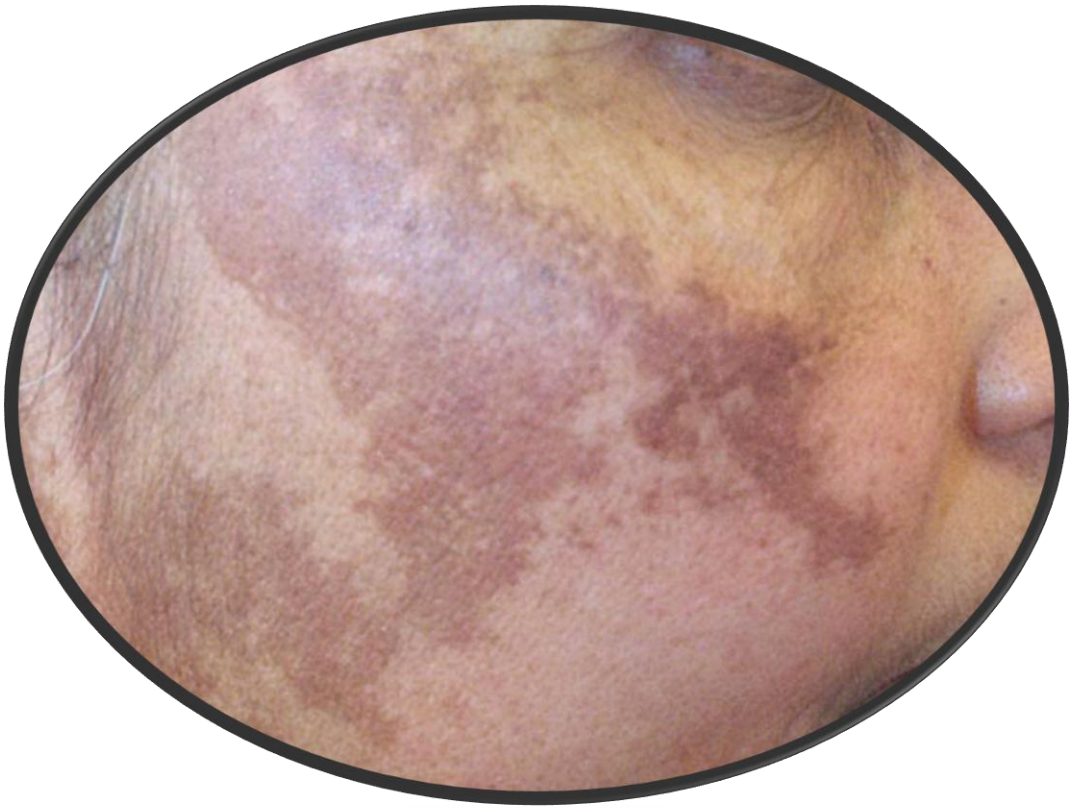
MELASMA AND HOMOEOPATHY

Miasmatic aspects of Melasma



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Melasma and Homoeopathy

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Definition

Melasma is a common acquired dermatosis (Psora/ Causa occasionalis), involving changes in normal skin pigmentation, characterized by symmetrical, blotchy, and light to dark brown macules (Psora), with irregular contour, but clear limits, on photo-exposed areas, especially the face, forehead, temples, and more rarely on the nose, eyelids, chin, and upper limbs, especially in fertile age women and those with intermediate skin phototypes, due to the hyperactivity of epidermal melanocytes, mostly induced by ultraviolet radiation with relation to genetic (Syphilitic) and hormonal factors, drugs and cosmetics use, endocrinopathies and sun exposure.

Synonyms

This form of facial pigmentation is sometimes called chloasma, but as this means green skin, the term melasma is preferred. Greek melas means black and cloazein, means greenish.

