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INCREASING THE BOTTOM LINE

In business it all comes down to the bottom line and how we can make more with what we have. In reality, we usually have only one hour with each patient to complete all of the “routine” procedures, and the thought of adding one more thing can be maddening. How many of you are pressed by your dentist for just a little more production? There are goals to be met and overhead to be covered, and this can become stressful for the entire staff.

In many offices there is a production bonus for exceeding set goals, and this benefits both the staff and dentist. We can increase the production and income of the oral hygiene department and practice without much added work. It all comes down to working smarter, but not necessarily harder.

A good leading question for patients is asking them what they would change about their smiles if they had the chance. The majority of patients I talk to say they would like a whiter smile, and many are unaware that it is possible and relatively inexpensive. It can be quick and easy to spark interest and generate extra income during conversations with patients. As clinicians, we should be performing intra-oral and extra-oral exams with each patient.

Next time, take an extra 30 seconds to show them the tooth shade guide for an initial shade to enter into the chart. Curious patients will question why you are taking a shade. Explain how teeth can change and darken over time with age, diet, and other factors, and that you are keeping track of changes for future reference. This will get patients thinking about their shade and smile.

When you show patients the shade guide and where their teeth are now, many want to know how to get a whiter shade. As you move on with the appointment, the foundation has been set to discuss whitening options, whether it is take-home bleach trays, in-office whitening, or something more involved such as veneers or crowns.

The cost to take impressions and make bleach trays is minimal; however, the potential for income is great for many reasons. Patients are happy to have the desired whitening effect, and the result is that many will talk to friends and hopefully generate referrals to the practice. In addition, they will purchase whitening gel during future visits to touch up their glowing smiles. Those 30 seconds of added effort during their oral hygiene visit have the potential to generate easy profits for years to come.

The majority of medical aid companies do not cover an in-office fluoride treatment for adults. In my experience, if the benefits of fluoride treatment are explained to patients who have hypersensitivity or a high caries rate, they often choose to receive in-office fluoride and understand that it is a small added expense for a good potential benefit. Paying a small amount for a fluoride varnish treatment to decrease sensitivity makes the expense worth it for most patients. We never know what a patient values or may be willing to pay unless we offer it.

We have all discussed the increased caries rate with people and looked for options to decrease the risk. What percentage of South Africans chews gum or mints throughout the day? How about recommending xylitol mints or gum? Studies have shown that xylitol can decrease the caries rate, and xylitol products are available in mints, gum, toothpaste, rinses, and nasal sprays. It is safe for all ages and for diabetics, as it is a sugar substitute that does not significantly raise glucose levels. Do not forget to warn people of the potential side effects if these products are consumed in large quantities. Most people do not experience side effects if they consume less than 5 mg per day. While there are many brands available over the counter, keep xylitol in the practice as well. We are a society of convenience, and having a few in the practice does not take up much space and allows for a quick and easy profit.

From my experience as a hygienist, the biggest added time is needed when we have a child or teenager in the chair in need of sealants. After you or the dentist has diagnosed the need for sealants, placing the sealants during the same visit as the prophylaxis is an added convenience for the parent or guardian. The additional five to 10 minutes to place fissure sealants has the potential to boost the daily production greatly. The extra time provides the opportunity to generate an additional few hundred rand for the practice, and potentially for the oral hygienist if it is a production-based pay rate or bonus system.

I used to recommend a power toothbrush as part of my home-care instructions for the majority of my patients, and I am sure many of you do too. Often a patient will ask for a brand recommendation and keeping two or three brands on hand and charged allows people to hold and feel the action the power toothbrushes offer. We have all explained the benefits of power toothbrushes versus manual brushes, and by allowing patients to hold and touch, they are able to see and feel the difference.

How many times have we put off buying something or forgotten to pick up something, even if it was recommended by a health-care professional? By keeping the brushes in the practice and having a professional recommend them, many patients will conveniently purchase on site. As consumers, we like to have choices in products without being overwhelmed by the options. Having choices on a few different models at different prices allows patients to purchase brushes within their budgets. This makes it a win-win for the patient and practice, adding easy production for the practice.

Now your patients have beautiful white teeth and are using power toothbrushes, but how is the caries rate? We all have those patients who have one or two new cavities every six months, and they just cannot figure out why. We discuss the options to reduce the caries rate, ranging from fluoride treatments to xylitol and chlorhexidine. Does your practice have these products on hand for patients to purchase, or do you give them a note with everything they should buy? Having a small supply in the practice is convenient for patients, freeing them from having to drive to pick up your recommended items, and increasing the likelihood of patient compliance.
Dear OHASA Members and Colleagues

The third quarter is on our doorstep and the start of spring. New beginnings! With the new publishers delivering a very professional looking *Journal* and a new interactive website we can only present a more professional image.

Some obstacles are opportunities. If you seek a way past them, you will eventually find success. Others are impenetrable. They are too big, broad, high or deep. No matter how much effort you make, you will be wasting your time. How are any of us supposed to know the differences between these two types of difficulties? There is only one way we can find out – we try for a while and see how far we get. One such challenge is Independent practice – we can now have a shot at it. Which leads to Ethics.

Ethics should be infused into all oral hygienists’ professional life and not only the lives of those who are practising independently. The public will be the greater beneficiary of the focus on ethics and the profession too will benefit from a reputational aspect.

The question is how to promote the culture of ethics among practising professionals. Professor Su Naidoo has, over the years, been writing the ethical articles in the *Journal*. The profession is defined among other things by the commitment of its members to standards of behaviour that are founded in ethics and best practice. These standards go beyond the general law of the land, in terms of which members of trades as opposed to professionals are held accountable. So as dental professionals we must continue to be held accountable to higher values than the average businessman or politician.

In striving for those higher values, what happens to practitioners that are faced with choices or decisions that need to be benchmarked against the rules of the professional conduct of the medical and dental profession?

Oral hygienists throughout the world are specialised professionals who place the interests of their patients above their own, and strive to obtain the best treatment for them. They have to combine a continuous update on dental developments with a service to their patients, and maintain a reasonable standard of living – between these elements there is often tension.

The rules of professional conduct, the demands of a practice and the need to earn a living – this is where an independent practitioner may need assistance in navigating the pressures. It will be possible to turn to the ethics committee of the HPCSA but in today’s fast-paced life, practitioners need an open, immediate and confidential channel that can provide real-time responses to ethical dilemmas. This is where the knowledge share on the new website will come in handy, with the experienced older practitioner providing guidance on a confidential basis. We can even have a database of questions and answers that is updated and accessible.

The database would serve a dual purpose by providing immediate information and serving as a resource to monitor trends and practice requirements.

Ethics, like morals, are intangible and cannot be taught. The impact of immoral and unethical actions in the profession is real and far reaching for the practitioner, the patient and the profession as a whole.

The promotion of a culture of ethics requires the commitment of every oral hygienist for it to remain a proficient profession.

God Bless

Stella

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**EDITORIAL**

**FROM THE PRESIDENT’S DESK**

Stella Lamprecht

OHASA president

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**OHASA’S VISION**

OHASA is a dedicated, dynamic, professional association representing hygienists as invaluable members of the health profession team.

**OHASA’S MISSION**

OHASA aims to promote quality oral health care by representing, protecting and advancing the profession in partnership with stakeholders.

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NUTRITION AND ORAL HEALTH:
OUR ROLE AS ORAL HYGIENISTS

Glynnis Vergotine: Dip OH, BA, B Ed (Hons), M Ed (Oiss)
Lecturer at the University of the Witwatersrand, Johannesburg

As South Africans, food is central to our identity. At mealtimes our families meet around the table, during holidays and special occasions we gather over a braai or the potjie which reflects the remarkable diversity of our country. However, there are pressing concerns linked to nutrition for our nation, with the rising incidence of food insecurity and non-communicable diseases (such as obesity, diabetes and coronary heart disease). Food insecurity findings in South Africa show that the nutrient density of the diet consumed by children is insufficient to meet daily nutrient requirements. Similarly, there is low food variety and household dietary diversity especially in poorer communities, with one child in four under the age of six years (translating to approximately 1.5 million children) stunted due to chronic malnutrition. Oral disease is classified as a non-communicable disease (NCD) by the World Health Organization (WHO), and adopting the common risk factor approach for NCDs through interventions with individuals and communities will assist in preventing a number of diseases. At-risk people can be accessed through private and public health clinics, social development centres and other government services. As the rate of systemic health problems such as malnutrition, diabetes, obesity, and cardiac disease continues to increase, the need for oral hygienists to provide dietary guidance is also growing.

So, when we think about the role of the Oral Hygienist in understanding the nutritional status and nutrition education of South Africans there are a number of questions which come to mind. What messages are being given to our patients and communities about nutrition? To what extent are Oral Hygienists addressing nutrition as part of their daily work? And, what type of interaction do we have with our colleagues in associated professions about the important link between oral health and general health.

In South Africa, dental caries is regarded as a condition of high prevalence and burden as it is the most common condition affecting children. Sixty percent of 6 year olds, in their primary dentition, have decay, and 55% untreated decay, therefore 91% goes untreated. Only 18% of 12 year olds have healthy gums and only 2% of 44 year olds have healthy gums. Outside of demineralisation and caries formation, many patients and health care providers do not realise the strong association that exists between nutrition and oral health. Oral hygienists are well positioned to educate both patients and medical colleagues about this link.

Oral hygienists have to be informed on the most current research in dentistry and oral hygiene, as well as allied fields. This will enable us to provide our patients with meaningful nutrition information, as well as prevent and rectify misconceptions about nutrition to improve both health literacy and patient compliance with dietary recommendations. This can be achieved through regular continued professional development courses and gaining access to evidence-based resources. Some resources which can be accessed by oral health professionals include the South African Food-based Dietary Guidelines for people aged >6 years, which takes into consideration both over-nutrition and under-nutrition. The guidelines related to oral health are highlighted to the left. Further details of South African food guidelines can be seen in policy documents and articles on nutrition in South Africa.

Other commonly used educational resources for delivering general nutrition advice and promoting healthy eating are the 2010 Dietary Guidelines for Americans, the Choose MyPlate, published by the United States Department of Agriculture and US Department of Health and Human Services and the Eatwell Plate used by the Food Standards Agency in the UK. These resources explain the basic guidelines for a healthy diet and take into consideration the correct balance of food intake.

Understanding the importance of added sugars in our diets and the effects on oral and general health is vital. Sugars are undoubtedly the most important dietary factor in the development of dental caries. The term ‘added sugars’ refers to monosaccharides and disaccharides. Sugar consumption must address the quantity, frequency, and timing of sugar consumption, with sugar intake <40 g/day in areas in which water is not fluoridated and <55 g/day in fluoridated areas. This equates to about 6–10% of energy intake. Messages such as ‘consume foods with fewer added sugars, and limit it to four times daily’ have to be consistently used by all health professionals. Teaching our patients to check food product labelling is important in getting them to realise the amount of added sugar and that sugars have various names.

Some other significant measures for oral hygienists to follow will include the following: taking a thorough medical and dental history can provide the necessary information to discuss patients’ oral health needs, as well as their nutritional requirements; routinely providing healthy eating advice to the public to promote good oral and general health;

- Enjoy a variety of foods
- Be active
- Make starchy foods the basis of most meals
- Eat plenty of vegetables and fruit every day
- Eat dry beans, peas, lentils, and soy regularly
- Chicken, fish, milk, meat, or eggs can be eaten daily
- Eat fats sparingly
- Drink lots of clean, safe water
- If you drink alcohol, drink sensibly
- Use salt sparingly
- Eat and drink food and drinks that contain sugar sparingly and not between meals
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Infections have been strongly associated with adverse pregnancy outcomes like pre-term and/or low birth weight (PTLBW). There is substantial evidence on the direct association of genito-urinary infections and the incidence of PTLBW. Numerous cases of adverse pregnancy outcomes without maternal genito-urinary infection but nevertheless high levels of the tumour necrosis factor alpha (TNF-α) and prostaglandin E2 (PGE) in the amniotic fluid, have been recorded. These findings alluded to the presence of infection elsewhere in the body. This paper reviews the literature on the association between the infective condition of periodontitis and PTLBW.

Key words: Adverse pregnancy outcomes, pre-term birth, low birth weight, periodontitis

INTRODUCTION
Numerous studies have been conducted to evaluate the relationship between periodontitis, pregnancy and adverse pregnancy outcomes such as pre-term birth, low birth weight and the combination of the two. The increase in research activity on this subject has been triggered by adverse pregnancy outcomes presenting a major public health problem worldwide, despite improvements in prenatal, antenatal, postnatal and maternal care including public awareness.2,3 Pre-term birth and/or low birth weight contribute significantly to the health care, economic and social burden imposed on the government, communities and families involved.2,4

According to the global action report on pre-term birth, there are approximately 15 million pre-term births annually.1 Global statistics published by the World Health Organisation (WHO) indicate that over 60% of pre-term births occur in Africa and South Asia. Some regions within sub-Saharan Africa record pre-term birth rates as high as over 15%.5

Whilst results from most studies have suggested that this association may exist, such findings have not been consistent. This is partly as result of the heterogeneity in the methodology of the studies conducted. The variations range from the diversity of populations, sample sizes, case definitions of periodontitis and pregnancy outcomes.

