



Public Health
England

Protecting and improving the nation's health

The role of smoking in the progressive decline of the body's major systems

A report commissioned by Public Health England

Author: Dr Rachael Murray

UK Centre for Tobacco and Alcohol Studies

Division of Epidemiology and Public Health, University of Nottingham



UKCTAS

UK Centre for Tobacco & Alcohol Studies



The University of
Nottingham

UNITED KINGDOM • CHINA • MALAYSIA

About Public Health England

Public Health England exists to protect and improve the nation's health and wellbeing, and reduce health inequalities. It does this through world-class science, knowledge and intelligence, advocacy, partnerships and the delivery of specialist public health services. PHE is an operationally autonomous executive agency of the Department of Health.

Public Health England
Wellington House
133-155 Waterloo Road
London SE1 8UG
Tel: 020 7654 8000
www.gov.uk/phe
Twitter: @PHE_uk
Facebook: www.facebook.com/PublicHealthEngland

Author: Dr Rachael Murray

UK Centre for Tobacco and Alcohol Studies
Division of Epidemiology and Public Health, University of Nottingham
Clinical Sciences Building
City Hospital
Hucknall Road
Nottingham
NG5 1PB

© Crown copyright 2014

You may re-use this information (excluding logos) free of charge in any format or medium, under the terms of the Open Government Licence v2.0. To view this licence, visit [OGL](https://www.ogil.io) or email psi@nationalarchives.gsi.gov.uk. Where we have identified any third party copyright information you will need to obtain permission from the copyright holders concerned. Any enquiries regarding this publication should be sent to phe.enquiries@phe.gov.uk

Published December 2014

PHE publications gateway number: 2014597



Contents

About Public Health England	2
1. Introduction	4
2. Smoking and the musculoskeletal system	6
3. Smoking and cognitive function	11
4. Smoking and oral health	13
5. Smoking and visual health	16
6. References	19

1. Introduction

The author was commissioned to review the evidence on the role of smoking in the progressive decline of the body's major systems including the respiratory, cardiovascular and musculoskeletal systems, cognitive function, oral health and vision. A brief review established that the evidence on harm to respiratory and cardiovascular health was so well established that there was little value in reciting this and so this report focuses on the latter four systems.

The author was asked, specifically, to determine whether it was justified to describe the effect of smoking as "to rot the body from the inside". This description is considered justified because of the impact on conditions such as peripheral arterial disease and dental caries. In addition, the review describes how smoking causes deterioration and degeneration of a number of body functions due to neglect, in a process akin to rotting, driven by the addictive nature of the habit.

Smoking does progressive harm to the musculoskeletal system. It has a negative impact on bone mineral density, with evidence of damage apparent from the late teens but is particularly pronounced among post-menopausal women. Smoking is associated with more bone fractures and slower healing, and is associated with up to a 40% increase in the risk of hip fractures among men. Smokers and ex-smokers experience 60% more pain in the back, neck and legs and a 114% increase in increase in disabling lower back pain. Smoking is a significant cause of rheumatoid arthritis, especially among men and can also reduce the impact of treatment. Much of the harm and risk caused by smoking arises from smoking in middle years and can be prevented by cessation as a young adult or partially reversed by quitting in later years.

Smoking also does progressive harm to the cognitive system, contributing to a decline in cognitive function as early as middle-age. Current smokers are over 50% more likely to develop cognitive impairment than non-smokers, and the effects are largest among those who smoke more and older age groups. Smoking is one of the main environmental risk factors for dementia, primarily through increasing vascular risk and the risk of dementia increases with increasing cigarette consumption. Heavy smokers are at the greatest risk. Much of the increased risk of dementia and cognitive decline can be reversed by quitting smoking.

Smoking is detrimental to oral health, causing a progressive degeneration of tooth enamel and gum tissue as early as the adolescent years. Current smokers have more than a three times greater risk of developing oral cancers and are three to four times more likely to have periodontal disease than non-smokers. The risk of periodontal disease is higher in heavier smokers, with nearly a six times greater risk than non-smokers and the risk is greater in male than female smokers. Current smokers are four and a half times more likely to experience tooth decay than never smokers, with a greater effect in heavier smokers and adolescent smokers are significantly more likely to experience tooth decay than never smokers. Quitting smoking reduces the risk of oral cancer, periodontal disease and tooth decay.

Smoking is detrimental to visual health, with current smokers having a 78%-358% increased risk of age-related macular degeneration (AMD) and are more likely to develop the disease up to ten years earlier than never smokers. Increasing cigarette consumption (both cigarettes per day and years of smoking) increases both the incidence and progression of AMD in current smokers, with heavy smokers at particular risk. In addition, ever smokers are at a 41%-57% increased risk of age-related cataracts. Quitting smoking decreases the risk of AMD and cataract, although early quitting is more beneficial.

2. Smoking and the musculoskeletal system

2.1 Summary

Smoking does progressive harm to the musculoskeletal system. The damage has been observed to start as early as the late teens and increases with age but both the risk and harm can be partially reversed by quitting.