Incidence

- *Women to men ratio 1-in-4 to 1-in-20.*
- *Common between the age of 20 and 40 years, but can begin in childhood or not until middle-age.*
- *Melasma is more common in people that tan well or have naturally brown skin (Fitzpatrick skin types 3 and 4) compared with those who have fair skin (skin types 1 and 2) or black skin (skin types 5 or 6).*

Causes

Melasma commonly arises in healthy, non-pregnant adults. Lifelong sun exposure causes deposition of pigment within the dermis and this often persists long term (Psora/ Sycosis). Exposure to ultraviolet radiation deepens the pigmentation because it activates the melanocytes to produce more melanin (Psora/ Causa occasionalis). The known causes are-

- *Sun exposure and sun damage (Causa occasionalis)*
- *Pregnancy (Causa occasionalis)*
- *Hormone treatments—oral contraceptive pills, hormone replacement, intrauterine devices (Causa occasionalis)*
- *Certain medications (including new targeted therapies for cancer), phototoxic drugs, scented or deodorant soaps, toiletries and cosmetics—these may cause a phototoxic reaction that triggers melasma (Causa occasionalis)*
- *Endocrinopathies like hypothyroidism (Psora/ Syphilis/ Sycosis)*
- *Genetic influences (Syphilis)*
- *Emotional factors (Psora)*

Pathophysiology

The pigmentation is due to overproduction (Psora/ Sycosis) of melanin by the melanocytes, which is taken up by the keratinocytes. This causes epidermal or dermal melanosis or both. There is a genetic predisposition to melasma. In most people melasma is a chronic disorder.

Skin Types

Thomas B. Fitzpatrick developed the most commonly used scheme to classify a person's skin type by their response to sun exposure in terms of the degree of burning and tanning. The classification is given here under-

Eye colour	Do you turn brown?
0. Light colours	0. Never
1. Blue, gray or green	1. Seldom
2. Dark	2. Sometimes
3. Brown	3. Often
4. Black	4. Always
Natural hair colour	**How brown do you get?**
0. Sandy red	0. Never
1. Blond	1. Light tan
2. Chestnut or dark blond	2. Medium tan
3. Brown	3. Dark tan
4. Black	4. Deep dark
Your skin colour (unexposed areas)	**Is your face sensitive to the sun?**
0. Reddish	0. Very sensitive
1. Pale	1. Sensitive
2. Beige or olive	2. Sometimes
3. Brown	3. Resistant
4. Dark brown	4. Never have a problem
Freckles (unexposed areas)	**How often do you tan?**
0. Many	0. Never
1. Several	1. Seldom
2. Few	2. Sometimes
3. Rare	3. Often
4. None	4. Always
If you stay in the sun too long?	**When was your last tan?**
0. Painful blisters, peeling	0. +3 months ago
1. Mild blisters, peeling	1. 2–3 months ago
2. Burn, mild peeling	2. 1–2 months ago
3. Rare	3. Weeks ago
4. No burning	4. Days

Score		
0–6	Skin Type I	Always burns, never tans (pale white skin)
7–13	Skin Type II	Always burns easily, tans minimally (white skin)
14–20	Skin Type III	Burns moderately, tans uniformly (light brown skin)
21–27	Skin Type IV	Burns minimally, always tans well (moderate brown skin)
28–34	Skin Type V	Rarely burns, tans profusely (dark brown skin)
35+	Skin Type VI	Never burns (deeply pigmented dark brown to black skin)

Fitzpatrick's skin types

Skin colour

The skin is the most visible aspect in human and its colour is one of its most flexible features. The synthesis of vitamin D on the skin, degradation of folic acid by UVR, resistance to direct sun exposure and cultural elements explain the phenotypical variation of skin colour in different latitudes.

Normal human skin colour is mainly influenced by the production of melanin, a dense high-molecular-weight brown pigment. Skin colour is also affected by-

- *The melanogenic activity inside melanocytes (Psora)*
- *Melanin synthesis rate (Psora)*
- *Density of melanin- The size, number, composition and distribution of cytoplasm particles of the melanocytes called melanosomes (Psora/ Sycosis)*
- *The chemical nature of the melanin*
- *Exogenous yellow pigments, the carotenoids (Causa occasionalis)*
- *Endogenous red, oxygenated haemoglobin in capillary vessels in the dermis*
- *Endogenous blue, reduced haemoglobin in venules*

Melanocytes

Melanocytes are the specialized cells, responsible for skin and hair pigmentation, skin tone and protection against the damages caused by UVR. Embryologically, melanocytes are dendritic cells, derived from melanoblasts, which originate from the neural crest, migrating to the skin shortly after closure of the neural tube. After being fully developed, they spread through different sites-

