A retrospective analysis of factors affecting levels of presenting Periodontitis of patients referred to the Royal London Dental Hospital

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Declaration Regarding Plagiarism

Except for the assistance mentioned in the acknowledgements, the Contents of this dissertation are entirely my own work. This work has not been previously submitted, in part or in full, for a degree or Diploma of this or any other university or examining board.

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List of Abbreviations

B	Regression weights for model
BoP	Bleeding on probing
BSP	British Society of Periodontology
CAL	Clinical attachment levels
CBCT	Cone beam Computer tomography
CVDs	Cardiovascular disease/s
DM	Diabetes mellitus
Df	Degrees of freedom
EFP	European federation of Periodontology
FI	Furcation involvement
GDP	General dental practitioner
IDB	Interdental brushes
NHANES	National Health and Nutrition Examination Survey
NS	Not significant
NSPT	Non-surgical periodontal therapy
OPG	Orthopantomogram
OR	Odds ratio
Pa	Periapical
Pg	Porphyromonos gingivalis
PHE	Public Health England
PPD	Probing pocket depth
RA	Rheumatoid Arthritis
RLDH	Royal London dental Hospital
RR	Relative risk
SE	Standard errors
SEA	South-East Asian
SEP	Socio-economic position
TNF-a	Tumor necrosis factor-alpha
Wald	Ratio of regression weight to SE

Abstract

Objectives: To describe the socio-demographic features of the typical periodontal referral cohort; the levels of presenting periodontitis at Royal London Dental Hospital (RLDH) with reference to the 2017 European Federation of Periodontology (EFP) classification; identifying the risk factors/risk indicators for Periodontitis in the population; use multivariate logistic regression modelling to determine the relative contribution of risk factor/indicators to levels of periodontitis determined by a focus on tooth loss as the primary outcome

Materials and Methods: This retrospective cross-sectional analytical study used a subset of data from subjects that were referred, clinically assessed on periodontal Consultation clinics at the RLDH and subsequently taken on for periodontal treatment. The sample consisted of 150 individuals (92 females/ 58 males), aged 16+ years (16-79 years old). Risk factors/indicators were assessed in relation to tooth loss and mean probing pocket depth (PPD) of ≥4mm.

Results:
Smoking, Age>40 years and self-declared stress at baseline assessment were significant predictors of tooth loss and only poor plaque control (defined as >50%) was a significant predictor of mean PPD≥4mm. A higher proportion of patients in the South-East Asian (SEA) cohort were younger, suggesting that they present earlier with severe disease, they presented with lower levels of self-reported stress and higher numbers of irregular dental attenders.

Conclusion:

Within the limitations of this retrospective cross-sectional study, it is unclear whether the SE-Asian group demonstrated with significant Periodontitis disease severity more or less than the other Ethnic groups combined. There is a lack of strong evidence to demonstrate that there are more diabetics amongst the SEA population, and as to whether this is controlled or uncontrolled DM. However, in this study the SEA group had lower levels of self-reported stress and higher numbers of irregular dental attenders compared with other ethnic groups combined.

Chapter 1. Introduction

Periodontitis is defined as a "chronic multifactorial inflammatory disease associated with dysbiotic plaque biofilms and characterised by the progressive destruction of the tooth supporting apparatus." (Papapanou *et al.*, 2018). The principal features are clinical attachment loss (CAL), increased probing pocket depths (PPD), the presence of gingival bleeding and radiographic evidence of alveolar bone loss (Papapanou *et al.*, 2018). The overall prevalence of severe periodontitis (1990-2010 Global burden of disease study) in the world is approximately 11%; being marked as the 6th most prevalent disease (Tonetti *et al.*, 2017; Kassebaum *et al.*, 2014). An individuals' self-esteem, quality of life and the socio-economic impacts are affected with the potential burden that comes with periodontitis and its progression (Chapple *et al.*, 2015).

A new classification system for Periodontitis has been accepted in 2018 which was revised at the 2017 Periodontology World Workshop. This consists of a staging and grading format, were the staging is referring to the severity, complexity and distribution of periodontitis. The grading section is the proposed rate at which the disease progresses. The stages are I to IV, they represent the degree of the lost supporting periodontal tissue structures by direct clinical examination and radiographic assessment, including tooth loss because of Periodontitis (Papapanou *et al.*, 2018).

The grading can be confirmed either through direct longitudinal evidence of clinical attachment loss or bone loss over 5 years if present. Or indirect evidence through the amount of bone loss through radiographic means and is calculated as the amount (percentage) of bone loss/age of the patient. The result is either in Grades A, B or C, with C representing rapid rate of progression. The correlation of plaque and distribution of periodontitis (localised <30%, generalised >30% or 1st Molar – incisor patterns of teeth affected) with the severity of periodontal destruction can also be factored in, to decide which grade will be given. Finally, there are grade modifiers which can contribute to periodontitis progression, these are diabetes and smoking (Papapanou *et al.*, 2018).

Periodontitis has a well-established set of associated risk factors e.g., bacterial plaque, smoking, uncontrolled diabetes etc. However, it remains unclear as to whether certain ethnic groups are more prone to disease or whether socioeconomic status or clustering of risk factors within certain populations

increases prevalence (Eke *et al.*, 2018; Eke, Borgnakke and Genco, 2020; Montero *et al.*, 2019; Jin *et al.*, 2011; Jepsen *et al.*, 2017).

The Royal London Dental Hospital (RLDH) is an NHS specialist referral centre for periodontitis and serves the surrounding areas of North-East London. It is situated within the London borough of Tower Hamlets, one of the most economically and socially deprived boroughs in the UK (PHE statistics) with a predominant Bangladeshi population (32% of the total borough population). Indeed, more widely, the Bangladeshi and Pakistani populations constitute a significant population of North-East London with high population proportions in Newham (Bangladeshi 12.4%, Pakistani 9.8%), Redbridge (Bangladeshi 5.7%, Pakistani 11.1%) and Waltham Forest (Bangladeshi 2.3%, Pakistani 10.2%). Public Health England data for the local borough demonstrates poorer general health with high levels of diabetes, obesity, cardiovascular disease, and smoking compared to the national average (PHE Statistics). Indeed, one previous study of the local population reported higher levels of periodontitis within these minority ethnic groups (Delgado-Angulo, Bernabé and Marcenes, 2016). Consequently, it follows that an understanding of the risk profile of our local ethnic groups is important in managing periodontitis more broadly. It is widely accepted that national dental health statistics (including The Adult Dental Health survey of 2009) under-represents ethnic minority populations (Steele *et al.*, 2012).

The aim of this retrospective study is to identify pre-operative risk factors and risk indicators associated with presenting levels of periodontitis in consecutively treated patients at the RLDH with a focus on the ethnicity of the population.

Chapter 2. Literature Review:

2.1) Risk Factors for Periodontitis:

A risk factor is defined as a 'behavioural, environmental or biological association with the disease and is confirmed by interventional or longitudinal studies.' (Lang NP and Lindhe, 2015). Risk indicators are potential risk factors. They can be considered true risk factors if proven to show significant association with the disease in question.

Several risk factors could contribute towards the periodontal disease, whether to its onset, progression or both and can be divided into modifiable (local or systemic) and non-modifiable systemic factors.

2.2) Local Modifiable Risk factors

2.2.1) Plaque:

The dental plaque biofilm is considered one of the most important risk factors in gingivitis and periodontitis. It contributes significantly to the progression of periodontal disease from gingivitis. A classical study identified a large group of Sri Lankan labourers that did not carry out common/basic oral hygiene

brushing resulting in high levels of plaque accumulation, calculus formation and staining of the teeth. The consequences of this were varying levels of periodontitis demonstrated. The subjects were grouped into no, moderate and rapid progression of periodontitis with respect to the rate of attachment loss (Löe *et al.*, 1986). The combination of specific, non-specific microflora along with the hosts risk factors contribute to periodontal disease (Marsh, 1994; Hajishengallis, Darveau and Curtis, 2012; Page and Schroeder, 1976).

2.2.1a & 1b) Factors affecting the plaque retention

2.2.1a) Manual vs Electric Toothbrushes:

It is important to extract from the patient, their knowledge, techniques/oral hygiene regime/habits; this is with respect to toothbrushing frequency and time spent brushing, methods of brushing, whether electric or manual including the brush design and how often they change their manual toothbrushes or electric toothbrush head. Systematic reviews (Slot *et al.*, 2012) have shown that brush designs and time is important in reducing plaque control. Moreover, studies (Van der Weijden *et al.*, 1996; Yaacob *et al.*, 2014) appear to have tipped the balance favouring electric over manual and sonic toothbrushes in reducing levels of supragingival plaque. Such information can be useful into understanding why and finding patterns on the presenting levels of periodontitis that have been assessed at the RLDH.

2.2.1b) Floss vs Interdental cleaning techniques:

The consensus report in 1998 of the European Workshop on Mechanical plaque controls identified that with the regularly performed toothbrushing techniques, there appeared to be undisturbed areas were the plaque was forming and this pattern was not altered due to the inefficacy of the toothbrush to access and remove the interdental plaque.

An overview paper (Ng and Lim, 2019) of the various interdental cleaning aids and how effective they are in contributing to achieving low plaque levels as well as a reduction in gingival inflammation concluded that interdental brushes (IDB) may potentially achieve lower plaque and gingival indices compared to dental floss, especially if the flossing technique is not carried out properly. There is also indication for IDB were periodontitis-based patients display with embrasures that are much wider.

Interdental cleaning techniques (Gjermo and Flötra, 1970) were even demonstrated some decades back to be significant in reducing plaque levels, so obtaining information from the patient on their awareness and application of this contributes further to forming a picture of the patients knowledge as well as their motivation and attitude towards maintaining good oral health, which can be reflected in the level of periodontitis they present with and critically one of the key elements in the management of patients with periodontitis.

2.2.2) Calculus:

Calculus also known as the mineralised form of dental plaque is a secondary etiological risk factor for Periodontitis (Akcalı and Lang, 2018). The main issue isn't the calculus itself; it is the bacterial plaque biofilm which can harbour itself onto the surface of the calculus whether supra or sub-gingivally (Mombelli *et al.*, 1995). It is essential to remove as much apparent calculus as possible, but the argument is more so that the disruption of the bacterial plaque biofilm in the vicinity or on the surface of the calculus is the greater imperative objective. As studies have shown cellular attachment between calculus and the junctional epithelium, (Listgarten and Ellegaard, 1973).

2.2.3) Removable dental prostheses:

Previously, partially removable dental prostheses were associated with higher presenting cases of gingival inflammation, Periodontitis and caries lesions due to the high levels of presenting dental plaque biofilm. However, those studies did not provide information of the subjects' knowledge and level of self-performed plaque, including control/oral hygiene regime. Information was also absent regarding the supportive periodontal maintenance programs these subjects were on, including the level of periodontitis disease they presented with (da Fonte Porto Carreiro *et al.*, 2017).

The conclusions so far state the following: If the removal partial prosthesis is designed correctly, respecting the patients' soft tissues and connection with the adjacent remaining dentition; the prostheses is reviewed on a regular

basis; the patients are demonstrating very good self-performed plaque control, including periodontal maintenance were required. Then these removable dental prostheses do not contribute to more plaque accumulation, thus not leading to increased periodontal clinical attachment loss and tooth mobility (Ercoli and Caton, 2018).

2.2.4) Overhanging margins/Poorly Contoured Restorations:

Defective marginal restorations and overhangs can lead to encroachment of the embrasure space and increased levels of plaque locally, causing build-up of plaque biofilm and changes to the microflora (particularly sub-gingivally) (Jeffcoat and Howell, 1980; Lang, Kiel and Anderhalden, 1983). Indeed, there is evidence that larger overhangs are associated with more alveolar bone destruction and potentially further clinical attachment loss compared to smaller ones (Jeffcoat and Howell, 1980).

2.2.5) Open Contacts

There is some evidence with respect to open contacts. In one paper there was a statistically significant relationship observed between contact type and food impaction and between food impaction and pocket depth but not between contact type and pocket depth or gingival index, (Hancock *et al.*, 1980). Another studied also focused on open contacts sites but also showed some statistical significance in food impaction in these areas and increased pocket depth (Jernberg, Bakdash and Keenan, 1983).

