DENTAL STAINS

Pigmented deposits on the tooth surface are called dental stains. The appearance of the dentition is of concern to a large number of people seeking dental treatment and the color of the teeth is of particular cosmetic importance. There has been a recent increase in interest in the treatment of tooth staining and discolorations as shown by a large number of tooth whitening agents appearing in the market. Some of these agents are sold as 'over-the-counter' products and have no professional involvement in their application. The correct diagnosis for the cause of discoloration is important as, invariably, it has a profound effect on treatment outcomes.

❖ Color and color perception:

A basic understanding of the elements of tooth color is important for many aspects of restorative dentistry. Teeth are typically composed of a number of colors and a gradation of color occurs in an individual tooth from the gingival margin to the incisal edge of the tooth. The gingival margin often has a darker appearance because of the close approximation of the dentine below the enamel. In most people canine teeth are darker than central and lateral incisors and younger people characteristically have lighter teeth, particularly in the primary dentition. Teeth become darker as a physiological age change; this may be partly caused by the laying down of secondary dentine, incorporation of extrinsic stains and gradual wear of enamel allowing a greater influence on color of the underlying dentine.

❖ Classification of tooth discoloration:

The coronal portion of the tooth consists of enamel, dentine and pulp. Any change to these structures is likely to cause an alteration in the outward appearance of the tooth caused by its light transmitting and reflecting properties. The appearance of tooth color is dependent on the quality of the reflected light and is also, as a consequence, dependent on the incident light.

Historically, tooth discoloration has been classified according to the location of the stain, which may be either intrinsic or extrinsic. It may also be of merit to consider a further category of internalized stain or discoloration.

➢ Intrinsic discoloration

Intrinsic discoloration occurs following a change to the structural composition or thickness of the dental hard tissues. The normal color of teeth is determined by the blue, green and pink tints of the enamel and is reinforced by the yellow through to brown shades of dentine beneath.

A number of metabolic diseases and systemic factors are known to affect the developing dentition and cause discoloration as a consequence. Local factors such as injury are also recognized.

1. Alkaptonuria
2. Congenital erythropoietic porphyria
3. Congenital hyperbilirubinemia
4. Amelogenesis imperfecta
5. Dentinogenesis imperfecta
6. Dentinal dysplasia
7. Tetracycline staining
8. Fluorosis
9. Enamel hypoplasia
10. Pulpal hemorrhagic products
11. Root resorption
12. Ageing

➢ Extrinsic discoloration:

Extrinsic discoloration is outside the tooth substance and lies on the tooth surface or in the acquired pellicle. The origin of the stain may be:

1. Metallic
2. Non-metallic

➢ Internalized discoloration:

Internalized discoloration is the incorporation of extrinsic stain within the tooth substance following dental development. It occurs in enamel defects and in the porous surface of exposed dentine. The routes by which pigments may become internalized are:

1. Developmental defects.
2. Acquired defects:

a) Tooth wear and gingival recession

b) Dental caries

c) Restorative materials

❖ The mechanisms of tooth discoloration

➢ Intrinsic tooth discoloration

The formation of intrinsically discolored teeth occurs during tooth development and results in an alteration of the light transmitting properties of the tooth structure. There are a number of metabolic disorders which affect the dentition during its formation, unlike the inherited disorders in which only the hard tissue forming at the time may be involved. These disorders will now be discussed individually.

1. **Alkaptonuria**: This inborn error of metabolism results in incomplete oxidation of tyrosine and phenylalanine, which promotes the buildup or increase level of homogentisic acid. This affects the permanent dentition by causing a brown discoloration.

2. **Congenital erythropoietic porphyria:** This is a rare, recessive, autosomal, metabolic disorder in which there is an error in porphyrin metabolism leading to the accumulation of porphyrins in bone marrow, red blood cells, urine, faeces and teeth. A red-brown discoloration of the teeth is the result and the affected teeth show a red fluorescence under ultra-violet light. King George III was said to have

suffered with acute intermittent porphyria but with the later onset of this disorder his teeth are unlikely to have been affected.

3. **Congenital hyperbilirubinemia:** The breakdown products of hemolysis will cause a yellow-green discoloration. Mild neonatal jaundice is relatively common, but in rhesus incompatibility massive hemolysis will lead to deposition of bile pigments in the calcifying dental hard tissues, particularly at the neonatal line.