- smoking has a negative impact on bone mineral density, reducing calcium absorption and lowering levels of vitamin D, changing hormone levels and reducing body mass
- smokers and ex-smokers experience 60% more pain in the back, neck and legs and 114% increase in disabling lower back pain
- smoking appears to be associated with poorer development of the hip, spine and neck and lower bone mineral density among men as young as 18-20
- smoking has a particularly greater impact on reduced bone mineral density among post-menopausal women
- smoking is associated with more bone fractures and slower healing and is associated with up to a 40% increase in the risk of hip fractures among men
- smoking is a significant cause of rheumatoid arthritis, especially among men. Smoking can also reduce the impact of treatment
- much of the harm and risk caused by smoking arises from smoking in middle years and can be prevented by cessation as a young adult or partially reversed by quitting in later years

2.2 Osteoporosis

Osteoporosis is the main disease associated with poor bone health. It is largely associated with older age although can affect people of any age, with almost three million people in the UK suffering with the disease.¹ Osteoporosis results from a loss of bone mineral density (BMD) and degeneration of bone tissue, leading to a weakening of the bone.² While the majority (60-80%) of BMD variation is explained by pre-determined factors such as genetics,³ the remaining 20-40% is attributable to non-modifiable risk factors such as age but also to modifiable risk factors, of which smoking is a long established contributing risk factor.⁴

There are a number of mechanisms by which smoking affects BMD, including decreased calcium absorption, lower levels of vitamin D, changes in hormone levels, reduced body mass, increased free radicals and oxidative stress, higher likelihood of peripheral vascular disease and direct effects of toxic components of tobacco smoke on bone cells.⁵⁻¹¹

There is little evidence to suggest that smoking at a young age has a physiologic influence on the development of osteoporosis in later life. Indeed, the 2004 Surgeon General's report concludes that the evidence is inadequate to infer a causal relationship between smoking and decreased bone density in pre-menopausal women and younger men.¹² Since smoking patterns established in early life are likely to persist in later life, and the evidence discussed below will highlight the negative effect of smoking on bone health in older individuals, the overall influence of youth smoking should not be ignored. Some evidence has begun to emerge, however, that the development in bone density may be reduced in smokers. One five year longitudinal study investigating the development of BMD in 833 young men aged 18-20 found that those who had started smoking had considerably smaller increases in total body and lumbar spine BMD, and substantially greater decreases at the total hip and femoral neck than men who were non-smokers at both baseline and follow up visits.¹³ Another study of 1,068 men aged 18-20 years found a significant mean difference in BMD of 3.3% in the spine and 5% in the trochanter after adjusting for age, height, weight, calcium intake and physical activity. This difference was evident despite the mean duration of smoking being just 4.1 years.¹⁴

In contrast to findings for younger age groups, smoking has a proven adverse effect on BMD in middle and later years of life. The 2004 Surgeon General's report concluded that there is sufficient evidence to infer a causal relationship between smoking and low bone density in post-menopausal women¹². The evidence is not as strong for men, with same report concluding that the evidence is suggestive but not sufficient to infer that there is a relationship between smoking and low bone density in older men¹². A meta-analysis of 29 cross sectional studies published in 1997 reported that bone density was lower in post-menopausal female smokers than non-smokers¹⁵. The difference increased linearly with age: for every ten year increase in age the BMD in smokers fell below that of non-smokers by approximately two percent of the average bone density at the time of menopause.¹⁵

A further systematic review published in 2001 reported similar reductions in both men and women, with a significantly lower bone mass in smokers than non-smokers and a particularly pronounced effect at the hip. Effects were greatest in men and smoking had a significant adverse effect in individuals aged over 60 years and in those with a higher smoking exposure (pack years, number of years as a smoker or cigarettes per day).¹⁶ The same review pooled the results of thirteen prospective studies and demonstrated that current and ever smokers had greater rates of bone loss than nonsmokers, independent of factors such as age, gender and body mass¹⁶. A recent cross sectional study of BMD at the lumbar spine and femoral neck in 41 pairs of female twins discordant for smoking habits showed an average deficit in BMD of 5-10% at the time of menopause in women who had smoked one pack of cigarettes per day throughout adulthood.¹⁷ It has also been suggested that bone loss shows a positive relationship with the daily number of cigarettes smoked and years of exposure, independent from age, sex, weight, BMI and other 'unhealthy' lifestyle factors.¹⁸

Quitting smoking may have positive effects on BMD. Levels of BMD in ex-smokers fall between those of current and ex-smokers, suggesting that smoking cessation may slow or partially

reverse the deleterious effects of smoking.^{16 19 20} In a study investigating the effect of smoking cessation on BMD in postmenopausal women, there was some suggestion of increases in BMD following cessation, although this was limited to a small number of sites.²¹

2.3 Fractures/bone repair

One of the main complications of osteoporosis and degeneration of bone tissue is an increased risk of fracture, with over 230,000 resulting fractures per year in the UK.² A systematic review comprising nearly 60,000 individuals concluded that current smokers were at a significantly increased risk of any fracture than non-smokers, and that this risk was significantly higher in men than women.²² Further, ex-smokers were at a significantly increased risk of fracture than never smokers, although this risk was less than for current smokers. Cigarette smoking leads to increased fracture rates of the hip, spine, distal radius and other osteoporosis-related fractures,²³ with reports that this increased risk across all bone sites is approximately 5% in women and 11% in men, with the greatest increases evident at the hip (31% for women, 40% for men) and at the lumbar spine (13% for women and 32% for men).¹⁶ The estimated proportion of hip and lumbar spine fractures attributable to smoking are 6.4% and 2.8% in women and 9.9% and 8.1% in men respectively.¹⁶ One systematic review reported that when comparing women who are current smokers with non-smokers, smokers were at a 17% greater risk of hip fracture at age 60, 41% at 70, 71% at 80 and 108% at 90.¹⁵ The data for men were limited but appear to suggest a similar pattern. It has been concluded that there is sufficient evidence to infer a causal relationship between smoking and hip fractures, although the evidence is inadequate to infer a causal relationship between smoking and fractures at sites other than the hip.¹²

