

# Smoking as a Risk Factor for Peri-Implantitis and Dental Implant Failure - A Literature Review

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**Abstract:** Background: Despite the fact that dental implant therapy is a very successful treatment, various studies have suggested higher Implant failure rates in smokers. The aim of this study is to assess whether smokers are at an increased risk of implant failure and peri-implantitis, as compared to non-smokers. Methods: A comprehensive search on PubMed, Cochrane library and Web of Science was conducted to identify studies investigating the association between smoking and peri-implantitis and implant failure. Only studies published between 1990 and 2016 were considered in this review. Results: From the 920 search results initially retrieved, only 20 were selected after analysis of the abstracts and titles. The quality of the included papers was assessed using the Quality Assessment Tool for Quantitative Studies. It was found that the quality rating for most of the studies included was moderate or strong. The majority of the included studies showed a relationship between cigarette smoking and dental implant failure. Conclusion: The results from the included studies showed that smoking is an important risk factor for dental implant failure. However high quality studies with additional robust epidemiological and clinical investigations are required to confirm the association between the two.

**Keywords:** Dental Implants, Peri implantitis, Smoking, Success, Tobacco

## 1. Introduction

Dental implants, with their high survival rates and predictability, have made a significant impact on dentistry. Their fixed nature and the lack of any need to involve adjacent teeth, has made them the option of choice for the restoration of most edentulous sites. The popularity of implants has increased over the past two decades, this has in part been due to studies showing their high survival rates. One such study (Jung et al., 2008) observed 96.8% survival after 5 years and similarly another more recent study, (Simonis et al., 2010) reported 89.23% and 82.94% survival rates after 10 and 16 years respectively.

In recent years the dental implant market has shown significant global growth. The rising ageing population as well as the increasing trend for general dentists to offer implants in their practice has played a role in the development of this growth. Data shows that there has been a 10 fold increase globally in the number of dental implants placed from 2002 to 2010 (Misch, 2014). According to the American Academy of Implant dentistry (AAID), the global market for dental Implants is anticipated to exceed \$4.2 billion by 2022. All of these statistics show the growing significance of implant treatment in general practice and suggest trends which confirm their increased usage.

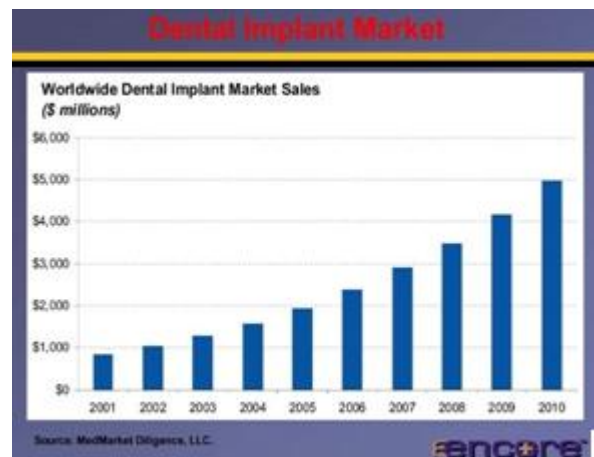


Figure 1: Dental Implant Market

Adapted from (Encore Medical Corporation.(ENMC) 2004

The success of implant treatment is important from the patient's perspective because firstly it is an expensive treatment and failure will represent a poor financial investment in their health and wellbeing and secondly because the placement of an implant involves an invasive, surgical procedure, subsequent failure of the implant would therefore have entailed the patient undergoing this traumatic procedure, and its associated surgical risks, without gaining the desired long term functional and/or aesthetic benefits.

Implant failure is important from the perspective of the clinician because they make the decision as to the suitability, for implant placement, of a case. An understanding of the factors that may compromise the success rate of implants is therefore crucial in this decision making process. Furthermore in gaining the patient's consent prior to embarking on implant placement, the clinician must be in a position to accurately inform the patient of any risk factors that might predispose the patient to a higher probability of implant failure.