2.2.6) Compliance/Attendance:

There is a well-established body of evidence supporting the importance of compliance and good long-term attendance in maintaining periodontal attachment (Axelsson and Lindhe, 1981b; Hirschfeld and Wasserman, 1978; Axelsson and Lindhe, 1981a) Regular attendance, periodontal health knowledge and dental practitioner instructions have proven important in maintaining periodontal attachment reported in cross-sectional surveys; one, identified a strong positive correlation between regular attendance and periodontal health knowledge (Varela-Centelles *et al.*, 2020). The other reported a moderate association between dental practitioner instructions, periodontal knowledge, homecare regimes and self-reported periodontal health (Hughes, Heo and Levin, 2018).

2.2.7) Cervical enamel projections and Enamel pearls

Enamel Pearls and cervical enamel projections have been considered to have an impact on periodontal health as their size, parameters and location on the tooth can provide a more opportunistic environment for the plaque biofilm to grow and incidentally compromise the ability for its removal.

For cervical enamel projections this is focused more about their extension into the furcation regions of teeth and has led to their associative contribution of increased pocket depth formations as well as the levels of clinical attachment loss. Some studies (Hou and Tsai, 1987; Hou and Tsai, 1997) have suggested the prevalence of cervical enamel projections in molars with and without furcation involvements to be around 82.5% and 17.5% respectively, but the

majority being found on the mandibular first molars. One study showed (Roussa, 1998) the incidence of these projections to be around 30% in molar teeth.

There can be a more damaging effect on the tooth apparatus (periodontal tissues) especially if these projections extend more into the furcation area, (Blanchard *et al.*, 2012) and in addition to this a narrow furcation area (Bower, 1979; Chiu *et al.*, 1991) serves a much bigger challenge to debride as the majority of their entrance width is less than that of a new Gracey curette.

Enamel pearls have a lower prevalence than cervical enamel projections, approximately in the range of 1.1-9.7%, (Moskow and Canut, 1990) but they can also harbour dental plaque biofilm, contribute to the aetiology of furcation involvements and to the progression of periodontitis in those localised areas. The order of the most common areas they are usually found is the maxillary third molars, mandibular third molars, the distal aspect of the maxillary second molars, (Goldstein, 1979; Moskow and Canut, 1990; Risnes, 1974). While another study identified them mainly in the mandibular than maxillary teeth predominately the buccal furcation aspect of the mandibular first molars, (Çolak *et al.*, 2014).

2.2.8) Root Concavities

Root concavities are usually identified during non-surgical periodontal therapy or surgical access mainly under the use of local anaesthetics making them hard to diagnose initially. Nonetheless these can also contribute to periodontal

disease and its progression as they provide an environment for dental plaque biofilm to thrive and are difficult to engage and remove. They are located in all molars up to a certain degree, as well as affecting the bicuspids (premolars) and anterior teeth. With regards to the premolars, (Dababneh and Rodan, 2013; Zhao *et al.*, 2014) depending on where the concavity is located (usually in the interproximal areas) either coronally or more apically in relation to the cemento-enamel junction, the ability to debride that area may be challenging and thus potentially causing areas of periodontitis. Even though the patient may have very good self-performed supragingival plaque control, dental plaque biofilm may still congregate in some of these concavities which could bring a rise to localised periodontitis.

2.2.9) Root proximity

Root proximity has been somewhat considered a potential risk marker for periodontitis. The main areas that have been identified for the maxillary arch seen bilaterally or unilaterally were between the maxillary molars, maxillary central and lateral incisors. For the mandibular teeth all the unilateral root proximities are the same as the maxillary arch, but the bilateral ones were predominantly seen between the central and lateral incisors. The study illustrated that the width and severity of the root proximities determined whether there is a higher chance of detecting angular or horizontal bone loss in the interdental space. (Loukideli *et al.*, 2011; Vermylen *et al.*, 2005a; Vermylen *et al.*, 2005b). However due to a heterogeneity in the definition and measurements of root proximity within the various literature, it has been difficult to ascertain its true effect on the periodontium.

2.2.10) Tooth crowding

Tooth crowding and positioning can create an environment for dental plaque biofilm to build-up an mature and as result of this give rise to gingival inflammation; with continued gingival inflammation and depending on the hosts response this could go on and progress to periodontal disease, but there have also been studies which showed no relationship between alveolar bone loss and crowded dentition subject to a high standard of plaque control by the patients. (Matthews and Tabesh, 2004).

2.2.11: Furcation Involvement

The degree and location of furcation involvement is considered a modifiable local anatomical risk factor for Periodontitis and tooth loss, (Ehnevid and Jansson, 2001). The main reason is the difficulty in accessibility to maintain good self-performed plaque control in those sites, in particular the proximal sites of the maxillary molars as well as access from the clinician to perform supra/subgingival debridement. Retrospective studies and systematic meta-analysis reviews have shown that there is a higher risk of tooth loss with molars with furcation involvement, (Helal *et al.*, 2019; Nibali *et al.*, 2016).

Moreover, not only the horizontal component but having vertical furcation involvement as well, has demonstrated an increase in the risk of tooth loss in the long-term demonstrated by retrospective studies and systematic reviews, (Helal *et al.*, 2019; Nibali *et al.*, 2018; Tonetti, Christiansen and Cortellini, 2017).

2.3) Systemic Modifiable risk factors

2.3.1) Diabetes:

Diabetes is defined by the World Health Organisation as, “a chronic, metabolic disease characterized by elevated levels of blood glucose (or blood sugar), which leads over time to serious damage to the tissues of the heart, blood vessels, eyes, kidneys, and nerves,” (Association, 2013); but importantly to include periodontal disease as an association with diabetes mellitus.

Diabetes Mellitus (DM) type I is the result of autoimmune pancreatic beta-cell destruction leading to insulin deficiency also referred to as the insulin dependent Diabetes, (Association, 2019). Whereas in type II DM there is a progressive loss in pancreatic beta-cell insulin secretion regularly concomitant with insulin resistance. (Association, 2019). Both these diseases can occur amongst children and adults. The presentation of clinical hyperglycaemia is what mainly influences the human immunological pathways that lead to the damage of tissue organs. The most frequent presenting symptoms are: polyuria, polydipsia, polyphagia, pruritis, weakness and fatigue, (Association, 2019).

Diabetes is now a well-established independent risk factor for periodontitis with evidence of increased attachment loss in Type 1 (Dicembrini *et al.*, 2020) *et al.*, 2020) and Type 2 diabetic patients regardless of gender and age (Shlossman *et al.*, 1990). Patients with poor metabolic control had 11 times more chance of progressive bone loss than non-diabetic patients (Taylor *et al.*, 1998). A recent consensus report and systematic review identified strong

evidence of a bi-directional relationship between diabetes and periodontitis (Sanz *et al.*, 2018; Badiger *et al.*, 2019). Furthermore, it has been recommended that diabetic control interventions comprising of the counselling of the patient's diet, patient education about diabetes and patient referral to achieve glycaemic control in cases of hyperglycaemia should be carried out to contribute in achieving realistic successful outcomes in periodontitis therapy (Ramseier *et al.*, 2020).

2.3.2) Smoking:

Smoking is now established as one of the single most important modifiable local and systemic risk factors for periodontitis. Until the mid-1980s, it was thought that the increased periodontitis in smokers was due to poorer oral hygiene, however this was dispelled by the experimental study that demonstrated similar rates of plaque formation in smokers and non-smokers, less bleeding and clinical inflammation in smokers (Bergström and Preber, 1986). Other studies have identified similar trends (Feldman, Bravacos and Rose, 1983; Luzzi *et al.*, 2007) and found that smoking may also promote dysbiotic biofilm formation (Hutcherson, Scott and Bagaitkar, 2015).

Furthermore, there are various mechanisms that have been reviewed underlying the effect of smoking on periodontitis. Smoking affects both the gingival epithelium and gingival connective tissue as well as components of the immune system. From the gingival epithelium aspect the nicotine component in the cigarettes contributes to an increased rate of proliferation of the gingival epithelium resulting in an epithelial thickness amongst smokers,

(Gültekin, Sengüven and Karaduman, 2008). The nicotine serves a role in connective tissue breakdown and reduced collagen production which is associated with the impaired wound healing in smokers, (Tipton and Dabbous, 1995). The nicotine also binds to nicotinic cholinergic receptors centrally and peripherally resulting in the release of various neurotransmitters including adrenaline and noradrenaline, vasopressin, calcitonin resulting in vasoconstriction of the blood vessels, thereby leading to a decrease in gingival bleeding and exudate production, (Yugar-Toledo *et al.*, 2005). The reports of reduced gingival bleeding and bleeding on probing (suppressed gingival inflammation in smokers) is potentially related to the reduced number of gingival blood vessels, (Palmer *et al.*, 2005).

Smoking can conceivably have a negative influence on the innate and immune response. With regards to Periodontitis, this can result in a heightened expression of tissue breakdown and impaired tissue repair. This is due reduced antibody production, inhibition of neutrophil function and impaired fibroblastic functions, (Söder, Jin and Wickholm, 2002; Matthews et al., 2012).

Knowing the significance of the effect smoking has on the periodontal tissues, it has been strongly recommended to patients to cease smoking tobacco, to increase the chances of a potentially successful outcome on the periodontal therapy treatment plan they are on (Ramseier *et al.*, 2020). Especially those with advanced forms of periodontitis (Tomasi, Leyland and Wennström, 2007b).

2.3.3) Obesity:

Obesity has been considered as a risk factor in connection with periodontitis in various studies, (Mealey, Oates and Periodontology, 2006; Nishimura and Murayama, 2001; Saito, Shimazaki and Sakamoto, 1998) there is suggestive pathological mechanisms connected with obesity causing insulin resistance resulting in the development of type 2 diabetes. The considered pathways include pro-inflammatory cytokine imbalance, immune response dysfunction/modulation and increased cellular stress which play a part in periodontal tissue destruction (Taylor, Preshaw and Lalla, 2013). However, according to the Bradford Hill (1971) criteria there hasn't been final conclusions in terms of mechanisms and temporality. Therefore, more studies are warranted especially longitudinal ones.

2.3.4) Stress:

Psychosocial stress has been considered another modifiable systemic risk factor for periodontitis. It is defined as, 'a state of mental or bodily tension resulting from factors that tend to alter an existent equilibrium.' (Lang NP and Lindhe, 2015).

A narrative review (Sabbah, Gomaa and Gireesh, 2018) discussed various biological reaction pathways that occur as a consequence of stress. It illustrates that the levels of cortisol produced as a result of the activation of the hypothalamus-pituitary-adrenal axis from a stressful situation, can trigger an immune response; including the release of substance P from the stimulation of autonomous nervous system, which has an indirect effect on the mast cells

and can also modulate the hosts inflammatory, cellular and humoral immune responses. It was concluded in the review that the stress hormone cortisol has been associated with increased periodontitis possibly through its action on the cellular and humoral immune response.

A systematic review (Castro *et al.*, 2020) following the preferred reporting items for systematic reviews and meta-analysis guide (PRISMA) which included the research papers in the above narrative review came to the conclusion that more studies are required to elucidate the connection between stress and periodontitis. There is little evidence to support stress reduction therapies as a means of improving periodontal outcomes at present.

2.3.5) Vitamins and Minerals:

Vitamins and minerals could play significant roles in periodontal health. Vitamin C (ascorbic acid) is considered one of the modulators of oxidative stress (Da Costa, Badawi and El-Sohemy, 2012). Oxidative stress (reactive oxygen species production) as a result of examples such as a hyperactive cell phenotype or impaired neutrophil function (Roberts *et al.*, 2015; White *et al.*, 2016), can contribute to collateral tissue damage. Vit C scavenges for reactive oxygen species (Nishida *et al.*, 2000), to try and reduce the levels of tissue damage, it is seen as mentioned above in polymorphonuclear leukocytes (neutrophils) and endothelial cells and its functions are expressed on periodontal fibroblasts as well as osteoclasts, (Mimori *et al.*, 2007). Iron has been considered an important mineral in ensuring there are good levels of

antioxidant enzymes (Chakraborty *et al.*, 2014) to counteract the actions of oxidative stress and avoid or aid in reducing damage to periodontal tissue. Zinc has shown significant contribution towards averting periodontitis caused through hyperglycaemic levels (Pushparani, 2014) by having an anti-oxidant mode of action.