Periodontitis is a chronic infectious disease, and when present is characterised by periods of exacerbation and remission. It is predominantly associated with gram negative organisms within the subgingival plaque biofilm,2 and can be modified by the host immuno-inflammatory response, environmental, genetic and systemic factors.

The tissue destruction seen in periodontitis is mainly related to the inflammatory process which, in an attempt to combat infection, results in tissue damage.4 The inflammatory mediators secreted in response to endotoxin/lipopolysaccharides (LPS) include pro-inflammatory cytokines e.g. IL-1β and TNF-α that bind to specific receptors on target cells; prostaglandins (PGE), which cause vasodilatation and matrix-metallo-proteases (MMPs), which are able to degrade collagen, gelatine and elastin.4,5 These inflammatory mediators are secreted locally within the periodontium and can be detected in the crevicular fluid and systemically following exposure to LPS.

Without intervention the diseased periodontium can serve as a reservoir for gram negative anaerobes, their virulence factor (LPS) and the associated inflammatory mediators which are able to enter the systemic circulation resulting in constant activation of the systemic host response. Hence periodontitis is associated with increased levels of c-reactive protein (CRP), an acute phase protein produced by hepatocytes in response to the circulating pro-inflammatory cytokines including TNF-α and IL-1β which is a diagnostic marker for clinical infections. Given the large epithelial surface that could be ulcerated in the periodontal pocket, periodontitis can be regarded as a constant pathogenic and inflammatory challenge at a systemic level.

Pre-term birth, as defined by the WHO, refers to all live births before 37 weeks of completed gestation. Pre-term birth can be further classified according to gestational period or age: extremely pre-term (<28 weeks), very pre-term (≥28 but <32 weeks), moderate pre-term (≥32 but <37 weeks); as determined by the last menstrual day and by ultrasound.6 Low birth weight has been defined as less than 2,500 g and is believed to reflect the health status of the immediate community into which the infant is born.7 A very low birth weight refers to a weight below 1,500 g.8 In developing countries, low birth weight is usually caused by intrauterine growth restriction, whereas in developed countries it is more often as a result of pre-term birth.1

Paradoxically, there are cases where infants born at > 37 weeks present with low birth weight, whilst in some cases premature infants may have a weight ≥2,500 g.12 Infected during pregnancy have been strongly associated with pre-term birth and/or low birth weight (PTLBW). Maternal genito-urinary tract infections have been most frequently associated with such outcomes.9 However, there have been a number of cases with these adverse outcomes, displaying high levels of TNF-α and PGE in the amniotic fluid, but in the absence of maternal genito-urinary tract infections. Such observations are suggestive of an infection arising from elsewhere in the body.8

An estimated 80% of all births at less than 30 weeks of gestation are due to infections which precede the pregnancy complications leading to pre-term birth.10 Recognition of risk factors facilitates the identification of those at risk of adverse pregnancy outcomes,1 and may thus inform policies on the prevention of PTLBW. This paper therefore seeks to review the literature on the association between periodontitis and PTLBW, highlighting the possible mechanisms of the association and the significance of oral health care in the prevention of these unfavourable outcomes of pregnancy.
pregnancy outcomes like pre-term birth, low birth weight and the combination of the two. The relationship between periodontitis and pregnancy is proposed to be bi-directional with one having an effect on the other.19

**EFFECT OF PREGNANCY ON PERIODONTITIS**

Pregnant women are said to be more susceptible to the development of periodontitis as a result of hormonal changes. The levels of ovarian hormones, namely, oestrogen and progesterone increase during pregnancy. These hormones increase, starting from the second month of the pregnancy and continue to rise until the eighth month, after which their levels begin to decline. This rise and fall pattern of the hormones coincides with the onset and peak of the gingival inflammation observed in pregnancy. When, after the eighth month, the levels of these hormones drop rapidly, a simultaneous reduction in the gingival inflammation may be observed. The timing and occurrence of these gingival changes in step with the fluctuations in hormone levels give credibility to the notion that hormonal changes cause the gingival inflammation.16 An increase in oestrogen and progesterone levels increases the risk of gingival inflammation. A number of mechanisms have been proposed for the effect of hormonal changes on gingival tissues: the known effects of these hormones is to increase capillary dilatation and permeability which would account for the gingival inflammation.17

Whilst this may explain only the inflammatory changes, unattended gingivitis may progress to periodontitis.

**Type and amount of subgingival flora present during pregnancy**

Kornman and Loesche and a study by Li et al showed a significant increase in the ratio of anaerobic to aerobic bacteria, particularly from the thirteenth to the sixteenth weeks of pregnancy18 which remained high till the third trimester. The subgingival concentrations of *Bacteroides intermedius* (now *Prevotella intermedia*) increased significantly, up to five-fold at the peak of gingival bleeding.16 Oestrogen and progesterone stimulate bacterial growth causing a shift in bacterial flora,18 and specifically substitute for menadione as an essential growth factor for *B. intermedius* with a resultant increased growth activity.16

Jensen et al observed an increase in the gingival crevicular fluid flow and in gingival score indices amongst pregnant women compared with non-pregnant controls. This difference was ascribed to alterations in the composition of the subgingival plaque, influenced by sex hormones.19

**Immunosuppression**

The role of oestrogen and progesterone among other hormones is to suppress the mother’s immune system to prevent the rejection of the foetus. Whilst the underlying mechanisms of immunosuppression are not clear, a number of plausible suggestions have been put forth. These include an altered T cell response and an impaired lymphocyte proliferation,20 blockage of K+ channels21 and depression of cell-mediated immunity.22

**Impaired collagen repair**

Pregnancy, through alterations in hormonal levels, alters the rate and pattern of collagen production in the gingivae with a resultant reduction in the body's ability to repair and maintain the gingival tissues. This compromised collagen repair may also be effected through folate deficiency caused by oestrogen and progesterone.8

**EFFECT OF PERIODONTITIS ON PREGNANCY OUTCOMES**

Periodontitis is claimed to be strongly associated with PTLBW, as it has been diagnosed more frequently among mothers with pre-term and/or low birth weight infants when compared with mothers who delivered full-term infants with normal birth weight.23 Mechanisms proposed and described to account for this association include the following:

**Common risk factors**

Specific bacteria within the dental plaque are etiologic for periodontitis. Whilst these bacteria may initiate the disease process, they alone are not sufficient to effect, to propagate and to sustain periodontitis. A susceptible host is necessary for disease to occur. Host susceptibility is influenced by certain factors, which may be environmental, genetic or systemic. These may include tobacco and alcohol use, stress, immunosuppression and diabetes, to name a few, and are referred to as risk factors. A risk factor by definition is that which increases the chances of an individual to contract a disease, or makes one more likely to have a disease. Periodontitis shares some of these risk factors with PTLBW e.g. tobacco and alcohol abuse, genetic factors, and stress. Therefore given this analogy, the possibility of a mother with periodontitis giving birth pre-term, or giving birth to an infant with low birth weight is quite high but is mostly likely a coincidence.

**Bacteraemia and inflammation**

Periodontitis is a common clinical finding amongst many pregnant women and is believed to provide an oral route of infectious burden to the foetal placental unit.1 The concept is not new that periodontal pathogens, their shed virulence factors and/or inflammatory cytokines from the periodontal pocket may gain access into the bloodstream and disseminate through the body. Miller reported this idea in 1891 when he published the theory of focal infection, but it became dormant due to lack of scientific evidence.25 Collins et al in the early 1990s rehashed the case and hypothesised that periodontitis could be a reservoir for bacteria that can access the systemic circulation and trans-locate into the foetal-placental unit where it may induce complications in the pregnancy.26 This bacteraemia can be elicited by processes as simple as chewing or tooth brushing. Translocated bacteria reaching a distant site with a susceptible environment trigger an inflammatory response with resultant complications.
This concept is supported by animal and clinical studies. Collins et al conducted a number of animal studies to test the hypothesis that periodontitis as an oral infection represents a significant source of infection and inflammation which would cause bacteraemia and pregnancy complications. The result of these studies in hamsters showed that *P. gingivalis* can reduce the foetal weight by over 15–16%, with an increase in PGE2 and TNF-α. Through these studies they also showed reduction of 22.5% in the foetal mean weight of hamsters that received plaque promoting diet and the exogenous *P. gingivalis* orally. These results suggest that low level oral infections can possibly produce adverse pregnancy outcomes.26–28

Forty percent of all pregnancies are associated with a certain degree of foetal IgM antibody response to a pathogen of maternal oral origin. Results from clinical studies on foetal cord blood from spontaneous pre-term births support this concept, having shown a significant increase of in-utero IgM antibody response which was specific to numerous periodontal pathogens including *P. gingivalis*. This antibody response induced an inflammatory response at the foetal-placental unit with resultant pre-term birth.27–29 A similar immuno-inflammatory response may ensue in the foetal circulation if the pathogens, their virulent components or inflammatory cytokine cross the placenta.2

Periodontitis is associated with increased serum levels of C-reactive protein (CRP), an acute phase protein produced by hepatocytes in response to the circulating pro-inflammatory cytokines including TNF-α and IL-1 that enter the circulation from the damaged periodontium.3 The CRP is an important diagnostic marker for clinical infections. A few possible mechanisms through which an inflammatory response can effect PTLBW have been proposed:2

- The normal blood flow between the foetus and the mother may be disrupted as a result of the structural damage of the placenta. This disruption affects the maternal blood pressure, thus leading to pre-eclampsia which may necessitate planned pre-term delivery.
- Whilst pro-inflammatory cytokines are necessary for normal parturition to occur, premature increase in production of these cytokines, as occurs in an inflammatory response due to infection, may contribute to pre-term rupture of the membranes and contraction of the uterus with resultant pre-term delivery or miscarriage depending on the severity of the response.
- In the foetus, local inflammatory response may cause structural damage of tissue and organ systems. The foetus may or may not survive the perinatal period, and if it does, it may have deficiencies that may negatively impact on the quality of life, depending on the extent of the tissue damage.

Whilst these mechanisms may sound plausible, further in-depth investigations are necessary for their confirmation.

Lin et al in their study on mice found that *P. gingivalis* restricted foetal growth and even resulted in foetal death in some. Their proposed mechanism was a shift in the TH1/TH2 ratio which led to TH1 cytokine dominance with adverse outcomes as a result of increased production of pro-inflammatory cytokines. In another study, toll like receptors, in particular TLR4 was shown to mediate an inflammatory response to specific pathogens in murines, which led to restricted foetal growth and foetal resorption. These receptors were over-expressed on trophoblast surfaces in mice that experienced these adverse outcomes.21,22

Mice that were deficient in TLR4 were protected against pre-term birth induced by bacterial influences and by LPS.

**Apoptosis**

Besides the inflammatory response concept, *P. gingivalis* can, through haematogenous spread, also invade trophoblasts in the placenta and cause apoptosis and cell cycle arrest, which may synergistically contribute to PTLBW.23

**EFFECT OF PERIODONTAL THERAPY ON PREGNANCY OUTCOMES**

The systemic impact of periodontal inflammation has been hypothesised as a plausible mechanism. It is on this basis that studies to assess the effect of periodontal therapy on levels of the biomarkers for inflammation and pregnancy outcomes have been conducted.

Interventional studies assessing the impact of periodontal therapy on pregnancy outcomes have yielded contradictory results. The hypothesis for these studies is that if periodontitis has an effect on the pregnancy outcomes, its treatment amongst pregnant women should therefore reduce the incidence of PTLBW. A number of studies have indeed reported a reduction in the incidence of PTLBW in women on whom periodontal therapy had been undertaken.34,35,36,37

Jeffcoat et al in their pilot study on women between gestational weeks 21 and 25, concluded that scaling and root planing in pregnant women with periodontitis may result in the reduction of pre-term births.35 These results were similar to those reported in an earlier study by Lopez on pregnant women in the same gestational period.36

Offenbacher et al reported a significant reduction in pre-term births following periodontal therapy.34 Whilst Mitchell-Lewis et al also reported similar findings, these were not significant in their study.37 This could be linked to the age of the subjects, who were in their teens. Low maternal age is a known risk factor for adverse pregnancy outcomes.