The burden of hip fractures should not be underestimated. Mortality rates of up to 20-24% in the year after a hip fracture have been reported,^{24 25} with an increased risk of death persisting for up to five years afterwards.²⁶ It has been reported that a 50 year old woman has an equal chance of dying from a hip fracture as breast cancer.²⁷ In addition to the increased risk of fracture, healing time is suggested to be prolonged in smokers.^{23 28-31} Smoking is also associated with non-union of fractures, infection and poor wound healing in addition to overall worse health outcomes from both fracture and fracture healing.²³

The association between smoking cessation the risk of fracture is less widely documented. The largest body of evidence exists for the risk on hip fractures, with a suggestion that successful smoking cessation for at least five years may reduce the risk in men,³² and a minimum of ten years cessation required to see a benefit in women.^{21 33} Current smokers have been reported to be at a 25% increased risk of any fracture, with no difference between males and females.¹⁰

2.4 Rheumatoid arthritis

Rheumatoid arthritis is a chronic inflammatory disease causing pain, joint stiffness and swelling. The cause is not fully understood but the condition affects an estimated 690,000 people in the UK, most commonly starts between the ages of 40 and 50 and is around three times more common in women than men.³⁴ It is estimated that smoking contributes to up to 25% of the population burden of rheumatoid arthritis.³⁵ One systematic review published in 2010 found the overall risk for developing rheumatoid arthritis was 1.35 (95% CI 1.17-1.55) for current smokers and 1.25 (95% CI 1.10-1.40) for past smokers compared to non-smokers.³⁶ Despite the overwhelming majority (94%) of patients included in the analysis being female, the risk appeared to be highest in males with an overall risk for current smokers of 1.89 (95% CI 1.56-2.28). A dose response relationship has been reported in women.^{35 36} A further meta-analysis published in 2012 supported the findings that smoking presents a stronger risk in males and is associated with longer duration of smoking.³⁷

After considering the evidence available, the Surgeon General 2014 report concluded that there is sufficient evidence to infer a causal relationship between cigarette smoking and rheumatoid arthritis.³⁸ Treatment for rheumatoid arthritis often incorporates TNF- α inhibitory drugs, and the evidence is sufficient to infer that cigarette smoking reduces the effectiveness of TNF-alpha inhibitors.³⁸

Smoking cessation has been shown to decrease the risk of developing rheumatoid arthritis,^{35 37 39} although one study reported that the risk remained elevated in smokers until 20 years or more after cessation.³⁵ There is insufficient evidence, however, to draw any conclusions as to the potential benefit of smoking cessation on rheumatoid arthritis.³⁸

2.5 Soft tissue

A recent review concluded that cigarette smoking is associated with an increased risk of back pain and degenerative disc disease.²³ A meta-analysis of 40 studies showed that current smoking was associated with a 33% increased prevalence of low back pain within the previous 12 months, 79% increased prevalence of chronic back pain and 114% increased prevalence of disabling lower back pain.⁴⁰ Results were higher for former smokers than never smokers, suggesting that smoking cessation has a positive effect on incidence. The review reported that the increased risk of low back pain in smokers was stronger in adolescents than adults (82% compared to 16%).⁴⁰

A study of nearly 13,000 subjects found that current and ex-smokers experienced up to 60% more musculoskeletal pain (low back, neck upper and lower limbs) than never smokers, and this prevalence was higher in current smokers than ex-smokers⁴¹ (Palmer 2003). One study has found that disc degeneration was 18% greater in heavy smokers than non-smokers, although there was no clinical difference.⁴² There is also some suggestion that cigarette smokers may have lower strength than non-smokers. Evidence from one study suggests that

quadriceps strength was reduced by 2.9% in men and 5% in women who smoked 100g tobacco per week.⁴³

Tendons are also affected by cigarette smoking: an association has been demonstrated between tendinopathy and persistent shoulder pain⁴⁴ and as a risk factor for the development of rotator cuff disease and tears.⁴⁴⁻⁴⁶ One study reported that among 584 patients presenting for a diagnostic ultrasound for shoulder pain, those with rotator cuff tears were significantly more likely to smoke daily, to have smoked regularly within ten years before attending the clinic, have a higher mean pack per day smoking history, have a longer duration of smoking and larger tobacco exposure than those without rotator cuff tears.⁴⁵ Smokers were 74% more likely than non-smokers to have a rotator cuff tear, and this was 424% more likely in those who had smoked within the past ten years and 335% more likely in heavy current smokers. In addition, increasing daily average number of cigarettes and total number of cigarettes smoked has been shown to be associated with the severity of tears.⁴⁷

3. Smoking and cognitive function

3.1 Summary

Smoking does progressive harm to the cognitive system, contributing to a decline in cognitive function as early as middle-age. The impact increases with age and amount smoked, although quitting can reduce the risk and harm caused.