- *Eyes- retina pigment epithelium, iris and choroid*
- *Ears- vascular strias*
- *Central nervous system- leptomeninges*
- *Hair matrix, mucosa, and skin*

The number of melanocytes goes down with age, in areas not exposed to light. Racial differences in pigmentation are due to level of activity of melanin viz. melanosomes and melanin synthesis, in the proportion of melanin subtypes like pheomelanin and eumelanin, their distribution, and environmental factors like sun exposure, which directly stimulates the synthesis of melanin (Psora/ Causa occasionalis). The melanin produced in melanocytes is stored in specific intracytoplasmatic structures called melanosomes.

Melanosomes

Melanosomes are highly specialized elliptical cell organelles, performing synthesis and storage of melanin as well as storage of the tyrosinase synthesized by ribosomes. Melanosomes are larger and more mature in black people than in white people and are stored more as units than in clusters.

Melanin and melanogenesis

Melanin is the main biological pigment involved in skin pigmentation causing differences in skin colour. In the presence of molecular oxygen, tyrosinase oxidizes tyrosine into dopa (dihydroxyphenylalanine) and this into dopaquinone. The presence or absence of cysteine determines the course of the reaction for the synthesis of its two types- eumelanin or pheomelanin. In this manner, melanogenesis has three different and important steps-

1. *Production of cysteinyldopa, which continues as intense as the quantity of cysteine present.*
2. *Oxidation of cysteinyldopa to form pheomelanin, a process that depends on the quantity of cysteinyldopa present.*
3. *Production of eumelanin, which only starts after the depletion of most of the cysteinyldopa. However, eumelanin seems to deposit on pre-formed pheomelanin. The ratio between pheomelanin and eumelanin is determined by the activity of tyrosinase and availability of cysteine.*

Eumelanin

Eumelanin is an insoluble brown alkaline polymer. It absorbs and disperses ultraviolet light, weakening its penetration on the skin and reducing the harmful effects of the sun. Thus, people with more pigmentation tend to get less sun burnt and to tan more than those who are lighter skinned.

Pheomelanin

Pheomelanin is a soluble yellowish alkaline pigment. It has a great potential to generate free radicals in response to UVR, which are capable of causing damage to DNA, and, in this manner, may contribute to the phototoxic effects of UVR. This explains why people with light skin, who have relatively high quantities of pheomelanin, are at greater risk of ultraviolet-induced epidermal damage, including neoplasms. A sulphur-containing pigment, derived from pheomelanin can be found in small quantities in red human hair, and is called trichrome.

Total skin melanin is the result of a combination of monomers of pheomelanin and eumelanin and the proportion between the two determines the final phenotypical expression of skin and hair colour.

After the complete synthesis of melanin, melanosomes filled with this pigment are injected in the interior of keratinocytes in the corresponding epidermal melanin unit through the dendritic extensions of melanocytes, so called cytocin activity. Melanosomes tend to spread through the cytoplasm, over the upper part of the nucleus, so as to protect it from ultraviolet radiations. It has been suggested that the pigment inside these cells also works as a scanner of photo-produced free radicals, always struggling to protect cell DNA.

Melanin has great affinity for DNA, and is capable of producing reactive oxygen species in response to ultraviolet radiation. In people with light skin, the greater incidence of melanomas seems to result not only from poor natural protection, but also from increased mutations promoting the formation of pheomelanin and/or melanin intermediates (Psora/ Sycosis/ Syphilis/ Causa occasionalis).

The main factors that regulate the quantity and quality of the melanin produced by melanocytes include UVR, α-MSH (α -type melanocyte stimulating hormone or melanocortin), ASP (AGOUTI signaling protein), and MC1-R gene.

The melanin pigmentation of human skin suffers intense hormonal control, called as melanocortin system. The melanocortin system consists of peptides with many forms of MSH (alpha, beta and gamma) and ACTH.

MC1-R gene is considered one of the main markers of susceptibility to malignant skin neoplasms, as gene variants are associated to an increased risk of melanoma and non-melanoma skin cancers.