Although implants are a predictable treatment and have high success rates, poor treatment planning, case selection and maintenance can result in their failure. The potential for implant failure is a significant concern for both patients and clinician, patients should be informed that implants can fail and that their noncompliance could be one of the main reasons for that failure. Patient selection and their physiologic and behavioural response to dental implant treatment is important in understanding their expectations, the findings of one study showed that almost all the patients expected implants to function in the mouth for rest of their lives. (Johannsen., 2012)

Several reasons are known for the failure of dental implants, one of these is the loss of bone around an integrated implant known as peri-implantitis. With the emergence of dental implants, peri implant mucositis and peri-implantitis have become an increasing problems in recent years. The term peri implant mucositis describes an inflammatory lesion that involves the mucosa adjacent to an implant (Filho et al., 2011) while peri-implantitis describes a non-reversible inflammatory condition surrounding an implant and resulting in the loss of supporting bone (Ashnagar et al., 2014).

Given that peri-implantitis is an emergent and common problem, a proper understanding of the risk factors that may predispose a patient to the disease, is required. Any attribute or exposure that increases the likelihood of developing a disease is known as its risk factor; there are several known risk factors for peri implantitis and implant failure. It is thought that peri-implantitis is more prevalent among smokers, thus exploration of the association between peri-implantitis and smoking is a subject of significant importance. Klekkod et al., (2007) reported that smokers with implants were diagnosed with 78% of peri- implantitis while only 64% in non-smokers.

Smoking has been linked with many diseases, including, coronary heart disease, COPD, pneumonia and cancer. Smoking has also been linked with loss of periodontal attachment, vertical bone height and tooth loss (Millar & Locker.,2007; Baljoon et al.,2004; Scabbia.,2001) The negative effects of smoking on the survival of dental implants has also been found (Esposito et al., 1998). The suggested mechanism for these effects on implants has been the toxic byproducts of cigarette tobacco namely nicotine, carbon monoxide, nitrosamines and hydrogen cyanide; these chemicals are known to be responsible for delayed wound healing (Sverzut et al., 2008 and Schwartz et al., 2002) Smoking is known to cause decreases in neutrophil elastase (Ataoglu et al.,2002) and vasoconstriction at the end of the arterial gingival vessels (Sham et al.,2003) resulting in a diminished peri-implant inflammatory reaction

## 2. Objectives

The aim of this review is to analyse the available literature to determine if smoking can be considered a risk factor for implant failure. The review will focus on the question: Are smokers at an increased risk of implant failure and peri implantitis as compared to non-smokers.?

## 3. Methods

### 3.1 Criteria for considering studies for review

**Types of studies:-** Eligible studies were included using the following criteria:

- Papers which evaluate the effect of smoking on dental implant outcome.
- Prospective, retrospective cohort, cross sectional studies, systemic reviews, control trials reporting on implant failure/ peri implant it is with history of smoking.
- Studies which were on humans.
- Studies which were published in the English language.

**Studies were excluded using following criteria**

- The study was not original research.
- The study did not involve human subjects.
- The study was a duplicate study.
- In vitro studies.
- Literature reviews
- Certain publication types (e.g. letters, technical reports, comments etc.)

**Types of participants: -** People who

- have at least one dental implant.
- Were 18 years or older.
- Animal studies will be excluded

**Types of intervention:-**

Included studies made a direct comparison between dental implant outcomes in smokers as compared to nonsmokers.

**Types of outcome measures:-**

Papers assessing dental implant failure and peri implantitis

**Search methods for identification of studies:-** A computerised search of the three databases, web of science, PubMed and Cochrane library from 1990 to 2016 was undertaken; searches were limited to English language publications. The search was carried out to extract studies providing statistical data on peri implantitis and implant failure due to smoking. The searches employed the following search terms (Medical Subject Headings in Medline):

- Peri-implantitis
- Peri-Implant\$
- Dental implants AND smoking
- Dental implant failure”
- Risk factors AND periimplantitis “
- Peri implantitis AND smoking
- Osseo integration
- Dental implant outcome
- Peri implantitis\$ OR periimplantitis\$ (Isolated or in different combinations using different Boolean operators)

**Study selection and Quality Assessment of the studies: -**

The titles and abstract of studies obtained from the searches were assessed to eliminate those studies that did not meet the inclusion criteria, full papers were retrieved for those studies that did meet the criteria. The exclusion criteria included

studies on animals, technical reports, in vitro studies, literature reviews and repetition of the same study through other databases. Other than these, studies which didn't match with the focused question and some which did not have assessable data for determining the effect of smoking on dental implant outcome, were excluded as well. All studies which met the inclusion criteria were discussed and their characteristics summarized in tabled form.