4. **Amelogenesis imperfecta:** In this hereditary condition, enamel formation is disturbed with regard to mineralization or matrix formation and is classified accordingly. There are 14 different subtypes; the majority is inherited as an autosomal dominant or x-linked trait with varying degrees of expressivity. The appearance depends upon the type of amelogenesis imperfecta, varying from the relatively mild hypomature 'snow-capped' enamel to the more severe hereditary hypoplasia with thin, hard enamel which has a yellow to yellow-brown appearance.

5. **Systemic syndromes:** Defects in enamel formation may also occur in a number of systemically involved clinical syndromes such as vitamin D dependent rickets, epidermolysis bullosa and pseudohypoparathyroidism. It had been reported areas of hypoplastic enamel, irregularities in the region of the amelo-dentinal and the cemento-dentinal junctions in Ehlers-Danlos Syndrome. In epidermolysis bullosa there is pitting of the enamel possibly caused by vesiculation of the ameloblast layer. However, the effect of these conditions depends on disease activity during the development of the dentition and is usually a minor element.

6. **Dentinogenesis imperfecta:** Dentine defects may occur genetically or through environmental influences. The genetically determined dentine defects may be in isolation or associated with a systemic disorder. The main condition related to the dentine alone is Dentinogenesis imperfecta II (hereditary opalescent dentine). Both dentitions are affected, the primary dentition usually more severely so. The

teeth are usually bluish or brown in color, and demonstrate opalescence on

transillumination. The pulp chambers often become obliterated and the dentine undergoes rapid wear, once the enamel has chipped away, to expose the amelo-dentinal junction. Once the dentine is exposed, teeth rapidly show brown discoloration, presumably by absorption of chromogens into the porous dentine. Dentinogenesis imperfecta I (associated with osteogenesis imperfecta, a mixed connective tissue disorder of type I collagen) may show bone fragility and deformity with blue sclera, lax joints and opalescent dentine. The inheritance may be dominant or recessive, the recessive being more severe and often fatal in early life. Opalescent teeth are more common in the dominant inheritance pattern, the primary teeth bear a strong resemblance to the teeth in Dentinogenesis imperfecta type I whereas the appearance of the secondary dentition is much more variable. The enamel is much less prone to fracture, the pulp chamber is seldom occluded by dentine (this may help to radiographically differentiate between types I and II), and the overall prognosis for the dentition is improved.

A third type of Dentinogenesis imperfecta (type III, brandy wine isolate hereditary opalescent dentine) was described by Wiktop. In this condition, the teeth may be outwardly similar to both types I and II of Dentinogenesis imperfecta; however, multiple pulpal exposures occur in the primary dentition. Radiographically, the teeth may take on the appearance of 'shell teeth' as dentine production ceases after the mantle layer has formed. This type of Dentinogenesis imperfecta is thought to be related more closely to type II.

7. Dentinal dysplasia:

Shields reclassified the inherited dentine defects in a review of the literature in 1973 and introduced the term dentinal dysplasia. This reclassification allows separation of the inherited types of dentine defects from Dentinogenesis imperfecta, with which they are often confused.

In type I dentine dysplasia the primary and secondary dentition are of normal shape and form but may have an amber translucency. Radiographically the teeth have short roots with conical apical constrictions. The pulp is commonly



obliterated in the primary dentition, leaving only a crescentic pulpal remnant in the adult dentition parallel to the cemento-enamel junction. There are characteristic periapical radiolucencies in many, otherwise disease free teeth. The condition is inherited as an autosomal dominant trait.

Type II dentine dysplasia is described through a small number of case reports in Shields review, the main characteristic is that of a thistle-shaped pulp chamber with numerous pulp stones and the teeth had brown discoloration.

8. Tetracycline staining:

Systemic administration of tetracyclines during development is associated with deposition of tetracycline within bone and the dental hard tissues. Tetracycline and its homologues have the ability to form complexes with calcium ions on the surface of hydroxy apatite crystals within bone and dental tissues. Dentine has been shown to be more heavily stained than enamel. Tetracycline is able to cross the placental barrier and should be avoided from 29 weeks in uterus until full term to prevent incorporation into the dental tissues. Since the permanent teeth continue to develop in the infant and young child until 12 years of age, tetracycline administration should be avoided in children below this age and in breast-feeding and expectant mothers. The color changes involved depend upon the precise medication used, the dosage and the period of time over which the medication was given. Teeth affected by tetracycline have a yellowish or brown-grey appearance which is worse on eruption and diminishes with time. Exposure to light changes the color to brown, the anterior teeth are particularly susceptible to light induced color changes. The various analogues of tetracycline produce different color changes, for instance chlortetracycline produces a slate grey color and oxy-tetracycline causes a creamy discoloration. Since tetracycline fluoresces under ultraviolet light so do affected teeth, giving off a bright yellow color. There have been recent reports of adults experiencing change in tooth color with the use of long term tetracycline therapy.

