

## TOOTH WHITENING & BLEACHING FACTOID

Humans have long attempted to beautify their teeth for various cultural reasons. In attempting to demonstrate their high social standing, some South American native cultures had the labial aspect of their maxillary anterior incisors drilled with certain tools to permit the cementation of semi-precious stones into the drill hole with various gum resins from trees & bushes—some of these cemented jewels remain cemented even today. Some ancient Malaysian cultures filed & chipped away enamel & dentine of young females anterior teeth into a sharp point using crude metal knives & stone tools to establish that they had reached the social age for marital consideration—even today these cultural habits are still practiced on few isolated Indonesian islands, even though they understand the process causes pain & may damage the teeth, which may eventually become lost due to trauma & pathology. In some Asian cultures e.g. Japan, married woman stained their teeth with black dye to identify her marital status—different than a single female—that social custom was employed into the late 1800's. Egyptians of the last millennium used powdered pumice & wine vinegar with a chew stick to remove stains & polish their teeth. In Africa, the *meswak* (chew stick) from trees & bushes contained certain essential oils e.g. eugenol, clove was used to clean & polish the teeth. Chew sticks remain in use today in many rural cultures where dental treatment is limited or unavailable.

### INTRINSIC & EXTRINSIC TOOTH STAINING

Hattab (1999) defined the staining of teeth as intrinsic, extrinsic or a combination of both. During tooth development, the enamel & dentine may incorporate various agents (e.g. antibiotics, fluoride) that are often bound within the substrates that show as dark stains. As secondary dentine is normally deposited in small increments throughout life, various agents e.g. tetracycline, fluoride may become incorporated, which cause increased darkening of dentine as part of the aging process. Environmental agents such as tea, coffee, wine, tobacco & certain foods may easily stain the surface biofilm & calculus to darken the teeth, which scaling, prophylaxis & tooth brushing may remove. On the other hand, tenacious stains in enamel lamella & other intrinsic blemishes may be lessened or removed by vital bleaching using various peroxides.

### NON-VITAL HUMAN TOOTH BLEACHING

The pulp & dentinal tubules of root canal treated teeth may contain remaining hemoglobin from hemorrhage due to: 1) a traumatic blow or death of the pulp, 2) iatrogenic treatment that allowed blood or other organic material to penetrate into the dentine tubules & 3) failure to remove a sealer paste—all 3-may cause discoloration (Dowson & Garber 1967). Gottlieb *et al*, 1950 suggested the use of two bleaching agents: 1) of 25% to 35% H<sub>2</sub>O<sub>2</sub> in 75% ether or 2) 30% H<sub>2</sub>O<sub>2</sub> in distilled H<sup>+</sup>OH<sup>-</sup>—he cautioned that both needed to be kept clear of the mucous membrane or skin to prevent burns that would turn the tissues white & eventually slough off, but they would eventually disappear without scarring. The so-called “walking bleach” of HCl, HNO<sub>3</sub>, H<sub>2</sub>O<sub>2</sub>, Na-perborate or silicate cement were popular throughout the 1950’s to the 1970’s, but they eventually fell into disfavor as root resorption (Lado 1983), crown fracture (Grevstad 1981), cervical root resorption (Dahl 2003), & PDL pathology were a common occurrence, especially if the patient failed to return for their definitive treatment. Today, in-office chair side bleaching is typically carried out under a rubber dam with 30% H<sub>2</sub>O<sub>2</sub>, which may cause tissue irritation of periapical tissues if leakage occurs. More favorable treatments are the use of Na-perborate & EDTA in combination, sometimes requiring several sessions. Regarding office storage & handling, certain high concentration H<sub>2</sub>O<sub>2</sub> solutions can easily become explosive & so they should be stored in tightly sealed & refrigerated dark bottles for safety & to maintain their chemical efficacy.

## UREA - AN OXIDIZING AGENT IMPORTED BY THE ROMANS

The Roman culture revered the color purple (indigo), which symbolized royalty. Pliny the Elder (23AD-79AD), the noted author, historian & naturalist described the use of human UREA to convert the insoluble *indigo white* for fabric dyeing in Mediterranean cultures. Scholars learned they could reduce the ground *insoluble indigo white* shell substance they extracted from the sea snail *Murex* exoskeleton—using human **urea**—also called **carbamide** to render the dye soluble. **Urea** is **highly soluble** in water, is non-toxic & tends to be neither acidic nor alkaline. The chemical process of urea causes the chemical oxidization of *indigo-white* of the Murex snail shell into the famous color of royal *Tyrain indigo purple*, which the Roman aristocracy highly favored.