A number of studies reported a reduction in the levels of the biomarkers in either the GCF or serum following periodontal therapy.34,36,38,39,40,41

Taylor et al and Michalowicz et al in their studies reported no reduction in PTLBW and no significant differences in the levels of biomarkers evaluated following periodontal therapy.42,43,44 The findings of this study, conducted in 2006, could have been influenced by the fact that the control group had regular examination recalls. Though there was no active treatment carried out in this group, their awareness of the recalls could have influenced their oral hygiene practices.

In the light of these conflicting results, the evidence in the literature is therefore inconclusive on the effect of periodontal therapy on pregnancy...
outcomes. More interventional studies are needed to validate the association between periodontitis and adverse pregnancy outcomes.

**RANDOMISED TRIALS AND PROGRESSIVE COHORT STUDIES**

The evidence from randomised trials, case-control and cohort studies that assessed the association between periodontitis and pregnancy outcomes including systematic reviews is conflicting.4,45,46,47,48,49,50 Most of the studies, however, reported a positive association. In a systematic review, Xiong et al reported that out of 22 cohort and case control studies reviewed, 17 found a positive association between periodontitis and adverse pregnancy outcomes and the remaining five found no association.45 Four out of the 17 positive studies reported periodontitis as an independent risk factor for PTLBW.38,50,51,52 In contradiction, two recent studies have reported no association between periodontitis and PTLBW.44,53

The challenge most often encountered in systematic reviews was the heterogeneity of the studies conducted, which sometimes made meta-analytical studies impossible. The differences ranged from population sample and case definitions, to the quality of the methodology, and where the variations were great the intensity of the association was small. Heterogeneity in methodology also reduced the pool of the studies reviewed.

**CONCLUSION**

Overall, the majority of studies have shown that maternal periodontitis is associated with PTLBW, and this association may even be independent of other risk factors. This association has further been supported by studies on interventional studies. The association however, does not imply causality and should be interpreted with caution. Notwithstanding the fact that findings contrary to this association have been reported, more studies with better methodologies and inclusive of other adverse pregnancy outcomes like stillbirths, late miscarriages etc. should be conducted.

Given the impact of PTLBW on the mother, the live births that survive, families and communities, the authors recommend that oral health care should be incorporated into existing maternal and child health care programmes in the private and public sectors. Bearing in mind that the results from intervention studies are inconclusive, focus should be placed on prevention of the onset of disease.

**REFERENCES**


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Hain Lifescience SA held its first dental study group on 26 July 2014 at its offices in Corporate Park, Midrand. The speakers, company, and of course the refreshments were excellent. Dr PJ Lofstedt, a dentist from Johannesburg, gave a very informative talk about the importance of peri-pathogenic organisms and the role of molecular testing in the treatment plan for periodontitis. Dr J Fourie is a specialist in oral medicine and periodontics and her presentation shed some light on the nature of periodontal infections. An oral hygienist, Christine de Sousa explained oral hygiene in layman’s terms and also displayed her new products from ‘Rollys’.

The study group clearly showed that there is a need to discuss ongoing developments in the dental diagnostic field and to educate patients on the risks associated with periodontitis. A demonstration of Hain Lifescience’s new periodontal tests, the Microident 11 and IL-1, was performed. These tests improved the approach to the treatment plan and also the final outcome of patients’ oral hygiene. The feedback from the users in the study group, on that day, confirmed this.

The day was a great success and dental professionals should be on the lookout for more Hain Lifescience study groups because this was definitely the first of many to come.

For more queries or demonstrations please contact Hain Lifescience at chrisna@hain-lifescience.co.za

**NOMADS**

**13 to 14 November 2014**

**Stand 26**

Are you prone to periodontitis?

Come and find out with the new **GenoType® IL-1**

Our new test will change the way you treat your patients and how your patients see you...

**FREE**

The first 75 dental practitioners will be tested for free!!!
GUIDELINES FOR THE SELECTION OF TOOTH WHITENING PRODUCTS AMONGST THOSE AVAILABLE ON THE MARKET

SUMMARY
Background: Several tooth whiteners are available on the market, and the ideal choice should be determined by efficacy and optimal clinical results.

Objectives: The purpose of this study was to compare the reported clinical success rates of different tooth whitening products.

Search strategy: The relevant literature (1998–2011) was studied, using as sources the databases: Google Scholar, Science Direct, Medline and Pubmed.

Selection criteria: The material was clearly identified, the manufacturers’ instructions were respected and the sample size stated.

Results and conclusions: This descriptive report on 49 papers focuses on the total colour change, measured with a calibrated shade guide and also numerically (colourimeter, chromameter or spectrophotometer), the relapse of the colour change and tooth sensitivity. In general, the dentist supervised at-home bleaching and the in-office treatment gave approximately the same initial percentage improvement of tooth whitening.

However, the relapse after a four week or longer period was significantly higher for the in-office treatment. The treatment of choice should be a dentist supervised at-home bleaching product which generally contains ~10% carbamide peroxide applied over about 14 days for about eight hours per night. Tooth sensitivity should not be a general problem although some subjects might choose to discontinue treatment as a result of sensitivity.

BACKGROUND

Dentists and oral hygienists in clinical practice are faced with a large number of tooth whiteners available and advertised on the market. Furthermore, they are bombarded with results based on laboratory studies and to a lesser extent on clinical studies. How then does the oral health professional select a tooth whitener which will be effective and provide optimal clinical results? Important also is the extent of colour relapse over time.

Peroxide is the chemical most frequently used as a tooth whitening agent with two types being generally employed: i.e. hydrogen peroxide (H₂O₂) or carbamide peroxide (CH₆N₂O₃). Due to the instability of hydrogen peroxide it is mostly added to the whitener just before the application process, while carbamide peroxide is the more stable oxidising agent and can be included in the whitener itself. Thus, the hydrogen peroxide can be applied directly on the tooth surface as such or is produced from carbamide peroxide which dissociates into hydrogen peroxide and urea upon contact with water. Urea further breaks down into ammonia and carbon dioxide.

It has been reported that a 10% carbamide peroxide solution can produce only about 3–3.35% hydrogen peroxide. Hydrogen peroxide is a very strong oxidising agent. When it decomposes it forms free radicals which are responsible for the strong bleaching effect on organic and inorganic chemicals in and on enamel and dentine. However, more of the stronger per-hydroxyl radical is formed in alkaline mediums with a resulting higher bleaching effect on teeth.

To the public, the most important aspects of tooth whiteners are the effectiveness of the whitener, how long-lasting the effect is, the cost of the treatment and the duration of the required treatment. Therefore, if money is not an issue, the in-office treatment would basically be the treatment of choice, since the resultant whitening effect can be observed just after the chair session.