Minocycline, a synthetic compound of tetracycline antibiotics, is also implicated in causing discoloration in an adult patient, following its long term use for treatment of acne. This phenomenon was described in a single case report in the literature by Cale et al. When the appearance of the dentine had altered following the long-term use of minocycline for acne, it was postulated that calcium-minocycline complexes were deposited in the dentine.

9. Fluorosis:

The association between fluoride intake and its effect on enamel was noted by Dean as long ago as 1932.This may arise endemically from naturally occurring water supplies or from fluoride delivered in mouth rinses, tablets or toothpastes as a supplement. The severity is related to age and dose, with the primary and secondary dentitions both being affected in endemic fluorosis. The enamel is often affected and may vary from areas of flecking to diffuse opacious mottling, whilst the color of the enamel ranges from chalky white to a dark brown/black appearance. The brown/black discoloration is post-eruptive and probably caused by the internalization of extrinsic stain into the porous enamel.

These features are often described as being pathognomonic of fluorosis, but care should be taken not to confuse the condition with the hypomaturation type of amelogenesis imperfecta. Fluoride only causes fluorosis in concentrations of greater than 1 ppm in drinking water and is not distinguishable, clinically or histologically, from any other type of hypo plastic or hypo mineralized enamel.

10. Enamel hypoplasia:

This condition may be localized or generalized. The most common localized cause of enamel hypoplasia is likely to occur following trauma or infection in the primary dentition. Such localized damage to the tooth-germ will often produce a hypoplastic enamel defect, which can be related chronologically to the injury.

Disturbance of the developing tooth germ may occur in a large number of fetal or maternal conditions (eg: maternal vitamin D deficiencies, rubella infection, drug

intake during pregnancy and in pediatric hypocalcemia conditions). Such defects will be chronologically laid down in the teeth depending on the state of development at the time of interference; the effect is directly related to the degree of systemic upset. There may be pitting or grooving which predisposes to extrinsic staining of the enamel in the region of tooth disturbed, often then becoming internalized.

11. Pulpal hemorrhagic products:

The discoloration of teeth following severe trauma was considered to be caused by pulpal hemorrhage. Hemolysis of the red blood cells would follow and release the hem group to combine with the putrefying pulpal tissue to form black iron sulphide. Grossman asserted in 1943 that the depth of dentinal penetration determines the degree of discoloration; there was little if any scientific investigation of this hypothesis. In vitro studies have recently shown that the major cause of discoloration of non-infected traumatized teeth is the accumulation of the hemoglobin molecule or other hematin molecules. In the absence of infection, the release of iron from the protoporphyrin ring is unlikely. This greater understanding of the nature of tooth staining following trauma to teeth may be of importance if the manufacture of bleaching agents, with specific activity, becomes possible. For instance, with further analysis it may become possible to develop a bleaching agent for use on teeth stained specifically by blood pigments. Incidentally, it has been shown that the pinkish hue seen initially after trauma may disappear in 2 to 3 months if the tooth becomes revascularized.

12. Root resorption:

Root resorption is often clinically asymptomatic; however, occasionally the initial presenting feature is a pink appearance at the amelo-cemental junction. Root resorption always begins at the root surface, either from the pulpal or periodontal aspect, as internal or external root resorption respectively. It can be difficult to locate a resorptive cavity on radiograph until it reaches a certain size.



13. Aging:

The natural laying down of secondary dentine affects the light-transmitting properties of teeth resulting in a gradual darkening of teeth with age.

❖ Extrinsic discoloration:

The causes of extrinsic staining can be divided into two categories; those compounds which are incorporated into the pellicle and produce a stain as a result of their basic color, and those which lead to staining caused by chemical interaction at the tooth surface.

Direct staining has a multi-factorial etiology with chromogens derived from dietary sources or habitually placed in the mouth. These organic chromogens are taken up by the pellicle and the color imparted is determined by the natural color of the chromogen. Tobacco smoking and chewing are known to cause staining, as are particular beverages such as tea and coffee. The color seen on the tooth is thought to be derived from polyphenolic compounds which provide the color in food.

Indirect extrinsic tooth staining is associated with cationic antiseptics and metal salts. The agent is without color or a different color from the stain produced on the tooth surface. Extrinsic tooth discoloration has usually been classified according to its origin, whether metallic or non-metallic.

