Periodontium includes:   
1) Gingiva 2) cementum 3) PDL 4) Alveolar bone

All are connective tissue except the gingival which is both a connective tissue and epithelium tissue. Their origin Is the **ectoderm**  
  
GINGIVA:   
part of the mucosa of the GI tract and oral cavity, and is masticatory (keratinized), unlike the lining type which is non-keratinized, it covers the bone till the mucogingival junction.

Free gingival: keratinized (masticatory process) -> not attached to the underlying tissue. Gingiva covers the alveolar process and surrounds the cervical part of the tooth.

The part that surrounds the tooth is called “epithelium”, whereas the one on the opposing side is called “gingival”.

Free gingival margin -> free gingiva-> Gingival groove-> attached gingival -> alveolar mucosa -> oral cavity

“sulcular epithelium, junctional epithelium

Q) What maintains the junction between the gingival and the tooth surface?  
Hemodesmosomes

Marginal gingival form free gingival margin -> marginal groove (unattached)

Gingiva: forms barrier agains microorganisms (**most important function**) as a defense mechanism.

Marginal gingival can be separated by periodontal probe, (1.5-2 mm) can be normal to the CEJ.

Gingival sulcus depth is zero, in germ-free animals. Histologically, its depth ranges from 07-1.8 mm, averaging 2 mm. 3 mm probing depth is considered healthy.

INTERDENTAL PAPILLAE:  
Shape depends on the **contacts between** **teeth, shape of teeth, width of proximal surface, course of CEJ**.

We have 2 types:

1. Pyramidal
2. Col (covered by non keratinized epithelium, feature is common with juntional epithelium(protective function))

ATTACHED GINGIVA:   
Film/tightly bound to underlying priostium/ resilient/stipplings (orange-shape appearance)

Stippling usually disappears with inflammation, but if we can’t see it doesn’t mean that th gingival is inflamed. They aren’t always related.

Width=span (cervical-apical), it increases with age.

Lingually, gingival terminates to the lingual alveolar mucosa. (Mandible -> lining epithelium).

Palatal there is no demarcation, since the palat is a masticatory mucosa  
  
EPITHELIUM:  
Stratified squamous epithelium

1. Oral epithelium (free and attached gingival)
2. Gingival epithelium   
    a. sulcular  
    b. junctional (no rete pege -> healthy situation)

* Rete peges increase surface area to increase exchange (nutrition), and for cells to pass through for signaling purposes.

Keratinization is differentiation process decreases in activity

Oral epithelium

* Towards oral cavity
* Smaller size
* Intercellular space is smalles
* More desmosomes

Juntional epithelium

* Towards the tooth
* Larger cell size (maintin dynamic physiologic contact.)
* Larger intercellular space

Describe the gingival:

Colour (vascularity)  
Width  
Keratinization   
Contour  
Shape  
Consistency

PDL:   
Has a glass appearance, connects tooth to root, increases with age.

Fibers:

1. Oblique fibers
2. Alveolar
3. Horizontal
4. Apical
5. Interradicular

PDL:

* maintains tooth against forces
* nutritional function
* sensory function
* transmitting occlusal forces to bone
* CEMENTUM:  
  no nerves, no vessels
* Acellular extrinsic fibers, cellular internsic fibers, cellular mix cementum (fibroblast from PDL and cementoblast)
* Coronally very thin, thick apically.
* CEJ:  
  alveolar bone
* Maxillary -> palatally -> thicker
* Mandible -> anteriorly -> lingually -> thicker  
   -> posteroirly -> buccally -> thicker
* NERVE SUPPLY:  
  maxilla:   
  infraorbital : 5-5 (mainly canines)  
  infraoribital and posterior superior: premolars  
  superior palatine: palatally anteriors   
  greater palatine: palatally posteriors
* Mandible:   
  mental: mental foramen to mental foramen  
  buccal nerve: lower 6 and behind it.  
  sublingual nerve: lingually.

Classification in periodontology is a very complicated issue : because

diseases are multifactorial and so complex and different diagnosis from periodentists   
  
  
Diagnosis is based solely on signs and symptoms, while classification is based on treatment approach, risk factors and many different things   
  
Periodontitis:

Major types are: aggressive and chronic periodontitis

How are they different? They are similar in the signs and symptoms, but this doesn’t mean signs and symptoms are found in each and every disease of them.

Periodontitis : any attachment loss in the periodontium, it can be aggressive or chronic depending on the degree of the attachment loss , amount of deposits , oral hygiene habits of the ptn , and if he’s smokers or not

Gingival diseasses :

-plaque induced (inflammation confined to the ginigiva ,no attachment loss)

-nonplaque induced

Plaque induced gingival diseases has many categories:

-without any local contributing factors ,, main cause of gingivitis is plaque (as well as chronic and aggressive periodontitis)

-the main etiological factor is plaque.