2.3.6) Cardiovascular diseases (CVDs)

CVDs are the biggest contributors for global mortality and morbidity rates and with the average lifetime expectancy increasing over almost the last 3 decades these diseases have been a non-communicable burden (Roth *et al.*, 2017) Hypertension preceding heart failure, atrial fibrillation, stroke, cardiomyopathy, rheumatic and ischemic heart disease making up almost all (95%) of the CVD-related deaths (Roth *et al.*, 2015).

There has been a strong association and independence of a severe of periodontitis and high cardiovascular related deaths in multiple populations (Linden *et al.*, 2012). There have also been mechanisms proposed associated with elevated levels in oxidative stress, C-reactive protein, the combined contribution of bacteraemia with the systemic inflammatory response (Schenkein and Loos, 2013).

Epidemiological studies have been conducted to find associations between periodontitis and CVD. There has been evidence identifying arterial stiffness, increased thickness in the carotid intima-media and endothelial dysfunction

coronary heart disease and cerebrovascular disease (Dietrich *et al.*, 2013). The evidence from these studies has been identified from case control and cohort studies.

Periodontitis patients were found to have a two-fold risk of either a thrombotic or cardioembolic stroke when compared to patients who were diagnosed as presenting with periodontal clinical health (Sen *et al.*, 2018). There have been higher incidences of atrial fibrillation and heart failure identified in periodontitis patients compared with non-periodontitis patients identified from a Taiwanese national health insurance research database (Chen *et al.*, 2016).

There have been quite a few biological plausible mechanisms which have been proposed connecting systemic inflammation and the periodonto-pathogenic bacterial microorganisms and their contribution especially to atherosclerosis more so than the other CVDs such as myocardial infarction and stokes (Reyes *et al.*, 2013; Schenkein and Loos, 2013).

In conclusion, although the evidence is not absolute, there is enough to make recommendations to the medical health professionals to inform periodontitis or non-periodontitis patients that the risk for cardiovascular diseases is much higher, therefore the management of the risk factors (blood pressure, excess weight, nutrition in terms of glucose and lipid management, smoking, exercise, efficient periodontal therapy and periodontal maintenance) needs to be managed (Sanz *et al.*, 2020).

2.3.7) Rheumatoid Arthritis (RA)

RA is a “systemic autoimmune chronic disease,” with characteristics of bone and cartilage destruction of the joints affecting up to 2% of the world population (Bartold and Lopez-Oliva, 2020). The aetiology is not known, but there are a lot of similarities in terms of its pathogenesis in comparison with periodontitis. RA Patients tend to have a heightened immune response with elevated levels of local and pro-inflammatory cytokines such as tumour-necrosis factor alpha (TNF-a), interleukins 1-beta, 6, 15 & 17. This are also found amongst active periodontitis patients with inflamed tissues. Osteoclastic activity proceeds to be greater with the constant inflammation in the joints leading to joint structure degradation. This causes signs related to RA such as chronic pain in the joints, fevers, disability/functional impairment of the joints (Bartold and Lopez-Oliva, 2020; Hussain *et al.*, 2020).

Even though the aetiology and biological link is not known regardless of the above similarities, the current projected theories have investigated the correlative risk factors, oral microbiome, immune-imbalance and protein citrullination for more clarity into the epidemiological correlation between periodontitis and RA (Chen *et al.*, 2013a; Chen *et al.*, 2013b; Joseph *et al.*, 2013).

The main biologically plausible attempt to link periodontitis and RA is with regards to the distinctive host antibody reaction to the presence of citrullinated proteins found in RA patients. (Bartold and Lopez-Oliva, 2020; Rodríguez-Lozano *et al.*, 2019). Citrullination is a physiological phenomenon that occurs

in healthy individuals. It involves an enzyme family called peptidyl-arginine deiminase which is responsible for the conversion of the amino acid arginine into citrulline (Bartold and Lopez-Oliva, 2020). Porphyromonos gingivalis (Pg) is the only known bacterium to citrullinate human and bacterial proteins in an unfavourable manner forming epitopes. The immune system identifies these foreign epitopes and produce anti-citrullinated protein antibodies (ACPAs) which are characteristics of RA patients (Bartold and Lopez-Oliva, 2020).

In the synovium of RA patients, DNA content of Pg has been found; patients with severe forms of periodontitis have displayed higher levels of citrullinated proteins, potentially from Pg. Therefore, it has been hypothesised that with periodontal intervention the Pg load could decrease, leading to decreased citrullination activity resulting in the weakening of the autoimmune response in RA patients (Bartold and Lopez-Oliva, 2020).

2.4) Systemic unmodifiable risk factors

2.4.1) Age:

Longitudinal research studies have shown that both prevalence and severity of periodontitis increases with age (Albandar, Brunelle and Kingman, 1999; Dye *et al.*, 2007). However, this relationship is likely due to older patients having been exposed to periodontitis risk factors for a longer period and so having an increased chance of periodontal breakdown with time. Risk factors

that may increase with age e.g. diabetes risk, obesity and immune-senescence may also explain this relationship (Hajishengallis, 2010).

2.4.2) Genetics:

Genetic predisposition has been a huge subject of discussion on whether it is a true risk factor for periodontitis. A lot of focus had been of the IL-1 genotype polymorphism and its association with periodontitis, however studies failed to show the link (Huynh-Ba *et al.*, 2007). The genetics of periodontitis is still poorly understood and further research is needed to identify association with periodontitis and its progression (Schaefer, 2018).

2.4.3) Pregnancy and Adolescence:

Pregnancy and adolescents have been connected with clinical signs of bleeding on probing, pseudo/false pocketing and increased gingival crevicular fluid but all can be minimized with good plaque control. The changing levels of progesterone, oestrogen and other hormones modulate vascular responses and can influence connective tissue turnover (Miyazaki *et al.*, 1991; Soory, 2000).

2.4.4) Race/Ethnicity:

Previous dental health surveys in the USA (NHANES) have reported that African Americans demonstrate higher prevalence of periodontitis compared with Mexican Americans and non-Hispanic Caucasians (Albandar, Brunelle and Kingman, 1999; Dye *et al.*, 2007; Borrell and Crawford, 2008). Furthermore, another study showed that the benefits from education and

income were less in terms of periodontal health amongst the African American subjects compared to the other aforementioned ethnicities (Borrell *et al.*, 2004).

A previous cross-sectional community based research study (Delgado-Angulo, Bernabé and Marcenes, 2016) focused on identifying if there were ethnic inequalities and disparities in Periodontitis among British adults including their socioeconomic position (SEP). Self-completed questionnaires were used and recorded teeth exhibiting PPDs and CAL levels ≥4mm. They found that all Asian groups (Pakistani, Indian, Bangladeshi and Asian Others) had higher PPDs compared to the White British. The results were compelling, as the level of education modulated the extent of the disease as opposed to the SEP. But from the methodology this was a random population study with partial, not full mouth assessments, which may underestimate the prevalence and severity of the periodontitis and therefore not conceivably represent its true reflection between ethnic groups (Kingman, Susin and Albandar, 2008; Müller, 1986; Papapanou, 1996; Susin, Kingman and Albandar, 2005).

The adult dental health survey 2009 illustrated only a small participation of the ethnic minority groups when analysing the data and this report did not include any ethnic categorical demonstration of oral health inequalities. This survey did not give representable generalisable data of the ethnic communities and no evidence of which minority groups were included (Steele *et al.*, 2012).

There is a lack of data and research to suggest that the severity of periodontitis presented at the RLDH is more than other London Boroughs and within the UK, bar the discussed epidemiological study above (Delgado-Angulo, Bernabé and Marcenes, 2016). In addition to this, the East London boroughs are highly populated by Asian communities with the majority being of Bangladeshi and Pakistani ethnic origins. Focusing more on the risk factors that are relevant to these populations as well as understanding whether risk is down to genetic differences or modifiable factors may help drive more focus on our interventions targeting an improvement in health-related quality of life.

Chapter 3. Hypotheses:

a) Periodontitis severity is significantly more in the South-East Asian populations compared to other referred ethnic groups combined (in connection with the presence of a higher number of established risk factors)
b) South-East Asian population are more likely to be diabetic compared with the other Ethnic groups combined

Chapter 4. Aims:

The aim of this retrospective study is to identify pre-operative risk factors and risk indicators associated with presenting levels of periodontitis in consecutively treated patients at the RLDH with a focus on the ethnicity of the population.

Chapter 5. Objectives:

i) To describe the socio-demographic features of the typical periodontal referral cohort

ii) To describe the levels of presenting periodontitis at the RLDH using both categorical and continuous definitions with reference to the 2017 EFP classification

iii) To identify the risk factors/risk indicators for Periodontitis in the population

iv) To use multivariate linear regression modelling to determine the relative contribution of risk factor/indicators to levels of periodontitis determined by a focus on tooth loss & mean probing pocket depths of ≥4mm

Chapter 6: Methodology

The study received local ethical approval through the Joint Management office Research (QMUL/Barts Health) (EDGE ID: 128856)

6.1) Study Type

Retrospective cross-sectional analysis

6.2) Study Subjects

All new patients referred and clinically assessed on periodontal Consultation clinics at the RLDH between March 2018 to February 2020 were consecutively assessed for inclusion in the study according to the criteria below. The first

150 patients meeting the inclusion criteria were analysed in this study. 7 records were not available for assessment and therefore were also excluded from the study.

6.3) Inclusion criteria

566 patients were consecutively assessed for inclusion and the first 150 patients meeting all inclusion criteria were included in the study

- ≥16 years
- Accepted for periodontal care by postgraduate students at the RLDH
- Received at least a full 6-point pocket periodontal examination and radiographic assessment of all teeth (OPG or full mouth peri-apicals)
- Cases - Periodontitis defined according to the 2017 EFP Classification

6.4) Exclusion criteria

- Not meeting inclusion criteria

6.5) Clinical Data

1. A data set was extracted from electronic and paper clinical records between March 2018 and February 2020

2. Clinical examination data were extracted from clinical paper records from cases examined and treated by periodontology postgraduate clinicians all of whom received standardised clinical examination training as part of their postgraduate training programme. Treating clinicians were uncalibrated for clinical measurements

3. All probing measurements (PPD and gingival recession) were recorded using a standard graduated Williams probe at six sites per tooth. Only fully erupted teeth were analysed. CAL was not accurate and therefore not used

4. The presence or absence of dental plaque was recorded at each of six sites per fully erupted tooth by using a plaque disclosing tablet. The proportion of sites with plaque was determined and expressed as a percentage of the total number of tooth surfaces examined

6.6) Risk factors/indicators measured (Independent predictor variables)

The following risk factors were assessed: gender, age, ethnicity, stress levels, smoking status, Type 1 or Type 2 diabetes mellitus, regular or irregular attender, family history of periodontal disease (self-declared), plaque levels & recorded data of whether patients used interdental brushes or not at baseline.

6.6.1) Ages – 16-19, 20-29, 30-39, 40-49, 50-59, 60-69, 70-79 years

6.6.2) Ethnicity - Asian Bangladeshi, Pakistani, Indian, Asian other, Black African, Black Caribbean, any other Black background, White – English, Welsh, Scottish, NI or British White, any other White and any other Ethnic group.

This is also broken down in the **results** section into 2 further combined ethnic groupings. 1 which is: White, Black, SEA and other. The other group is: SEA (Asian Bangladeshi, Pakistani and Indian) and others.