- **Non-metallic stains:** The non-metallic extrinsic stains are adsorbed onto tooth surface deposits such as plaque or the acquired pellicle. The possible etiological agents include dietary components, beverages, tobacco, mouth rinses and other medicaments. Chromogenic bacteria have been cited in children. Particular colors of staining are said to be associated with certain mouths, for instance, green stain caused by penicillium and Aspergillus species, orange in children with poor oral hygiene and black/brown stains in children with good oral hygiene and low caries caused by Actinomyces species.

The most convincing evidence for the extrinsic method of tooth staining comes from the differing amount of stain found in a comparison of smokers and non-smokers. The staining effect of prolonged rinsing with chlorhexidine mouth rinses and quaternary ammonium compounds used in mouth rinses is of considerable interest to the dental profession.

- **Metallic stains:**

Extrinsic staining of teeth may be associated with occupational exposure to metallic salts and with a number of medicines containing metal salts. The characteristic black staining of teeth in people using iron supplements and iron foundry workers is well documented. Copper causes a green stain in mouth rinses containing copper salts and in workers in contact with the metal in industrial circumstances. A number of other metals have associated colors such as potassium permangenate producing a violet to black color when used in mouth rinses; silver nitrate salt used in dentistry causes a grey color, and stannous fluoride causes a golden brown discoloration. It was previously thought that the mechanism of stain production was related to the production of the sulphide salt of the particular metal involved. This is perhaps not surprising since the extrinsic stain coincided with the color of the sulphide of the metal concerned. However, those proposing the hypothesis appeared not to consider the complexity of the chemical process necessary to produce a metal sulphide.

Chlorhexidine or iron sulphate followed by tea rinse produced immediately the characteristic brown and black discoloration of the teeth and tongue reported for chlorhexidine and iron respectively.

- **Internalized discoloration:**

The stains taken up into the body of enamel or dentine are the same as that causing extrinsic tooth discoloration, including in particular dietary chromogens and the by-products of tobacco smoking. Dental defects permitting the entry of chromogenic material can be classified under the headings of 'developmental and acquired'.

1. Developmental defects: The most important defects are considered under the 'intrinsic tooth discoloration' section of this review. As described these developmental defects create their own color change in the tooth caused by influences on light transmission through the dentine and enamel. Post-eruptively, however, either caused by increased enamel porosity, or the presence of enamel defects, extrinsic stains can penetrate into the enamel. Such examples would include fluorosis and other enamel conditions resulting in enamel hypoplasia or hypocalcification. Alternatively, developmental defects may expose dentine either directly or later caused by early loss of enamel as in dentinogenesis imperfecta. Chromogens are then able to enter the dentine directly or facilitated almost certainly by the tubule system.

2. Acquired defects: Wear and tear and disease of the teeth and supporting tissues occur throughout life, all of which can lead directly or indirectly to tooth discoloration. Additionally, repairs on restorations of teeth can influence the color of teeth.

a) Tooth wear and gingival recession: Both conditions appear to have multifactorial etiologies but to date are poorly understood, there being limited research on the topics. Tooth wear is usually considered to be a progressive loss of enamel and dentine due to erosion, abrasion and attrition. As enamel thins the teeth become darker as the color of dentine becomes more apparent. Once dentine is exposed the potential of chromogens to enter the body of the tooth is increased. Physical trauma can also result in bulk loss of enamel or enamel cracks, both of which facilitate internalisation of extrinsic stains. Although tooth wear occurs at the cervical area of teeth, where enamel is most thin, exposure of dentine is more likely caused by gingival recession. Again, the net result is dentine exposure and the increased potential for the uptake of chromogens into the tooth.

b) Dental caries: The various stages of the carious process can be recognized by changes in color as the disease progresses. For instance, the initial lesion is characterized by an opaque, white spot. The white spot lesion differs in color from

the adjacent enamel by virtue of its increased porosity and the effect this has on the refractive index. Enamel has a Refractive Index of 1.62, compared with 1.33 for water and 1.0 for air. Air-drying removes water from the pores in partially dematerialized enamel leaving air and makes the 'white spot lesion' conspicuous by the alteration in its light transmitting properties. The hard, arrested lesion is black having picked up stain from exogenous sources. Early investigation into the change in color with the carious process centered around the amino-acids released during proteolysis, as a result of the proteolysis-chelation theory of cavity formation.

c) Restorative materials including amalgam: Some of the materials used in restorative dental treatment may have an effect on the color of teeth. Eugenol and phenolic compounds used during root canal therapy contain pigments which may stain dentine. Some of the poly antibiotic pastes used as root canal medicaments may cause a darkening of the root dentine. Clinicians are familiar with the dark grey to black color of dentine following the removal of a long-standing amalgam restoration. It was previously thought that mercury was penetrating the dentinal tubules and reacting with sulphide ions. Electron microscopic studies have shown that this discoloration is caused by the migration of tin into the tubules.

