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An Overview of Tooth Discoloration: Extrinsic, Intrinsic and Internalized Stains

Abstract: The causes of tooth discoloration are varied and complex but are usually classified as being either intrinsic, extrinsic or internalized in nature. Dietary chromogens and other external elements deposit on the tooth surface or within the pellicle layer either directly or indirectly to form extrinsic discoloration.

Stains within the dentine or intrinsic discoloration often results from systemic or pulpal origin, while internalized stains are the result of extrinsic stains entering the dentine via tooth defects such as cracks on the tooth surface.

Clinical Relevance: Tooth discoloration creates a wide range of cosmetic problems and the dental profession and the public expend considerable amounts of time and money in attempts to improve the appearance of discoloured teeth. The methods available to manage discoloured teeth range from removal of surface stain, bleaching or tooth whitening techniques and operative techniques to camouflage the underlying discoloration, such as veneers and crowns. It is also hoped that the reader will be able to decide when and how to treat various types of discoloration by understanding their mechanism of development.

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The causes of tooth discoloration are varied and complex but are usually classified as being either intrinsic or extrinsic in nature. Extrinsic discoloration arises when external chromogens are deposited on the tooth surface or within the pellicle layer. Intrinsic discoloration occurs when the chromogens are deposited within the bulk of the tooth, usually in the dentine, and are often of systemic or pulpal origin.[1,2] A third category of 'stain internalization' has recently been described to include those circumstances where extrinsic stain enters the tooth through defects in the tooth structure (Table 1).[1]

Figure 1. Amelogenesis imperfecta. (Courtesy of Dr Peter Crawford.)

Figure 2. Dentinogenesis imperfecta. (Courtesy of Dr Peter Crawford.)

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Natural colour of teeth

Teeth are made up of many colours with a natural gradation from the darker gingival to the lighter incisal third of the tooth. This variation is affected by the thickness and translucency of enamel and dentine as well as the reflectance of different colours. Typically, canine teeth are darker than central and lateral incisors and teeth become darker with age, whereas lighter teeth are common in younger people,

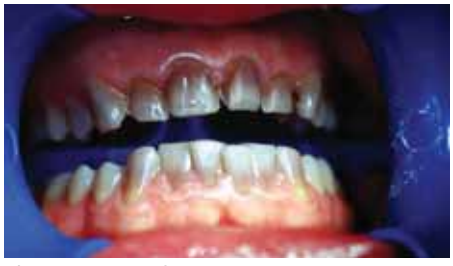

Figure 3. Tetracycline staining.

especially in the primary dentition.

The colour of teeth is primarily determined by the dentine but is influenced by the colour, translucency and varying degrees of calcification of enamel, as well as its thickness, notably greatest at the occlusal or incisal edge. The normal colour of teeth is determined by the blue, green and pink tints of enamel and reinforced by the yellow through to brown shades of dentine beneath.[3]

Visual assessment of tooth discoloration and the perception of colour are affected by individual interpretation, duration of viewing and difficulty in quantifying. The viewing conditions are also very important, with variables such as lighting, surroundings, operator age, fatigue, skin tone or make up, hydration of the tooth and the angle from which the tooth is viewed all contributing to the final perception of the colour.

Classification of tooth discoloration

The appearance of teeth depends on their absorptive or reflective properties of light and is influenced by all the structures that make up the tooth, including the enamel, dentine and pulp. Any changes to these structures during formation or throughout development and post eruption (3 months *in utero* to 20 years) can cause a change in the light transmission properties and hence discoloration.

Intrinsic discoloration

Intrinsic discoloration occurs following a change to the structural composition or thickness of the dentinal hard tissues during tooth development.

This classification can be further subdivided into:

- Metabolic causes
- Inherited causes
- Iatrogenic causes
- Traumatic causes
- Idiopathic causes
- Ageing causes.