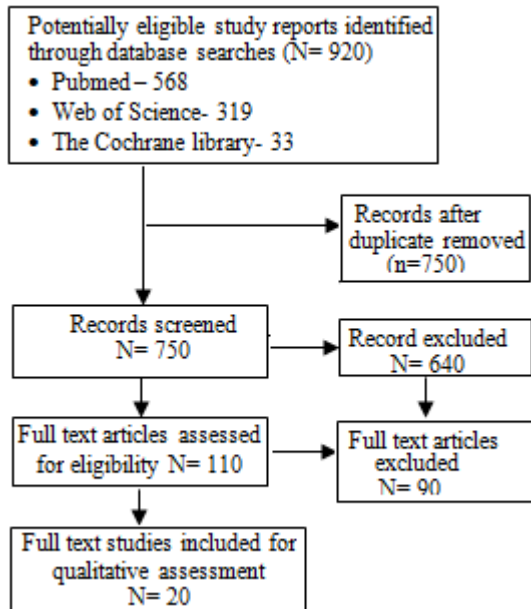


Figure 2: Prisma 2009 flow diagram

**Based on flow chart adapted by Moher et al (2009)**

**4. Results**

The keywords search yielded total of 920 papers from the initial search of three databases, 568 from PubMed: 319 from web of science and 33 from Cochrane library. After initial screening several papers were rejected on the basis of their title and abstract while others were excluded due to the unavailability of full texts or because they were duplicated in different publications. In addition some papers were

excluded for having topics of investigation not consistent with the primary focus of this review. These selection processes resulted in 20 papers being retained for inclusion in this review. The selected papers comprised of study designs as follows: Four systemic reviews, one RCT, nine retrospective, three prospective studies and three cross sectional studies.

The methodological quality score of all included papers was assessed using an appraisal tool developed by *The Effective Public Health Practice Project* (EPHPP), the results being summarised in table 2.

The appraisal tool used, sometimes known as Hamilton Tool, is considered an appropriate means for the evaluation of the study design of RCT's, non-randomized trials, and case control studies. This tool was developed by EPHPP specifically to enable knowledge synthesis and provides a standardised means of assessing study quality; it has been reported to have good content and inter rater reliability. This appraisal tool, developed by EPHPP, is a discrete step within the review process and has been considered suitable, by various authors, for use in systemic reviews (Deeks et al., 2003).

Quality Assessment tool has the following component

- 1) Selection bias (representative sample, response rate)
- 2) Study design (with optional questions on randomisation)
- 3) Confounders (control group comparability)
- 4) Blinding (assessor and participants)
- 5) Data collection methods (validity, reliability)
- 6) Withdrawals/drop outs (Reported? Explained? Attrition rates?)

An accompanying dictionary explains and clarifies each of the questions in more detail and provides guidance on scoring, the dictionary also helps to maintain standardised results. The components are graded as "strong", "moderate" or "weak" based on the features of each component described in the study. The mechanism for scoring is illustrated in table 1 below.

**Table 1: Mechanism of scoring**

Components	Strong	Moderate	Weak
Design	RCT and CCT	Cohort analytic, case control, cohort, or an interrupted time series	All other design or designs not stated
Confounders	Controlled for atleast 80% of confounders	Controlled for 60-79% of confounders	Confounders not controlled for or not stated
Blinding	Blinding of outcome assessor and study participants to intervention status	Blinding of either outcome assessor or study participants	Outcome assessor and study participants are aware of intervention status and/or research question
Data collection methods	Tools are valid and reliable	Tools are valid but reliability not described	No evidence of validity or reliability
Withdrawals and dropouts	Follow-up rate of >80% of participants	Follow up rate of 60-79% of participants	Follow-up of rate of <60% of participants or withdrawals and dropouts not described

Table 1: Adapted from (Thomas et al., 2004)

The scores for the included studies is summarised in table 2. The majority of studies included in this paper showed high level of scientific evidence. The qualities of the included

studies were mainly rated moderate & strong, only two studies were graded as weak.

