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Gingival Pigmentation – A Review

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ABSTRACT

Of all the colours in the animal kingdom, the wide spread and most important to man is melanin. Besides providing pigmentation, it also plays an essential role in defending the body against harmful Ultraviolet (UV) rays and other environmental challenges. Other than physiological pigmentation, various disorders can also cause hyperpigmentation. Gingival pigmentation plays a key role in facial esthetics and before undergoing any depigmentation procedure it is absolutely necessary to exclude any other pathological conditions affecting gingival pigmentation. In this review, we discuss the physiology and biochemistry of pigmentation, racial and intra oral variations in gingival pigmentation, disorders of pigmentation and various methods for depigmentation.

Keywords: Gingiva, melanin pigmentation, melanocytes, gingival depigmentation techniques

Introduction

Colours abound throughout the animal kingdom. The wide spread and most important to man is melanin, which also gives colour to the feathers of the birds, beetles and slugs, and fills the ink sacs of the octopus and the squids. Melanin pigmentation is highly heritable, being regulated by genetic, environmental, and endocrine factors that modulate the amount, type, and distribution of melanin in the skin, hair, and iris of eyes.[1] Melanin also plays an essential role in defending the body against harmful UV rays and other environmental challenges. It has been traditionally believed that skin pigmentation is the most important photo protective factor, as melanin, besides functioning as a broadband UV absorbent, has antioxidant and radical scavenging properties.[2] In this review we discuss the physiology and biochemistry of pigmentation, various indices used for gingival pigmentation, disorders of pigmentation and various depigmentation procedures.

Physiology of Gingival Pigmentation

Melanin is a non–hemoglobin-derived pigment formed by the melanocytes, which are dendritic cells of neuroectodermal origin located in the basal and spinous layers of the gingival epithelium.[3] Melanin pigmentation appears as early as three hours after birth in the oral tissues and in some cases is the only sign of pigmentation on the body.[4] The number of melanocytes in the mucosa corresponds numerically to that of skin; however, in the mucosa their activity is reduced. Melanin can be of three types. Eumelanin which imparts black or brown colour, Phaeomelanin which imparts red colour and Neuromelanin which

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is present in nervous system. Melanin from natural sources has reported to possess protection against UV radiation, enzymatic lysis and damage by oxidants.[5]

Biochemistry of Pigmentation

The process of pigmentation consists of three phases[6]:

I) Activation of melanocytes: The activation phase occurs when the melanocytes are stimulated by factors like stress hormones, sunlight etc. leading to production of chemical messengers like melanocyte stimulating hormone.

II) Synthesis of melanin: In synthesis phase, melanocytes make granules called melanosomes. Both eumelanins and phaeomelanins are derived from amino acid tyrosine. Tyrosine isoxidized to 3,4-dihydroxyphenylalanine (DOPA) by the copper containing enzyme tyrosinase, which also catalyses the future oxidation of dopaquinone. Tyrosinase catalyses the first two steps of melanin production. (Figure 1)

III) Expression of melanin: In expression phase, melanosomes are transferred from the melanocytes to the keratinocytes which are the skin cells located above melanocytes in the epidermis. After this, melanin colour eventually becomes visible on the surface of skin.

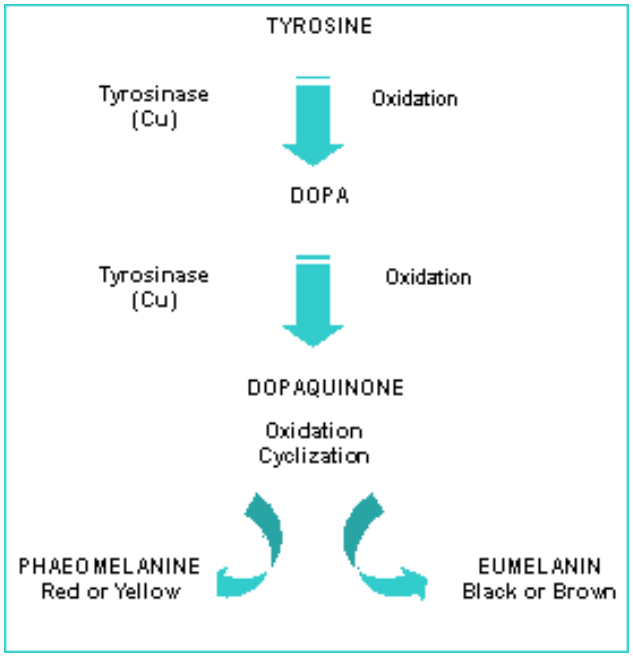

Figure 1. Melanin Synthesis

It is generally agreed that pigmented areas are present only when melanin granules synthesized by melanocytes are transferred to the keratinocytes. Dendritic melanocytes account for only 1% of epidermal cells. Each basal layer melanocyte is associated with about thirty six keratinocytes and one Langerhans cell (epidermal melanin unit).[7]