Circa 200-BC, Rome annexed Portugal into their empire from which they imported much of the their urea—they felt Iberian human **urea** was the best to reduce *indigo*

*white* into *indigo blue* (Pliny The Elder). Someone realized that urea also whitened human teeth—**history suggests the Romans were the first culture to whiten teeth with UREA**. Today we know that bleaching teeth with a low concentration of **urea** also reverses gingivitis & slows alveolar bone loss, which also stabilizes teeth—besides keeping teeth free of plaque & free of caries.

In the European Middle Ages, to treat patient complaints, barber-dentistas would file various tooth defects to smooth the rough or broken edges of a persons tooth with a small metal file, called an *iron-grater*. Then they would rub the area with a highly-corrosive  $\text{HNO}_3$  (nitric acid) to smooth & whiten the teeth, but in doing so, it deeply etched the enamel surface by altering the underlying substrate & also damaged the adjacent epithelial tissue attachment that led to eventual loss of attachment, which in turn led to plaque buildup & progressive development of cervical & root caries.

### **EARLY BLEACHING OF VITAL TEETH IN THE US**

In 1850-Dwinelle suggested the use of chloride of lime ( $\text{CaOCl}$ ) & soda for vital bleaching, in 1860—Fitch used nitric acid, in 1861—White used sulfuric acid & in 1864-Truman bleached discolored teeth with chlorine & a weak acid—suggesting to saturate the entire pulp canal with an acid before inserting powdered chlorinated lime or to pack a paste of water & lime into the tooth followed by an acid saturated cotton. In the late 1860's, Dr. Wright, of Richmond Virginia forced a continuous stream of chlorine gas into the pulp-canal & tooth cavity by an elaborate apparatus made especially for the purpose. The method was rapid & efficient, but device maintenance & gas preparation safety prohibited its general acceptance, so it was abandoned.

In 1895, Westlake published his observations in the Am Jour of Dent Sci regarding his use of 35%  $\text{H}_2\text{O}_2$  & weak concentrations of  $\text{HCl}$  to remove extrinsic stains—his procedure became popular for general dentists to bleach vital teeth. In the 20<sup>th</sup> century, Dr. Herman Prinz advocated the use of a stabilized mixture of 30% superoxol ( $\text{H}_2\text{O}_2$ ) to bleach intrinsically fluoride stained teeth—in 1937, Dr. Ames gained some notoriety by using a 30%  $\text{H}_2\text{O}_2$  mixed with ether in the presence of heat as a clinical treatment that lasted 30-mins, sometimes with 20 or more sessions.

In 1965, Zach & Cohen reported on the histological effects of externally applied heat onto the labial surface of vital monkey teeth—15% of teeth heated by  $5.5^\circ\text{C}$  & 60% of the teeth heated by  $11^\circ\text{C}$  suffered irreversible cell damage at the pulp-dentine wall—

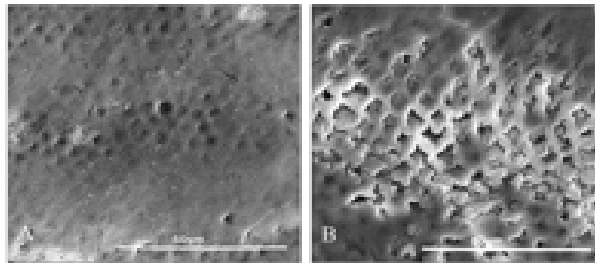
those results were later confirmed by Nyborg & Brännström (1970) & in 1987 Seale & Wilson described complete obliteration of the odontoblastic layer, loss of predentine, a dense infiltrate of inflammatory cells, areas of internal resorption & pulpal hemorrhage after 3-days.

In 1960's, Dr. William Klusmier, an orthodontist from Fort Smith Arkansas recognized that a number of his patients had developed gingivitis due to poor hygiene—from his reading, he used a 10% carbamide peroxide in a removable nighttime appliance in a young orthodontic patients, resulting in a reversal to a near normal gingival. Following their treatment, Dr. Klusmier noted the gingival enamel 1/3 was lighter & that even intrinsic tetracycline stains had disappeared.

In 1968, Dr. John Munro, a general dentist from Tennessee used a 10% carbamide peroxide & also noted the gingiva was less inflamed & the teeth were whiter—based on his observations, the first commercial 10% carbamide peroxide home bleaching agent called White & Brite was developed by Howard Kerr & his colleagues—produced by Omni Company & sold only through dental offices. Almost immediately, the carbamide peroxide the Omni nighttime home bleaching tray system became adopted & recommend by clinicians. However, after the emergence of Omni's White & Brite, a number of uncontrolled high concentration peroxide products appeared on the worldwide market. Due to certain publications—those high peroxide concentrations—over 10%-created many patient concerns to the FDA as gingival irritation & post-treatment hypersensitivity to cold stimuli were quite common outcomes.