DISCOLORATION	COLOUR
EXTRINSIC	
Direct stains	
Tea coffee and other foods	Brown to black
Cigarettes/cigars	Yellow/brown to black
Plaque/poor oral hygiene/chromogenic bacteria	Yellow/brown/green
Indirect stains	
Polyvalent metal salts and Cationic antiseptics, eg chlorhexidine	Black and brown to black
Copper salts in mouthrinses	Green
Potassium permanganate in mouthrinses	Violet to black
INTRINSIC	
Metabolic causes	
eg Congenital erythropoietic porphyria	Purple/brown
Inherited causes	
Amelogenesis imperfecta	Yellow-brown to dark yellow affecting both dentitions
Dentinogenesis imperfecta	Blue-brown (opalescent)
Iatrogenic causes	
Tetracycline	Banding appearance Classically yellow, brown, blue, black or grey
Fluorosis	White, yellow, grey or black
Traumatic causes	
Enamel hypoplasia	Yellow-brown or white subsurface decalcification following trauma/ infection
Pulpal haemorrhage products	Grey-brown to black
Internal resorption	Pink
Idiopathic causes	
MIH	White to yellow or brownish defect
Ageing causes	Yellow
INTERNALIZED	
Caries	White spot lesion to orange-brown to black arrested decay
Restorations	Brown, grey, black

Table 1. Causes of tooth discoloration.

Metabolic causes of discoloration

There are a number of metabolic disorders such as alkaptonuria, congenital erthropoietic porphyria and congenital hyperbilirubinaemia that cause

tooth discoloration. Alkaptonuria is an inborn error of metabolism that results in a brown discoloration of the permanent dentition[4] while congenital erythropoietic porphyria, a rare, recessive, autosomal metabolic disorder gives a red/purple-brown discoloration of teeth.[5] Congenital hyperbilirubinaemia is characterized by yellow-green discoloration resulting from the deposition of bile pigments in calcifying dental hard tissues, particularly at the neonatal line as a result of massive haemolysis in rhesus incompatibility.

Inherited causes of discoloration

Amelogenesis imperfecta (Figure 1) is a hereditary condition in which the mineralization or matrix of enamel formation is disturbed. There are 14 different subtypes based on clinical appearance, with the majority being inherited as an autosomal dominant or x-linked trait with varying degrees of expressivity.[6-8] The appearance can be severe with hypoplastic thin enamel yellow to yellow-brown in colour, or can be quite mild hypomineralized 'snow-capped' enamel, depending on the type of amelogenesis imperfecta present. The colour of the teeth is presumed to reflect the degree of hypomineralization of the enamel – the darker the colour the more severe the degree of hypomineralization.[9]

Dentine defects can be inherited or caused by environmental factors,[10] genetically determined defects may occur in isolation or associated with a systemic disorder.

Dentinogenesis imperfecta (Figure 2) is an inherited disorder of dentine which may or may not be associated with osteogenesis imperfecta.[9] Dentinogenesis imperfecta type I is associated with osteogenesis imperfecta (mixed connective tissue disorder of type I collagen) and is characterized by opalescent primary teeth, especially when the condition is the result of a dominant inheritance pattern. Dentinogenesis imperfecta type II or hereditary opalescent dentine may be more severe in the primary rather than the secondary dentition; the pulp chambers often become obliterated and the dentine undergoes rapid wear once the enamel has chipped away. Clinically, the appearance is an amber, grey to purple-bluish discoloration or opalescence thought to be the result of absorption of chromogens into the porous dentine after exposure of the dentine. This condition is clearly demonstrated by opalescence on transillumination.

Unlike type II, dentinogenesis imperfecta type I shows enamel that is much less prone to fracture and the dentine seldom obliterates pulp chambers, hence, radiographic examination can differentiate between the two types. A third type of dentinogenesis imperfecta has been described,[11] similar in appearance to types I and II but with the radiographic appearance of 'shell teeth' with multiple pulpal exposures in the primary dentition.