- smoking is one of the main environmental risk factors for dementia, primarily through increasing vascular risk
- the risk of dementia increases with increasing consumption, with the greatest risk in heavy smokers
- current smokers are over 50% more likely to develop cognitive impairment than non-smokers.
- faster declines in verbal memory and lower visual search speeds have been reported in smokers aged between 43 and 53
- the effects of smoking on cognitive decline are largest among those who smoke more
- much of the increased risk of dementia and cognitive decline can be reversed by quitting smoking

3.2 Dementia

Dementia is a syndrome resulting from a progressive deterioration in higher cognitive function (including memory, language, concentration and reasoning) that impairs daily activities.⁴⁸

Dementia develops as a result of degeneration and death of brain cells and is caused by a number of diseases. Alzheimer's disease is the most common cause of dementia; brain cells are surrounded by an abnormal protein and their internal structure is damaged. Ultimately, chemical connections between brain cells are lost and some cells die⁴⁹ (Alzheimer's Society 2014). Vascular dementia is the second most common type of dementia and results from a reduced oxygen supply to the brain, resulting in damage to or death of brain cells.⁴⁹ Dementia has a number of genetic and environmental risk factors, the main ones being those that influence vascular risk including smoking, obesity, high blood pressure and diabetes.^{50 51} Two recent reviews calculated that an estimated 13-14% of Alzheimer's disease cases worldwide were potentially attributable to smoking.^{50 52}

Two systematic reviews have been published investigating the link between smoking and dementia.^{53 54} A meta-analysis of eight studies published in 2008 reported that current smokers were 59% more likely than never smokers to suffer Alzheimer's disease and 35% more likely to suffer vascular dementia.⁵⁴ These findings were consistent with a meta-analysis of 19 studies published in 2007 that reported increased risks of 79% and 78% respectively.⁵³ Three studies

reported exposure-related effects, with the risk of dementia increasing with increasing cigarette consumption, with a marked effect in heavy smokers.⁵⁵⁻⁵⁷

There are currently no treatments available to cure or alter the progression of dementia, thus changing modifiable risk factors is of key importance. A recent modelling estimate has suggested that every 5% reduction in smoking prevalence would result in a 2% reduction in the risk of dementia.⁵⁸ A further review estimated that if smoking were eliminated, 13.9% of Alzheimer's disease could be prevented.⁵⁰

Two meta-analyses reported no association between former smoking and the risk of dementia, suggesting that smoking cessation could play a role in reducing the incidence of dementia.^{53 54}

3.3 Cognitive impairment

Cognitive impairment (as opposed to degeneration and death of brain cells) is a natural part of ageing⁵⁹ but may develop into dementia and this decline is suggested to be accelerated in current smokers.⁶⁰ A recent study reported that current smokers were 53% more likely to develop cognitive impairment than non-smokers.⁶¹ When looking at differential effects by gender, it has been reported that a significant effect was only present in women; female smokers were 62% more likely to develop cognitive impairment than never-smokers, and those with high pack years were more than twice as likely to develop cognitive impairment than never-smokers.⁶¹ Other studies have reported a stronger effect in men, with reports that middle aged male smokers experienced faster declines in global cognition and executive function than never smokers.⁶⁰ Further, smoking was consistently associated with lower performance on cognitive outcomes in adults aged 50 and over.⁶²

There is evidence to suggest a significant dose-response relationship between amount smoked and rate of decline in cognitive function in smokers with a higher cigarette pack-year exposure.^{61 63} One study also reported higher rates of decline in male and female smokers aged over 65.⁶⁰ Faster declines in verbal memory and lower visual search speeds have been reported in male and female smokers aged between 43 and 53, with the effect largest in those who smoked more than 20 cigarettes per day, independent of other potentially confounding factors.⁶⁴ It has been demonstrated that ex-smokers who have not smoked for at least ten years were at no increased risk of cognitive decline as a result of their past smoking.⁶⁰

4. Smoking and oral health

4.1 Summary

Smoking is detrimental to oral health, causing a progressive degeneration of tooth enamel and gum tissue as early as the adolescent years. The risk and harm can, however, be reduced by quitting.

- current smokers have more than a three times greater risk of developing oral cancers than non-smokers
- current smokers are three to four times more likely to have periodontal disease than non-smokers
- the risk is higher in heavier smokers, with nearly a six times greater risk than non-smokers
- male smokers are at a greater risk of periodontal disease than female smokers
- current smokers are four and a half times more likely to experience tooth decay than never smokers, with a greater effect in heavier smokers
- adolescent smokers are significantly more likely to experience tooth decay than never smokers
- quitting smoking reduces the risk of oral cancer, periodontal disease and tooth decay

4.2 Oral cancers

Cancer is a disease caused by changes in normal cells so that they grow in an uncontrolled way; oral cancer includes cancers of a number of sites including the lip, tongue, mouth, oral cavity and pharynx.⁶⁵ The 2004 Surgeon General's report on the health consequences of smoking concluded that smoking is the major cause of oral cancer.¹² Smoked and smokeless tobacco use are linked to a wide range of cancerous, precancerous and non-malignant conditions affecting the soft and hard tissues of the oral cavity.⁶⁶ It is estimated that current smokers have more than three times greater risk of developing oral cancers⁶⁷ and an estimated 65% of oral and pharyngeal cancers in the UK are linked to smoking.⁶⁸ Oral cancer is two to three times more common in men than women, however, trends over the last 15 years showed an increase in oral cancer mortality rates in women in industrialised countries.⁶⁹⁻⁷¹