**Table 2: The quality of the studies**

Author and date	Selection bias	Study design	confounders	Blinding	Data collection methods	Withdrawals/ dropouts	Overall ratings
Bain and Moy., (1993):	Strong	Moderate	Strong	Weak	Strong	Moderate	Moderate
Van steenberghe et al., (2002):	Moderate	Moderate	Strong	Weak	Strong	Moderate	Moderate
Sverzut et al., (2008):	Moderate	Moderate	Moderate	Weak	Moderate	Moderate	Moderate
Klokkevold et al.,(2007):	Strong	Strong	Moderate	Moderate	Strong	Moderate	Strong
Crawford Bain.,(1996).	Strong	Moderate	Strong	Weak	Strong	Moderate	Moderate
Lindquist et al., (1996)	Strong	Moderate	Strong	Weak	Moderate	Moderate	Moderate
Strietzel et al., (2007)	Strong	Strong	Moderate	Strong	Strong	Moderate	Strong
Cavalcanti et al., (2011)	Strong	Moderate	Moderate	Weak	Moderate	Strong	Moderate
Sanchez –Perez et al., (2007)	Moderate	Moderate	Moderate	Weak	Moderate	Moderate	Moderate
Deluca et al., (2006)	Strong	Moderate	Moderate	Moderate	Weak	Moderate	Moderate
Clementini et al., (2013)	Strong	Strong	Strong	Moderate	Moderate	Moderate	Strong
Stoker et al., (2012)	Strong	Strong	Moderate	Strong	Strong	Moderate	Strong
Schwartz Arad et al., (2002)	Strong	Moderate	Strong	Moderate	Moderate	Moderate	Strong
Alsaadi et al., 2008	Strong	Moderate	Weak	Moderate	Moderate	Strong	Moderate
Haas et al., (1996)	Moderate	Moderate	Moderate	Weak	Moderate	Strong	Moderate
Costa et al., (2012)	Strong	Moderate	Weak	Weak	Strong	Moderate	Weak
Gorman et al., (1994)	Moderate	Moderate	Weak	Moderate	Moderate	Moderate	Moderate
Kumar et al., (2002)	Moderate	Moderate	Strong	Moderate	Moderate	Moderate	Strong
Vandheweghe et al., (2011)	Strong	Moderate	Strong	Moderate	Weak	Moderate	Moderate
Sgolastra et al., (2014)	Weak	Moderate	Strong	Moderate	Weak	Moderate	Weak

**Table 3:** Characteristics of included studies

Study & Reference	Study type	Sample size	Observational period	Evidence Level	conclusion
Bain and Moy., (J Oral Maxillofac Imp 1993)	Retrospective	2194 implants in 540 patients NS-1804 S-390	6 years	2b	Smoking as a Statistically significant factor in failure of dental implant Maxilla is more affected
Van steenberghe et al., (Clin Oral implants 2002)	Prospective	1263 implants in 399 patients	2 years	2b	Heavy smoking increased implant failure rate.
Sverzut et al., (J Oral Maxillofacial 2008)	Retrospective	1628 Implants in 650 patients		2b	No relation found between tobacco smoking and implant failure.
Klokkevold et al., (J Oral Maxillofac 2007)	Systemic review	---	5 years	2a	Smoking effects the outcome of implant treatment adversely.
Crawford Bain, ( Int J Oral Maxillofac Implants 1996).	Prospective	223 Implants in 78 patients, Non Smokers-176, SmokingNot Quitted-13, SmokingQuitted-34	Less than 1 year	3b	Smoking as a significant factor in the failure of dental implant. Cessation of smoking improves the success rate in smokers.
Lindquist et al., (Clin Oral Implants Res 1996)	Prospective	273 implants in 45 patients, Nonsmokers- 24, Smokers 21	12- 15 Years	2b	Significantly greater marginal bone loss in smokers than in non-smokers
Strietzel et al., (J Clin Periodontal 2007)	Meta-Analysis	NA	16 years	2a	Smoking identified as a significant attribute for implant failure
Cavalcanti et al., ( Euro J oral implantology 2011)	Retrospective cohort study	6720 implants in 1727 patients, Nonsmokers-4460, Smokers- 2260	5 years after loading	2b	Smokers have significantly more implant failure than non-smokers
Sanchez –Perez et al., (J Periodontology 2007)	Retrospective	165 implants in 66 patients, Max-105, Mand-60, Nonsmokers-70, Smokers-95	5 years	3b	Increased risk of implant failure found in heavy smokers. P<, 0.05.
Moy et al., ( Int j oral Maxillofacial 2005)	Retrospective	4680 implants in 1140 patients, Nonsmokers 967, Smokers 173	21 years	2b	Statistically higher rate of failure of implants experienced in smokers rather than Non Smokers.
Deluca et al., (Int J prosthodont 2006)	Retrospective	1852 implants in 464 patients, Non Smokers- 285, Smokers- 104	20 years	2b	Smoking at the time of implant placement increases risk of implant failure. Heavy smokers at higher risk of implant failure and Marginal bone loss
Clementini et al., (Inj J oral maxillofacial Surgery2013)	Meta-Analysis	5730 implants in 1883 patients	6-20years	2a	Peri implant bone loss is higher in smokers as compared to nonsmokers.
Stoker et al., (clin oral implant res 2012)	RCT	256 implants in 94 patients, Non Smokers- 59, Smokers-35	8 years	1b	Smokers have double Marginal Bone Loss
Schwartz Arad et al., (J Periodontol 2002)	Retrospective	959 Implants in 261 patients, Non Smokers-172, Smokers-89	3 years	2b	Smokers have more complication than Non Smokers. Incidence of complication was increased with increased duration of smoking.
Alsaadi et al., (Clinical Oral implant Res 2008)	Retrospective	6949 implants in 2004 patients	2 years	2b	Failure rates increased with cigarette consumption