Racial Differences in Melanosomes

Although the number of melanocytes is essentially constant, the number, size, and the manner in which the melanosomes are distributed within the keratinocytes vary. In general, more deeply pigmented skin contains numerous single large melanosomal particles that are ellipsoidal and intensely melanotic. Lighter pigmentation is associated with smaller and less dense melanosomes that are clustered in membrane bound groups.[8] (Figure 2)

Intra Oral Variations in Pigmentation

There are two basic colour zones in the oral cavities of most people which comprise the attached and marginal gingiva on one hand and the adjacent alveolar mucosa on the other hand.[9] Studying gingival colour using the Munsell colour system in dentistry, Ibusuki (1975) reported that gingival colour varied with the position of the papillary, marginal

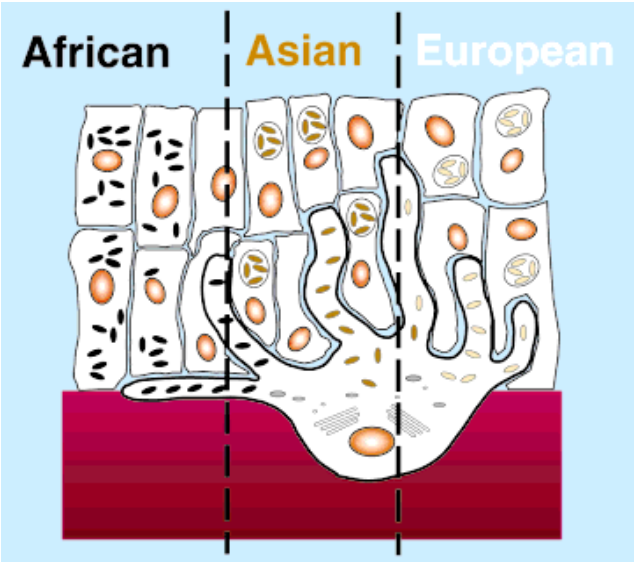

Figure 2. Schematic representation of a single melanocyte in different races

and attached gingiva.[10] A study to correlate skin colour and gender with intensity and distribution of gingival melanin pigmentation in a group of South Indians by Ponnaiyan et al found that South Indians predominantly have pigmentation in attached gingiva and interdental papilla. It appeared that the degree of gingival pigmentation of the gingiva and skin was reciprocally related. The highest rate of gingival pigmentation was observed in the area of the incisors. Incidence of pigmentation did not differ between the sexes.[11]

Disorders of Pigmentation

Apart from physiologic pigmentation, pigmentation can occur due to systemic or local causes.

Systemic and local causes of pigmentation[16]

Localized Pigmentations
Amalgam tattoo, graphite or other tattoos, nevus, melanotic macules, melanoacanthoma, malignant melanoma, Kaposi's sarcoma, verruciform xanthoma
Multiple or Generalized Pigmentations
Genetics: Idiopathic melanin pigmentation (racial or physiologic pigmentation), Peutz-Jegher's syndrome, Laugier-Hunziker syndrome, spotty pigmentation, endocrine overactivity, Carney syndrome, Leopard syndrome, and lentiginosisprofusa
Drugs: Smoking, betel, anti-malarials, antimicrobials, minocycline, amiodarone, clorpromazine, ACTH, zidovudine, ketoconazole, methyldopa, busulphan, menthol, contraceptive pills, and heavy metals exposure (gold, bismuth, mercury, silver, lead, copper)
Endocrine: Addison's disease, Albright's syndrome, Acanthosis nigricans, pregnancy, hyperthyroidism
Post inflammatory: Periodontal disease, postsurgical gingival repigmentation
Others: Haemochromatosis, generalized neurofibromatosis, incontinentiapigmenti, Whipple's disease, Wilson's disease, Gaucher's disease, HIV disease, thalassemia, pigmented gingival cyst, and nutritional deficiencies

INDICES USED FOR GINGIVAL PIGMENTATION

Various indices used are:

ORAL PIGMENTATION INDEX (DOPI)[12]

(Dummett 1964)

This index of oral pigmentation is the commonly used index due to its simplicity and ease of use. The scores are as follows:

0	No clinical pigmentation (pink-colored gingiva)
1	Mild clinical pigmentation (mild light brown color)
2	Moderate clinical pigmentation (medium brown or mixed pink and brown color)
3	Heavy clinical pigmentation (deep brown or bluish black color)