There is some controversy as to whether dentinal dysplasias are a separate entity from dentinogenesis imperfecta.[9] The primary and secondary dentitions in type I dentinal dysplasia are of normal shape and form but have an amber translucency. The pulp chambers in the primary dentition are frequently obliterated while only crescentic pulpal remnants are found parallel to the cemento-enamel junction of the secondary dentition. Type II dentinal dysplasias are thought to involve thistle-shaped pulp chambers and pulp stones with a brown tooth discoloration.[12]

Iatrogenic causes

Tetracycline staining (Figure 3) results from systemic administration of the drug and its subsequent chelation to form complexes with calcium ions on the surface of hydroxy apatite crystals, primarily within dentine, although enamel is also affected.[13] Tetracycline should be avoided for expectant and breast-feeding mothers and in children up to the age of 12 years to avoid discoloration of the developing teeth. The discoloration produced depends on the type of tetracycline used, the dosage and period of time it was taken for, as well as the age at the time of administration. Generally, the affected teeth tend to be yellow or brown-grey in colour and the appearance is worse on eruption and diminishes with time. The teeth are then susceptible to colour changes by photo oxidation (exposure to light), especially the anterior teeth that turn brown, but with continued exposure over a period of time lighten. Various analogues of tetracycline produce different colour changes, for example, chloretetracycline produces a slate grey colour and a creamy discoloration is produced by oxytetracycline.[14,15]

Tetracycline staining has also been classified according to the extent, degree and location of the tetracycline involvement:[16]

- Degree I: there is minimal expression of tetracycline stain uniformly confined to the incisal three-quarters of the crown and it is light yellow in colour.
- Degree II: there is more variability in staining ranging from a highly uniform deep yellow to a grey banded discoloration in which a distinctive difference in discoloration is noted between the cervical region and the incisal four-fifths of the crown.
- Degree III: there is very dark blue or grey uniform discoloration.

Post-eruptive staining with minocycline, used for acne treatments in adolescents and adults, has been described resulting from chelation with iron to form insoluble complexes,[17] or by the formation of complexes of the drug with secondary dentine.[18]

Fluorosis (Figure 4) may be the result of naturally occurring water supplies or from fluoride in mouthrinses, tablets or toothpastes as a supplement. This type of staining is most often confined to the enamel, varying from areas of flecking to diffuse opaque mottling superimposed on to a chalky white or dark brown/black appearance. The dark discoloration is

Figure 4. Dental fluorosis. (Courtesy of Dr Peter Crawford.)

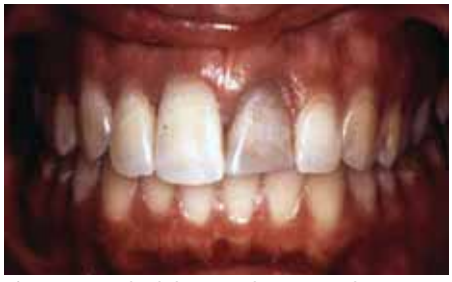

Figure 5. Pulpal haemorrhagic products in a non-vital upper left central incisor. (Courtesy of Professor Martin Addy.)

Figure 6. Internal resorption: pink spot lesion on the upper right central incisor.

Figure 7. Enamel hypoplasia affecting the upper left lateral incisor. (Courtesy of Dr Peter Crawford.)

thought to be post-eruptive by a process of internalization of extrinsic stain into the porous enamel.[19] The severity of the discoloration is related to the age and dose administered and can affect both the primary and secondary dentitions in endemic causes of fluorosis.

Traumatic causes of discoloration

One of the most common causes of tooth discoloration seen in every day general practice is that resulting from pulpal haemorrhagic products following trauma (Figure 5). The major cause of the discoloration is thought to be the accumulation of the haemoglobin molecule or haematin molecules in the traumatized tooth and their penetration into the dentine determines the severity of the discoloration.

Root resorption following trauma often presents as a pink spot lesion at the cemento-enamel junction in an otherwise symptomless tooth (Figure 6). The resorption always begins at the root surface but may be internal, being of pulpal origin or external of periodontal origin.