Haas et al., (Prosthetic Dent 1996)	Retrospective study	1336 implants in 421 patients, Non Smokers-314, Smokers-107	22 months	2b	Bone loss have higher scores especially in maxilla of smokers as compared with mandible and implants in maxilla of non-smokers
Costa et al., (J Clin periodontal 2012)	Cross sectional	336 Implants in 80 patients, Smoking 21, Nonsmoking 59	5 years	2c	smoking Cessation eliminated the risk of peri-implantitis.
Gorman et al., (Impl Dent; 1994)	Cross sectional	2066 Implants in 310 patients, Non Smokers-228, Smokers 82	More than 3 years	2c	Smokers have higher implant failure rate than Non Smokers
Kumar et al., (Int j Maxillofacial Implant 2002)	retrospective	1183 implants in 461 patients, Non Smokers- 389, Smokers-72	18 Months	2b	No significant difference on implant failure between smokers and non-smokers
Vandeweghe et al., ClinDent (2011)	Retrospective	712 implants in 329 patients, Non Smokers-288, Smokers-41	1 – 2 years	2b	Smoking could not be related to bone loss in implants
Sgolastra et al., (J Clin Oral Implant 2014)	Meta-analysis	NR	15 years	2a	T No significant difference between smokers and nonsmokers (patient based analysis)

**5. Discussion**

This review attempts to assess if the risk of implant failure or peri implantitis is significantly higher in smokers than in nonsmokers. The negative effects of smoking and its effects on implants observed in the included studies are presented under the following headings:

**Negative effects of smoking:-**

Despite the fact that smoking is a significant risk factor for many chronic diseases, it still is common throughout the world. Smoking has also been shown to have deleterious effects on oral health and in particular on periodontal disease. In addition, smoking can also cause delayed bone healing, reduced bone heights and peri- implantitis (Bain and Moy, 1993)

Bain and Moy, (1993) were the first to suggest the effect of smoking on implant failure reporting that smoking caused both systemic and local effects and negatively influences wound healing through decreased tissue oxygenation. There are various other studies which suggest impaired wound healing but the accurate phenomena still remains unknown. According to some studies the cytotoxic effect of hydrogen cyanide and nicotine causes vasoconstriction leading to weakened tissue perfusion and this altered response of tissue leads to ineffective healing (Bain, 1996) Others suggest that the mechanism behind compromised wound healing may be due to impaired polymorphonuclear cell function, increased levels of carboxyhemoglobin in blood and increased levels of fibrinogen (Klokkevold et al., 2007). Van Steenberghe et al., (2002) and Sverzut et al., (2008) reported that failure of implants occurs at the bone–implant interface due to the deposition of fibrous tissue. According to these authors, the components present in cigarette smoke affect the differentiation of pluripotent mesenchymal cells which in turn affects the fibroblasts and osteoblasts resulting in inadequate healing around the implant. Similarly Gorman et al (1994) found that significant number of implants were lost in smokers as compared to nonsmokers after osseo-integration and prosthetic loading(p=0.002)

Apart from the systemic vasoconstriction effect reported by Klokklord et al., (2007) nicotine also shows localized vasoconstriction effects (Bain & Moy, 1993) by being absorbed through the oral mucosa during smoking.