Solar ephelis and lentigines are different types of pigmented lesions that present significant differences in their aetiologies, but gene variants of MC1-R are a necessary factor for the development of ephelis, while they play a less important role in the case of lentigines.

UVR and Pigmentation

The solar radiation spectrum is broad, ranging from cosmic rays to infrared radiation. Shorter wavelength radiations, up to 200 nm, do not reach the Earth, because they are absorbed by atmospheric oxygen and ozone. UVR and visible light are between 200 nm and 760 nm and form the photo-biological spectrum, with ultraviolet between 200 and 400 nm and visible light between 400 and 760 nm. Beyond this limit, up to 17000 nm is infrared, which is a heat inducer.

The acute effects of exposure to UVR are basically skin burn and/or tanning. After even one single exposure to UVR, size of melanocytes is increased, followed by an increase in tyrosinase activity (Causa occasionalis/ Psora). Repeated exposures to UVR lead to increase in the number of stage IV melanosomes transferred to keratinocytes, as well as an increase in the number of active melanocytes (Psora/ Sycosis).

Thus, UVR is a competent stimulant of skin pigmentation in humans and is responsible for the initiation of the tanning response. Many mechanisms may be involved including different signals acting both directly and indirectly in melanocytes. Indirect UVR action involves the release of keratinocyte mediators in the skin (Psora).

Ultraviolet- A Radiation (UVA)

Ultraviolet A (UVA) (320-400 nm) penetrates deeper in the skin, causes pigmentation and carcinogenic alterations, and is the main inducer de photosensitivity (Psora/ Sycosis/ Syphilis/ Causa occasionalis).

Ultraviolet-B Radiation (UVB)

Ultraviolet-B radiation (UVB) (290-320 nm) on human skin induces the production of α-MSH and ACTH in melanocytes and keratinocytes. α-MSH stimulates the activity of tyrosinase and thus melanin synthesis, and the synthesis of melanocytes through MC1-R. Moreover, the synthesis of many epidermal factors, including α-MSH, ACTH and endothelin-1, is increased by the exposure to UVR, suggesting an important influence of these mediators on the response of melanocytes to sun light. UVB is the chief cause of sun burn, causing erythema after a latency period of 2 to 7 hours. UVA causes erythema that appears later and may gradually become more intense (Psora/ Sycosis/ Syphilis/ Causa occasionalis).

Ultraviolet-C Radiation (UVC)

Ultraviolet C (UVC) (200-290 nm) is basically germicide; UVB causes erythema, pigmentation and especially alterations that induce skin cancer (Psora/ Sycosis/ Syphilis/ Causa occasionalis).

The interaction of hormones and UVR can be illustrated in melasma. UVR stimulates the production of melanocortin inside melanocytes and keratinocytes, which explains the involvement of this hormone in the pathogenesis of melasma that is basically characterized by increased epidermal melanization in melanocytic proliferation.

Clinical features

Melasma presents as macules (freckle-like spots) and larger flat brown patches. These are found on both sides of the face and have an irregular border. There are several distinct patterns.

- *Centrofacial pattern: forehead, cheeks, nose and upper lips*
- *Malar pattern: cheeks and nose*
- *Lateral cheek pattern*
- *Mandibular pattern: jawline*
- *Reddened or inflamed forms of melasma ,also called erythrosis pigmentosa faciei*
- *Poikiloderma of Civatte: reddened, photo-aging changes seen on the sides of the neck, mostly affecting patients older than 50 years*
- *Brachial type of melasma affecting shoulders and upper arms, also called acquired brachial cutaneous dyschromatosis.*

Types of Melasma

There is no harmony as to the clinical classification of melasma. Three patterns of facial melasma are recognized-

1- Central-facial

Affecting the central region of the forehead, mouth, lips, supra labial area, and chin