Cases of oral cancer in the UK have increased by 32% from 2000-2002 to 2009-2011.⁶⁵ Incidence increases with age in both genders and across countries with smokers aged 50 and above having a higher risk of developing oral cancers. Socioeconomic inequalities were found to influence the mortality rates in oral cancer with nearly doubled mortality rates in blacks compared to whites.^{69 70} Alcohol consumption is a risk factor for oral cancer in smokers⁷⁰⁻⁷² and is almost tripled in alcohol drinkers who smoke.⁶⁵

Smoking cessation has major and immediate health benefits for smokers of all ages and both genders.^{73 74} A cohort study of over 12,000 smokers and former smokers with a ten year follow up found that cessation of tobacco use lead to a substantial decrease in incidence of mucosal lesions in smokers who ceased tobacco use, implying a reduced risk for oral cancer.⁷⁵ Findings from the Australian National Survey of Adult Oral Health also confirmed that smoking cessation may reduce the prevalence of oral mucosal lesions in the population following tobacco cessation interventions.⁷⁶

4.3 Periodontitis and tooth loss

Periodontitis is an inflammatory response that causes inflammation, bleeding gums and ultimately the degeneration of gum, bone and connective tissue that support the teeth, ultimately leading to tooth loss.^{77 78} In addition to the impact on oral health, it has also been shown that individuals with periodontal disease are at a 1.24-1.35 times greater risk of coronary heart disease⁷⁹ and 1.47-2.63 times greater risk of stroke,⁸⁰ independent of other risk factors. Smokers are more likely than non-smokers to develop periodontitis,⁸¹ indeed the 2004 Surgeon General's report on the health consequences of smoking concluded that there is a causal relationship between smoking and periodontal disease.¹² Adult smokers are suggested to be approximately three to four times as likely as non-smokers to have periodontitis^{82 83} and among post-menopausal women, heavy smokers were 82% more likely to report tooth loss than never smokers.⁸⁴ Smoking appears to have considerable adverse effects on the inflammatory process, thereby, promoting the progression of periodontal disease in smokers.⁸⁵

The severity of periodontal disease increases with the number of cigarettes smoked per day and the duration of the habit⁸⁶ and is more evident after ten years of smoking.⁸⁷ One study reported that in those smoking less than nine cigarettes per day, the risk of developing periodontal disease more than doubled but was nearly six times higher in those who smoked more than 31 cigarettes per day.⁸³ The effect of tobacco on periodontal tissues seem to be more pronounced in men than women.⁸⁷ Smoking is a risk indicator for missing teeth in older adults in both current and previous smokers,⁸⁸ and the progression of periodontal disease and tooth loss is independently influenced by smoking and disadvantaged socioeconomic status.⁸⁹

Tobacco cessation is associated with improvement in periodontal status and former smokers have lower rates of periodontal disease than current smokers^{82 87 90} and respond significantly better to non-surgical periodontal therapy after quitting compared to those who continue to smoke.⁹¹ While the rate of periodontal disease progression is suggested to be faster in smokers,⁹² it decreases to that of a nonsmoker following tobacco cessation.⁸² The elevated risk for tooth loss in smokers who quit also decreases over time. A longitudinal cohort study on tobacco use and tooth loss in 43,112 male health professionals found that risk decreased with the increase in time since cessation but remained elevated by 20% for smokers who had quit ten or more years before.⁹³ Similarly, another study reported that individuals who had quit smoking for between 0 and 2 years were almost three times more likely experience periodontitis than non-smokers, compared to just 15% more likely more among those who had

quit smoking for more than 11 years⁸³ Although the risk of experiencing tooth loss declines with time since smoking cessation, the effects of smoking may persist for at least 30 years.⁹⁴

4.4 Dental caries

Dental caries (or tooth decay) is an infection where bacterial produced acids dissolve enamel,⁹⁵ leading to a degeneration of hard tissues of the teeth. There are a limited number of research studies investigating the relationship between smoking and dental caries. Based on 16 studies, the 2014 Surgeon General's Report concluded that the evidence is suggestive but not sufficient to infer a causal relationship between smoking and dental caries.³⁸ Four studies have reported a significantly higher prevalence of untreated dental caries in smokers than non-smokers but no significant difference in the overall prevalence of dental caries.⁹⁶⁻⁹⁹ One study that investigated the relationship between smoking and tooth decay found that current smokers were around four and a half times more likely to experience tooth decay than never smokers, with former smokers over two and a half times more likely.¹⁰⁰ Smoking is also shown to have an effect in adolescents, with one study showing significantly higher proportions of decayed, missing and filled teeth than non-smokers,¹⁰¹ with another showing that among those adolescents with at least four decayed, missing or filled tooth surface 85% more likely to be current smokers than those with no history of dental caries in their permanent teeth.¹⁰² The data also indicate that the prevalence of dental caries in smokers increases with the increase in daily consumption of cigarettes.^{101 103 104} Although the association between smoking and prevalence of dental caries can be attributed to poor dental care and oral hygiene,¹⁰⁵ a cross-sectional study with a four-year follow-up found that daily smoking independently predicts caries development in smokers.¹⁰⁶

5. Smoking and visual health

5.1 Summary

Smoking is detrimental to visual health, contributing to visual impairment earlier and to a greater severity in smokers than non-smokers. Quitting smoking can decrease the risk, although early quitting is more beneficial.