Therefore negative effects of smoking doubles the risk factor on the oral tissues.

**Smoking as a risk factor:-**

The relationship between smoking and dental implant outcome was first implicated by Bain and Moy, (1993). Majority of studies show statistically significant associations between implant failure and smoking suggesting it as an important cause in their loss (Sgolastra et al., 2015; Bain & Moy, 1996; Gorman et al., 1994; Moy et al., 2005; Strietzel et al., 2007; Vandeweghe et al., 2011; Cavalcanti et al., 2011).

In a systemic review, Strietzel et al., (2007), determined smoking as an important risk factor in dental implant therapy. The authors included 35 studies in their systemic review and 29 for meta –analysis. The meta-analysis found that smokers had significantly higher risk for implant failure as compared to non-smoker. The systematic review analysed an increasing risk of periimplant inflammatory complications in periodontally compromised smokers. According to the authors an increased risk of inflammatory peri-implant complication is expected in smokers because of the significant decrease of neutrophil elastase as well as vasoconstriction which effects the end arterial gingival vessels hence resulting in diminishing the inflammatory peri-implant reaction. (Strietzel et al., 2007)

In a 5 year retrospective study, Cavalcanti et al.,(2011) found that failure of implants were doubled in smokers (5.5%) as compared to nonsmokers (2.9%). Similarly, Sanchez Perez et al.,(2007) also reported a much higher failure rate in smokers as compared to non-smokers suggesting smoking as an important risk factor in implant failure.

Several studies of over periods of 20 years or greater (Moy et al, 2005 and Deluca et al., 2006) also confirmed smoking as an important risk factor for implant failure. Gorman et al.,(2014) reported smoking as a significant factor for implant failure between the time of implant placement and second stage of surgery. Although Implant failure rate of smokers was found to be twice as that of non-smokers this study did not specify the amount of smoking and type of smoking at the time of history taking.

**Smokers experiencing more marginal bone loss:-**

Clementini et al., (2014) reported that smokers presented a higher level of peri-implant bone loss (0.164 mm/year) than non-smokers. Similarly smokers experienced more marginal bone loss as compared to non-smokers in the results of a 10 years prospective study of mandibular implants by Lindquist et al., (1996). This significant peri-implant bone loss in the mandible was also found in their 15 years results. Contrary to this Bain and Moy, (1993) reported higher failure rates in maxilla as compared to mandible suggesting that this may be due to the fact that maxillary bone is of lower quality than mandibular bone and thus can be more susceptible to have harmful effects of smoking.

Similarly, Stoker et al., (2012), also showed considerably more loss of marginal bone in smokers than the nonsmokers finding that the marginal bone loss was more extreme in patients having four implants than in patients having two implants. In this study marginal bone loss was seen to be almost twice as great in smokers, independent of the treatment strategy. Similarly studies like Haas et al., (1996); Bain and Moy, (1996) and Schwartz -Arad et al., (2002) stated that smokers suffered increased marginal bone loss. Haas et al., (1996) showed that smokers have adverse results around the integrated maxillary implants with more frequent peri-implant inflammation and higher mesial and distal bone loss radiographically. However the definition of smokers was not specific e.g. all the patients were considered smokers who smoked at the time of examination. Many patients who had complicated smoking histories and did not smoke at the time of examination were considered as nonsmokers; this can definitely obscure possible difference between the two groups and can lead to variable results. Moreover the smoking habits were assessed by the patient's own statement which can lead to unreliable information.

**Effect of frequency and duration of smoking on implant**

The majority of studies found that increased frequency and number of cigarettes smoked caused increased rates of implant failure (Schwartz-Arad et al., 2002; Deluca et al., 2006; Alsaadi et al., 2008). Schwartz -Arad et al., (2002) found significantly more complications in groups of mild and heavy smokers than in nonsmokers. Similarly Alsaadi et al., (2008) observed that an increase in the number of cigarettes smoked increased the risk of early implant failure. Significantly higher rates were seen in heavy smokers (>20 cigarettes/day) as compared to no smoker groups {p-value <0.001, Odds ratio (95% CI): 2.72(1.63-4.54)} (Alsaadi et al., 2008)

DeLuca et al., (2006) observed statistically significant failure rates with increased use of cigarette smoking thus establishing a strong relation between the amount of cigarettes smoked and implant failure. This 20 years of follow up retrospective study proved by linear regression model that a history of smoking > 25 cigarettes per day was a predisposing factor for a higher risk of late implant failure.