2- Malar

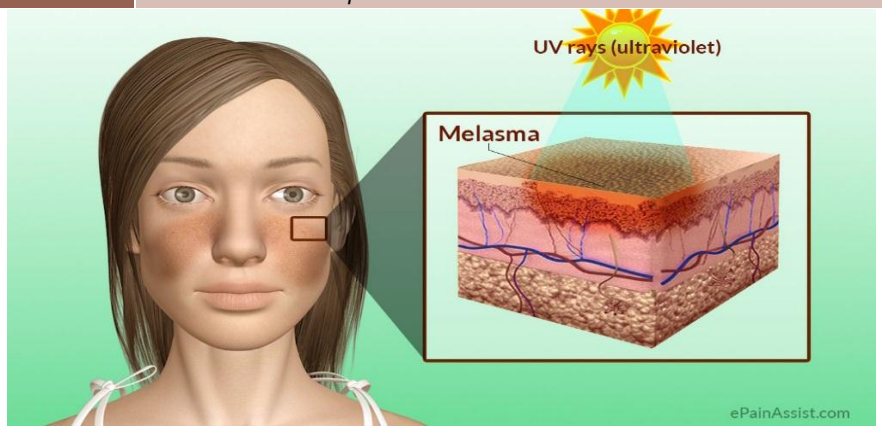
Affecting the zygomatic region

3- Mandibular

Less common

Melasma is sometimes divided into epidermal, dermal and mixed types. A Wood lamp that emits black light (UVA1) may be used to identify the depth of the pigment.

Type of melasma	Clinical features
Epidermal	• Well-defined border • Dark brown colour • Appears more obvious under black light • Responds well to treatment
Dermal	• Ill-defined border • Light brown or bluish in colour • Unchanged under black light • Responds poorly to treatment
Mixed	• The most common type • Combination of bluish, light and dark brown patches • Mixed pattern seen under black light • Partial improvement with treatment



Diagnosis

The characteristic appearance of melasma is self-diagnostic. Occasionally, skin biopsy may be performed to confirm the diagnosis. Histology varies with the type of melasma-

- *Melanin deposited in basal and suprabasal keratinocytes*
- *Highly dendritic deeply pigmented melanocytes*
- *Melanin in the dermis within melanophages*
- *Solar elastosis and elastic fibre fragmentation*

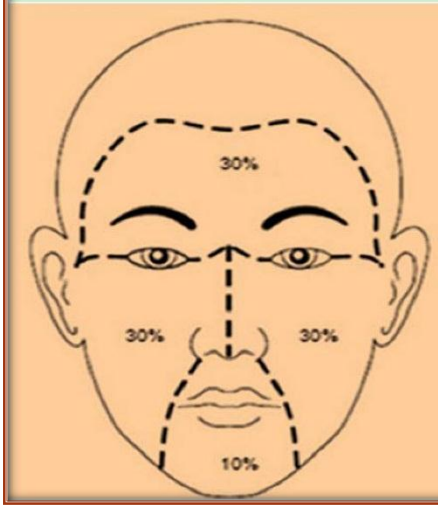
The evaluation of the expression of α-MSH and MC1-R in the epidermis with melasma lesions, compared to healthy perilesional skin, may permit estimating the role of MC1-R pathway in the physiopathogenesis of the disease.

The extent and severity of melasma can be described using the Melasma Area and Severity Index (MASI).

Melasma Area and Severity Index (MASI)

This tool is mainly used in research for measuring severity in patients who have melasma (chloasma, pregnancy mask). It would be useful in the measurement of the melasma before and after a treatment for example.

Modified MASI Scoring



Part affected	Score- Right	Score- Left	Total Score
Forehead	30%	30%	30%
Left and right Malar	30%	30%	60%
Chin	10%	10%	10%
Total	50%	50%	100%

0.3A(f)D(f)+0.3A(lm)D(lm)+0.3A(rm)D(rm)+0.1A(c)D(c)

Score 0-24

Area		Darkness	
0	No involvement	0	Absent
1	>10%	1	Slight
2	10-29%	2	Mild
3	30-49%	3	Marked
4	50-69%	4	Severe
5	70-89%		
6	90-100%		

- Its measurement relies on the measurement in 4 areas-
  - (F) forehead which accounts for 30% of the score
  - (RMR) right and (LMR) left malar region which each account for 30% of the score
  - (M) chin which accounts for 10% of the score
- The area (A) of melasma involvement is graded from 0 to 6-
  - 0 = no involvement
  - 1 = less than 10% involvement
  - 2 = 10% to 29% involvement
  - 3 = 30% to 49% involvement
  - 4 = 50% to 69% involvement
  - 5 = 70% to 89% involvement
  - 6 = 90% to 100% involvement
- The degree of pigmentation (P) and homogeneity (H) graded from 0 to 4-
  - 0 = absent
  - 1 = slight
  - 2 = mild
  - 3= marked
  - 4 = maximum
- MASI score = 0.3 A(F) [P(F) + H(F)] + 0.3 A(RMR) [P(RMR) + H(RMR)] + 0.3 A(LMR) [P(LMR) + H(LMR)] + 0.1 A(M) [P (M) + H (M)].