6.6.3) Stress Levels – 0-5, 6-10 (0 lowest, 10 highest)

6.6.4) Smoking status – Current smoker, ex-smoker, never smoker

6.6.5) Oral hygiene - Interdental brush use

6.6.6) Plaque levels (%) – 0-9, 10-19, 20-29, 30-39, 40-49, 50-59, 60-69, 70-79, 80-89, 90-100

Compliant: plaque score <20%,

Non-complaint: (poor) 20-49%, very poor (>50%)

6.7) Radiographic data: Bone level Assessment – Peri-apical vs Panoramic Radiographs vs CBCT

Long cone peri-apicals (PA) are more accurate than panoramic radiography (OPG) but both techniques tended to underestimate early disease and over-estimate advanced disease (Pepelassi and Diamanti-Kipioti, 1997). The underestimations of bone loss can reach up to 30% in OPGs and 20% in PAs respectively (Akesson, Håkansson and Rohlin, 1992). There is a consensus that peri-apicals are the gold standard and more suitable as a radiographic assessment protocol for patients with Periodontitis (Jenkins *et al.*, 2005; Persson *et al.*, 2003; White *et al.*, 2001). However, it can be argued that the osseous defect location and dimensions can dictate the extent of concurrence between OPG and PAs (Pepelassi, Tsiklakis and Diamanti-Kipioti, 2000).

Cone Beam CT is another method aimed at assessing hard tissues, however, a relatively recent systematic review concluded that there is deficiency in scientific evidence to validate its use for radiographic assessment that is

limited to periodontal non-surgical or surgical treatment provisions (Nikolic-Jakoba, Spin-Neto and Wenzel, 2016).

At the RLDH, full mouth digital radiographs are typically taken at initial examination either as an OPG or full mouth peri-apicals to assess alveolar bone levels around teeth.

In this study the radiographs were used to contribute to the determination of the levels of presenting periodontitis in terms of staging and grading.

6.8 Periodontal Probing

The probe should be 'walked around' all surfaces following the root contour in line with the long axis of the tooth with light forces of approximately 20-25g/blanching of a finger-nail pressure. The tip of the probe comes to a stop at the most coronal connective tissue fibres (Listgarten, Mao and Robinson, 1976), the pocket depth is less likely to vary in healthy tissues, however with greater inflammation there is likely to be a greater probing depth. There are many influential probing errors (1st generation periodontal probe, which is what has been used and made reference to) that can occur such as the thickness of the probe, the pressure applied and angulation of the probe, plus the degree of tissue inflammation (Armitage, 1996). It is more important to have a standardised area to be probed as well as the direction than the force used. (van der Velden, 1979).

In cases of severe periodontitis there can be up to 2mm of clinical probing measurement error, including averages of 1.5mm of inter-examiner error when

measuring the same site (Hassell, Germann and Saxer, 1973). Therefore, in this retrospective study using the clinical periodontal data of uncalibrated postgraduate students would be a limitation when it comes to the accuracy of interpreting the results.

6.9: Primary Outcomes (Dependant Variables)

6.9.1: Probing pocket depth:

Recession has been poorly recorded across the data extracted, therefore the use of PPD plus recession as an approximation of calculating CAL is not feasible in this research and its limitation will be further discussed in the discussion part of the thesis.

PPD measures the depth of the periodontal pocket from the gingival margin to the base of the residual pocket. Despite in the EFP 17' Periodontal classification the PPD (Tonetti, Greenwell and Kornman, 2018) comes as one of the local clinical parameters under the complexity part of the staging section we can only use the EFP classification crudely, not by definition as the CAL parameter is missing.

PPD has been used in a few studies in the past as an indicator for the current presenting levels of periodontal inflammation and more importantly as a threshold for Periodontitis (Savage *et al.*, 2009) with a minimum threshold of ≥3mm (Borrell and Papapanou, 2005; Craig *et al.*, 2001) to a maximum of ≥6mm (Anagnou-Vareltzides *et al.*, 1996; Machtei *et al.*, 1992); calculated as

either a mean of sites, the worst site or up to 4 sites. In this study the threshold for Periodontitis was used as a mean probing pocket depth of ≥4mm.

6.9.2: Tooth loss:

There are studies that have used tooth loss as a primary outcome; taking into consideration associative risk factors/indicators for the severity of periodontal disease (Periodontitis) and the contributing tooth loss risk due to periodontal reasonings, (Matuliene *et al.*, 2008; König *et al.*, 2002; Moore *et al.*, 2001; McGuire and Nunn, 1999; Holm, 1994; Kocher *et al.*, 2000b; Kocher *et al.*, 2000a). In this study tooth loss was used as a primary dependant variable outcome to identify which known risk factors are associated with higher tooth loss for those patients seen at the RLDH, particularly those of South-East Asian ethnic background.

6.10. Data Analysis

1. Descriptive and inferential statistics were used to describe periodontitis population.
    a. Chi-Squared (χ^2) test was used to compare whether the SEA ethnic groups demonstrate higher frequency of the risk factors/indicators for Periodontitis compared with all the other Ethnic groups combined
    b. Odds ratios with 95% confidence intervals were calculated for each risk factor /indicator analysed comparing SEA to other Ethnic groups combined

c. Odds ratios with 95% confidence intervals were calculated for mean PPD≥4 & ≥6mm and single worst site ≥8 &10mm comparing SEA to other Ethnic groups combined
d. Analysing risk factors and risk indicators as a function of tooth loss

2. Multiple Logistic regression (multivariable) analysis to model the relationship between the statistically significant risk factors/indicators and:
   I. Tooth loss
   II. Mean PPD ≥4mm

6.11) Sample size

"Based on Delgado-Angulo *et al*. (2016), in the East London adult population the mean difference in the number of teeth with PPD ≥ 4mm between South-Asian (Pakistani, Indian, Bangladeshi) individuals and "Other" ethnicities is 6.68 teeth, and the standard deviation is 12.48. Therefore, we would require 110 participants (55 South-Asian and 55 Other) to detect a true difference between the groups with a power of 80% and a level of significance of 5% (two sided)." This number was increased to 150 to ensure a minimum of South-Asian subjects were included in the study. However, the other main dependant variable in this study is tooth loss and this has not been powered which will be discussed as part of the limitations of the research study.

6.12) Procedure Employed

1) The patients that were consecutively accepted for Periodontal treatment at the RLDH from March 2018 - February 2020, had their baseline 6PPC data recorded on an excel file with the following data extracted for all 150 patients.

i. Teeth present
ii. PPD
iii. CAL
iv. BoP (0 or 1)
v. Furcation involvement degree (FI)
vi. Tooth mobility degree
vii. Plaque (0 or 1)

2) Three surveys were completed from the patients' clinical notes, please see the Appendix for each one respectively:

a) Survey 1 - Medical and Social history
b) Survey 2 - Dental/Periodontal History and related factors
c) Survey 3 – Periodontal Diagnosis

Chapter 7. Results

The results containing the statistical analysis have been spilt into 4 parts. The **first** focuses on the descriptive raw data collected in the study including the risk factors and risk indicators.

The **second** part which is in **chapter 8** focuses on the statistical analysis (bivariate analysis) of demographic and clinical variables.

The **third** part which is in **chapter 9** focuses on the relative influence of risk factors and risk indicators on tooth loss at baseline (defined as <28 teeth for this analysis).

The **fourth part** which is in **chapter 10** looks at the multiple regression analysis of the statistically significant risk factors and risk indicators in association with the dependant variable outcomes (tooth loss and mean PPD ≥4mm).

The main raw data looked at in the **first part** were the following:

1) **Periodontitis disease severity distribution** in the cohort according to the EFP 2017' classification which illustrated a greater percentage of the cohort had Generalised Periodontitis Stage 3 Grade C, (77%, 76% and 71.3% respectively).

2) The **age distribution** of the population expanding from 16 years of age up to 79 years. With higher percentages in the 40-49 and 50-59 age groups, (25.5% and 36% respectively).

3) The **number of erupted teeth**, illustrating the number of missing teeth grouped into no tooth loss, less than 5 missing teeth and more than 5 missing teeth. 1, 2 or 3 missing teeth contributed to the most percentages of missing teeth when excluding wisdom teeth, (7.3%, 12% and 7.3% respectively).

4) The **bleeding on probing** percentage of sites in the mouth, were the greatest percentage was in the 10-19% an 20-29% brackets, contributing to 27% in each of those in the cohort studied.

5) The **BoP (<20%) comparing SEA to all other Ethnic groups combined** were a similar percentage of the SEA and the other Ethnic groups combined had BoP sites greater than 20%, (77% and 73% respectively).

6) The **mean periodontal pocket depth** in the cohort were the most common mean PPDs were 43% of the cohort that had a mean of 2-3mm and 61% that had a mean PPD of 3-4mm.

7) The **distribution of all the ethnicities** identified in the study population and then ethnicities combined into 2 further categories.

8) The distribution of **risk factors and risk indicators** (plaque smoking, diabetes mellitus, stress, and attendance) of the study cohort.

Descriptive Data:

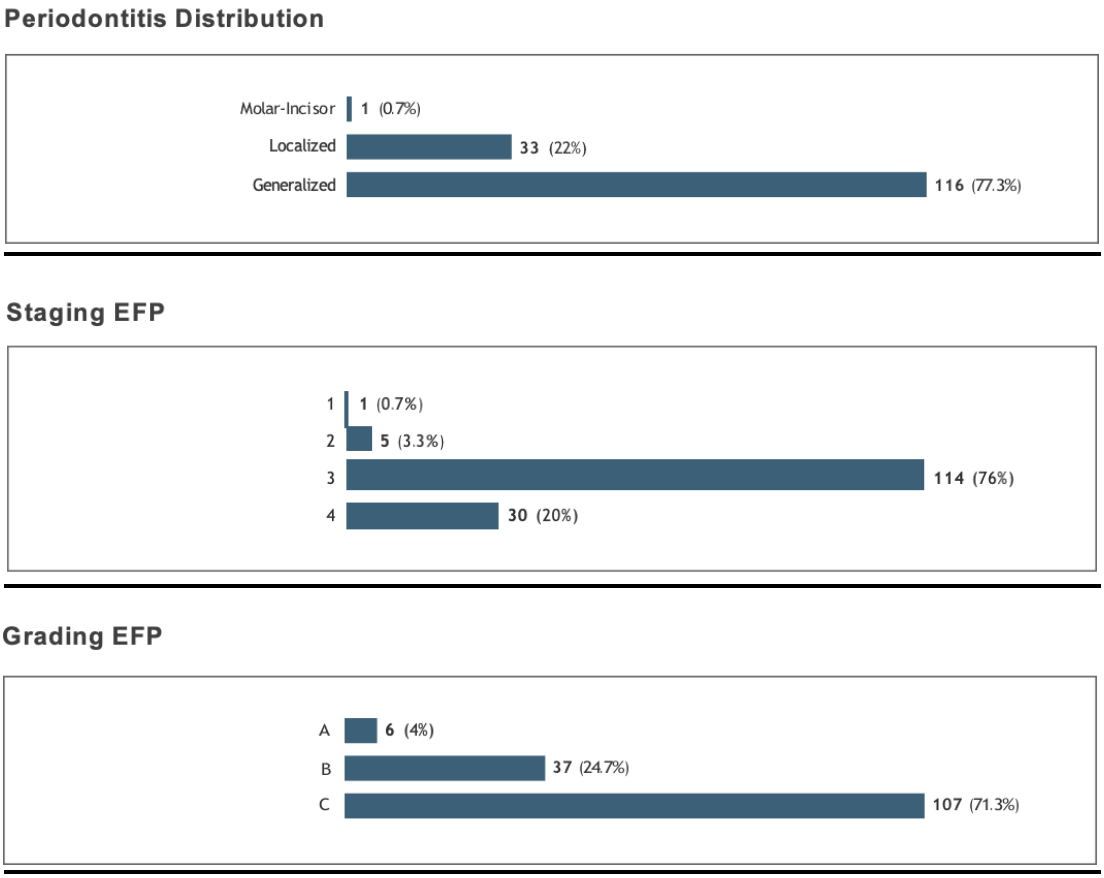

Figure 1: Distribution of Periodontitis in this study population including the staging and grading according to the EFP 2017' Classification. 77.3% of subjects had generalised periodontitis with 76% of the population with stage 3 and 71.3% with Grade C (rapid rate of disease progression) diagnosis. 20% of subjects were in the stage 4 of disease severity category. 24.7% of the population had Grade B for moderate rate of progression of the periodontal disease. 1 subject had Molar-incisor pattern periodontitis with 22% of subjects with localized periodontitis. 1 subject had Stage 1 (mild) and 5 stage 2 (moderate) Periodontitis disease respectively. 4% had grade A (slow rate of disease progression)

Table 1: Age distribution of the population

The table demonstrates the percentages of subjects in each age category from 16-19 up to 70-79. 25% of subjects were in the 40-49 age group and 36% of the total population was in the 50-59 age bracket. The 30-39 & 60-69 shared in the 12% of subjects. The 70-79 group was at 10.7% with the 16-19 & 20-29 age groups with lower percentages of the total population, 4% & 7.3% respectively. n=150, total number of subjects (58 Male, 92 Female)

Mean Age: 47.3 years +/- 12.9 (SD)

Age (Years)	n (%)
16-19	6 (4.0%)
20-29	11 (7.3%)
30-39	19 (12.7%)
40-49	38 (25.3%)
50-59	54 (36.0%)
60-69	18 (12.0%)
70-79	4 (10.7%)

Table 2: Number of erupted teeth

The table has 3 categories of missing teeth. The first no periodontal tooth loss, the second ≤5 missing teeth and the third >5 missing teeth. 20.7% of the total population 28 teeth. 52% of the total population had 28 teeth or more. 36% of the total population have lost <5 teeth and 12% have lost >5 teeth respectively, excluding third molars.