Studies like Van Steenberghe et al., (2002) show that higher implant failure is associated with the amount of citrate consumption and of the quality of bone as well. Van Steenberghe et al., (2002) reported that heavy smokers have higher implant failure with type IV bone while Crawford et

al., (1996) reported implant failure is more in the posterior maxilla and least in the anterior mandible. Lindquist et al., (1996) reported heavy smokers (>14 cigarettes/day) have significantly greater marginal bone loss than those having low cigarette consumption (<14 cigarettes /day).

**Smoking cessation causes reverse effect of peri implantitis:-**

Costa et al., (2012) suggested that smoking cessation prevents a major risk factor for the incidence of peri-implantitis. In their study, former smokers were passed through an extensive period of smoking cessation ( $9.2 \pm 6.3$  years) and were not significantly associated with the occurrence of peri-implantitis. Gorman et al., (1994) also reported the same and observed that quitting smoking gives implant outcome similar to that of nonsmokers. Similarly, Bain, (1996), observed a statistically pronounced difference between smokers quitting (SQ) and smokers non quitting (SNQ). The failure rate of SQ was 12% while the failure rate of SNQ was reported as 38% thus proving the reverse effects of implant outcome by smoking cessation. Although Bain et al., (1996) showed that there is a reduction of effects of smoking on dental implant outcome on patients who quit smoking, the length of time after quitting was not sufficiently investigated. Bain and Moy, (1993) also hypothesized that if smoking is stopped, prior to implant placement, for a period of 8 weeks, it would enable healing of bone and osseointegration. Deluca et al., (2006) also suggested that smoking cessation improves chances of successful osseointegration. According to the study patients who stopped smoking one week prior to implant surgery showed lower incidence of early implant failures as compared to smokers who showed 1.69 times higher incidence of early implant failure (DeLuca et al., 2006)

**Studies showing no association between smoking and implants:-**

In contrast to most of the above studies some studies failed to show a significant negative effect of smoking on implant outcome. Kumar et al., (2002); Sverzut et al., (2008) and Vandheweghe et al., (2011) did not find any statistical differentiation between smokers and non-smokers thus contradicting smoking as an important factor with early implant failure. Sverzut et al., (2008) concluded that smoking alone cannot be considered as a risk factor related to early implant failure. However the retrospective design of this study and the small size of this study must have led to biases. Moreover the patient selection could have influenced as the participants were informed of the harmful effects of tobacco prior to the treatment being started. Similarly, Kumar et al., (2002) did not find any statistical difference between smokers and non-smokers. However there was a difference in placement of implant e.g. some were placed immediately after extraction, some placed with or without bone grafting or membrane. Moreover the classification of bone type could have been varied between the two operators. All these factors could lead to a bias and affect the results adversely.

In another review, while assessing smoking as a risk factor, Sgolastra et al., (2014) found that there is not much available information that confirms smoking as a risk factor for peri

implantitis. The patient-based analysis did not report any remarkable variation for the risk of peri-implantitis in smokers (Sgolastra et al., 2014). But some important points should be taken into consideration while analyzing the results. Firstly, the quantitative analysis could have been underpowered as the included studies were small in number causing a limitation of important results in the patient-based analysis. Secondly, the definition for smoking status was included only in a small number of studies and the included definitions varied greatly, which could have affected the meta-analysis result adversely. Finally all the included studies failed to reach the maximum score for the Newcastle-Ottawa scale hence high risk of bias in most of the studies.

## 6. Conclusion

This review has tried to assess the significance of smoking as a risk factor in peri-implantitis and implant failure. The harmful effects of smoking on general health have long been known but its effect on dental implants has only been the focus of research for the past two decades, in particular since the work of Bain and Moy, (1993). The interest in this area of research is evidenced by the number of studies that were retrieved for this review, not only the included papers but also many literature reviews, animal studies and other design papers, which were excluded but also suggested an association between smoking and peri-implantitis.