Tooth-bleaching or tooth-whitening is mainly performed according to four different protocols: 1) dentist/oral hygienist-supervised at-home bleaching, 2) in-office bleaching, 3) over-the-counter whitening products for self-application and 4) combination therapy (in-office followed by at-home treatment). In general, the most effective would be the first two, as the peroxide concentrations and application time of over-the-counter products are simply too low. In contrast, the peroxide concentrations (~35%) of the in-office bleaching whiteners are high (Table 1) and applied for short bursts of time (~30 minutes) which might be repeated in the same session (or occasionally over more sessions) to show the effect before the patient goes home. Alternatively, the dentist/oral hygienist-supervised at-home bleaching process normally makes use of lower peroxide concentrations (~10% carbamide peroxide) and patient self-application at home, mostly over-night and over several days, for a good result.
<table>
<thead>
<tr>
<th>Study #</th>
<th>Reference</th>
<th>Product Description</th>
<th>% CP</th>
<th>% HP</th>
<th>Category</th>
<th>Application</th>
<th>Tooth Sensitivity</th>
<th>% Colour Improvement</th>
<th>Colour Relapse</th>
<th>After 4 weeks</th>
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<td>1</td>
<td>Browning, 2008</td>
<td>Opalescence PF (plus nitrate and fluoride)</td>
<td>10%</td>
<td>-</td>
<td>At-home</td>
<td>2 weeks/6 hrs per night</td>
<td>45%</td>
<td>88%</td>
<td>14%</td>
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<tr>
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<td>Opalescence formulation (F)</td>
<td>10%</td>
<td>-</td>
<td>At-home</td>
<td>2 weeks/6 hrs per night</td>
<td>62%</td>
<td>83%</td>
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<td>Opalescence Tooth Whitening Gel</td>
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<td>-</td>
<td>At-home</td>
<td>2 weeks/8 hrs per night</td>
<td>Mild</td>
<td>73%</td>
<td>41%</td>
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<tr>
<td>4</td>
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<td>Opalescence F</td>
<td>15%</td>
<td>-</td>
<td>At-home</td>
<td>2 weeks/8 hrs per night</td>
<td>-</td>
<td>69%</td>
<td>18%</td>
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<td>15%</td>
<td>-</td>
<td>At-home</td>
<td>2 weeks/8 hrs per night</td>
<td>Mild</td>
<td>67%</td>
<td>24%</td>
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<td>6</td>
<td>Mokhlis, 2000</td>
<td>Day White</td>
<td>-</td>
<td>7.5%</td>
<td>At-home</td>
<td>2 weeks/1 hr twice a day</td>
<td>Mild</td>
<td>67%</td>
<td>13%</td>
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<td>20%</td>
<td>-</td>
<td>At-home</td>
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<td>-</td>
<td>67%</td>
<td>11%</td>
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<td>8</td>
<td>Zekonis, 2003</td>
<td>Opalescence Tooth Whitening Gel</td>
<td>10%</td>
<td>-</td>
<td>At-home</td>
<td>2 weeks/8 hrs per night</td>
<td>Mild</td>
<td>67%</td>
<td>46%</td>
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<td>2 weeks/8 hrs per night</td>
<td>-</td>
<td>64%</td>
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<td>16%</td>
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<td>2 weeks/8 hrs per night</td>
<td>Mild</td>
<td>63%</td>
<td>55%</td>
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<tr>
<td>11</td>
<td>Deliperi, 2004</td>
<td>Opalescence Xtra Boost plus Opalescence PF</td>
<td>10%</td>
<td>38%</td>
<td>In-office &amp; At-home</td>
<td>3 days (30 min in-office/60 min at home)</td>
<td>None</td>
<td>56%</td>
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<td>12</td>
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<td>-</td>
<td>38%</td>
<td>In-office</td>
<td>x2/15 min</td>
<td>-</td>
<td>54%</td>
<td>-</td>
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<td>Matis, 2007</td>
<td>Brite Smile</td>
<td>-</td>
<td>15%</td>
<td>In-office</td>
<td>3x20 min</td>
<td>-</td>
<td>54%</td>
<td>68%</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>Gurgan, 2009</td>
<td>Laser-White 1-Laser Smile</td>
<td>-</td>
<td>37%</td>
<td>In-office</td>
<td>x3/8 min</td>
<td>Mild</td>
<td>54%</td>
<td>-</td>
<td></td>
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<tr>
<td>15</td>
<td>Gurgan, 2005</td>
<td>By White-Biowhite</td>
<td>-</td>
<td>38%</td>
<td>In-office</td>
<td>x2/20 min</td>
<td>-</td>
<td>53%</td>
<td>-</td>
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<tr>
<td>16</td>
<td>Deliperi, 2004</td>
<td>Opalescence Xtra plus Opalescence PF</td>
<td>10%</td>
<td>35%</td>
<td>In-office &amp; At-home</td>
<td>3 days (30 min in-office/60 min at home)</td>
<td>None</td>
<td>53%</td>
<td>-</td>
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<tr>
<td>17</td>
<td>Gurgan, 2009</td>
<td>Remnewhite-Remecure</td>
<td>-</td>
<td>35%</td>
<td>In-office</td>
<td>x3/20 min</td>
<td>-</td>
<td>53%</td>
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<tr>
<td>18</td>
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<td>ACP containing bleaching gel</td>
<td>16%</td>
<td>-</td>
<td>At-home</td>
<td>2 weeks/3 hrs per day</td>
<td>Mild</td>
<td>51%</td>
<td>-</td>
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<td>19</td>
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<td>10%</td>
<td>-</td>
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<tr>
<td>20</td>
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<td>Nite White Excel 3 Regular</td>
<td>16%</td>
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<td>2 weeks/3 hrs per day</td>
<td>-</td>
<td>48%</td>
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<td>Nupro Gold bleaching gel</td>
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<td>2 weeks/at least 4 hrs per night</td>
<td>-</td>
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<td>1 week/8 hrs per night</td>
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<td>47%</td>
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<td>PolaOffice</td>
<td>-</td>
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<td>x3/12 min</td>
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<td>69%</td>
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<td>-</td>
<td>35%</td>
<td>In-office</td>
<td>x3/15 min</td>
<td>-</td>
<td>44%</td>
<td>54%</td>
<td></td>
</tr>
<tr>
<td>26</td>
<td>Cibrika, 1999</td>
<td>StarBrite</td>
<td>35%</td>
<td>-</td>
<td>In-office</td>
<td>x2/3x10 min</td>
<td>Mild</td>
<td>44%</td>
<td>32%</td>
<td></td>
</tr>
<tr>
<td>27</td>
<td>Matis, 2007</td>
<td>Opalescence</td>
<td>10%</td>
<td>-</td>
<td>At-home</td>
<td>2 weeks/8 hrs per night</td>
<td>-</td>
<td>44%</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>28</td>
<td>Matis, 2007</td>
<td>Niveous</td>
<td>-</td>
<td>25%</td>
<td>In-office</td>
<td>3x15 min</td>
<td>-</td>
<td>43%</td>
<td>72%</td>
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</tr>
<tr>
<td>29</td>
<td>Matis, 2007</td>
<td>ArcBrite</td>
<td>-</td>
<td>30%</td>
<td>In-office</td>
<td>3x20 min</td>
<td>-</td>
<td>41%</td>
<td>53%</td>
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<tr>
<td>30</td>
<td>Matis, 2007</td>
<td>Zoom!</td>
<td>-</td>
<td>25%</td>
<td>In-office</td>
<td>3x20 min</td>
<td>-</td>
<td>39%</td>
<td>50%</td>
<td></td>
</tr>
<tr>
<td>31</td>
<td>Matis, 2007</td>
<td>Accelerated</td>
<td>-</td>
<td>40% and 30%</td>
<td>In-office</td>
<td>5x3 min</td>
<td>-</td>
<td>39%</td>
<td>81%</td>
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<tr>
<td>32</td>
<td>Li, 2005</td>
<td>Opalescence ThreeWhite</td>
<td>-</td>
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<td>10 days /1 hr daily</td>
<td>Mild</td>
<td>38%</td>
<td>-</td>
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<tr>
<td>33</td>
<td>Ausschil, 2005</td>
<td>Opalescence PF</td>
<td>10%</td>
<td>-</td>
<td>At-home</td>
<td>1 week/8 hrs per night</td>
<td>-</td>
<td>38%</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>34</td>
<td>Ausschil, 2005</td>
<td>Opalescence Xtra Boost</td>
<td>-</td>
<td>38%</td>
<td>In-office</td>
<td>3x15 min</td>
<td>-</td>
<td>38%</td>
<td>-</td>
<td></td>
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<tr>
<td>35</td>
<td>Ausschil, 2005</td>
<td>Whitenstrip</td>
<td>-</td>
<td>5.3%</td>
<td>At-home</td>
<td>2 weeks/30 min 2x daily</td>
<td>-</td>
<td>38%</td>
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<td>Zantner, 2006</td>
<td>Odo-med3 gel</td>
<td>-</td>
<td>-</td>
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<td>0%</td>
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<td>37</td>
<td>de la Penya, 2006</td>
<td>Opalescence Ultradent</td>
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<td>-</td>
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<td>4 weeks/3 hrs daily</td>
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<td>-</td>
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<td>38</td>
<td>de la Penya, 2006</td>
<td>FKS (Kin Lab)</td>
<td>-</td>
<td>3.5%</td>
<td>At-home</td>
<td>4 weeks/3 hrs daily</td>
<td>Mild</td>
<td>31%</td>
<td>-</td>
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<tr>
<td>39</td>
<td>Calayayu, 2009</td>
<td>Vivastyle Paint on Plus</td>
<td>-</td>
<td>6%</td>
<td>At-home</td>
<td>10 days/10 min 1x daily</td>
<td>-</td>
<td>30%</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>40</td>
<td>Calayayu, 2009</td>
<td>Vivastyle Paint on Plus</td>
<td>-</td>
<td>6%</td>
<td>In-office</td>
<td>2 sessions 1 week apart/5x10 min</td>
<td>-</td>
<td>29%</td>
<td>-</td>
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<td>41</td>
<td>Kugel, 2000</td>
<td>Crest Whitestrips</td>
<td>-</td>
<td>5.3%</td>
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<td>2 weeks/30 min 2x daily</td>
<td>-</td>
<td>28%</td>
<td>-</td>
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<td>Study</td>
<td>Reference</td>
<td>Product</td>
<td>% CP</td>
<td>% HP</td>
<td>Category</td>
<td>Application</td>
<td>Tooth Sensitivity</td>
<td>% Colour Improvement</td>
<td>% Colour relapse after 4 weeks</td>
<td></td>
</tr>
<tr>
<td>-------</td>
<td>----------------------------</td>
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<td>------</td>
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<td>------------------</td>
<td>----------------------</td>
<td>-------------------------------</td>
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<tr>
<td>42</td>
<td>Zantner, 2006²⁵</td>
<td>Colgate Simply White</td>
<td></td>
<td>5.9</td>
<td>At-home</td>
<td>2 weeks/15 min 2x daily</td>
<td>None</td>
<td>28%</td>
<td>3%</td>
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<td>43</td>
<td>Abu Alenain, 2009²⁹</td>
<td>Opalescence Treswhite</td>
<td></td>
<td>9%</td>
<td>At-home</td>
<td>1 week/60 min per day</td>
<td>Mild</td>
<td>28%</td>
<td>-</td>
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<tr>
<td>44</td>
<td>Matis, 2007²⁶</td>
<td>Illumine</td>
<td></td>
<td>15%</td>
<td>In-office</td>
<td>x3/20 min</td>
<td>-</td>
<td>25%</td>
<td>18%</td>
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<tr>
<td>45</td>
<td>Cibrika, 1999²¹</td>
<td>Nite White Excel</td>
<td>10%</td>
<td></td>
<td>At-home</td>
<td>2 weeks/8 hrs per night</td>
<td>-</td>
<td>25%</td>
<td>-</td>
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<tr>
<td>46</td>
<td>Hannig, 2007²⁷</td>
<td>Whitestrips</td>
<td></td>
<td>6%</td>
<td>At-home</td>
<td>2 weeks/2x daily for 30 min</td>
<td>Mild</td>
<td>24%</td>
<td>-</td>
<td></td>
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<tr>
<td>47</td>
<td>Hannig, 2007²⁷</td>
<td>Vivadent Vivastyle</td>
<td>10%</td>
<td></td>
<td>At-home</td>
<td>2 weeks/tx daily for 60 min</td>
<td>Mild</td>
<td>24%</td>
<td>-</td>
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<tr>
<td>48</td>
<td>Abu Alenain, 2009²⁹</td>
<td>White-Smile</td>
<td>10%</td>
<td></td>
<td>At-home</td>
<td>1 week/2 hrs daily</td>
<td>Mild</td>
<td>21%</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>49</td>
<td>Al Shethri, 2003³¹</td>
<td>Star Brite</td>
<td></td>
<td>35%</td>
<td>In-office</td>
<td>2 treatments 1 week apart/ x3 daily for 10 min</td>
<td>Mild</td>
<td>21%</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>50</td>
<td>Bernardon, 2010²²</td>
<td>Whiteness HP max FGM</td>
<td></td>
<td>35%</td>
<td>In-office</td>
<td>2 sessions, 3 applications per session, 15 day interval</td>
<td>-</td>
<td>21%</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>51</td>
<td>Al Shethri, 2003³¹</td>
<td>Opalescence Xtra Boost</td>
<td></td>
<td>38%</td>
<td>In-office</td>
<td>2 treatments 1 week apart/ x3 daily for 10 min</td>
<td>Mild</td>
<td>20%</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>52</td>
<td>Bernardon, 2010²²</td>
<td>Whiteness HP max FGM</td>
<td></td>
<td>35%</td>
<td>In-office</td>
<td>2 sessions/3 applications of 15 min/15 day interval</td>
<td>-</td>
<td>19%</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>53</td>
<td>Bernardon, 2010²²</td>
<td>Whiteness Perfect FGM</td>
<td>10%</td>
<td></td>
<td>At-home</td>
<td>2 weeks/8 hrs per night</td>
<td>19%</td>
<td>0%</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>54</td>
<td>Leonard, 2001³³</td>
<td>Nightguard vital bleaching</td>
<td>10%</td>
<td></td>
<td>At-home</td>
<td>2 weeks/8 hrs per night</td>
<td>Mild</td>
<td>19%</td>
<td>0%</td>
<td></td>
</tr>
<tr>
<td>55</td>
<td>Mereless, 2008³⁴</td>
<td>Whiteness Perfect</td>
<td>10%</td>
<td></td>
<td>At-home</td>
<td>3 weeks/2 hrs daily</td>
<td>-</td>
<td>14%</td>
<td>9%</td>
<td></td>
</tr>
<tr>
<td>56</td>
<td>Mereless, 2008³⁴</td>
<td>Whiteness Perfect</td>
<td>16%</td>
<td></td>
<td>At-home</td>
<td>3 weeks/2 hrs daily</td>
<td>-</td>
<td>13%</td>
<td>2%</td>
<td></td>
</tr>
<tr>
<td>57</td>
<td>dos Santos, 2008³⁸</td>
<td>Opalescence PF</td>
<td>10%</td>
<td></td>
<td>At-home</td>
<td>3 weeks/8 hrs per night</td>
<td>36%</td>
<td>13%</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>58</td>
<td>Braun, 2006³⁸</td>
<td>Voco CP solution</td>
<td></td>
<td>(Control)</td>
<td>At-home</td>
<td>1 week/2 hrs daily</td>
<td>10%</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>59</td>
<td>Abu, 2009³⁹</td>
<td>CleverWhite over-the-counter</td>
<td>6%</td>
<td></td>
<td>At-home</td>
<td>1 week/30 min per day</td>
<td>Mild</td>
<td>8%</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>60</td>
<td>Braun, 2006³⁸</td>
<td>Voco CP solution</td>
<td>10%</td>
<td></td>
<td>At-home</td>
<td>1 week/2 hrs daily</td>
<td>8%</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>61</td>
<td>Braun, 2006³⁸</td>
<td>Voco CP solution</td>
<td>17%</td>
<td></td>
<td>At-home</td>
<td>1 week/2 hrs daily</td>
<td>7%</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>

**Objectives**

The objective of this study was firstly to evaluate and compare the clinical success rates of different commercially available tooth whitening products as reported in selected clinical trials in which the application was effected according to the instructions of the manufacturer. Secondly, to thereby enable clinicians to make an informed decision regarding the choice of the products which are most clinically effective.

**Search Strategy**

This was accomplished by means of a comprehensive study of the literature which had reported on relevant clinical trials between 1998 and 2011, using the databases: Google Scholar, Science Direct, Medline and Pubmed respectively, with any combination of the keywords: tooth whitening, tooth bleaching, clinical studies, at-home, and in-office. The search was performed only on published, peer-reviewed articles.

**Criteria for Selection of Studies**

The papers included were clinical studies in which:
- the whiteners had been applied according to the manufacturers’ instructions. (The performance of the product could then be fairly evaluated in the circumstances under which it was expected to be applied in practice);
- the brand name of the tooth whitener was clearly specified;
- the sample size had been recorded.

Reporting the degree of relapse was not a criterion for inclusion, nor was assessment of tooth sensitivity, although, in the small number of cases where it was measured, the latter data were recorded on a word ordinal scale and in some cases as a percentage.

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**Figure 1:** Representation of colour solid for L*a*b* colour space from Minolate.
ASSESSING COLOUR CHANGES

Basically, there are two different ways to measure the effect of tooth whiteners, namely, by means of matching with a calibrated shade guide or numerically (colourimeter, chromameter or spectrophotometer).

Some papers provided the shade guide assessments and some, numerical values, while a few provided both. These varied methods of reporting complicated the summary of the research results. Evaluations using a shade guide are subjective, while the numerical measurements are objective, providing more reliable and accurate results.1,2 Most important are the improved accuracy and the quantification of colours by measurement in a three dimensional colour space, of which the L*a*b* (also known as the CIELAB) is presently the most popular for the measurement of tooth colour (Figure 1). In this space L* indicates lightness/darkness (white/black), a* varies from green (negative side) to red (positive side), while the b* value varies from blue (negative side) to yellow (positive side).4 (The asterisk is used to differentiate the CIELAB system from previous colour space descriptions.) As with the shade guide, colour change measurements with the spectrophotometer can be given in one value, namely the ∆E*ab (Minolta), where ∆E*ab = [(ΔL*)2 + (Δa*)2 + (Δb*)2]1/2 and ΔL*, Δa* and Δb* provide the changes which occurred in these components.

