

Dental stain

Pigmented deposits on the tooth surface are called **dental stains**. The correct diagnosis for the cause of discoloration is important as, it has a profound effect on treatment outcomes. It would seem reasonable, therefore, that dental practitioners have an understanding of the etiology of tooth discoloration in order to make a diagnosis and enable the appropriate treatment to be carried out.

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 hyperbilirubinaemia**
- 4. Amelogenesis imperfecta**
- 5. Dentinogenesis imperfecta**
- 6. Tetracycline staining**
- 7. Fluorosis**
- 8. Enamel hypoplasia**
- 9. Pulpal haemorrhagic products**
- 10. Root resorption**
- 11. 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.

1. Alkaptonuria: is rare inherited disorder of amino acids metabolism results in incomplete breakdown of tyrosine and phenylalanine , which promotes the buildup of homogentisic acid. This affects the permanent dentition by causing a **brown discolouration**.



Fig. :- Alkaptonuria symptoms.

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 discolouration** of the teeth is the result and the affected teeth show a red fluorescence under ultra-violet light.

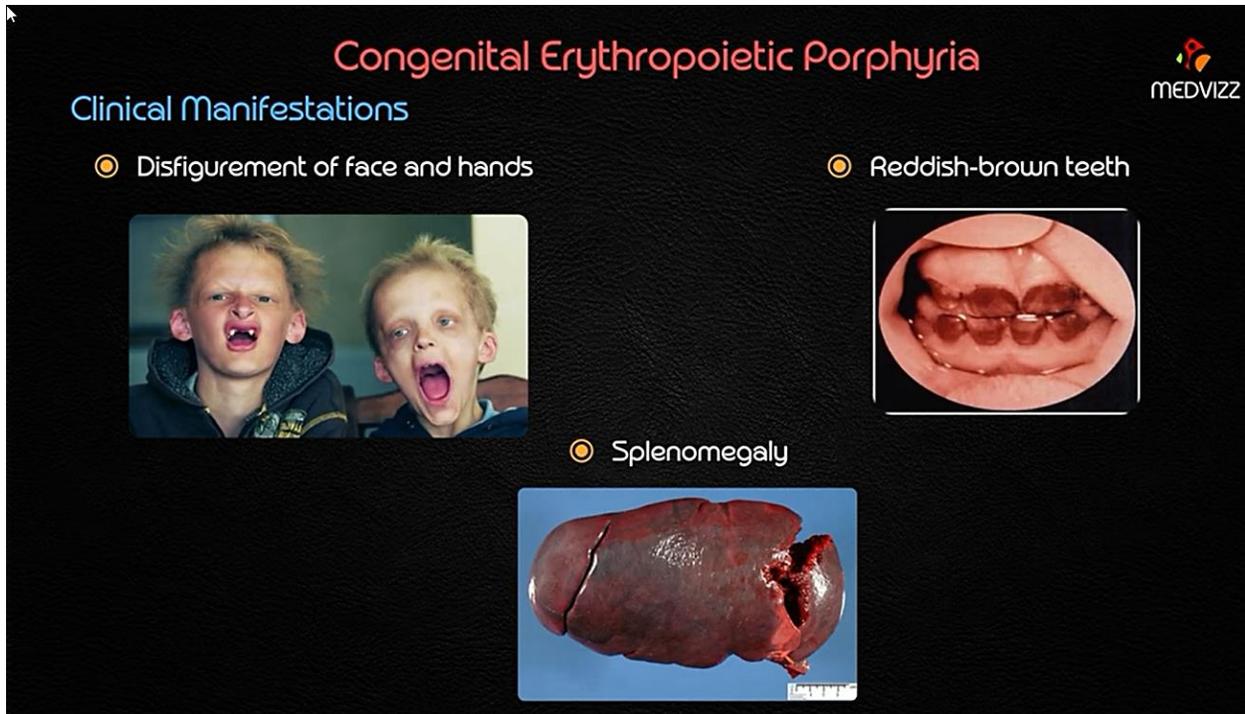


Fig. :- Congenital erythropoietic porphyria clinical manifestations.

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



Fig. :- Congenital hyperbilirubinaemia.

4. Amelogenesis imperfecta: In this hereditary condition, enamel formation is disturbed with regard to mineralization or matrix formation and is classified into **14 different subtypes**; the majority is inherited as an autosomal dominant or x linked trait with varying degrees of expressivity. The appearance depends on 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.



Fig. :- Amelogenesis imperfect.

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 amelodentinal 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 factors. 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 affected. The teeth are usually **bluish or brown** in color. 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.