Most of the literature in this review concluded that smoking is linked with peri-implantitis and implant failure while only a few of studies were unable to establish an association (Kumar et al., 2002; Sverzut et al., 2008 and Vandheweghe et al., 2011). Some of the studies show that the failure rate of implants in smokers is twice that of non-smokers (Haas et al., 1996; Bain and Moy, 1996). However most of the studies included were retrospective in nature and a clear definition of a smoker was variable between the studies. These variations can lead to miscalculations in evaluating and assessing the papers moreover most of the statistics collected relating to smoking habits was acquired from the patients. It is not uncommon for this data to be misreported by patients and consequently in many cases, can be unreliable; biochemical variation could have given much more reliable data without any bias. Similarly Bain and Moy, (1993) and Strietzel et al., (2007) described a patient as a smoker with no consideration to the quality or quantity of cigarettes smoked. The different types of cigarette smoked and the quantity of smoke can have different toxic effects thus resulting in variable results due to bias.

The current review also identified some papers with other risk factors which could have led to increased implant failure. These confounding risk factors were seen in papers like Moy et al., (2005) Medical conditions such as Crohn's disease, diabetes, SLE etc. were not totally accounted for.

Another point was that some of the studies like Sgolastra et al., (2014) included only small numbers of studies leading to them being underpowered in turn causing a lack of significant results in the patient-based analysis. Moreover only a few studies provided a definition for smoking status and some studies also utilised various definitions for peri-

implantitis, which may have influenced the meta-analysis outcomes in unpredictable ways. Finally none of the studies reached the maximum score on the Newcastle-Ottawa Scale thus most of the included studies seemed to be moderate to high risk of bias. (Sgolastra et al., 2014). Hence there is a need for prospective studies in future to validate such results.

Moreover the various studies included in this paper did not mention the quantity of cigarettes smoked per day or for how many years the patient have been smoking (Haas et al., 1992; Strietzel et al., 2007). The dose effect of smoking has shown significant effect on osseointegration (Lindquist et al., 1996). The lack of these factors restricted the degree to which robust conclusions could be drawn in this review of the literature.

Within the limitations of this review, it can be presumed that smoking is associated with increased marginal bone loss and peri-implantitis. Therefore a detailed history of smoking including its duration and intensity should be considered as an important aspect of the investigation stage of patient assessments and its findings should inform the treatment planning process. Patients should be informed about the susceptibility for implant failure and smoking cessation should be encouraged.

Despite the fact that smoking is considered an important risk factor associated to peri-implantitis, it should not act as a contraindication for implant therapy. Instead patients should be advised about the benefits of smoking cessation rather than depriving them of implant therapy, smoking should be regarded as a controllable risk factor.

Smoking cessation definitely enhanced the dental implant outcome, moreover the duration and frequency of smoking increased the susceptibility to peri-implantitis. These factors should be explained to the patient in detail and smoking cessation should be encouraged. Those patients who seem to be motivated in stopping should be referred to more intensive counselling services so as to increase their chances of quitting successfully. Patients who stop smoking prior to the implant procedure can attain similar outcomes to nonsmokers. For those who do not agree to quit smoking the likelihood of failure and complications should be explained as part of the consent process.

This paper presents an overview of the knowledge of the effects of smoking on the dental implant outcome. The majority of papers confirm that heavy smokers have higher risk of developing peri-implantitis and of suffering implant failure, this knowledge help the general practitioner during the decision making process that accompanies treatment planning.

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Level	Type of evidence
1a	Systematic review with homogeneity of randomized control trials
1b	Individual randomized control trial with a narrow confidence interval
1c	All or none related outcome
2a	Systematic review with homogeneity of cohort studies
2b	Individual cohort study (including low-quality randomized control trials, e.g., <80% follow-up)
2c	“Outcomes” Research; Ecological studies
3a	Systematic review with homogeneity of case-control studies
3b	Individual case-control study
4	Case-series (and poor-quality cohort and case-control studies)
5	Expert opinion without explicit critical appraisal, or based on physiology, bench research or “first principles”
<b>Grades of recommendation</b>	
A	Consistent level 1 studies
B	Consistent level 2 or 3 studies or extrapolations from level 1 studies
C	Level 4 studies or extrapolations from level 2 or 3 studies
D	Level 5 evidence or troublingly inconsistent or inconclusive studies of any level

Figure 3: Level of evidence  
(Source from <http://www.cebm.net/>)