MATERIALS AND METHODS

Based on the above selection criteria a total of ~49 full-length published articles were included. Most of these articles reported on two or more studies on different products and some 45 products had been tested. All data were recorded in Excel® tables. The variables for which data were sought are as follows: Product name; percentage carbamide peroxide (CP) and hydrogen peroxide (HP); Category (in-office or at-home bleaching); Application method; Tooth sensitivity; Percentage colour improvement; Total colour change obtained immediately after the treatment process; Percentage colour relapse after 4 weeks; and ∆E*ab after treatment. Where the percentage colour improvement or relapse was not given, it was calculated from the results. These results were summarised in two tables (1 and 2). Table 1 reflects data according to shade guide measurements and Table 2 those according to numerical values (spectrophotometer, chromameter and colourimeter).

Each study included a number of trials from which the specific authors had calculated an average for their sample for colour changes. To gain an impression of the average efficacy in tooth whitening achieved by the products under test, the reported averages were added and an average of the averages derived. These data were used to compare the general efficacy of in-office and at-home treatments.

RESULTS

Table 1 provides a summary of the results reported (or calculated) in the selected clinical studies assessing the efficacy of tooth bleaching through shade guide assessments.3-36 The data were sorted from the highest to lowest percentage perceived colour improvement just after bleaching according to column 9 and the studies were numbered (column 1). The percentage colour relapse after a four week or longer period had been assessed and was recorded in column 10. In a few studies where both the shade guide and ∆E*ab values were given in a study, the average percentage improvement of the two was calculated and noted (Table 1). Table 22, 4,9-13,15,16,25,31,32,34,37-45 includes the same columns as for Table 1, except Column 9 now shows the ∆E*ab values (total colour change) which indicate the colour improvement as measured numerically and column 10 the percentage relapse as calculated from the ∆E*ab values. This table was also sorted according to highest to lowest ∆E*ab values (column 9) immediately after treatment.

<table>
<thead>
<tr>
<th>Study #</th>
<th>Reference</th>
<th>Product</th>
<th>% CP</th>
<th>% HP</th>
<th>Category</th>
<th>Application</th>
<th>Tooth Sensitivity</th>
<th>Colour Improvement</th>
<th>% Colour Relapse after 4 weeks+</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Zekonis, 200336</td>
<td>Opalescence Tooth Whitening gel</td>
<td>10%</td>
<td>-</td>
<td>At-home</td>
<td>2 weeks/8 hrs per night</td>
<td>Mild</td>
<td>12.32</td>
<td>46</td>
</tr>
<tr>
<td>2</td>
<td>Matis, 200030</td>
<td>Opalescence F</td>
<td>15%</td>
<td>-</td>
<td>At-home</td>
<td>2 weeks/8 hrs per night</td>
<td>-</td>
<td>11.03</td>
<td>-</td>
</tr>
<tr>
<td>3</td>
<td>Matis, 200731</td>
<td>Opalescence PF</td>
<td>15%</td>
<td>-</td>
<td>At-home</td>
<td>2 weeks/8 hrs per night</td>
<td>Mild</td>
<td>9.57</td>
<td>57</td>
</tr>
<tr>
<td>4</td>
<td>Tsrburu, 200532</td>
<td>Polanight</td>
<td>10%</td>
<td>-</td>
<td>At-home</td>
<td>2 weeks/8 hrs per night</td>
<td>Mild</td>
<td>9.23</td>
<td>-</td>
</tr>
<tr>
<td>5</td>
<td>Mokhli, 200233</td>
<td>Day White</td>
<td>-</td>
<td>7.50%</td>
<td>At-home</td>
<td>2 weeks/1 hour twice a day</td>
<td>Mild</td>
<td>9.2</td>
<td>-</td>
</tr>
<tr>
<td>6</td>
<td>Matis, 200034</td>
<td>Opalescence Dental Whitening Agent</td>
<td>10%</td>
<td>-</td>
<td>At-home</td>
<td>2 weeks/8 hrs per night</td>
<td>-</td>
<td>8.79</td>
<td>-</td>
</tr>
<tr>
<td>7</td>
<td>Matis, 199835</td>
<td>Opalescence Whitening Gel</td>
<td>10%</td>
<td>-</td>
<td>At-home</td>
<td>2 weeks/8 hrs per night</td>
<td>Mild</td>
<td>8.6</td>
<td>41</td>
</tr>
<tr>
<td>8</td>
<td>Bernardon, 201036</td>
<td>Whiteness Perfect FGM</td>
<td>10%</td>
<td>-</td>
<td>At-home</td>
<td>2 weeks/8 hrs per night</td>
<td>-</td>
<td>8.4</td>
<td>0</td>
</tr>
<tr>
<td>9</td>
<td>Zantner, 200637</td>
<td>Colgate Simply White</td>
<td>-</td>
<td>5.90%</td>
<td>At-home</td>
<td>2 weeks/2 daily for 15 min</td>
<td>None</td>
<td>8.38</td>
<td>3</td>
</tr>
<tr>
<td>10</td>
<td>Zantner, 200638</td>
<td>Odol-med3 Gel</td>
<td>Chlorten</td>
<td>chlorten</td>
<td>At-home</td>
<td>2 weeks/2 daily for 10 min</td>
<td>None</td>
<td>8.22</td>
<td>0</td>
</tr>
<tr>
<td>11</td>
<td>Matis, 200739</td>
<td>Niveous</td>
<td>-</td>
<td>25%</td>
<td>In-office</td>
<td>3x15 min</td>
<td>-</td>
<td>8.1</td>
<td>72</td>
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<tr>
<td>12</td>
<td>Matis, 200740</td>
<td>Nite White</td>
<td>16%</td>
<td>-</td>
<td>At-home</td>
<td>2 weeks/8 hrs per night</td>
<td>Mild</td>
<td>8.04</td>
<td>55</td>
</tr>
<tr>
<td>13</td>
<td>Matis, 200741</td>
<td>Brite Smile</td>
<td>-</td>
<td>15%</td>
<td>In-office</td>
<td>3x20 min</td>
<td>-</td>
<td>7.8</td>
<td>68</td>
</tr>
<tr>
<td>14</td>
<td>Tsrburu, 200542</td>
<td>Opalescence</td>
<td>10%</td>
<td>-</td>
<td>At-home</td>
<td>2 weeks/8 hrs per night</td>
<td>Mild</td>
<td>7.78</td>
<td>-</td>
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<tr>
<td>15</td>
<td>Mokhli, 200243</td>
<td>Opalescence Tooth Whitening Gel PF</td>
<td>20%</td>
<td>-</td>
<td>At-home</td>
<td>2 weeks/7 hr x 2 daily</td>
<td>-</td>
<td>7.6</td>
<td>-</td>
</tr>
<tr>
<td>Study #</td>
<td>Reference</td>
<td>Product</td>
<td>% CP</td>
<td>% HP</td>
<td>Category</td>
<td>Application</td>
<td>Tooth Sensitivity</td>
<td>% Colour improvement</td>
<td>% Colour relapse after 4 weeks</td>
</tr>
<tr>
<td>--------</td>
<td>----------------------------</td>
<td>--------------------------</td>
<td>------</td>
<td>------</td>
<td>-------------------</td>
<td>---------------------------------</td>
<td>-------------------</td>
<td>----------------------</td>
<td>-------------------------</td>
</tr>
<tr>
<td>16</td>
<td>Matis, 2007</td>
<td>ArcBrite</td>
<td>-</td>
<td>30%</td>
<td>In-office</td>
<td>3x20 min</td>
<td>-</td>
<td>6.8</td>
<td>53</td>
</tr>
<tr>
<td>17</td>
<td>Bernardon, 2010</td>
<td>Whiteness HP maxx FGM</td>
<td>-</td>
<td>35%</td>
<td>In-office</td>
<td>2 sessions/3 applications of 15 min/15 day interval</td>
<td>-</td>
<td>6.64</td>
<td>-</td>
</tr>
<tr>
<td>18</td>
<td>Matis, 2007</td>
<td>Accelerated</td>
<td>-</td>
<td>40%</td>
<td>In-office</td>
<td>5x3 min</td>
<td>-</td>
<td>6.6</td>
<td>82</td>
</tr>
<tr>
<td>19</td>
<td>Bizhang, 2009</td>
<td>Illumine Home</td>
<td>10%</td>
<td>-</td>
<td>At-home</td>
<td>2 weeks/8 hrs per night</td>
<td>-</td>
<td>6.57</td>
<td>24</td>
</tr>
<tr>
<td>20</td>
<td>Matis, 2007</td>
<td>Zoom!</td>
<td>-</td>
<td>25%</td>
<td>In-office</td>
<td>x3/20 min</td>
<td>-</td>
<td>6.4</td>
<td>50</td>
</tr>
<tr>
<td>21</td>
<td>Bernardon, 2010</td>
<td>Whiteness HP maxx FGM</td>
<td>-</td>
<td>35%</td>
<td>In-office</td>
<td>2 sessions, 3 applications per session, 15 day interval</td>
<td>-</td>
<td>6.17</td>
<td>-</td>
</tr>
<tr>
<td>22</td>
<td>Matis, 2007</td>
<td>PolaOffice</td>
<td>-</td>
<td>35%</td>
<td>In-office</td>
<td>x3/12 min</td>
<td>-</td>
<td>5.9</td>
<td>69</td>
</tr>
<tr>
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<td>Opalescence PF</td>
<td>10%</td>
<td>-</td>
<td>At-home</td>
<td>2 weeks/4 hrs daily</td>
<td>Mild</td>
<td>5.84</td>
<td>-</td>
</tr>
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<td>24</td>
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<td>Illumine Office</td>
<td>15%</td>
<td>-</td>
<td>In-office</td>
<td>45 min/x3 over 3 weeks</td>
<td>-</td>
<td>5.77</td>
<td>20</td>
</tr>
<tr>
<td>25</td>
<td>Gurgan, 2009</td>
<td>Laser-White 1–Laser Smile</td>
<td>-</td>
<td>37%</td>
<td>In-office</td>
<td>x3/8 min</td>
<td>Mild</td>
<td>5.69</td>
<td>-</td>
</tr>
<tr>
<td>26</td>
<td>Gurgan, 2009</td>
<td>Opalescence Xtra Boost</td>
<td>-</td>
<td>38%</td>
<td>In-office</td>
<td>x2/15 min</td>
<td>-</td>
<td>5.54</td>
<td>-</td>
</tr>
<tr>
<td>27</td>
<td>Matis, 2007</td>
<td>Illumine</td>
<td>-</td>
<td>15%</td>
<td>In-office</td>
<td>x3/20 min</td>
<td>Mild</td>
<td>5.5</td>
<td>36</td>
</tr>
<tr>
<td>28</td>
<td>Gurgan, 2009</td>
<td>By White- Biowhite</td>
<td>-</td>
<td>38%</td>
<td>In-office</td>
<td>x2/20 min</td>
<td>-</td>
<td>5.43</td>
<td>-</td>
</tr>
<tr>
<td>29</td>
<td>Matis, 2007</td>
<td>One-hour Smile</td>
<td>-</td>
<td>35%</td>
<td>In-office</td>
<td>x3/15 min</td>
<td>-</td>
<td>5.4</td>
<td>54</td>
</tr>
<tr>
<td>30</td>
<td>Zekonis, 2003</td>
<td>StarBrite</td>
<td>35%</td>
<td>-</td>
<td>In-office</td>
<td>x2/3x10 min</td>
<td>Mild</td>
<td>5.32</td>
<td>32</td>
</tr>
<tr>
<td>31</td>
<td>Grobler, 2010</td>
<td>Nite White ACP</td>
<td>10%</td>
<td>-</td>
<td>At-home</td>
<td>2 weeks/8 hrs per night</td>
<td>Low</td>
<td>5.29</td>
<td>27</td>
</tr>
<tr>
<td>32</td>
<td>Grobler, 2011</td>
<td>Nite White ACP</td>
<td>10%</td>
<td>-</td>
<td>At-home</td>
<td>2 weeks/8 hrs per night</td>
<td>Low</td>
<td>5.29</td>
<td>31</td>
</tr>
<tr>
<td>33</td>
<td>Gurgan, 2009</td>
<td>Remewhite-Remecure</td>
<td>-</td>
<td>35%</td>
<td>In-office</td>
<td>x3/20 min</td>
<td>-</td>
<td>5.28</td>
<td>-</td>
</tr>
<tr>
<td>34</td>
<td>Benbachr, 2008</td>
<td>Vivastyle Paint On Plus</td>
<td>-</td>
<td>6%</td>
<td>In-office</td>
<td>3 days over 2 weeks/10 min x5 times per session</td>
<td>-</td>
<td>5.25</td>
<td>-</td>
</tr>
<tr>
<td>35</td>
<td>Grobler, 2011</td>
<td>Opalescence PF</td>
<td>10%</td>
<td>-</td>
<td>At-home</td>
<td>2 weeks/8 hrs per night</td>
<td>Low</td>
<td>5.2</td>
<td>18</td>
</tr>
<tr>
<td>36</td>
<td>Ishikawa-Nagal, 2004</td>
<td>Nite White Excel</td>
<td>10%</td>
<td>-</td>
<td>At-home</td>
<td>2 weeks/4 hrs daily</td>
<td>Mild</td>
<td>5.03</td>
<td>-</td>
</tr>
<tr>
<td>37</td>
<td>Luo, 2007</td>
<td>Crest White Strips</td>
<td>-</td>
<td>6%</td>
<td>At-home</td>
<td>2 weeks/30 min x2 daily</td>
<td>-</td>
<td>4.95</td>
<td>-</td>
</tr>
<tr>
<td>38</td>
<td>Meireless, 2008</td>
<td>Whiteness Perfect</td>
<td>16%</td>
<td>-</td>
<td>At-home</td>
<td>3 weeks/30 min x2 daily</td>
<td>-</td>
<td>4.6</td>
<td>2</td>
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<tr>
<td>39</td>
<td>Gerlach, 2002</td>
<td>Crest Professional Whitestrips</td>
<td>-</td>
<td>6.50%</td>
<td>At-home</td>
<td>2 weeks/x2 hrs daily</td>
<td>-</td>
<td>4.55</td>
<td>-</td>
</tr>
<tr>
<td>40</td>
<td>Meireless, 2008</td>
<td>Whiteness Perfect</td>
<td>10%</td>
<td>-</td>
<td>At-home</td>
<td>3 weeks/x2 hrs daily</td>
<td>-</td>
<td>4.3</td>
<td>9</td>
</tr>
<tr>
<td>41</td>
<td>Bizhang, 2009</td>
<td>Whitestrips</td>
<td>-</td>
<td>6%</td>
<td>At-home</td>
<td>2 weeks/30 min x2 daily</td>
<td>-</td>
<td>3.58</td>
<td>16</td>
</tr>
<tr>
<td>42</td>
<td>Salem, 2010</td>
<td>Yotuel Special</td>
<td>-</td>
<td>35%</td>
<td>In-office</td>
<td>20 min x 3 (1 session)</td>
<td>Mild</td>
<td>3.56</td>
<td>53</td>
</tr>
<tr>
<td>43</td>
<td>Grobler, 2010</td>
<td>Opalescence PF</td>
<td>10%</td>
<td>-</td>
<td>At-home</td>
<td>2 weeks/8 hrs per night</td>
<td>Low</td>
<td>3.25</td>
<td>8</td>
</tr>
<tr>
<td>44</td>
<td>Karpinia, 2002</td>
<td>Professional crest Whitestrips</td>
<td>-</td>
<td>6.50%</td>
<td>At-home</td>
<td>3 weeks/30 min x2 daily</td>
<td>Mild</td>
<td>3.15</td>
<td>-</td>
</tr>
<tr>
<td>45</td>
<td>Gerlach, 2002</td>
<td>Nite White Excel 2-tray system</td>
<td>10%</td>
<td>-</td>
<td>At-home</td>
<td>2 weeks/x2 hr daily</td>
<td>-</td>
<td>2.55</td>
<td>-</td>
</tr>
<tr>
<td>46</td>
<td>Al Shethri, 2003</td>
<td>Opalescence Xtra Boost</td>
<td>-</td>
<td>38%</td>
<td>In-office</td>
<td>2 treatments 1 week apart/ x3 daily for 10 min</td>
<td>Mild</td>
<td>2.45</td>
<td>-</td>
</tr>
<tr>
<td>47</td>
<td>Al Shethri, 2003</td>
<td>Star Brite</td>
<td>-</td>
<td>35%</td>
<td>In-office</td>
<td>2 treatments 1 week apart/ x3 daily for 10 min</td>
<td>Mild</td>
<td>2.31</td>
<td>-</td>
</tr>
<tr>
<td>48</td>
<td>Karpinia, 2002</td>
<td>Nite White Excel2</td>
<td>10%</td>
<td>-</td>
<td>At-home</td>
<td>2 weeks/2 hrs daily</td>
<td>Mild</td>
<td>1.94</td>
<td>-</td>
</tr>
<tr>
<td>49</td>
<td>Luo, 2007</td>
<td>Colgate Great regular Flavour</td>
<td>-</td>
<td>0% (Control)</td>
<td>At-home</td>
<td>2 weeks/x1 min x 2 daily</td>
<td>-</td>
<td>1.27</td>
<td>-</td>
</tr>
</tbody>
</table>
An Oral-B symposium and product launch was held in Cape Town on 24 June 2014. The event was attended by 74 external participants (3 Dental Deans of Universities, 3 Heads of Periodontal Departments at Universities, 5 Private Practicing Periodontist, 14 Heads and Lecturers of Oral Hygiene departments from Universities, 14 Private Dental Specialists, and the balance were top influencers, recommenders and sellers of our existing range of dental products from private practices, mostly Practicing Oral Hygienists).