Differential Diagnosis

These may include-

- *Postinflammatory pigmentation*
- *Solar lentigines and other forms of lentigo*
- *Drug-induced pigmentation, as in minocycline or nonsteroidal antiinflammatory drugs*
- *Lichen planus*
- *Naevus of Ota and naevus of Hori*
- *Guttate hypomelanosis, characterized by prominent pale spots*

Treatment

Melasma can be very slow to react to treatment, especially if it has been present for a long time.

General measures

- *Topical therapy*
- *Stopping hormonal contraception*
- *Year-round life-long sun protection. Use of broad-spectrum very high protection factor (SPF 50+) sunscreen applied to the whole face every day. It should be reapplied every 2 hours if outdoors during the summer months. Alternatively or as well, use of a make-up that contains sunscreen. Wearing a broad-brimmed hat.*
- *Use of a mild cleanser and a light moisturiser if the skin is dry*
- *Cosmetic camouflage (make-up) is invaluable to mask the pigment*
- *Tyrosinase inhibitors are the mainstay of treatment. The aim is to prevent new pigment formation by inhibiting formation of melanin by the melanocytes.*
- *Ascorbic acid (vitamin C) also acts through copper to inhibit pigment production. It is well tolerated but highly unstable, so is usually combined with other agents.*
- *Superficial or epidermal pigment can be peeled off. Peeling can also allow tyrosinase inhibitors to penetrate more effectively. These must be done carefully as peels may also induce postinflammatory pigmentation.*

Devices used to treat melasma

The ideal treatment for melasma would be to destroy the pigment, while leaving the cells intact. Fractional lasers, Q-switched Nd:YAG lasers and intense pulsed light (IPL) appear to be the most suitable options. Several treatments may be necessary and postinflammatory hyperpigmentation may complicate recovery.

Carbon dioxide or erbium:YAG resurfacing lasers, pigment lasers (Q-switched ruby and Alexandrite devices) and mechanical dermabrasion and microdermabrasion should be used with caution in the treatment of melasma. Unfortunately, even in those that get a good result from treatment, pigmentation may reappear on exposure to summer sun and/or because of hormonal factors. New topical and oral agents are being studied and offer hope for effective treatments in the future.

Homoeopathic Treatment

CHLOASMA IN GENERAL- ant-c. Arg-n. Ars. aur. cadm-s. card-m. CAUL. cob. coch. Con. cur. ferr. guar. kali-p. laur. LYC. merc-i-r. Nat-hp. nit-ac. nux-v. paull. petr. plb. raph. rob. SEP. sul-ac. Sulph. syph. thuj.
FACE – CHLOASMA ant-c. Ars. cadm-s. card-m. caul. coch. guar. kali-p. lyc. merc-i-r. nux-v. plb. raph. rob. sep. sul-ac. Sulph. syph.
FACE - CHLOASMA - pregnancy agg.; during caul. Con. ferr. nit-ac. SEP.
FACE - CHLOASMA - sun and wind agg.; exposure to cadm-s.
SKIN - DISCOLORATION – chloasma Arg-n. aur. cadm-s. card-m. CAUL. cob. cur. guar. laur. LYC. Nat-hp. nux-v. paull. petr. plb. raph. rob. SEP. sulph. thuj.

Bibliography


Chapter 197. Melasma The Color Atlas of Family Medicine, 2e


Chapter 6. Normal Pregnancy and Prenatal Care > a. Chloasma— CURRENT Diagnosis & Treatment: Obstetrics & Gynecology, 11e .... Chloasma is exacerbated by sunlight. ...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