	Number of erupted teeth	n (%)
No periodontal tooth loss (n=78, 52.0%)	32	11 (7.3%)
	31	6 (4.0%)
	30	15 (10%)
	29	15 (10%)
	28	31 (20.7%)
<5 missing teeth (n=54, 36.0%)	27	11 (7.3%)
	26	18 (12%)
	25	11 (7.3%)
	24	8 (5.3%)
	23	6 (4.0%)
>5 missing teeth (n=18, 12.0%)	22	5 (3.3%)
	21	3 (2.0%)
	20	5 (3.3%)
	19	1 (0.7%)
	18	1 (0.7%)
	17	1 (0.7%)
	16	0 (0.0%)
	15	1 (0.7%)
	14	1 (0.7%)
		150 (100%)

Table 3: Bleeding on Probing

The table displays the percentage of sites with BoP from 0 up to 100%. 29 % of the total population had ≥50% of sites with BoP. Whereas 71% of the total number of subjects (n=150) had sites with BoP from 0-49%. 8% of the population had <10% of sites with BoP.

Mean BoP (%) = 37.1% +/- 22.4(SD)

% sites BoP	n
0-9	12
10-19	27
20-29	27
30-39	23
40-49	17
50-59	20
60-69	9
70-79	8
80-89	5
90-100	2

Table 4: BoP (<20%) comparing SEA to all other Ethnic groups combined

The table demonstrates that 10 SEA (Bangladeshi, Indian & Pakistani) and 29 of all the other ethnic groups combined demonstrated sites with BoP less than 20%. Whereas 74% (111/150) of the population had >20% sites with BoP.

		BoP (<20%)		
		Yes	No	Total
Ethnic group	South-East Asian	10	33	43
	All Others	29	78	107
		39	111	150

Table 5: Mean Periodontal pocket depth

The table illustrates the mean PPD ranging from 0 to 8mm. 41% of the total population had a mean PPD of 3-4mm. 71% of the total population had mean PPD of up to 4mm, while the remaining 29% had a mean PPD over >4mm.

Mean PPD (mm) = 3.65+/-1.05

Mean PPD (mm)	n
0-1	0
1-2	2
2-3	43
3-4	61
4-5	29
5-6	11
6-7	1
7-8	3

Table 6: Distribution of Ethnic groups 1

The table shows the raw distribution of all the Ethnic groups categorised into 4 main categories and subcategories for each one. In the White category, the White British/Irish subcategory contributed to 38.7% of the total population.

The Asian ethnic group make up 34.7% of the total population, 20% for the Black background based ethnic groups and 0.7% for other ethnic groups.

Ethnic Group		n (%)
White (n=67, 44.7%)	White British/Irish	58 (38.7%)
	White Other	9 (6.0%)
Asian or Asian British (n=52, 34.7%)	Indian	14 (9.3%)
	Pakistani	9 (6.0%)
	Bangladeshi	20 (13.3%)
	Chinese	3 (2.0%)
	Any other Asian background	6 (4.0%)
Black, Black British, Caribbean, African (n=30, 20%)	Black African	20 (13.3%)
	Black Caribbean	7 (4.7%)
	Any other Black, Black British or Caribbean background	2 (1.3%)
	Mixed	1 (0.7%)
Other ethnic group (n=1, 0.7%)	Arab	0 (0.0%)
	Any other ethnic group	1 (0.7%)
Total		150 (100%)

Table 7: Combined Ethnic groups 2

The table demonstrates 4 categories of Ethnic groups. Those of White ethnic origin contributed to 44.7% of the total population. South-East Asians 28.7% of the subjects in this study. Black and other ethnic origins displayed lower

percentages, 19.3% & 7.3% of patients assessed and then taken on for further periodontal treatment at the RLDH.

Combined Ethnic groups	n
White	67 (44.7%)
Black	29 (19.3%)
South-East Asian	43 (28.7%)
Other	11 (7.3%)
Total	150 (100%)

Table 8: Combined Ethnic groups 3

The combined Ethnic grouping in this study shows that 28.7% make up the South-East Asian ethnic group in comparison to the other ethnic groups combined making up the remaining 71.3%.

Combined Ethnic groups	n
South-East Asian	43 (28.7%)
Other	107 (71.3%)
Total	150 (100%)

7.1: Risk Factors

7.1.1) Plaque control: Mean 57.1% +/- 18.55 (SD)

At baseline only **4/150** (2.7%) patients were considered compliant (i.e. <20% plaque score). **146/150** (97.3%) patients were considered non-compliant (i.e. >20% plaque scores)

Table 9: Percentage of plaque scores categorised into Compliant and Non-complaint subjects:

The table illustrates full mouth plaque score levels of the subjects assessed. 70% of the population had plaque scores ranging from 50-100%. 27% of the total population had plaque scores between 20-49%. Only 3% had plaque scores <20%.

	% Plaque score	n
Compliant (Plaque score <20%) N=4	0-9	2 (1.3%)
	10-19	2 (1.3%)
Non-compliant (Poor) – 20-49% n=41	20-29	6 (4.0%)
	30-39	14 (9.3%)
	40-49	21 (14.0%)
Non-compliant Very poor (>50%) N=105	50-59	42 (28.0%)
	60-69	32 (21.3%)
	70-79	10 (6.7%)
	80-89	12 (8.0%)
	90-100	9 (6.0%)
	Total	150 (100%)

7.1.2) Smoking

Table 10: Smoking status and distribution

The tables show the smoking status and overall smoking exposure of the population in this cross-sectional study. Never smokers contributed to 57.3% of the total population compared to 42.7% of those with smoking exposure. 12% of the current population still smoke with 30.7% of the subjects in this study being ex-smokers.

	n
Current smoker	18 (12.0%)
Ex-smoker	46 (30.7%)
Never smoker	86 (57.3%)
Total	150 (100%)

	n
Smoking exposure	64 (42.7%)
Never smoker	86 (57.3%)
Total	150 (100%)

7.1.3) Diabetes Mellitus:

Table 11: Diabetes Mellitus presence or absence (self-reported)

The table illustrates the percentage of subjects with diabetes mellitus and those without. 14.7% of the population have DM. Of those subjects with DM, 13.3% out of the 14.7% have DM type II, with 1.3% out 14.7% with DM type 1. 85.3% of the total population self-reported as not have DM.

	n (%)	Diabetes type	n (%)
Diabetes Mellitus	22 (14.7%)	Type 1	2 (1.3%)
		Type 2	20 (13.3%)
No Diabetes Mellitus			128 (85.3%)
Total			150 (100%)

7.1.4) Stress:

Table 12: Stress (self-declared stress levels 0-10)

81.3% of subjects self-reported to have stress levels ≤5, compared to 18.7% with ≥6 out of 10. (10 being the greatest stress level).

	n (%)
Low stress (0-5)	122 (81.3%)
High Stress (6-10)	28 (18.7%)
Total	150 (100%)

7.1.5) Attendance:

Table 13: Irregular dental attendance (Self-declared)

78% of the total population in this cross-sectional study self-reported as being regular attenders compared with the 22% that attend irregularly to their general dental practitioners.

	n (%)
Irregular attender	33(22.0%)
Regular attender	117(78.0%)
Total	100 (100%)

Chapter 8. Statistical Analysis I (Bivariate analysis) of Demographic and Clinical Variables:

Risk Factors and Risk indicators: Are certain established periodontitis risk factors and some risk indicators more common in the South-East Asian ethnic group compared to all other groups combined?

A one-tailed test is used throughout for all chi-squared tests performed as we are testing in the alternative hypothesis whether SE Asian ethnic groups have higher proportions compared to all other ethnic groups (presumption is that SE Asian population are more likely to have higher levels of the risk factor in question. A chi-squared test is used to analyse each 2x2 table as sample size is high (n=150).

8.1: Diabetes

Diabetes in this analysis is a diagnosis of either Type 1 or Type 2 diabetes (No reference is made to amount of control).

Research Question: Is diabetes prevalence higher in SEA ethnic groups compared to other ethnic groups?

		Diabetes		
		Yes	No	Total
Ethnic group	South-East Asian	10	33	43
	All Others	12	95	107
		22	128	150

Chi-squared = 3.553, z= 1.885, p=0.0297* (1-tailed test).

Effect size and 95% CIs shown: Relative Risk (RR) = 2.074 (0.9722-4.319), Odds Ratio (OR) = 2.399 (0.9621-5.923)

Figure 2: **Diabetes in the SEA population compared to all other Ethnic groups**. 10 out of the 22 subjects having diabetes is from the SEA group with the remaining 12 out of the 22 to all the ethnic groups combined. The mean relative risk and mean odds ratio for SEA subjects having diabetes in this study is 2.074 & 2.399 respectively.

Conclusion: Diabetes occurs at **significantly** higher levels in the SE Asian ethnic group compared to all other groups combined (p=0.0297).

8.2: Age <40 years in cohort

Research Question: Within the periodontitis cohort referred for Specialist assessment, Is the proportion of patients <40 years greater in the SE Asian ethnic group i.e. Do a higher proportion of the SE Asian ethnic groups get referred at a younger age?

		Age <40 years		
		Yes	No	Total
Ethnic group	South-East Asian	15	28	43
	All Others	21	86	107
		115	36	150

Chi-squared = 4.038, z= 2.009, p=0.0222* (1-tailed test).

Effect size and 95% CIs shown:

Relative Risk=1.794 (1.015-3.086), Odds Ratio=2.219 (1.042-4.953)

Figure 3: Age of subjects in the study <40 years old - comparing SEA group to other Ethnic groups. 15 out of 115 (SEA group) were <40years old compared to 21 out of the 115 of the other ethnic groups combined. The mean RR & mean OR for SEA subjects being referred earlier for periodontitis disease were 1.794 & 2.219 respectively.

A **significant** larger proportion of the SE Asian ethnic group get referred earlier compared to other ethnic groups (p=0.0222).

8.3 Smoking exposure (Ex or current smoker)

Research question: Is smoking exposure higher in the SE Asian ethnic group compared to all other groups combined?

		Smoking exposure		
		Yes	No	Total
Ethnic group	South-East Asian	17	26	43
	All Others	47	60	107
		64	86	150

Chi-squared = 0.2417, z= 0.4916, p=0.3115 (ns) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 0.9000 (0.5730-1.341), Odds Ratio = 0.8347 (0.4098-1.727)

Figure 4: Smoking exposure comparison of SEA group to other ethnic groups combined. 17 out of the 64 (SEA group) had smoking exposure compared to the remaining 47 out of 64 of the other Ethnic groups combined. The mean RR & mean OR for SEA subjects being smokers compared to the other ethnic groups combined was 0.900 and 0.834 respectively.