The event commenced with a luxury bus tour of areas of the Winelands of the Western Cape, with a stop to purchase wines at a farm stall, then on to Asara Wine Farm for a guided wine tour, wine and chocolate tasting, a three-course lunch at the restaurant overlooking a lake and the beautiful blue mountains of the Western Cape Winelands, and entertainment by Saxophonist Debbie Parkinson. The bus departed after lunch for the modern upmarket 15 on Orange Hotel in Gardens Cape Town, where KOL’s then registered for the Symposium and were escorted through to the Judges lounge for canapés and drinks. An hour later the symposium began with the Opening Speaker Lorna Carneiro, President of the IADR for Africa, giving an overview of the dental landscape in Africa. Next up was Mr. Francois Facomprez, followed by Mr. Guy Goffin and Mr. Adam Boulding, together revealing the science, research and development behind Stabilised Stannous technology of the Oral-B Pro-Expert Paste.

For the product reveal, Pro Expert samples of paste and manual brushes, patient leaflets, product overviews and books of Clinical Trials were magically brought to the tables under cover of darkness in glass boxes with blue LED lights.

Some of the KOLs tried the product after the product reveal and gave us interviews and feedback on the survey forms.

Product samples were also delivered to the rooms of the delegates.

Dinner was served and delegates were entertained during dinner by Sterling EQ, a classy trio of ladies who played the violin and other instruments. The evening wrapped up at around 10:00 pm after a dessert buffet and coffees.

Delegates were so excited to have the new product and are looking forward to recommending it to their patients!
The values of shade guides cannot be directly compared with those numerically measured. A shade guide provides only a combination value for the three colour components \( L^*, a^* \) & \( b^* \). The distances between ‘before’ and ‘after’ colour measurements on each treated tooth differ when estimated with shade guides or taken numerically and these values could not be directly compared. Hence these data are assembled separately in two tables (Tables 1 and 2). As \( \Delta E^* \) is a difference between two values the percentage change could only be determined in the relapse phase when there are indeed two such \( \Delta E^* \) values. In relapse calculations where both methods had been used to determine the changes (a few cases only), the percentage relapses determined by shade guide and by numerical methods were first separately calculated and the average of the two then noted.

The percentage improvement (Table 2) could not be calculated for studies where only \( \Delta E^* \)-ab values were given as \( \Delta E^* \)-ab gives the difference between the tooth colour at base-line and that after treatment.

For the shade guide values, (Table 1), the top ten achievers were at-home products and likewise according to \( \Delta E^* \)-ab (Table 2) the top ten achievers were also at-home products. The highest values reported (Tables 1 and 2) were with a 10% or 15% carbamide peroxide treatment over a relatively long treatment period (two weeks 6/8 hours per night). In general, the treatment periods for top achievers (Table 1 and 2) were all scheduled over relatively long time-periods which, as seen in Table 1, varied from two hours/day for two weeks, to six hours/night for two weeks and to eight hours/night for two weeks. For Table 2 the first eight study achievers were for eight hours/night for two weeks. The shortest treatment period (Table 1) within the ten top achievers (studies # 6 and 7) was one hour/twice-a-day for 14 days but this was a trial using 7.5% HP ("25% CP) and 20% CP in comparison with the more general treatment which uses 10% carbamide peroxide. In Table 2 the shortest treatment period within the top ten at-home achievers was twice a day for 15 min (study # 9) but as for Table 1, this study relied on a high peroxide concentration (5.9% HP). The other treatment with a short application time was with chlorite (study # 10). Therefore, it can be deduced that to obtain the same success rate as with 10% carbamide peroxide it seems that a shorter treatment period might be indicated but using a higher peroxide concentration.

In general, it can be seen that in-office treatments (even with high peroxide concentrations) were far less successful (Table 1 and 2) than at-home treatments with ~10% carbamide peroxide. On the ranking list, in-office applications came in well below the highest achievers at study # 11 (Table 1) and study # 11 (Table 2). Why then is the high peroxide concentration treatment unexpectedly found to be less successful? The reason may be found in the length of the application period. In-office applications (Table 1, studies # 11–17; Table 2, study # 11 and others) were performed over a short period in comparison with the dentist/oral hygienist supervised at-home treatment procedures. The in-office treatment periods were normally short bursts, for example three x 15 minutes/session, three x 20 minutes/session, etc. (Tables 1 and 2). However, there were a few examples (Table 1, studies # 40 and 49–52) where the in-office sessions were repeated over days but only two of these studies (Table 1, studies # 11 and 16) were further extended to an at-home treatment. However, to allow subjects to personally handle such a high peroxide concentration is very risky and should not be recommended.

There are probably two main reasons for the short application period of in-office treatments: the first reason is financial, in that the longer the in-chair session, the more expensive the treatment becomes;

| TABLE 3: Descriptive statistics of colour improvement (shade guide assessment) immediately after ‘At-home’ and ‘In-office’ treatment. |
|---|---|---|
| Values | At-home | In-office |
| Number of studies | 40 | 19 |
| Number of studies indicating % colour improvement | 19 | 9 |
| Average of percentage of % colour improvements | 39.3% | 38.8% |
| Std Dev of % colour improvement | 22.4% | 17.2% |
| Min % colour improvement | 6.9% | 19.1% |
| Max % colour improvement | 88.1% | 54.4% |

| TABLE 4: Descriptive statistics of colour relapse (shade guide assessment) after four weeks for ‘At-home’ and ‘In-office’ treatment. |
|---|---|---|
| Values | At-home | In-office |
| Number of studies | 40 | 19 |
| Number of studies indicating % colour relapse after 4 weeks | 19 | 9 |
| Average of percentage of % colour relapses after 4 weeks | 13.1% | 55.2% |
| Std Dev of % colour relapse after 4 weeks | 17.0% | 20.2% |
| Min % colour relapse after 4 weeks | 0.0% | 18.0% |
| Max % colour relapse after 4 weeks | 55.0% | 81.0% |

| TABLE 5: Descriptive statistics of colour improvement (\( \Delta E^* \)-ab-scale) immediately after treatment for ‘At-home’ and ‘In-office’ treatment. |
|---|---|---|
| Values | At-home | In-office |
| Number of studies | 29 | 20 |
| Number of studies indicating colour improvement | 13 | 11 |
| Average of average \( \Delta E^* \)-ab after treatments | 6.36 | 5.60 |
| Std Dev of \( \Delta E^* \)-ab after treatment | 2.80 | 1.48 |
| Min \( \Delta E^* \)-ab after treatment | 1.27 | 2.31 |
| Max \( \Delta E^* \)-ab after treatment | 12.32 | 8.1 |

| TABLE 6: Descriptive statistics of colour relapse (\( \Delta E^* \)-ab-scale) after 4 weeks for ‘At-home’ and ‘In-office’ treatment. |
|---|---|---|
| Values | At-home | In-office |
| Number of studies | 29 | 20 |
| Number of studies indicating % colour relapse | 13 | 11 |
| Average of average % colour relapses | 26.02% | 53.60% |
| Std Dev of % colour relapses | 18.98% | 18.65% |
| Min % colour relapse | 2.17% | 20.45% |
| Max % colour relapse | 57.37% | 81.82% |
The second is that high peroxide application has a possible hazardous effect (related to the oxidising strength) on the soft tissue of the oral cavity. From Table 3 (shade guide) it can be deduced that overall, both at-home (39.3%) and in-office (38.8%) treatments showed more or less the same initial colour improvement. However, a major difference could be seen in the relapse after a four week or longer period (at-home 13.1%; in-office 55.2%; Table 4). From the ΔE'ab (Table 5) numerical values, the initial colour improvements for in-office and at-home were also about the same (5.60 against 6.36).

When the relapse was calculated from ΔE'ab values (Table 6), in-office treatment gave a value of 53.6%, similar to that found with the shade guide assessments (55.2%) (Table 4). However, the at-home relapse was 26.02% (Table 6) which is about double that when calculated from shade guide measurements (13.1%) (Table 4).