- current smokers have a 78%-358% increased risk of age-related macular degeneration (AMD), and are more likely to develop the disease up to ten years earlier than never smokers
- increasing cigarette consumption (both cigarettes per day and years of smoking) increases both the incidence and progression of AMD in current smokers, with heavy smokers particularly at risk
- ever smokers are at a 41%-57% increased risk of age-related cataracts
- quitting smoking decreases the risk of AMD and cataract, although early quitting is more beneficial

5.2 Age-related macular degeneration

Age-related macular degeneration (AMD) is an eye condition that leads to a gradual loss of central vision.¹⁰⁷ Based on a review of evidence the 2014 Surgeon General's report concluded that there is a causal relationship between cigarette smoking and neovascular and atrophic forms of AMD³⁸. A recent meta-analysis reported significant increases in AMD risk for current versus never smokers of between 78% and 358%, depending on the study design¹⁰⁸ (Chukravathy et al 2010). In addition, smokers are more likely to develop the disease ten years earlier than non-smokers.¹⁰⁹ The number of cigarettes and a greater number of pack-years smoked increased the risk of both incidence and progression of AMD in current smokers.^{110 111} The findings of a large French case-control study confirmed that heavy smokers (at least 25 pack-years of smoking) were particularly at risk.¹¹² A recent longitudinal study reported that current smoking was associated with a 36% increased risk of transitioning from minimal to moderate early AMD, and a greater number of pack-years smoked was associated with a 3% increased risk of developing early AMD and transitioning from severe early AMD to late AMD.¹¹⁰

Evidence suggests, but is not sufficient to infer, that smoking cessation decreases the risk of age-related macular degeneration.³⁸ The results of a number of studies showed that in former smokers the risk of AMD after quitting remained high compared to never smokers and only began to decrease after more than 20 years after quitting.^{112 113} Early quitting, therefore, is more beneficial.

5.3 Cataract

Cataracts are cloudy patches that develop in the lens of the eye, causing blurred vision and sometimes stopping some light reaching the back of the eye. Cataracts become progressively worse over time and often require surgery to remove and replace the affected lens (cataract extraction).¹¹⁴

It has been concluded that there is sufficient evidence to infer a causal relationship between smoking and nuclear cataracts¹². Findings from cohort and case-control studies showed that ever smoking was associated with a statistically significant 41%-57% increased risk of age-related cataracts; in current smokers, this increased risk is between 47% and 55%.¹¹⁵ Age and gender have been found to be independent risk factors for the development of mixed lens opacity.¹¹⁶

Smoking cessation reduces risk over time, however, the larger the exposure the longer it takes for the risk to reduce and this risk is unlikely to return to that of a never smoker,^{117 118} although one study just in women has demonstrated this possibility.¹¹⁹

Male and female smokers who had quit for at least 25 years had a 20% lower risk of cataract extraction than current smokers after adjusting for other risk factors, although this risk did not decrease to the level of never smokers.¹¹⁸ A prospective cohort study of 20,907 men found that those who quit smoking were at a 21-27% reduced risk of cataract, with the lowest reduction in risk evident in those who had quit smoking less than ten years previously although the risk did not reduce to that of never smokers in any category.¹²⁰ Similar results have recently been reported by Lindblad and colleagues, who found that smoking cessation in men significantly decreased the risk of cataract extraction with time, although risks did not reduce to that of never smokers.¹¹⁷ The effect of smoking cessation was observed earlier in lighter smokers (≤ 15 cigarettes per day) than heavy smokers.¹¹⁷

In a prospective cohort study of 34,595 women, smoking cessation was associated with decreasing risk over time.¹¹⁹ For women with a moderate intensity of smoking (6-10 cigarettes/day), the risk of cataract extraction after ten years of smoking cessation decreased to the same level as that among never smokers. Risk among women with a higher smoking intensity (>10 cigarettes/day) also decreased significantly over time but it took 20 years of cessation until the risk was no longer significantly increased in comparison with never smokers.

5.4 Other eye conditions

There are a number of other eye conditions that have a very limited evidence base addressing their relationship with smoking. Glaucoma is a condition usually caused by a build up of pressure within the eye which can damage the optic nerve.¹²¹ The evidence is inadequate to infer a relationship between smoking and glaucoma,¹² with a small number of studies reporting no association between smoking and glaucoma.¹²²⁻¹²⁵ However, there is a strong link between

smoking and high blood pressure, cataracts and diabetes all of which are risk factors for glaucoma.

Retinal vein occlusion (RVO) is a major cause of vision loss. A basic risk factor for RVO is age, although cigarette smoking increases the risk of RVO as do systemic conditions such as arteriosclerosis and vascular cerebral stroke (both of which are increased in smokers).¹²⁶ Smoking is significantly related to vision loss from retinal vasculitis (inflammation of the vascular branches of the retinal artery), and cigarette smoking predicted vision loss in a study of 56 patients followed for 25 years.¹²⁷ A cohort study of the association of smoking cessation on the retinal vessels in women found that the impact of smoking on the retinal vessel calibre is reversible after ten or more years of smoking cessation.¹²⁸