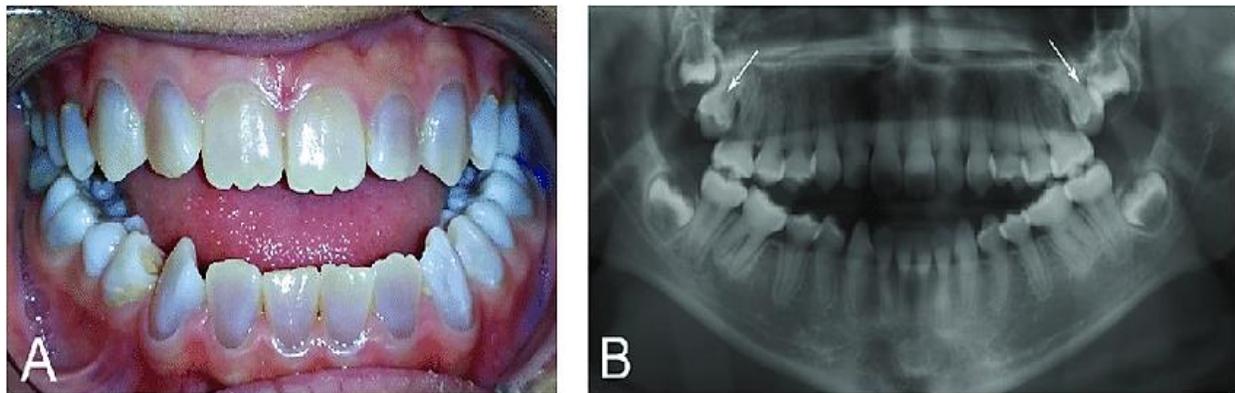


Fig. :- Dentinogenesis imperfecta type II.

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 enamel is much less prone to fracture, the pulp chamber is rarely occluded by dentine (this may help to radiographically differentiate between types I and II), and the overall prognosis for the dentition is improved.



Fig. :- blue sclera associated with osteogenesis imperfect.

Dentinogenesis imperfecta III (brandywine 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 stops 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 dysplasias. 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 have 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.



Fig.:- Dentine dysplasia type I.

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



Fig.:- Dentine dysplasia Type II with numerous pulp stones.

8.Tetracycline staining: Systemic administration of tetracycline 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**.



Fig. :- Tetracycline staining.

The various analogues of tetracycline produce different color changes, for example **chlortetracycline** produces a slate grey color and **oxy-tetracycline** causes a creamy discoloration. 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 opacous 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.

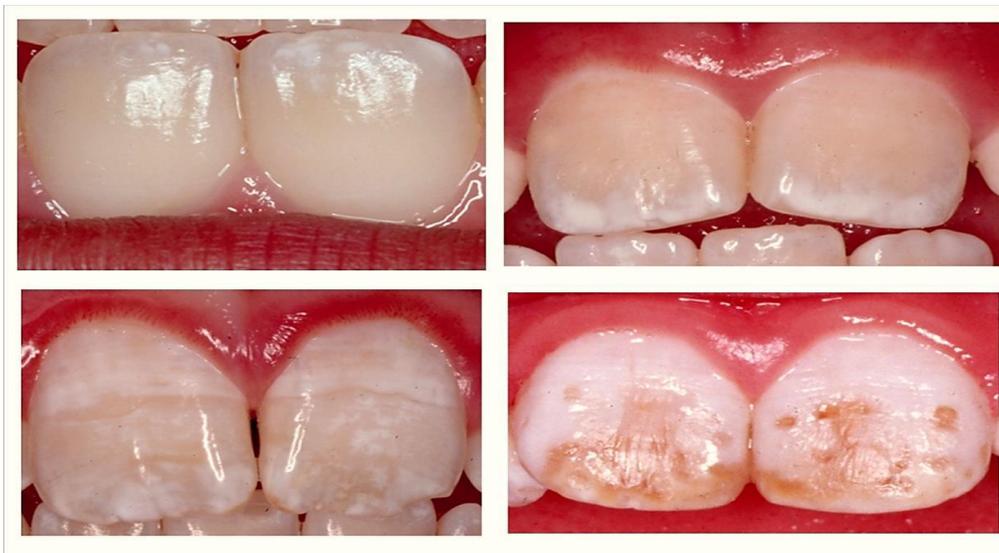


Fig.:- Fluorosis.

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 e.g. maternal vitamin D deficiencies, rubella infection, drug intake during pregnancy and in pediatric hypocalcaemic 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.



Fig.:- Enamel hypoplasia after primary tooth trauma.

11. Pulpal hemorrhagic products: The discoloration of teeth following severe trauma was considered to be caused by pulpal hemorrhage. Haemolysis of the red blood cells would follow and release the hem group to combine with the putrefying pulpal tissue to form black iron sulphide. 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 haematin molecules. 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 amelocemental 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.



Fig.:- Root resorption.

13. Ageing: 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:-

- ☒ Compounds incorporated into the pellicle and produce a stain.
- ☒ Chemical interaction of a specific compounds at the tooth surface lead to staining.

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 discolouration has usually been classified according to its origin, whether metallic or nonmetallic.

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 **Actenomyces 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 permanganate** 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.

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:

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, restorations of teeth can influence the color of teeth.

a) Tooth wear and gingival recession:

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 internalization 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.

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.

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 non-vital.

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 mouth guard.

- ☒ Allergic reactions to the bleaching agent are 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.