Other findings reported on the combination of dentally supervised at-home treatments: Leonard et al.13 revealed that the whitening effected by 10% carbamide peroxide (Nite White Classic; eight to ten hours per day for 14 days) reported five tabs lighter teeth, an effect which lasted over a 3.8 year period. In two different articles44–46 reporting on 10% carbamide peroxide which was applied for two hours per day for three weeks, no relapse was reported after one year. No comparable results were reported for any in-office treatment. Only two studies reported results for a combination of in-office and at-home treatments (studies # 11 and 16). Study # 11 showed a colour improvement of 56% (Table 1) and study # 16 also showed a high colour improvement of 53%. These results may indicate that a combination of the in-office and at-home treatments could produce the most positive results. Unfortunately no assessment on any colour relapse was reported in these two studies.

The most commonly tested product was Opalescence PF, a material evaluated in 11 studies. The average of the overall colour improvements as reflected on the shade guide scale, was 54% compared with 36% for all the other products. With regard to numerical measurements, the five studies using that method of assessment recorded 6.9 (ΔE') for the average of the overall colour improvements for Opalescence PF and an average of 5.95 (ΔE') for all the other products. These values can be considered high spectrophotometer readings, possibly highlighting the accuracy of the technology in comparison with shade guide measurements.

The mean relapse measured numerically after four weeks was 27.8% for Opalescence (n=3) which is considerably lower than the mean of 40.2% recorded for all the other products (n=21). Not all studies recorded relapse values (Opalescence 3 of 5; other products: 21 of 44) and these findings should therefore be regarded only as an indication of relapse potentials.

The next question which may be posed is which of the in-office or dentists/oral hygienist supervised at-home treatments would give the lowest tooth sensitivity scores? Not all studies reported on this, but the data recorded in Tables 1 and 2 reveals no significant differences in tooth sensitivities between the routines, and overall these effects were low – with an exception of the two top achievers in bleaching (Table 1), where a higher sensitivity was in fact noted (62% and 45%). However, it should also be observed that in some instances during treatments, the sensitivity might be so high that the individual prefers to terminate the application. Some of the manufacturers (studies # 1 and 4) added chemicals (potassium nitrate and fluoride) to their bleaching products in an attempt to counteract sensitivity. However, the clinical data (Table) indicates uncertainty on the possible positive effects of those materials, or on amorphous calcium phosphate and no general conclusion could be reached.11,16,17,38,48,49 Furthermore, from the published results (Tables 1 and 2), it can also be concluded that in general, where tooth sensitivity was noted during the whitening process, it disappeared spontaneously after the treatment period.

CONCLUSION

This analysis of the data shows that the dentist/oral hygienist-supervised at-home-bleaching and the in-office treatment gave nearly the same initial tooth whitening improvement. However, the relapse after a four week or longer period was found to be much higher for the in-office treatment at a relapse of “55% on average, while the at-home treatment showed a much lower relapse over the same period of about 13% to 26%.

It can be concluded that the treatment of choice should be a dentist/oral hygienist-supervised at-home bleaching process using a product which in general contains about 10% carbamide peroxide applied over “14 days and for “8 hours per night on average. Tooth sensitivity should not be seen as a consistent problem although some subjects might choose to discontinue treatment as a result of experiencing the problem.

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Disclaimer: The South African Dental Association does not necessarily support the findings or conclusions made in this article.

Declaration: No conflict of interest declared.
OHASA LOGO COMPETITION

WINNER ANNOUNCED

The winner of the OHASA Logo Competition run by the Professional and Public Relations Committee is Emma Coulter from the Western Cape Branch. The Competition was very tight with a variety of entries but in the end it was Emma’s that won.
Since the 1950s, the practice of prescribing antibiotics for the prophylaxis of endocarditis prior to invasive procedures has been widely accepted by the dental profession. The rationale for this is to reduce or eliminate the bacteraemia that may result from such procedures. The paradigm of this model is the prevention of bacterial endocarditis – a rare, but life-threatening disease. In developed countries, empiric guidelines for antibiotic prophylaxis for endocarditis, based primarily on pathophysiology and expert opinion, are put forward by committees of the American Heart Association (AHA), European Society of Cardiology (ESC) and British Society for Antimicrobial Chemotherapy (BSAC). These commonly-used guidelines, which are not in the first instance based on clinical evidence, have been periodically updated and we reviewed the latest recommendations of the AHA and BSAC in the SADJ in 2008. In that paper we referred to the lack of scientific evidence for linking infective endocarditis to dental procedures and the uncertainty regarding the effectiveness of prophylaxis – both these factors challenging the entire concept of antibiotic prophylaxis in dentistry. Subsequent to our review of 2008, important communications have appeared in the literature. In 2008, an updated version of the Cochrane review of 2004 on antibiotics for the prophylaxis of bacterial endocarditis in dentistry was published, as well as a guideline by the United Kingdom’s National Institute for Health and Clinical Excellence (NICE). Furthermore, a survey was conducted among dental practitioners in South Africa to determine their knowledge of the guidelines of the AHA and NICE 4 Although this survey had a low response rate, it was clear that knowledge in the profession of the use of infective endocarditis prophylaxis and compliance with existing guidelines was generally poor. In the current update we attempt to clarify some of the prevailing confusion among South African dental professionals regarding when and for whom antibiotic prophylaxis is indicated.

GUIDELINES

The traditionally accepted guidelines of the BSAC and the AHA as modified in 2006 and 2007 respectively, are recommended for use by South African dental practitioners. Their advantages and disadvantages, as well as the proposed antibiotic regimens of both these sets of guidelines have been discussed in some detail in our previous review. As mentioned in that review, the main characteristics of both guidelines are a simplification, in terms of complexity and breadth, of the published pre-2006 and pre-2007 guidelines, resulting in a reduction in the indications for antibiotic prophylaxis. Only a few categories of high-risk patients, who require antibiotic prophylaxis prior to dental procedures, are identified in the 2006 and 2007 guidelines. The dental procedures requiring prophylaxis are those involving manipulation of gingival tissue or the periapical region of teeth, or perforation of the oral mucosa. Of significance is that the updated Cochrane review of 2008 has not provided conclusive evidence about whether penicillin prophylaxis is effective against bacterial endocarditis in people at risk who are about to undergo an invasive dental procedure. Evidence from that review is insufficient to support previously published guidelines, such as those of the BSAC and AHA. Additionally, it is of interest that the overall incidence of infective endocarditis has remained stable from 1950 to 2000, i.e. approximately 3.6–7.0 cases per 100 000 patient-years. This incidence has remained unchanged over half a century despite improvements in cardiac imaging techniques, which may have enhanced the detection of endocarditis.

In March 2008, the National Institute for Health and Clinical Excellence (NICE) in the United Kingdom published a controversial new guideline, which was a radical departure in that it recommends complete abolition of antibiotic prophylaxis for patients at risk of infective endocarditis undergoing dental and a wide range of other invasive procedures. The rationale of the NICE guidelines is the absence of a consistent association between interventional procedures, dental or otherwise, and the development of endocarditis as well as the unproven effectiveness, the potential mortality of anaphylaxis and increased expense. NICE guidelines recommend that antibiotic cover should be offered to patients only if the procedure is at a site where there is already a suspected infection. Furthermore, the NICE guidelines do not recommend the use of chlorhexidine mouthrinses prior to dental procedures. Although the guidelines do recognise certain cardiac conditions which present a high risk for developing infective endocarditis, these are mainly listed to emphasise the need for good oral hygiene and awareness of infective endocarditis. Although the AHA and ESC for Cardiology modified their guidelines more or less simultaneously, both of these authoritative bodies still recommended administering antibiotic prophylaxis in certain high-risk groups.
CONTROVERSIES
Theoretically, antibiotic prophylaxis in patients with cardiac risk factors should decrease the incidence of infective endocarditis; however, to date this principle has not been underpinned by sound scientific evidence. Several factors contribute to this failure of what appears to be a linear-logic approach. While it has been shown that invasive procedures, e.g. dental extractions, cause bacteraemias, other common daily activities, such as toothbrushing, interdental flossing and mastication, do so as well. The transient bacteraemias which may be caused by surgical dental procedures are several orders of magnitude higher than those resulting from common daily activities. The latter, however, may cumulatively be several million times higher than those resulting from single invasive procedures (so-called cumulative bacteraemia). Additionally, it has been shown that only a small proportion, if any, of cases of infective endocarditis were causally linked to dental procedures. Conflicting evidence has been reported regarding the reduction or prevention of bacteraemias by means of antibiotic prophylaxis.

It is contentious whether antibiotic prophylaxis is cost-effective for at-risk patients. Strains of antibiotic-resistant organisms may emerge and antibiotic-related side-effects do occur, but these phenomena are both rare following high, single-dose antibiotic administration on an infrequent basis, such as during prophylaxis. While minor unwanted effects may occur during prophylactic antibiotic usage, no cases of anaphylaxis have been reported to the AHA during the 50 years that they have recommended using a penicillin for the prophylaxis of infective endocarditis. The AHA believes that it is safe to administer a single dose of a broad-spectrum penicillin, e.g. amoxicillin or ampicillin, to persons who do not have a history of hypersensitivity to a penicillin, such as anaphylaxis, urticaria or angioedema. While the NICE committee quotes the potential of fatal anaphylaxis to penicillins as one of the reasons for their stance, reports of oral amoxicillin causing this condition have never been reported in the world literature.

DISCUSSION
Publication of the NICE guidelines in the UK in 2008 has challenged the standard of care for prevention of infective endocarditis. That these guidelines have been accepted by many practitioners in the UK is evidenced by a 78.6% reduction in antibiotic prophylaxis. NICE is unique in recommending no antibiotic prophylaxis for cardiac patients undergoing dental or non-dental procedures, except for manipulations at an infected site while most national or international guidelines from the USA, Europe and Australia have pared down their indications, they still recommend prophylaxis for certain dental procedures in high-risk cardiac patients (Table 1). Even though the NICE committee correctly stated that, in the absence of prospective, randomised trials, the clinical effectiveness of antibiotic prophylaxis remains unproven, some clinical and animal studies reviewed by the AHA and BSAC have suggested that there are benefits. It has been estimated that a randomised, placebo-controlled trial to demonstrate the effectiveness of antibiotic prophylaxis for infective endocarditis would require the participation of 60,000 individuals, making such a study unlikely to transpire.

The South African context
A variety of arguments have been advanced that the guidelines emanating from the USA and Europe cannot be extrapolated to the local situation, namely: (i) the high prevalence of rheumatic heart disease predisposes to IE; (ii) HIV might predispose to infective endocarditis; and (iii) the local microbiological profile of infective endocarditis differs.

The theory of cumulative bacteraemia takes rheumatic heart disease into account. However, there is no evidence that it poses a significantly higher risk for development of infective endocarditis than other, accepted, risk factors or that the severity and consequences of the disease are far worse than those seen with other risk factors. Therefore, it should not be seen as an exception. The evidence that HIV predisposes to infective endocarditis is scant. Although the data are scarce, the most common pathogenic organisms in a South African setting are oral streptococci, and the antibiotic choice should therefore be no different to that of the international guidelines. This is an issue unrelated to the threshold/indications for prophylaxis.

The opinion of the authors, as evidenced in our previous review, is that in the absence of any data to the contrary, the guidelines as set forth by the AHA, ESC and BSAC are appropriate and apply also in the South African context. It is clear that the risk of developing infective endocarditis following dental

<table>
<thead>
<tr>
<th>TABLE 1: Cardiac conditions requiring antibiotic prophylaxis for high-risk dental procedures included in international guidelines, but excluded by NICE</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Replacement valves or prosthetic material used for cardiac valve repair</td>
</tr>
<tr>
<td>• Previous episodes of infective endocarditis</td>
</tr>
<tr>
<td>• Congenital heart disease</td>
</tr>
<tr>
<td>• Unrepaired cyanotic congenital heart disease including palliative shunts and conduits</td>
</tr>
<tr>
<td>• Completely repaired using prosthetic material or device during the first 6 months after procedure (surgical or percutaneous)</td>
</tr>
<tr>
<td>• Repaired with residual defect at the site or adjacent to the site of a prosthetic patch or device</td>
</tr>
<tr>
<td>• Cardiac transplantation with valvular regurgitation due to a structurally abnormal valve*</td>
</tr>
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</table>

* Included in the AHA guideline, but excluded in the ESC guideline
procedures is low. There is little scientific evidence that prophylactic administration of antibiotics prior to dental procedures would lower the risk of developing the disease and it should be kept in mind that the above guidelines are not infallible. However, in the absence of hard evidence on prophylactic efficacy and being mindful of the potential legal consequences, we recommend adherence to the AHA, ESC and BSAC guidelines in high-risk cardiac patients (Table 1) who are undergoing manipulation of dento-gingival tissue, procedures involving the periapical region of teeth and endodontics. Of further importance is that dental treatment plans be drawn up and executed in such a manner that patients are not unnecessarily exposed to prophylactic antibiotics — this might include concurrent execution of procedures. Where that is not possible, the AHA and BSAC respectively recommended intervals of at least 10 and 14 days. Otherwise, amoxicillin may be alternated with clindamycin and, in patients being treated with these two antibiotics for other infections, azithromycin or clarithromycin may be substituted.