Enamel hypoplasia (Figure 7) may be the result of disturbance of the developing tooth germ following trauma or infection of the deciduous tooth giving rise to a localized enamel defect. Pitting or grooving may predispose to later internalization of external chromogens. Generalized hypoplasia may result from any disturbance of the developing tooth germ by many different foetal or maternal conditions, such as rubella infection or even drug intake during pregnancy. The defect is usually directly related to the degree of the systemic upset and is chronologically traced back to the time of the upset.

Dentine hypercalcification may result following trauma temporarily disturbing the blood supply of a tooth and affecting the odontoblasts, giving rise to excessive irregular dentine deposition in the pulp chamber and canal walls. The tooth gradually decreases in translucency and becomes yellow or yellow/brown in colour but is still vital.[20]

Idiopathic causes of discoloration

Molar incisor hypomineralization (MIH) is a condition of unknown aetiology characterized by severe hypomineralized enamel affecting incisors and permanent first molars (Figure 8).[21] The appearance of the hypomineralized enamel is asymmetrical, affecting one molar severely while leaving the contra-lateral molar relatively unaffected or only with minor subsurface defects.[22] The incisors also show asymmetry but not usually with loss of enamel substance. The enamel defects can vary from white to yellow or brownish areas but they always show a sharp demarcation between sound and affected enamel.[23] The nature of the enamel is porous and brittle, breaking down shortly after eruption under masticatory forces, often resembling enamel hypoplasia, but distinguished by having irregular borders with normal enamel as apposed to smooth borders with hypoplastic lesions.[23]

The suggestions as to possible aetiologies, including environmental changes during a limited time period, infections during early childhood, dioxin in breast milk and genetic factors, have yet to be eliminated from the possible causes.

Ageing causes of discoloration

The natural darkening and yellowing of teeth with age and the change in their light transmission properties is due to a combination of factors involving both enamel and dentine. The enamel undergoes both thinning and

a

b

Figure 8. MIH: (**a**) enamel defects seen as white-yellow discoloration on the lower central and lateral incisors. Upper incisors are not affected but seen with plaque accumulation; (**b**) affected first molars with typical asymmetry – severe hypomineralization and breakdown of the upper right molar and relative lack of an effect on the upper left molar. (Courtesy of Dr Peter Crawford.)

Figure 9. Extrinsic discoloration in a pipe smoker. (Courtesy of Professor Martin Addy.)

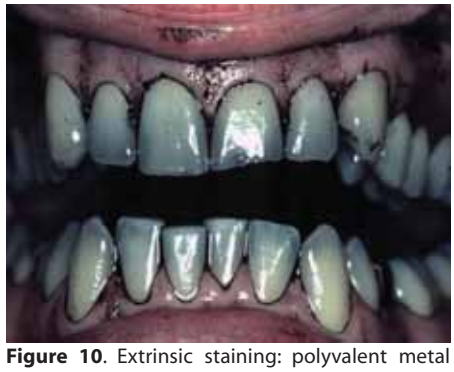

Figure 10. Extrinsic staining: polyvalent metal salts. (Courtesy of Professor Martin Addy.)

Figure 11. Extrinsic discoloration: chlorhexidine mouthrinse. (Courtesy of Professor Martin Addy.)

textural changes,[24] while the deposition of secondary and tertiary dentine and pulp stones all contribute to the darkening process of ageing.

Extrinsic discoloration

External staining may be divided into two main categories: direct staining by compounds incorporated into the pellicle layer and producing a stain as a result of the basic colour of the chromogens, and indirect staining where there is chemical interaction at the tooth surface with another compound that produces the stain.