Due to the commercial popularization that the rapid non-regulated tooth bleaching caused in the late 1980's, the FDA received many complaints from people who reported they had been treated with the high concentration (15% to 35%) of H<sub>2</sub>O<sub>2</sub> products—stating they had experienced harmful consequences due to a high acid content that often burned the gingiva & caused increased levels of post treatment sensitivity to cold & air stimuli. In 1991, the FDA requested all manufacturers to submit all their research documentation on the safety of their peroxide product, prior to its marketing. However, when the FDA was challenged by a certain major dental manufacturer, their action was recalled—conversely the ADA immediately took notice of the absence of bleaching product regulations & so without any delay, the ADA proceeded to develop guidelines for approval of vital H<sub>2</sub>O<sub>2</sub> tooth bleaching.

Today, it seems that certain human segments of the world wide culture are vainly attempting to make an impression on their friends with a dazzling white personalized smile, & they are led to believe that nothing can ruin their white teeth after bleaching. As an answer—in a more preventive mode after bleaching treatments, dentists & hygienists are now advising concerned patients to avoid chromogenic staining agents such as tea, coffee, red wine, smoking & chewing tobacco as well as certain foods that create extrinsic staining that will rapidly darken their bleached teeth. In reality, it's not very practical to expect most people to stay away from all those potential staining agents, **but** at least we are able to properly inform any patient as to both pre & post bleaching expectations especially as to what causes tooth discoloration.



The above SEM's are from the enamel surface of extracted human teeth from Holmen *et al*, (1985). Photo A on the left, shows the normal oral surface with enamel rods & different levels of morphology, some rod heads are slightly concave while other areas are covered with oral debris. Photo B on the right shows an enamel surface that was exposed to a H<sub>2</sub>O<sub>2</sub> gel 2 X a day with a rinse & storage in water each day. At the end of the *in-vitro* 3-week treatment, the bleached surface (B) was prepared for SEM, showing obvious enamel surface alteration caused by the extended bleaching. Other studies (Shannon *et al*, 1993, Ernst *et al*, 1996, Oltu & Gurgan 2000) also demonstrated that extended clinical bleaching with 15% to 30% H<sub>2</sub>O<sub>2</sub> alone, or when mixed with a 30% Na-perborate would dramatically change & remove the inorganic–organic composition of enamel. On the other hand, bleaching treatments with the 10% carbamide peroxide did not dramatically alter the enamel surface. These studies reinforce an early study of Bartelstone (1951) whose data demonstrated that enamel is a porous substrate, which permitted the rapid passage of radiolabelled I-131 through the enamel lamella & dentine tubules to the pulp & into the vascular system to reach the thyroid gland within 20-minutes from the beginning of the procedure.

In light of the various published studies in the literature, several clinical consequences should be considered regarding tooth bleaching expectations:

1) Swift & Perdigo (1998) reported that high concentrations of H<sub>2</sub>O<sub>2</sub> may increase the solubility & eventual breakdown of certain glass-ionomer systems & other luting cements. 2) Dishmann *et al*, (1994) have reported a reduction in bond-strength between enamel & resin-based fillings after 24-hours of H<sub>2</sub>O<sub>2</sub> bleaching. 3) Lai *et al*, (2002) reported that remaining H<sub>2</sub>O<sub>2</sub> bleaching residuals in human enamel would inhibit the polymerization of resin-based materials that would eventually reduce bond-strengths. Consequently, they suggested tooth bleaching with H<sub>2</sub>O<sub>2</sub> should not be used before enamel restoration with any of the resin-based materials.

In 1999, the International Programme on Chemical Safety; In the Environmental Health Criteria, Geneva SU, defined 4-Risk Assessments for **EXTERNAL TOOTH BLEACHING**:

1) Dose-response relationship, 2) hazard identification, 3) exposure assessment, & 4) risk characterization as a comparison of the dose-response association.

A 1995 *in vivo* study by Dahl & Becher reported: To prevent possible pulp damage, the concentration of H<sub>2</sub>O<sub>2</sub> should not exceed 3.5%—equivalent to 10% carbamide peroxide. Evaluating additional *in vivo* studies, Dahl & Pallesen (2003) concluded: Optimal levels of any patient peroxide bleaching system should not exceed 10%; the delivery tray should not be overfilled; excess overflow must be removed; only 1-arch should be bleached at a time—to avoid possible ingestion or tissue irritation of the H<sub>2</sub>O<sub>2</sub> agent for no more than 2-hours at a time (Matis *et al*, 2002).