There is **no significant** difference in the proportion of smokers in the SE Asian population compared to all other ethnic groups combined.

8.4: Gender

Research question: Are there higher proportions of a particular gender in SE Asian ethnic groups compared to all other ethnic groups combined?

		Gender		
		M	F	Total
Ethnic group	South-East Asian	18	25	43
	All Others	40	67	107
		58	92	150

Chi-squared = 0.009842, z= 0.09921, p=0.9210 (2-tailed test – used as no presumed direction of effect)

Effect size and 95% CIs shown:

Relative Risk = 0.9765 (0.6341-1.436), Odds Ratio = 0.9648 (0.4891-1.957)

Figure 5: The gender categories of the SEA population compared to all other Ethnic groups. SEA group: 18 out of 58 Males and 25 out of 92 females All other ethnic groups: 40 out of 58 Males and 67 out of 92 females. The mean RR & mean OR for SEA subject proportion of Male/Female was 0.976 & 0.964 respectively.

There is **no significant** difference in proportion of presenting Male/females in the SE Asian ethnic group compared to all other groups combined (p=0.9210)

8.5: Plaque (Poor plaque control >50%)

Research question: Are there higher proportions of patients with poorer plaque control in SE Asian ethnic groups compared to all other ethnic groups combined?

		Poor plaque control (>50%)		
		Y	N	Total
Ethnic group	South-East Asian	31	12	43
	All Others	74	33	107
		105	45	150

Chi-squared = 0.1257, z= 0.3546, p=0.3614 (ns) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 1.042 (0.8079-1.284), Odds Ratio = 1.152 (0.5447-2.577)

Figure 6: The number of subjects with poor plaque control >50%. 29.5% (SEA group) had poor plaque control compared with the 70.5% of the other Ethnic groups combined. The mean RR & mean OR for SEA patients to have poorer plaque control (>50%) compared with all other ethnic groups combined is 1.042 and 1.152 respectively.

There is **no significant** difference in the proportion of patients with baseline poor plaque control (>50% score) in the SE Asian ethnic group compared to all other groups combined (p=0.3614).

8.6: Family History of periodontal disease (self-declared)

Research question: Are there higher proportions of patients with a FH of periodontitis in SE Asian ethnic groups compared to all other ethnic groups combined?

		FH of periodontal disease		
		Y	N	Total
Ethnic group	South-East Asian	9	34	43
	All Others	18	89	107
		27	123	150

Chi-squared = 0.3507, z= 0.5922, p=0.2769 (ns) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 1.244 (0.6038-2.465), Odds Ratio = 1.309 (0.5535-3.144)

Figure 7: The number of patients with self-declared family history of Periodontal disease. SEA group: 9 out of 27 did have FH of periodontal disease compared with the 18 out of 27 for all the other Ethnic groups combined. The mean RR & mean OR for SEA patients to have a positive family history of periodontal disease are 1.244 & 1.309 respectively.

There is **no significant** difference in the proportion of patients with a known family history of periodontal disease in the SE Asian ethnic group compared to all other groups combined (p=0.2769).

8.7: Interdental brush use

Research question: Are there higher proportions of patients using interdental brushes in SE Asian ethnic groups compared to all other ethnic groups combined?

		Interdental brush use		
		Y	N	Total
Ethnic group	South-East Asian	15	28	43
	All Others	47	60	107
		62	88	150

Chi-squared = 1.034, z= 1.017, p=0.1546 (ns) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 0.7942 (0.4888-1.219), Odds Ratio = 0.6839 (0.3195-1.434)

Figure 8: The number of subjects which use interdental brush identified at baseline assessment. SEA group: 15 out of 43 said they use interdental brushes compared with 47 out of 107 for all other ethnic groups combined. The mean RR & mean OR for SEA groups using interdental brushes compared with all other ethnic groups is 0.794 & 0.683 respectively.

There is **no significant** difference in the proportion of patients that use interdental brushes in the SE Asian ethnic group compared to all other groups combined (p=0.1546).

8.8: Irregular dental attender (Self-declared)

Research question: Are there higher proportions of irregular dental attenders in the SE Asian ethnic groups compared to all other ethnic groups combined?

		Irregular attender		
		Y	N	Total
Ethnic group	South-East Asian	17	26	43
	All Others	16	91	107
		33	117	150

Chi-squared = 10.80, z= 3.286, p=0.0005 (ns) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 2.644 (1.472-4.685), Odds Ratio = 3.719 (1.696-8.193)

Figure 9: The number of subjects that self-declare irregular dental attendance to their GDP. 17 out of the 33 irregular attenders are of the SEA group compared with 16 out of 33 of all the other Ethnic groups combined. The mean RR & mean OR for a subject from a SEA group to be an irregular attender to their GDP were 2.644 & 3.719 respectively.

There is a **significantly** higher levels of irregular dental attendance in the SE Asian ethnic group compared to all other groups combined (p=0.0005).

8.9: Stress (Self-reported on scale 0-10)

Research question: Are there higher amounts of self-reported stress in the SE Asian ethnic groups compared to all other ethnic groups combined at baseline?

		Stress Levels		
		High (6-10)	Low (0-5)	Total
Ethnic group	SE- Asian	3	40	43
	All Others	25	82	107
		28	122	150

Chi-squared = 5.426, z= 2.329, p=0.0099** (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 0.2986 (0.09837-0.8477), Odds Ratio = 0.2460 (0.07504-0.7913)

Figure 10: Self-reported stress levels in the study population. 3 out of 28 (SEA subjects) compared with 25 out of 28 of all other Ethnic groups combined reported stress levels ≥6 (scale of 0-10). 33% of SEA group compared with 77% of all other ethnic groups had stress levels between 0-5. The mean RR & mean OR of a SEA subject to have a higher amount of self-reported stress was 0.298 & 0.246 respectively.

There are **significantly** lower levels of self-reported stress in the SE Asian ethnic group compared to all other groups combined (p=0.0099**).

8.10: Tooth Loss (<28 teeth)

Research question: Are there higher proportions of tooth loss in the SE Asian group compared to all other Ethnic groups combined at baseline?

		Tooth Loss (<28 teeth)		
		Yes	No	Total
Ethnic group	South-East Asian	18	25	43
	All Others	54	53	107
		72	78	150

Chi-squared = 0.9103, z= 0.9541, p=0.1700 (ns) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 0.8295 (0.5418-1.200), Odds Ratio = 0.7067 (0.3537-1.452)

Figure 11: The number of subjects with <28 teeth at baseline assessment. 25% (18/72) SEA group compared with 75% of all the other ethnic groups combined had <28 teeth at baseline. The mean RR & mean OR of SEA subject to have more tooth loss (<28 teeth) at baseline was 0.829 & 0.706 respectively.

Tooth loss is **not significantly** increased in the SE Asian population compared to other groups

8.11: Tooth Mobility

Research question: Is there a greater number of tooth mobility in the SE Asian group compared with all the other Ethnic groups combined?

		Tooth Mobility		
		Yes	No	Total
Ethnic group	South-East Asian	31	12	43
	All Others	87	20	107
		118	32	150

Chi-squared = 1.552, z= 1.246, p=0.1064 (ns) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 0.8867 (0.6956-1.064), Odds Ratio = 0.5939 (0.2711-1.425)

Figure 12: The number of subjects presenting with tooth mobility at baseline assessment. 26% (31/118) which is the SEA group had tooth

mobility compared to the remaining 74% of all the other ethnic groups combined. The mean RR and mean OR of a SEA having higher numbers of tooth mobility was 0.886 and 0.593 respectively.

Tooth mobility is **not significantly** increased in the SE Asian population compared to other groups

8.12) Furcation Involvement

(NB: 2 patients had lost all molars and were excluded – n=148)

Research question: Are there greater numbers of furcation involvement in the SE Asian ethnic groups compared to all other ethnic groups combined?

		Furcation involvement		
		Yes	No	Total
Ethnic group	South-East Asian	42	1	43
	All Others	99	6	105
		141	7	148

Chi-squared = 0.7774, z= 0.8817, p=0.1890 (ns) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 1.036 (0.9293-1.114), Odds Ratio = 2.545 (0.3919-29.86)

Figure 13: The number of subjects having teeth with molar furcation involvement at baseline. SE-Asian group: 30% have furcation involvement

compared with 70% of all other ethnic groups combined. The mean RR & mean OR for SEA patient to have increased molar furcation involvement is 1.035 & 2.545 respectively.

Molar furcation involvement is **not significantly** increased in the SE Asian population compared to other groups

8.13.1) Mean PPD ≥4mm

		Mean PPD ≥4mm		
		Yes	No	Total
Ethnic group	South-East Asian	15	28	43
	All Others	29	78	107
		44	106	150

Chi-squared = 0.8959, z= 0.9465, p=0.1719 (ns) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 1.287 (0.7581-2.102), Odds Ratio = 1.441 (0.6802-2.985)

Figure 14: The number of patients with mean PPD≥4mm. 15 out of the total 43 SEA group, and for the other ethnic groups combined they showed 29 out of the 107 subjects having a mean PPD ≥4mm. The mean RR & mean OR for a SEA subject to have a mean PPD ≥4mm was 1.287 & 1.441 respectively.

Mean PPD≥4mm is **not significantly** increased in the SE Asian population compared to other groups

8.13.2) Mean PPD ≥6mm

		Mean PPD≥6mm		
		Yes	No	Total
Ethnic group	South-East Asian	2	41	43
	All Others	2	105	107
		4	146	150

Chi-squared = 0.9146, z= 0.9564, p=0.1694 (ns) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 2.488 (0.4464-13.69), Odds Ratio = 2.561 (0.3884-16.65)

Figure 15: The number of patients with mean PPD≥6mm. 2 SEA and 2 from all the other ethnic groups combined patients demonstrated mean PPD ≥6mm. The remaining 41/43 and 105/107 patients of both groups respectively did not have a mean PPD ≥6mm. The mean RR & mean OR for a SEA subject to have a mean PPD ≥6mm was 2.488 & 2.561

6mm sites and above are **not significantly** increased in the SE Asian population compared to other groups

8.14) Single worst site ≥8mm

		Single worst site ≥8mm		
		Yes	No	Total
Ethnic group	South-East Asian	29	14	43
	All Others	79	28	105
		108	32	148

Chi-squared = 0.6212, z= 0.7882, p=0.2153 (ns) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 0.9135 (0.6978-1.130), Odds Ratio = 0.7342 (0.3484-1.542)

Figure 16: The number of subjects with single worst site ≥8mm. 27% of subjects with single worst site ≥8mm belonged to the SEA group compared to 73% of all the other ethnic groups combined. The mean RR & mean OR of an SEA subject having ≥8mm sites in this study was 0.913 & 0.734 respectively.

8mm sites and above are **not significantly** increased in the SE Asian population compared to other groups

8.15) Single worst site ≥10mm

		Single worst site ≥10mm		
		Yes	No	Total
Ethnic group	South-East Asian	15	28	43
	All Others	44	63	107
		59	91	150

Chi-squared = 0.5002, z= 0.7072, p=0.2397 (ns) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 0.8483 (0.5195-1.312), Odds Ratio = 0.7670 (0.3573-1.616)

Figure 17: The number of subjects with single worst site ≥10mm. SEA group: 15 out 43 and all other Ethnic group had 44 out of 107 subjects that had single worst site ≥10mm. The mean RR & mean OR of an SEA subject having ≥10mm sites in this study was 0.848 & 0.767 respectively.