❖ How can we prevent teeth discoloration?

By making a few simple lifestyle changes, you may be able to prevent teeth discoloration. For example, if you are a coffee drinker and/or smoker, consider cutting back or quitting all together. Also, improve your dental hygiene by brushing and flossing regularly and getting your teeth cleaned by a dental hygienist every 6 months.

What treatment options are available to whiten teeth?

Dental treatment of tooth discoloration involves identifying the etiology and implementing therapy. Medical treatment also may be warranted, depending on the etiology of the tooth discoloration.

- **Diet and habits**: Extrinsic staining caused by foods, beverages, or habits (e.g., smoking, chewing tobacco) is treated with a thorough dental prophylaxis and cessation of dietary or other contributory habits to prevent further staining.
- **Tooth brushing**: Effective tooth brushing twice a day with a dentifrice helps to prevent extrinsic staining. Most dentifrices contain an abrasive, a detergent, and an anti-tartar agent. In addition, some dentifrices now contain tooth-whitening agents.
- **Professional tooth cleaning**: Some extrinsic stains may be removed with ultrasonic cleaning, rotary polishing with an abrasive prophylactic paste, or air-jet polishing with an abrasive powder. However, these modalities can lead to enamel removal; therefore, their repeated use is undesirable.
- **Enamel microabrasion**: This technique involves the rotary application of a mixture of weak hydrochloric acid and silicon carbide particles in a water-soluble paste. The resultant surface is smooth and has a glazed appearance. Enamel microabrasion is indicated for the removal of superficial intrinsic tooth discoloration, including that caused by fluorosis and decalcifications secondary to orthodontic brackets or bands. Enamel microabrasion may be used in conjunction with bleaching.
- **Bleaching (tooth whitening)**: Early bleaching techniques were developed almost a century ago, and all of the techniques involved a process of oxidation. Today, with proper patient selection, bleaching is a safe, easy, and inexpensive modality that is used to treat many types of tooth discoloration. Usually, bleaching is not indicated for the treatment of discoloration of the primary teeth. Bleaching includes 2 types of techniques: vital and nonvital.
  - **Vital bleaching**
    - Bleaching of vital teeth is indicated primarily for patients with generalized yellow, orange, or light brown extrinsic discoloration (including chlorhexidine staining), although it may be helpful in ameliorating mild cases of tetracycline-induced intrinsic discoloration and fluorosis.



- Currently, the bleaching agents most commonly used are carbamide and hydrogen peroxide. When applied in higher concentrations, the agents produce more significant bleaching than they do without these measures.
- In office "power" bleaching involves the use of a 15-40% hydrogen peroxide solution and must be performed by a dental professional because careful isolation of the teeth is required to protect the soft tissues from the caustic effects of the bleaching agent.
- The use of home bleaching systems is currently popular; they may be used alone or in combination with in-office bleaching. The systems must be used under the careful supervision of dentists or dental hygienists. Patients apply a 10-22% carbamide peroxide solution into a custom-made mouth guard. After repeated daily and/or nightly (often while patients sleep) applications for 2-6 weeks, the teeth are gradually bleached. Whitening strips, using a 5.3% hydrogen peroxide–impregnated polyethylene strip, offer an at-home alternative to the above methods and can be recommended for maintaining already whitened teeth.
- Whitening toothpastes, containing 1% or less peroxide, are minimally effective.
- With darker stains, the best results are achieved by using a combination of office and home bleaching systems. Most patients also require periodic re-treatment.
- Clinicians should be aware of potential adverse reactions and contraindications for bleaching. Approximately two thirds of patients have short-term, minor tooth sensitivity to cold and/or gingival irritation. Tooth surfaces, particularly exposed roots or enamel surfaces with defects secondary to incomplete amelogenesis, are porous to the bleaching agent and are more likely to develop cold sensitivity. Gingival irritation usually is related to improper fitting of the custom-made mouthguard.
- Allergic reactions to the bleaching agent are exceedingly rare.
- No adverse reactions are documented in pregnant or breastfeeding women or in patients who smoke; however, bleaching is not advised in these patients.

- Non vital bleaching

- **Non vital bleaching** is indicated for the treatment of teeth with discoloration secondary to pulpal degeneration. This technique involves placing a mixture of 30% hydrogen peroxide and sodium perborate into the pulp chamber for as long as 1 week.
- For non vital bleaching, a tooth with an un restored crown is ideal.