References

1. National Osteoporosis Society [Internet]. 2014. Available from: <http://www.nos.org.uk>. (accessed 12th October 2014)
2. Poole KE, Compston JE. Osteoporosis and its management. *BMJ* 2006;333(7581):1251-6.
3. Eisman JA. Genetics of osteoporosis. *Endocr Rev* 1999;20(6):788-804.
4. Wong PK, Christie JJ, Wark JD. The effects of smoking on bone health. *Clin Sci (Lond)* 2007;113(5):233-41.
5. Brot C, Jorgensen NR, Sorensen OH. The influence of smoking on vitamin D status and calcium metabolism. *Eur J Clin Nutr* 1999;53(12):920-6.
6. Daniel M, Martin AD, Drinkwater DT. Cigarette smoking, steroid hormones, and bone mineral density in young women. *Calcif Tissue Int* 1992;50(4):300-5.
7. Duthie GG, Arthur JR, James WP. Effects of smoking and vitamin E on blood antioxidant status. *Am J Clin Nutr* 1991;53(4 Suppl):1061S-63S.
8. Kapoor D, Jones TH. Smoking and hormones in health and endocrine disorders. *Eur J Endocrinol* 2005;152(4):491-9.
9. Nelson HD, Nevitt MC, Scott JC, Stone KL, Cummings SR. Smoking, alcohol, and neuromuscular and physical function of older women. Study of Osteoporotic Fractures Research Group. *JAMA* 1994;272(23):1825-31.
10. Vestergaard P, Mosekilde L. Fracture risk associated with smoking: a meta-analysis. *J Intern Med* 2003;254(6):572-83.
11. Broulik PD, Jarab J. The effect of chronic nicotine administration on bone mineral content in mice. *Horm Metab Res* 1993;25(4):219-21.
12. U.S. Department of Health and Human Services. *The Health Consequences of Smoking: A Report of the Surgeon General*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, 2004.
13. Rudang R, Darelid A, Nilsson M, Nilsson S, Mellstrom D, Ohlsson C, et al. Smoking is associated with impaired bone mass development in young adult men: a 5-year longitudinal study. *J Bone Miner Res* 2012;27(10):2189-97.
14. Lorentzon M, Mellstrom D, Haug E, Ohlsson C. Smoking is associated with lower bone mineral density and reduced cortical thickness in young men. *J Clin Endocrinol Metab* 2007;92(2):497-503.
15. Law MR, Hackshaw AK. A meta-analysis of cigarette smoking, bone mineral density and risk of hip fracture: recognition of a major effect. *BMJ* 1997;315(7112):841-6.
16. Ward KD, Klesges RC. A meta-analysis of the effects of cigarette smoking on bone mineral density. *Calcif Tissue Int* 2001;68(5):259-70.
17. Baczyk G, Opala T, Kleka P, Chuchracki M. Multifactorial analysis of risk factors for reduced bone mineral density among postmenopausal women. *Arch Med Sci* 2012;8(2):332-41.
18. Abate M, Vanni D, Pantalone A, Salini V. Cigarette smoking and musculoskeletal disorders. *Muscles Ligaments Tendons J* 2013;3(2):63-9.
19. Nguyen TV, Kelly PJ, Sambrook PN, Gilbert C, Pocock NA, Eisman JA. Lifestyle factors and bone density in the elderly: implications for osteoporosis prevention. *J Bone Miner Res* 1994;9(9):1339-46.
20. Szulc P, Garnero P, Clausturat B, Marchand F, Duboeuf F, Delmas PD. Increased bone resorption in moderate smokers with low body weight: the Minos study. *J Clin Endocrinol Metab* 2002;87(2):666-74.

21. Oncken C, Prestwood K, Kleppinger A, Wang Y, Cooney J, Raisz L. Impact of smoking cessation on bone mineral density in postmenopausal women. *J Womens Health (Larchmt)* 2006;15(10):1141-50.
22. Kanis JA, Johnell O, Oden A, Johansson H, De Laet C, Eisman JA, et al. Smoking and fracture risk: a meta-analysis. *Osteoporos Int* 2005;16(2):155-62.
23. Lee JJ, Patel R, Biermann JS, Dougherty PJ. The musculoskeletal effects of cigarette smoking. *J Bone Joint Surg Am* 2013;95(9):850-9.
24. Cooper C, Atkinson EJ, Jacobsen SJ, O'Fallon WM, Melton LJ, 3rd. Population-based study of survival after osteoporotic fractures. *Am J Epidemiol* 1993;137(9):1001-5.
25. Leibson CL, Tosteson AN, Gabriel SE, Ransom JE, Melton LJ. Mortality, disability, and nursing home use for persons with and without hip fracture: a population-based study. *J Am Geriatr Soc* 2002;50(10):1644-50.
26. Magaziner J, Lydick E, Hawkes W, Fox KM, Zimmerman SI, Epstein RS, et al. Excess mortality attributable to hip fracture in white women aged 70 years and older. *Am J Public Health* 1997;87(10):1630-6.
27. Cummings SR, Black DM, Rubin SM. Lifetime risks of hip, Colles', or vertebral fracture and coronary heart disease among white postmenopausal women. *Arch Intern Med* 1989;149(11):2445-8.
28. Adams CI, Keating JF, Court-Brown CM. Cigarette smoking and open tibial fractures. *Injury* 2001;32(1):61-5.
29. Al-Hadithy N, Sewell MD, Bhavikatti M, Gikas PD. The effect of smoking on fracture healing and on various orthopaedic procedures. *Acta Orthop Belg* 2012;78(3):285-90.
30. Schmitz MA, Finnegan M, Natarajan R, Champine J. Effect of smoking on tibial shaft fracture healing. *Clin Orthop Relat Res* 1999(365):184-200.
31. Sloan A, Hussain I, Maqsood M, Eremin O, El-Sheemy M. The effects of smoking on fracture healing. *Surgeon* 2010;8(2):111-6.
32. Hoidrup S, Prescott E, Sorensen TI, Gottschau A, Lauritzen JB, Schroll M, et al. Tobacco smoking and risk of hip fracture in men and women. *Int J Epidemiol* 2000;29(2):253-9.
33. Cornuz J, Feskanich D, Willett WC, Colditz GA. Smoking, smoking cessation, and risk of hip fracture in women. *Am J Med* 1999;106(3):311-4.
34. Bosworth, A. What is rheumatoid arthritis? [Internet] 2011 [updated October 2011]. Available from: <http://www.nras.org.uk/what-is-ra--what-is-ra--295> [accessed 09 October 2014].
35. Costenbader KH, Feskanich D, Mandl LA, Karlson EW. Smoking intensity, duration, and cessation, and the risk of rheumatoid arthritis in women. *Am J Med* 2006;119(6):503 e1-9.
36. Sugiyama D, Nishimura K, Tamaki K, Tsuji G, Nakazawa T, Morinobu A, et al. Impact of smoking as a risk factor for developing rheumatoid arthritis: a meta-analysis of observational studies. *Ann Rheum Dis* 2010;69(1):70-81.
37. Lahiri M, Morgan C, Symmons DP, Bruce IN. Modifiable risk factors for RA: prevention, better than cure? *Rheumatology (Oxford)* 2012;51(3):499-512.
38. U.S. Department of Health and Human Services. *The Health Consequences of Smoking—50 Years of Progress: A Report of the Surgeon General*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, 2014.
39. Di Giuseppe D, Orsini N, Alfredsson L, Askling J, Wolk A. Cigarette smoking and smoking cessation in relation to risk of rheumatoid arthritis in women. *Arthritis Res Ther* 2013;15(2):R56.