10mm sites and above are **not significantly** increased in the SE Asian population compared to other groups

Chapter 9) Statistical Analysis II:

What is the relative influence of Risk Factors and risk indicators on **tooth loss at baseline?** (**Defined as <28 teeth for this analysis)**

9.1) Diabetes

		Tooth Loss		
		Yes	No	Total
Diabetes	Yes	9	13	22
	No	63	65	128
		72	78	150

Chi-squared = 0.5194, z= 0.7207, p=0.2356 (ns) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 0.8312 (0.4616-1.307), Odds Ratio = 0.7143 (0.3018-1.687)

Figure 18: The distribution of patients with or without DM presenting with or without tooth loss at baseline. The table shows 9 patients with DM & tooth loss compared with, 63 subjects with no DM at baseline but presenting with tooth loss. 13 patients with DM but no tooth loss and 65 subjects with no DM and tooth loss at baseline assessment. The mean RR & mean OR for patients for DM on tooth loss in this study is 0.831 & 0.714 respectively.

Patients with diabetes at baseline **DO NOT** have significantly greater amounts of tooth loss compared to those without.

9.2) Smoking (Ex or Current)

		Tooth Loss		
		Yes	No	Total
Smoking Exposure	Yes	44	20	64
	No	28	58	86
		72	78	150

Chi-squared = 19.26, z= 4.388, p=<0.0001**** (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 2.112 (1.507-3.013), Odds Ratio = 4.557 (2.2227-8.826)

Figure 19: The distribution of patients with or without smoking exposure presenting with or without tooth loss at baseline. 44 subjects presented with smoking exposure and tooth loss compared with 28 with no smoking exposure but tooth loss at baseline. 20 subjects with smoking exposure but no tooth loss and 58 subjects with no smoking exposure and no tooth loss at baseline assessment. The mean RR & mean OR for smoking exposure on tooth loss is 2.112 & 4.557 respectively.

Patients with a smoking history at baseline have **significantly** greater tooth loss compared to those without.

9.3) High Plaque (>50%)

		Tooth Loss		
		Yes	No	Total
High Plaque (>50%)	Yes	55	50	105
	No	17	28	45
		72	78	150

Chi-squared = 2.691, z= 1.641, p=0.0504 (ns) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 1.387 (0.9429-2.158), Odds Ratio = 1.812 (0.8925-3.653)

Figure 20: The distribution of patients with or without high plaque scores (>50%) presenting with or without tooth loss at baseline. 55 subjects presenting with high plaque scores and tooth loss compared with 17 that did not have high plaque score but tooth loss at baseline. 50 patients did not have tooth loss despite high plaque scores and 28 did not have high plaque scores or tooth loss. The mean RR & mean OR for having high plaque scores (>50%) on tooth loss was 1.387 & 1.812 respectively.

High amounts of plaque at baseline are **NOT** associated with increased tooth loss

9.4) South-East Asian Ethnicity

		Tooth Loss		
		Yes	No	Total
SE Asian ethnicity	Yes	18	25	43
	No	54	53	107
		72	78	150

Chi-squared = 0.9103, z= 0.0.9541, p=0.1700 (ns) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 0.8295 (0.5418-1.200), Odds Ratio = 0.7067 (0.3537-1.452)

Figure 21: The distribution of patients that are or are not of SEA Ethnic origin presenting with or without tooth loss at baseline. 18 patients in the SEA ethnic group presenting with loss compared with 54 that are not of SEA ethnic origin but presented with tooth loss. 25 subjects of SEA origin and no tooth loss. 53 patients not of SEA origin and no tooth loss at baseline assessment. The mean RR & mean OR of SEA ethnicity origin on tooth loss was 0.829 & 0.706 respectively.

Patients from a SE Asian ethnic group do **NOT** have significantly more tooth loss compared to other ethnic groups.

9.5) Irregular Attendance

		Tooth Loss		
		Yes	No	Total
Irregular attender	Yes	14	19	33
	No	58	59	117
		72	78	150

Chi-squared = 0.5270, z= 0.7259, p=0.2339 (ns) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 0.8558 (0.5324-1.267), Odds Ratio = 0.7465 (0.3530-1.637)

Figure 22: The distribution of patients that are or are not irregular attenders to their general dental practitioner presenting with or without tooth loss at baseline. 14 patients self-reported irregular attendance and had tooth loss at baseline compared with 58 that had tooth loss but are not irregular attenders. 19 patients had no tooth loss and were irregular attenders. 59 subjects had no tooth loss and were not irregular attenders. The mean RR & mean OR for irregular attendance on tooth loss was 0.855 and 0.746 respectively.

Patients declaring irregular attendance did **NOT** have significantly more tooth loss compared to regular attenders

9.6) Older age (>40 years)

		Tooth Loss		
		Yes	No	Total
Age >40 years	Yes	64	50	50
	No	8	28	28
		72	78	150

Chi-squared = 12.61, z= 3.551, p=0.0002*** (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 2.526 (1.431-4.878), Odds Ratio = 4.480 (1.897-10.96)

Figure 23: The distribution of patients that are or are not greater than 40 years of age presenting with or without tooth loss at baseline. 64 patients older than 40 years of age presented with tooth loss compared with 8 patients that were younger than 40 and had tooth loss. 50 subjects presented with no tooth loss that were >40 years compared with 28 patients that were younger than 40 and had tooth loss at baseline assessment. The mean RR & mean OR of Age >40years on tooth loss was 2.526 & 4.480 respectively.

Patients aged >40 years at baseline had **significantly** greater tooth loss compared to younger patients

9.7) High Stress (Self-declared 6-10/10)

		Tooth Loss		
		Yes	No	Total
High Stress	Yes	21	7	28
	No	51	71	122
		72	78	150

Chi-squared = 10.05, z= 3.171, p=0.0008*** (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 1.794 (1.282-2.380), Odds Ratio = 4.176 (1.717-10.49)

Figure 24: The distribution of patients that have or do not have high stress levels (6-10/10) presenting with or without tooth loss at baseline. 21 patients had high stress levels and tooth loss compared with 51 lower stress levels but still had tooth loss. 7 subjects had high stress levels and no tooth loss compared with 71 subjects with no high stress levels or tooth loss at baseline assessment. The mean RR & mean OR of high stress levels (6-10/10) on tooth loss at baseline was 1.794 and 4.176 respectively.

Patients declaring higher stress levels (self-rated 6-10/10) had **significantly higher** levels of tooth loss compared to those rating stress levels low (self-rated-0-5/10)

Chapter 10. Statistical Analysis III: Multiple regression models

Risk Factors and Periodontal outcome measures

10.1) Model 1: Logistic Regression

Dependent Outcome variable = Tooth Loss

Dichotomous (binary) outcome:

- No tooth loss = 28 teeth or greater
- Tooth loss = 27 teeth or fewer

7 Predictor (Independent variables) – all with dichotomous outcomes:

- Diabetes (p=0.2356) - Univariate
    - Yes
    - No
- Smoking Exposure (p<0.0001) - Univariate
    - Yes (Current or Former smokers combined)
    - Never previously smoked
- High Plaque (>50%) (p=0.0504) - Univariate
    - Yes >=50%
    - No <50%
- South-East Asian Ethnicity (p=0.170) - Univariate
    - SE Asian Ethnic group
    - All other ethnic groups combined
- Irregular attendance (Self-declared) (p=0.2339)
    - Yes

- No

- Age >40 years (p=0.0002) - Univariate
  - Yes 40 years and older
  - <40 years
- High Stress (p=0.0003) - Univariate
  - High Stress levels (6-10)
  - Low Stress levels (0-5)

Enter 7 predictor variables (Univariate p<=0.2) into the binary logistic regression model. As we have n=150 in the sample it is reasonable to have up to 7 variables (20/predictor variables)

Predictors							95% CI for EXP(B)	
Factor	B	S. E	Wald	df	Sig	Exp(B)	Lower	Upper
Age >40 years	1.769	0.517	11.691	1	<0.001	5.867	2.128	16.175
High Stress	1.562	0.577	7.330	1	0.007	4.770	1.539	9.516
Smoking exposure	1.486	0.391	14.425	1	<0.001	4.420	2.053	9.516
Irregular attendance	-0.459	0.482	0.906	1	0.341	0.632	0.246	1.626
SE Asian ethnicity	0.265	0.463	0.328	1	0.567	1.303	0.526	3.227
Diabetes	-0.482	0.482	0.906	1	0.375	0.617	0.212	1.794
High Plaque scores >50%	0.505	0.434	1.358	1	0.244	1.658	0.708	3.879
Constant	-2.619	0.626	17.478	1	<0.001	0.073		

*All variables entered into the equation

Figure 25: Model 1 Summary:

- Model chi-square = 44.073 p<0.001 – significant – model good model

- Hosmer and Lemeshow Test chi-square 4.440 p=0.815 ns – indicates goodness of fit of model i.e., a non-significant p>0.05 result here shows a good model.

- Nagelkerke R-square = 0.340 (model explains 34% of the variation in tooth loss).

- Classification Accuracy of model - Model successfully predicts 73.3% of tooth loss. The model better predicts cases without tooth loss (80.8%) compared to a lower success rate of model to predict tooth loss (65.3%)

- Age>40years, High Stress and Smoking exposure are significant predictor variables in the multiple binary logistic regression model

- B=regression weights for model – not intuitive to understand
- SE = Standard errors
- Wald = Ratio of regression weight to SE i.e., B/S. E
- Df = degrees of freedom
- Exp(B) = Odds Ratio – Change in odds for every unit change of predictor variable

- Patients presenting over age of 40 are 5.9xmore likely to have tooth loss compared to those <40 (P<0.001, Exp(B)=5.867)
- Patients self-declaring high levels of stress (i.e., who score stress as 6-10 at baseline) are 4.7x more likely to have tooth loss compared to those declaring lower levels of stress (self-rate 0-5 stress score) (P=0.007, Exp(B)=4.770)

- Ex-smokers/current smokers i.e., any patient with a previous history of smoking is 4.4x more likely to have tooth loss compared to never smokers (p<0.001, Exp(B)=4.42)

- All other factors are non-significant and have a 95% Exp(B) CI that straddles/crosses 1 i.e., we cannot be sure that the odds ratio for these factors are significantly different from 1

10.2) Model 2 – Logistic Regression

Dependent Outcome variable = Mean PPD>=4mm

Dichotomous (binary) outcome:

- Mean PPD >=4mm
- Mean PPD <=4mm

Predictor (Independent variables) – all with dichotomous outcomes:

- Diabetes (p=0.2165) – Univariate 1-tailed
  - Yes
  - No

- Smoking Exposure (p=0.2095) – Univariate 1-tailed
    - Yes (Current or Former smokers combined)
    - Never previously smoked
- High Plaque (>50%) (p=0.0005) – Univariate 1-tailed
    - Yes >=50%
    - No <50%
- South-East Asian Ethnicity (p=0.172) – Univariate 1-tailed
    - SE Asian Ethnic group
    - All other ethnic groups combined
- Irregular attendance (Self-declared) (p=0.0305) 1-tailed
    - Yes
    - No
- Age >40 years (p=0.4265) – Univariate 1-tailed – Not included in model
    - Yes 40 years and older
    - <40 years
- High Stress (p=0.3585) – Univariate 1-tailed – Not included in model
    - High Stress levels (6-10)
    - Low Stress levels (0-5)

Enter 5 predictor variables (Univariate p<=0.2) into the binary logistic regression model. 2 predictor variables removed as univariate analysis demonstrated p value >0.2

As we have n=150 in the sample it is reasonable to have up to 7 variables (20/predictor variables), 5 in this model.

