreated the amount of tissue destruction extend deeper toward the periodontal ligament and alveolar bone. A form of repair is initiated, resulting in fibrosis coexisting at foci of inflame.
The process of periodontal regeneration is complex and involves significant communication between all of the cellular and matrix components of the periodontium to induce the regeneration or repair capacity of this tissue. A major reason why periodontal regeneration is such a challenge is the need for restoration of not one, but four different tissues (gingival, periodontal ligament, cementum and bone.

Mechanisms of repair and regeneration of periodontal connective tissues:

Regeneration of periodontium is need new conn. Tissue fibers that insert into cementum and bone. Fiber insertion requires the healing components of all the soft and hard conn. T. of the periodontium to be integrated fully. These events include stimulation of an initial infl. Response, followed by specific cell populations, induction of their proliferation, cellular differentiation and combined of several cell types including fibroblasts for soft conn. T. , cementoblasts for cementogenesis, osteoblasts for bone formation and endothelial cells for angiogenesis. Repair of the period. Ligament involves the same mechanism as that found in skin without scar formation.

The reason is that although the repair mechanism is identical to that in skin, this scar tissue is remodeled by the ligament fibroblasts to restore normal architecture. For regeneration to occur the cells responsible for each tissue must be able to participate in these process at right location and in the correct temporal sequence. Also new cementum formation must occur for periodontal repair to take place. Thus cementum components may be capable of providing informational signals for the proliferation and

differentiation of periodontal cells and may regulate the regeneration of cementum and adjacent periodontal components.