Periodontitis as a manifestation of systemic diseases has nothing to do with plaque

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gingivitis plaque induced, is affected by the endocrine system, mainly puberty, menstruation, pregnancy, DM)

Inflammation of the gingiva due to menstruation and pregnancy (disturbance in the hormones) which increase the risk of gingival diseases.

Progesterone, decreases the inflammatory protection mechanism of the host’s body, and so it increases the inflammation of the gingiva.

[Diabetes Mellitus](https://www.google.jo/url?sa=t&rct=j&q=&esrc=s&source=web&cd=1&ved=0CCsQFjAAahUKEwjf9Yub79rIAhWj_HIKHVZABU0&url=http%3A%2F%2Fwww.webmd.com%2Fdiabetes%2Fguide%2Ftypes-of-diabetes-mellitus&usg=AFQjCNG7YsY8j_Dl2iEC7Z-MFL0U9tpF2w&bvm=bv.105841590,d.bGQ) as a factor that increase the inflammation present due to plaque accumulation.( In gingivitis )

But diabetes mellitus in chronic periodontits is a risk factor .  
  
  
Modifying factors for plaque induced gingivitis:

Blood dyscrasias :

20-30% leukemia cases,,,can be diagnosed for the first time on the dental chair ( the gym become bluish , atrophic and you may find multiple ulcers )Modified by medication:1- drug induced gingivitis ,2- drug influenced gingival enlargement , caused mainly by 3 drugs :  
 1- phenytoin 2-calcium channel blocker 3- cyclosporine

-the main cause of gingivitis is plaque but is exacerbated by other factors  
  
Non plaque induced gingivitis mainly affect low socioeconomic group of people, developing countries, and immunocompromised people  
  
Periodontitis : is an inflammatory disease of the supporting tissues of the teeth caused by specific microorganism or a group of microorganisms resulting in progressive destruction of the periodontal ligaments and the alveolar bone with pocket formation , recession or both.

Inflammatory disease happen due to destruction caused by an inflammatory response.

Clinically we see periodontitis as an enlargement in the gingiva, pocketing alone or with gingival recession  
  
In periodontitis mainly bone loss is horizontal  
  
Risk factors of periodontitis : main one is plaque accumulation (main etiological factor )

Others : smoking ,age (with increasing age risk increases),,we mean here a longer time span spent under the risk ,,not the age itself as a factor,,,DM, obesity , genetics, stress .

Aggressive periodontitis : bone loss can occur with pockets but no gingival recession ,,a lot of pain and bleeding in the gingiva , this happens as a result of a very fast loss of attachment in the periodontal tissues that the gingiva had no time to recess along with them .

Only pocketing , there is no plaque accumulation

Primary factors:

-medically fit pockets

-rapid attachment loss and more destruction

-familail aggregation of cases (a lot of aggressive periodontitis cases in the family ),, clustering in the family.  
  
Down syndrome and cyclic neutropenia ,,might be associated with certain degree of periodontitis (different degrees) that has nothing to do with plaque accumulation.

Necrotizing ulcerative periodontal diseases : clinical characteristics: pain, fetid breath, bleeding spontaneously, blunting ,pseudo membrane)

Abscess of the periodontium ,,,gingival abscess ,periodontal abscess, pericoronal abscess

Periodontitis associated with endodontic lesion ,,,endo-perio (I treat the endo problem first ,as a result, the perio problem will disappear ) ,,or perio-endo lesion ( the problem is mainly perio problem ,,very difficult to treat ),,or combined (extract the tooth).

Developmental or acquired deformity and conditions :

1 -localized factors that predispose to plaque induced diseases ,ex: palatal grooves, enamel projections ,enamel curvatures, teeth location, teeth anatomic factors , root fracture, restorations ,appliances cervical root resorption ,dental restorations).

2- mucogingival deformity and conditions around the tooth ,ex: soft tissue recession ,lack of keratinized gingiva , it has nothing to do with plaque  
  
Occlusal trauma can be: primary or secondary

the most common prevalent disease in the world is **Chronic Gingivitis**and the next most prevalent disease is **Caries**, and the third most prevalent is **Chronic Periodontitis**Epidemiology; is a Greek word that means observation upon people like what we just did in the example above.

*How we can utilize Epidemiology?*

1. Collecting data to describe normal biological process that each of us passes through in his life. Eg; A-heights (We have now corresponding charts for heights in every maternal or children clinics and based on these they came up with a fact that is if you measure a 2 year old child’s height and you multiply it by 2 you would get his height at adulthood). B- Chronology of tooth eruption. C- Blood groups.  
     
     
   2- Identify determinants of the disease (Risk Factors). This is important for preventive approaches for patients with risk factors such as obesity, lack of exercise, smoking…
2. Testing hypothesis for prevention and control of diseases through special studies in population, clinical studies and clinical drugs  
     
     
   4 - Planning and evaluating health care services.