Predictors							95% CI for EXP(B)	
Factor	B	S. E	Wald	df	Sig	Exp(B)	Lower	Upper
Smoking exposure	0.350	0.381	0.845	1	0.358	1.419	0.673	2.992
Irregular attendance	0.548	0.444	1.526	1	0.217	1.730	0.725	4.125
SE Asian ethnicity	0.142	0.430	0.109	1	0.741	1.153	0.497	2.676
Diabetes	0.628	0.546	1.324	1	0.250	1.873	0.643	5.458
High Plaque scores >50%	1.587	0.533	8.863	1	0.003**	4.887	1.720	13.890
Constant	-2.533	0.554	20.906	1	<0.001	0.079		

*5/7 variables entered into the equation

Figure 26: Model 2 Summary:

- Model chi-square = 15.999 p=0.007 – significant – model good model

- Hosmer and Lemeshow Test chi-square 7.950 p=0.337 ns – indicates goodness of fit of model i.e., a non-significant p>0.05 result here shows a good model

- Nagelkerke R-square = 0.144 (model only explains 14% of the variation in tooth loss) perhaps due to only one significant predictor variable

- Classification Accuracy of model - Model successfully predicts 100% of cases with PPD <4mm but fails to predict any of the cases with mean PPD>4mm the model seems to only predict those cases with PPD <4mm

- High plaque levels >50% is the sole significant predictor variable in the multiple binary logistic regression model with 5 variables entered
- B=regression weights for model – not intuitive to understand
- SE = Standard errors
- Wald = Ratio of regression weight to SE i.e., B/S. E
- Df = degrees of freedom

- Easier to understand Exp(B) = Odds Ratio – Change in odds for every unit change of predictor variable

    - Patients with high plaque levels (>50% at baseline) are 4.9xmore likely to have an overall mean PPD >=4mm (P=0.003, Exp(B)=4.887)
    - All other factors are non-significant and have a 95% Exp(B) CI that straddles/crosses 1 i.e., we cannot be sure that the odds ratio for these factors are significantly different from 1

- Only poor plaque control has significant effect on Mean PPD

Outcomes:

- In a range of periodontal outcome measures SE Asians did not have significantly worse disease compared to other ethnic groups. (See previous but perhaps present younger?)

Risk Factors as predictors of tooth loss:

- Smoking, Age>40 years and self-declared stress were significant predictors of tooth loss at baseline assessment
- Only poor plaque control (defined as >50%) was a significant predictor of mean PPD>=4mm

Chapter 11. DISCUSSION, FUTURE WORK AND CONCLUSIONS

11.1: Discussion

Identifying if there is greater periodontitis disease severity in minor ethnic groups within the UK and the risk factors/indictors associated with this is of great interest. Particularly in London areas were the Public Health England data demonstrates poorer general health with higher levels of diabetes, obesity, cardiovascular disease, and smoking compared to the national average (PHE Statistics). Also there has been limited studies and limitations within those studies too (Steele *et al.*, 2012; Delgado-Angulo, Bernabé and Marcenes, 2016; Al-Haboubi *et al.*, 2014) looking into specific minor ethnic groups in the UK.

This cross-sectional study had been carried out retrospectively on a referred population that had been consecutively assessed and taken on for periodontal treatment at the RLDH by the postgraduate periodontal trainees. There is selection bias in the population already as we only receive the most severe periodontitis disease-based cases. It may have been more rationale and better to focus only on a specific disease severity of subjects such as Generalised Periodontitis Stage III Grade C (EFP 2017'). Further to this, another limitation is that there was no calibration in the diagnosis of Periodontitis when extracting the data from the clinical records. That may have changed the overall raw data findings in the cohort studied with respect to the percentages of subjects in each staging and grading category of their periodontitis disease severity.

Another avenue that could have also been considered, is adding the 2018 BSP implementation of the classification and observe if different outcomes would have come to fruition.

The consistency of the measurements in particular probing pockets depths (including lack of clinical attachment levels) is not likely to be as high in prospective studies due to a probable higher intra- and inter-examiner variability. In this study, several uncalibrated examiners (postgraduate dental trainees) carried out the baseline assessments which may further reduce the accuracy of recording the probing pocket depths. Significant relationships involving probing depths may potentially be overlooked if there is a limit to the number of observations. The limitation here also is that we cannot tell if there was a consistency in those measurements. If we could, then statistically significant relationships would not be as critically invalidated, especially as there were several postgraduate periodontal students carrying out the assessments in this present study. This in turn could have increased the external validity of this study, (Cox DR., 1958). In retrospective studies there is also potentially less bias amongst the assessors in comparison with prospective studies.

Primary guidance of the independent variables was on the evidence based literature showing that these variables would have an influence on the periodontal probing depths and tooth loss, (Helal *et al.*, 2019).

In the study it was found that smoking, and subjects older than 40 years of age were significant predictors of tooth loss at baseline assessment. This is in line with a recent systematic and meta-analysis review, (Helal *et al.*, 2019) which

also identified these 2 factors amongst others to be associated with higher risk of tooth loss. Although we must take into consideration that smoking has causal associations with the pathological mechanisms of periodontitis, (Rivera-Hidalgo, 2003).

Self-declared high stress levels (≥6 out 10) was also a significant predictor of tooth loss in this study. However, as undermentioned despite a number of subjects having high levels of stress in this study, the lack of information (raw data) demonstrating that tooth loss was due to periodontitis limits this statistical significant finding despite the continuing emerging evidence, (Decker, Kapila and Wang, 2021; Castro *et al.*, 2020; Decker *et al.*, 2020) that links stress with periodontal and peri-implant diseases as well as wound healing.

Nonetheless, this is another systemic risk factor of paramount that we as clinicians need to gain knowledge and understanding to subsume into more predictable periodontal treatment plans as well as making patients understand the effects of stress and the importance of its management towards general health and oral health related care.

Tooth loss which was used as a primary outcome can be considered cruder as teeth could have also been lost due to endodontics and other restorative reasons and not just periodontal ones. Also, the subjects in this study were those in the stage 3 and 4 categories of periodontal disease severity. Taking into account that the annual rate of tooth loss for each patient have not automatically been found to be different from those with more advanced/very

severe forms of periodontitis (previously diagnosed as aggressive periodontitis) to those with moderate forms (previously diagnosed as chronic periodontitis), (Graetz *et al.*, 2017; Nibali, 2014).

Elaborating further on tooth loss in this study, another limitation is that tooth loss was recorded as being yes or no in association with risk factors and risk indicators, but it is completely different when a patient loses 1 tooth compared to one that loses 5 or more teeth. The disadvantage of dichotomous outcomes is partly due to the size groups. We would need to look at a far larger sample size to be able to do further sub-analysis. Therefore, here the risk is likely to be different in no tooth loss vs 1-5 tooth loss vs 5+ tooth loss. By undertaking a simplified analysis which was done here, this does not allow us to delve further into a stronger association between tooth loss and the risk factors and risk indicators. This, therefore, needs to be taken into consideration when interpreting the findings in this study.

In the multiple regression analysis model 2 were PPD was used as alternative primary outcome dependant variable only poor plaque control (>50% plaque scores) demonstrated a statistically significant effect on the mean PPD. We are aware that dental plaque biofilm is a primary etiological risk factor for periodontitis, (Darveau and Curtis, 2021). It was interesting to see that in the Model 1 regression model, when tooth loss was used as the dependant primary outcome, poorer plaque controls did not bear statistical significance on tooth loss. This can be related to the explanation earlier on that there are

multiple causes for tooth loss other than periodontal reasoning. Also, the sample size is small which can under or overestimate results in the findings.

A significant limitation in this study however is not being able to use the CAL as a primary outcome instead of PPD as a parameter to diagnose Periodontitis severity which is what the disease is classified with. This means that the EFP classification was used crudely in some ways. This, therefore, creates a drawback in being conclusive on which ethnic groups had Periodontitis disease severity more or less than the other if any. Using the BSP 2018' adaptation of the new classification, might potentially have been even better for this study considering the limitations of the data retrieved.

From the data analysis in this study the SE Asian population were found to more likely be diabetic than other ethnic groups combined. A limitation here is that DM was recorded in the data as Yes or No, however it does not take into consideration the level of control of the glycosylated haemoglobin levels in these subjects. So, yes some of the SEA patients may have DM I or II, however this may be controlled, and this does not have the same significant impact as uncontrolled DM I or II on the associations with Periodontitis.

There is a higher proportion of SE Asians presenting <40 years old compared to all other ethnic groups combined. SE Asian population perhaps present younger? Is this because they have disease earlier compared to non – SE Asians. Significantly more of the SE Asian cohort self-declared as irregular attenders compared to other ethnic groups combined. Furthermore, SE Asians

reported lower levels of “stress” when ask to rate stress from 0-10. Also, the SE Asians did not appear to have worse periodontal disease than the other Ethnic groups combined. However, it can be noted that only 28.7% percent of the population in this study are from the SE Asian ethnic background. It would be difficult to ascertain as to why in a densely populated London Borough (Tower Hamlets) of SE Asians, not as many are taken on for further periodontal treatment at the RLDH.

Furthermore, the cohort in this cross-sectional study overall demonstrated with poor levels of compliance in terms of plaque control at baseline assessment which many studies have demonstrated over several decades, that dental biofilm is a major risk factor for periodontitis, the impacts of poor dental biofilm control on initial non-surgical periodontal therapy (NSPT) and long term periodontitis disease progression, (Lertpimonchai *et al.*, 2017; Tomasi, Leyland and Wennström, 2007a; Sanz-Martín *et al.*, 2019).

The patients that are usually supposed to be accepted for further periodontal treatment at the RLDH are those expressing plaque control levels ≤20% at baseline assessment. It has been requested to the GDP that patients should be complaint at least in terms of plaque control (≤20%) prior to being referred to the RLDH. However, it is widely known and accepted at the Hospital and transparently in this study that this is not the case. Patients are taken on for treatment despite the poor plaque control levels. However, part of the NHS policy at the RLDH is that if patients fail to demonstrate compliance in attendance and significant improvement and consistency in their plaque

control, they would be discharged back to the continued care of their GDP. They can only be re-referred to the RLDH if their plaque control has improved to the required levels prior to undertaking NSPT.

The sample size in this study has been powered so that a true difference between the SEA and all other Ethnic groups combined can be detected for the primary variables assessed. However, another limitation was that powering was only done for one of the primary variables (PPD≥4mm and not for Tooth loss), therefore, it is quite possible that with a much larger sample size the non-significant findings in this study may be significant or the significant findings may also be non-significant. This means there is also a higher risk of type I and type 2 errors in this study and the sample size may be considered small and not fully demonstrate sound external validity to other Dental hospitals and primary dental care centres with a magnified focus on minor ethnic groups.

Some of the benefits of this study are that a specific period of point in time is chosen to assess for meaningful information to contribute to more exploration of the results with further in-depth research. There is reduced risk of bias slipping in while the raw data is being gathered as variable are not manipulated. It is a cost-effective way of conducting research. Several variables/characteristics can be observed simultaneously. However, some of the disadvantages with this type of study design are that it doesn't necessarily demonstrate the entire demographic (especially minor ethnic origins). Larger sample sizes are often required to be able to generate information which can be more generalisable. With smaller samples coincidence/chance may

potentially influence the results. In such study designs we are unable to investigate for temporal consistency/relationship between the risk factors and outcomes.

11.2: Implications for further research

Multi-centre research with much larger sample sizes of the minor ethnic group should be conducted across the UK Dental Hospital clinics to gather further data regarding the risk factors and periodontitis disease severity. This is so that we can make some comparisons on the results identified in this study as there is a lack of existing quantitative study-based evidence to collate with for the minor ethnic groups. Although, the difficulty here would be in trying to isolate the effect of ethnicity when so many other variables are different between the patients when you examine them.

A study published back in 2016 concluded that despite the highlighted health inequalities amongst various ethnic groups, “oral health was better among non-White groups,” (Arora *et al.*, 2016) with a strong limitation that only 5.7% of the population was of minor ethnic backgrounds. Therefore, we need to consider further qualitative studies to identify amongst the ethnic groups (in particular significantly greater numbers from the minor ethnic group communities), their cultural beliefs, behaviour variables contributing towards their dental care philosophies, the ease of their accessibility to primary and secondary oral health care, and other elements that may contribute to the percentage of those being taken on for further periodontal treatment at other

respective UK Dental hospitals; to see if there is also a trend in a lower percentages elsewhere and potential rationales for this.

11.3: Conclusions

Within the limitations of this retrospective cross-sectional study, it is unclear whether the SE-Asian group demonstrated with significant Periodontitis disease severity more or less than the other Ethnic groups combined. There is a lack of strong evidence to demonstrate that there are more diabetics amongst the SEA population, and as to whether this is controlled or uncontrolled DM. However, in this study the SEA group had lower levels of self-reported stress and higher numbers of irregular dental attenders compared with other ethnic groups combined.

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