Direct staining

Direct staining chromogens derived from dietary sources such as tea and coffee are taken up into the pellicle and their natural colour imparts the stain on to the tooth. Smoking or chewing tobacco, medicines, spices, vegetables and red wine are also known to cause direct staining. It is the polyphenolic compounds found in food that are thought to give rise to the colour of the stain.[25] The actual mechanism of staining is not understood, but certainly involves pellicle constituents. Naked enamel does not take up chromogens easily, pellicle proteins are often cited as reacting with chromogens but how specific this reaction is has not been clarified and some degree of non-specificity is probable: the chromogen merely being incorporated within the pellicle layer much as a sponge can soak up and hold fluids. For dentine, chromogens may be absorbed into the tissue itself as dentine is more porous than enamel, both in respect of intertubular dentine and the tubules themselves.[26]

Traditionally, extrinsic tooth discoloration has been classified according to its origin and whether it is metallic or non-metallic.[27]

Non-metallic extrinsic stains caused by beverages, tobacco (Figure 9), mouthrinses and other medicines are adsorbed on to the tooth surface by incorporating into the plaque or acquired pellicle .Chromogenic bacteria have also been implicated in extrinsic staining in children who have poor oral hygiene (green and orange stains) and in those that have good oral hygiene (black/brown stain), but the mechanism involved has not been proven.[2]

Indirect staining

Indirect stains are associated with cationic antiseptics and metal salts that are either colourless or a different colour to the stain produced as a result of a chemical interaction with another compound.

Polyvalent metal salts (Figure 10) are known to be associated with extrinsic staining, such as the black discoloration seen in people using iron supplements and in iron foundry workers occupationally exposed to these metal salts. Other examples include the green discoloration resulting from the use of mouthrinses containing copper salts, or violet to black colour in potassium permanganate containing mouthrinses.[28]

Cationic antiseptics such as chlorhexidine, hexetidine, cetylpyridinium chloride (CPC) and other mouthrinses also cause staining after prolonged use. Chlorhexidine, for example, produces the brown to black discoloration (Figure 11) seen around the labial and lingual surfaces of anterior teeth after only about a week to ten days' use.

The mechanism of staining has attracted a lot of interest from the dental profession and is thought to be due to the precipitation of anionic dietary chromogens such as polyphenols on to adsorbed cationic antiseptics or polyvalent metal salts on the tooth surface.

Internalized discoloration

Extrinsic stains are taken up into the enamel or dentine via a developmental defect or as a result of trauma. The incorporation of extrinsic stain into the porous tooth structure of developmental defects has already been discussed under the intrinsic staining section above.

Acquired defects of teeth resulting from function and para-function, dentinal caries and restorative materials can all lead to tooth discoloration directly or indirectly. Consider the following:

- Toothwear and gingival recession: the loss of enamel and dentine by erosion, abrasion and attrition results in potential exposure of dentine to the extrinsic chromogens. The exposure of dentine or loss of enamel gives rise to darker looking teeth as more of the yellow colour of dentine is apparent. Cracks as a result of trauma to the enamel or gingival recession and exposure of dentine predispose the teeth to extrinsic stain internalization.
- Dentinal caries: the progression of the

carious lesion is usually associated with changes in colour ranging from the initial white spot lesion to the black arrested lesion which picks up stain from an extrinsic source.[29]

■ Restorative materials: the classic grey to black discoloration seen around old amalgam restorations is thought to be caused by the migration of tin into the dentinal tubules.[30] Eugenol-containing medicaments cause an orange/yellow stain and silver points in root canals give a grey or pink appearance to a root-treated tooth.

Conclusion

Tooth discoloration has a multifactorial aetiology and the dental practitioner must be aware of the various mechanisms of staining in order to manage the patient with tooth discoloration. The management may involve a simple prophylaxis for extrinsic staining, or a combination of bleaching and microabrasion techniques for various intrinsic stains or even more invasive veneers and crowns. Internalized stains may be managed by bleaching, but more usually require restorative procedures ranging from the placement of simple composites to veneers and crowns.

In addition, the practitioner will be able to help his/her patients in limiting the incidence of staining by giving appropriate dietary advice, especially when using cationic mouthrinses.

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