Patients should be well-informed by their dental practitioner, with the option of obtaining written consent for the administration/omission of prophylaxis being very reasonable. They should be advised to report any adverse effects following prophylaxis to their practitioner via a direct line of communication.

Declaration: No conflict of interest declared.

REFERENCES
A long-standing patient attended a practice and said that she plans to report her practitioner to the Health Professions Council for failing to adequately treat her periodontal condition. At her initial examination some ten years earlier, she presented with advanced periodontal disease and radiographs clearly showed the extent of the severe bone loss throughout the patient’s mouth. The practitioner did a thorough examination and charting, and discussed at length both the short- and long-term treatment plans. The patient was fully informed of the situation, given appropriate advice and consented to undergo extensive periodontal therapy. It was recommended that she attend every three months for examinations, scaling and oral hygiene instruction. She agreed to the treatment plan and her progress was carefully monitored and her periodontal status gradually improved over the years.

Although the practitioner saw the patient regularly and carried out such care and treatment as was deemed necessary, the periodontal condition began to deteriorate resulting in mobility of a number of anterior teeth and the patient was then referred to a periodontist for a second opinion. During the visit to the periodontist, a perhaps inappropriate comment was made to the effect that something could have been done to save a number of these teeth had the appropriate advanced treatment been instituted at an earlier stage. The patient was naturally upset by this statement and decided to make a complaint against her dentist and oral hygienist.

**COMMENTARY**

People are living longer and retaining their teeth into later life. The percentage of individuals with moderate to severe periodontitis, in which the destruction of supporting tissue can cause loosening and even loss of teeth, increases with age. The most common form of adult periodontitis is described as ‘general and moderately progressing’. It is characterised by a gradual loss of attachment of the periodontal ligament to both the gingiva and bone, progressing to actual loss of the supporting bone. It is most often accompanied by gingivitis

The severity of periodontal disease is determined through a series of measurements, including the extent of gingival inflammation and bleeding, the probing depth of the pocket to the point of resistance, clinical evidence of attachment loss of the periodontal ligament and the loss of adjacent alveolar bone as measured by radiographs. Severity is also determined by the rate of disease progression over time and the response of the tissues to treatment. The prevalence and severity of periodontal disease increases, but does not accelerate with age. The current view is that the disease process may not be continuous but rather progresses in random bursts in which short periods of breakdown of periodontal ligament and bone alternate with periods of quiescence. These episodes occur randomly over time and at random sites in the mouth. Part of the difficulty in determining the pattern of progression reflects variation in the sensitivity of the instruments used to measure the loss of soft tissue and bone.

While there is no doubt that the existence of bacteria plays an important role in the aetiology of periodontal disease, studies suggest that it is the combination of the presence of these bacteria and the host response of the individual that determines the development and rate of progression of periodontal disease. The main risk factors of the disease are often outside the control of the clinician. In addition, familial history, genetic susceptibility, systemic disease and smoking are known to play a part in the aetiology and rate of progression of the disease.

Again, the clinician cannot be held responsible for the existence of these factors and patients need to understand that their periodontal disease is also their problem. However, it is an ethical responsibility of the clinician to educate the patients, to make them aware of the condition and assist them to reduce the impact, giving advice, guiding, monitoring and encouraging the patient to maintain the best levels of oral hygiene they can achieve.

Complaints regarding undiagnosed and untreated periodontal disease are on the increase. The most common allegation is that the patient was unaware of the presence of periodontal disease or that the extent and implications of the disease had not been adequately explained to them. In these instances, two questions are usually posed: firstly, did the oral health professional properly diagnose, treat and monitor the periodontal disease and secondly, was there adequate communication and discussion regarding the diagnosis between the oral health professional and the patient?
A patient-centred approach is in keeping with the principle of respect for autonomy. Listening to the patient enables the oral health professional to decide what information the patient needs, how this information should be transmitted to the patient and what the patient’s preferences are. Good communication makes it possible to compile a complete and accurate patient history, and makes the patient feel reassured and cared for. Furthermore, good communication is a necessary pre-requisite for responsible decision-making. In order to exercise their right to informed consent, patients must understand their diagnoses, the various treatment options, and the possible consequences of undergoing or refusing treatment.7 In the above-mentioned case scenario, it is clear that the dentist and oral hygienist acted in the best interests of the patient. She was informed at her first visit that her periodontal condition was compromised and that periodontal disease can manifest by years of quiescence and occasional bouts of sporadic activity. Radiographic evidence did not show any major deterioration until the final visit and when a second opinion was required she was referred to a specialist.

In many cases, the levels of periodontal disease in a patient’s mouth are due to factors beyond the oral health professional’s control and do not reflect any fault or the part of the oral health professional. However, it is easier to demonstrate that a high standard of care was provided, if dental records are comprehensively written up.

An oral health professional that is able to communicate effectively and compassionately is able to dissipate fear and allay anxiety. This, in turn, leads to better patient satisfaction and to better treatment adherence. Research has demonstrated a relationship between communication skills and complaints lodged against oral health care workers. Oral health professionals who focus on technical procedures or technology, who spend little time talking to patients and who give minimal explanations to patients are at higher risk of litigation. Risk of litigation appears to be related to “patients’ dissatisfaction with their physicians’ ability to establish rapport, provide access, administer care and treatment consistent with expectation and communicate effectively”.

It is worth taking the time to schedule a face-to-face conversation with patients to discuss complex disease like periodontal disease.5 Such communication goes a long way to encourage the patient to ‘internalise’ the problem, take responsibility and importantly to adhere to oral hygiene instructions and oral health education messages. These discussions need to be carefully documented in the clinical records, together with copies of any written correspondence. Oral health professionals who do not keep adequate records are placed in an invidious position when a patient makes a claim about the standard of care which has been provided. It has been recommended that a clinical audit be carried out to monitor patients with moderate or severe periodontal disease. Any gaps or weaknesses in record keeping should be identified and improved upon.

REFERENCES
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ACKNOWLEDGEMENTS
Acknowledgements persons who have made substantive contributions to the study. Authors are expected to disclose any commercial or other relationships that could constitute a conflict of interest.

REFERENCES
The author is responsible for the accuracy of the reference list at the end of the paper. All references cited, and only these, must be listed at the end of the paper. This should include the names and initials of all authors unless they are more than six when only the first three should be given followed by et al. in italics. The authors’ names are followed by the title of the article; the title of the journal abbreviated according to the style of Index Medicus or the Index to Dental Literature; the year of publication; the volume number; first and last page numbers in full. Titles of books should be followed by the place of publication, the publisher and the year.

EXAMPLES
Reference to a journal article

Reference to a book

Reference to a chapter in a book

The Editor reserves the right to edit material for clarity of style and to suit the space available. A full copy of Guidelines for Authors is available on request from the Editor.
The association between periodontitis and pre-term birth and/or low birth weight: a literature review

1. Pre-term birth and/or low birth weight contribute significantly to the health care, economic and social burden imposed on the government, communities and families involved.
   a. True
   b. False

2. Periodontitis can be modified by:
   a. The type of organisms
   b. Host immuno-inflammatory response
   c. Environmental, genetic and systemic factors
   d. b and c
   e. a, b and c

3. Periodontitis can be regarded as a constant pathogenic and inflammatory challenge at a systemic level because of the large epithelial surface that could be ulcerated in the periodontal pocket.
   a. True
   b. False

4. The WHO defines pre-term birth as all live births before 36 weeks of complete gestation.
   a. True
   b. False

5. In developed countries, low birth weight is usually caused by intra-uterine growth restriction, whereas in developing countries it is more often as a result of pre-term birth.
   a. True
   b. False

6. The rise and fall pattern of the hormones coincides with the onset and peak of the gingival inflammation observed in pregnancy.
   a. True
   b. False

7. Pregnancy alters the rate and pattern of collagen production in the gingiva but collagen repair may also be affected due to folate deficiency caused by oestrogen and progesterone.
   a. True
   b. False

8. Periodontal pathogens, their shed virulence factors and/or inflammatory cytokines from the periodontal pocket may gain access into the bloodstream and disseminate through the body.
   a. True
   b. False

9. Mechanisms through which an inflammatory response can affect PTLBW are:
   a. Damage to the placental tissue
   b. Pre-eclampsia
   c. Pre-term rupture of the membrane and contraction of the uterus
   d. b and c
   e. a, b and c

10. Scaling and root planing in pregnant women with periodontitis may result in the increase of pre-term births.
    a. True
    b. False

Guidelines for the selection of tooth whitening products amongst those available on the market

11. Peroxide is the chemical most frequently used as a tooth whitening agent with two types being generally employed namely hydrogen peroxide or carbamide peroxide.
    a. True
    b. False

12. The public sees the following aspects as important with regards to tooth whiteners:
    a. Effectiveness of the whitener
    b. How long lasting is the effect
    c. The cost of the treatment
    d. All of the above
    e. None of the above

13. There are two ways to measure the effect of tooth whiteners namely by means of matching with a calibrated shade guide or numerically (colourimeter, chromameter or spectrophotometer).
    a. True
    b. False
14. The best results with the highest values according to Tables 1 and 2 were with a 10% or 15% carbamide peroxide treatment over a relatively long treatment period (two weeks 6/8 hours per night).
   a. True
   b. False

15. In general in-office treatments were far more successful than at-home treatments with "10% carbamide peroxide.
   a. True
   b. False

16. There is a major difference in the relapse after a four week or longer period between at-home whitening and in-office whitening namely 13.1% for at-home and 55.2% for in-office.
   a. True
   b. False

17. Opalescence PF was the most commonly tested product and showed 45% improvement as reflected on the shade guide scale.
   a. True
   b. False

18. Tooth sensitivity during the whitening process does not disappear spontaneously after the treatment period.
   a. True
   b. False

19. This article concludes that the whitening treatment of choice should be:
   a. At-home whitening under supervision of a dentist/oral hygienist
   b. Using a 10% carbamide peroxide
   c. "14 days and "8 hours per night on average
   d. a, b and c

20. The two factors challenging the concept of antibiotic prophylaxis in dentistry are the lack of scientific evidence for linking infective endocarditis to dental procedures and the uncertainty regarding the effectiveness of prophylaxis.
   a. True
   b. False

21. According to the 2006 and 2007 guidelines the dental procedures requiring prophylaxis are those involving manipulation of gingival tissues or the peri-apical regions of the teeth or perforation of the oral mucosa.
   a. True
   b. False

22. The rationale of the NICE guidelines is:
   a. The absence of a consistent association between interventional procedures, dental or otherwise and the development of endocarditis
   b. The potential mortality of anaphylaxis
   c. Increased expense
   d. All of the above
   e. None of the above

23. The cumulative bacteraemia refers to bacteraemia resulting from common daily activities and may cumulatively be several million times higher than those resulting from single invasive procedures.
   a. True
   b. False

24. The most common pathogenic organisms in a South African setting are oral streptococci and the antibiotic choice should therefore be no different to that of the international guidelines.
   a. True
   b. False

25. The guidelines as set forth by the AHA, ESC and BSAC are appropriate and apply also in the South African context.
   a. True
   b. False

26. The factors playing a part in the etiology and rate of progression of periodontal disease are:
   a. Bacteria
   b. Genetic susceptibility
   c. Practitioner’s neglect
   d. a and b
   e. a, b and c

27. Listening to the patient enables the oral health practitioner:
   a. To decide what information the patient needs
   b. To know how this information should be transmitted to the patient
   c. To know what the patient’s preferences are
   d. All of the above
   e. None of the above

28. In order to exercise their right to informed consent, patients must understand their diagnoses, the various treatment options and the possible consequences of undergoing or refusing treatment.
   a. True
   b. False

29. Dental records that are comprehensively written up demonstrate that a high standard of care was provided.
   a. True
   b. False

30. Oral health professionals who focus on technical procedures or technology, who spend little time talking to patients and who give minimal explanations to patients are at lower risk for litigation.
   a. True
   b. False
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1. Euromonitor International. 2. LISTERINE® ZERO™ approved package insert. 3. Data on file D, microbiology dossier, McNEIL-PPC, Inc. 

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