DOPI Assessment - Sum of assigned estimates of components ÷ 32 unit spaces

The DOPI Assessment is scaled according to the following designations:

0	No clinical pigmentation of the gingiva
0.031 - 0.97	Mild gingival pigmentation
1.0 - 1.9	Medium gingival pigmentation
2.0 - 3.0	Heavy gingival pigmentation

MELANIN INDEX [13] (Hedin 1977)

This index has classified pigmentation as follows:

Degree I	No pigmentation
Degree II	One or two solitary unit(s) of pigmentation in papillary gingiva without the formation of a continuous ribbon between solitary units
Degree III	More than three units of pigmentation in papillary gingiva without the formation of a continuous ribbon
Degree IV	One or more short continuous ribbons of pigmentation
Degree V	One continuous ribbon including the entire area between canines

MELANIN PIGMENTATION INDEX[14] (Takashi *et al* 2005)

The index is as follows:

Score 0	No pigmentation
Score 1	Solitary unit(s) of pigmentation in papillary gingiva without extension between neighbouring solitary units
Score 2	Formation of continuous ribbon extending from neighbouring solitary units

According to Peeran et al, in 2014[15] the above indices seemed to lack the capacity to relate various aspects of gingival pigmentation. They are also not determining the patient's treatment need. Moreover, other gingival-pigmented lesions are beyond their scope, as they were intended only for racial pigmentation.

Proposed gingival melanin pigmentation and pigmented lesions index by Peeran et al

Score 0	Coral pink-colored gingiva, no gingival pigmentation, and/or pigmented lesions
Score 1	Mild, solitary/diffuse, gingival melanin pigmentation involving anterior gingiva, with or without the involvement of posterior gingiva
Score 2	Moderate to severe, solitary or diffuse, gingival melanin pigmentation involving anterior gingiva with or without the involvement of posterior gingiva
Score 3	Gingival melanin pigmentation only in posterior gingiva
Score 4	Tobacco-associated pigmentation: Smoker's melanosis, chewing tobacco
Score 5	Gingival pigmentation due to exogenous pigments-Amalgam tattoos, arsenic, bismuth, chewing betel nut, cultural gingival tattooing, drinks, food colors, lead-burtonian line, mercury, silver, topical medications and idiopathic.
Score 6	Gingival pigmentation due to other endogenous pigments: Bilirubin, blood breakdown products, ecchymosis, hemochromatosis, hemosiderin, petechiae.
Score 7	Drug-associated gingival pigmentation: Antimalarial drugs, minocycline, oral contraceptives
Score 8	Gingival pigmentation associated with other causes: Addison's disease, Albright's syndrome, basilar melanosis with incontinence, hereditary hemorrhagic telangiectasia, HIV patients, lichen planus, neurofibromatosis, Peutz-Jeghers syndrome, pyogenic granuloma/ granulomatous epulis
Score 9	Pigmented benign lesions: hemangioma, melanocytic nevus, pigmented macule
Score 10	Pigmented malignant lesions: Angiosarcoma, Kaposi's sarcoma, malignant melanoma

A clinician may recommend a depigmentation procedure when the patient scores 1-2 score in the index and has up to class 2 of **Liebart and Deruelle**[15] Smile line classification, which is as follows:

Class 1	Very high smile line - more than 2 mm of the marginal gingiva visible
Class 2	High smile line - between 0 and 2 mm of the marginal gingiva visible
Class 3	Average smile line - only gingival embrasures visible
Class 4	Low smile line - gingival embrasures and cemento-enamel junction not visible.

Depigmentation Methods

Though not a medical problem, cosmetic demands by patients warrant depigmentation procedures. Before depigmentation procedures, it is important to exclude any pathologic reasons for pigmentation. **Roshna & Nandakumar** in 2005[17] classified different gingival depigmentation methods as:

I. Methods used to remove the gingival pigmentation:

A. Surgical methods:

a. Scalpel surgical technique

b. Bur abrasion method

c. Electro-surgery

d. Cryosurgery,

e. Lasers,

f. Radiosurgery.

B. Chemical methods.

II. Methods used to mask the gingival pigmentation:

a. Free gingival graft.

b. Acellular dermal matrix allograft

Conclusion

Facial aesthetics involves the interaction of many elements of the periodontium, of which gingival pigmentation is one. Patient awareness and expectations have increased to a point where less than optimal esthetics is no longer acceptable. Before undergoing any depigmentation procedure, it is absolutely necessary to exclude any other pathological conditions affecting the gingival pigmentation. Success of the depigmentation procedure may be weighed only by the extent of depigmentation achieved and by the time taken for reappearance of pigments.

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