40. Shiri R, Karppinen J, Leino-Arjas P, Solovieva S, Viikari-Juntura E. The association between smoking and low back pain: a meta-analysis. *Am J Med* 2010;123(1):87 e7-35.
41. Palmer KT, Syddall H, Cooper C, Coggon D. Smoking and musculoskeletal disorders: findings from a British national survey. *Ann Rheum Dis* 2003;62(1):33-6.
42. Battie MC, Videman T, Kaprio J, Gibbons LE, Gill K, Manninen H, et al. The Twin Spine Study: contributions to a changing view of disc degeneration. *Spine J* 2009;9(1):47-59.
43. Kok MO, Hoekstra T, Twisk JW. The longitudinal relation between smoking and muscle strength in healthy adults. *Eur Addict Res* 2012;18(2):70-5.
44. Bodin J, Ha C, Serazin C, Descatha A, Leclerc A, Goldberg M, et al. Effects of individual and work-related factors on incidence of shoulder pain in a large working population. *J Occup Health* 2012;54(4):278-88.
45. Baumgarten KM, Gerlach D, Galatz LM, Teefey SA, Middleton WD, Ditsios K, et al. Cigarette smoking increases the risk for rotator cuff tears. *Clin Orthop Relat Res* 2010;468(6):1534-41.
46. Kane SM, Dave A, Haque A, Langston K. The incidence of rotator cuff disease in smoking and non-smoking patients: a cadaveric study. *Orthopedics* 2006;29(4):363-6.
47. Carbone S, Gumina S, Arceri V, Campagna V, Fagnani C, Postacchini F. The impact of preoperative smoking habit on rotator cuff tear: cigarette smoking influences rotator cuff tear sizes. *J Shoulder Elbow Surg* 2012;21(1):56-60.
48. *Smoking and mental health- A joint report by the Royal College of Physicians and the Royal College of Psychiatrists*. 2013, Royal College of Physicians/ Royal College of Psychiatrists: London.
49. National Alzheimers Society. What is dementia? [Internet] 2014. Available from http://www.alzheimers.org.uk/site/scripts/documents_info.php?documentID=106. Accessed October 10 2014.
50. Barnes DE, Yaffe K. The projected effect of risk factor reduction on Alzheimer's disease prevalence. *Lancet Neurol* 2011;10(9):819-28.
51. Stewart R. Cardiovascular factors in Alzheimer's disease. *J Neurol Neurosurg Psychiatry* 1998;65(2):143-7.
52. Norton S, Matthews FE, Barnes DE, Yaffe K, Brayne C. Potential for primary prevention of Alzheimer's disease: an analysis of population-based data. *Lancet Neurol* 2014;13(8):788-94.
53. Anstey KJ, von Sanden C, Salim A, O'Kearney R. Smoking as a risk factor for dementia and cognitive decline: a meta-analysis of prospective studies. *Am J Epidemiol* 2007;166(4):367-78.
54. Peters R, Poulter R, Warner J, Beckett N, Burch L, Bulpitt C. Smoking, dementia and cognitive decline in the elderly, a systematic review. *BMC Geriatr* 2008;8:36.
55. Juan D, Zhou DH, Li J, Wang JY, Gao C, Chen M. A 2-year follow-up study of cigarette smoking and risk of dementia. *Eur J Neurol* 2004;11(4):277-82.
56. Rusanen M, Kivipelto M, Quesenberry CP, Jr., Zhou J, Whitmer RA. Heavy smoking in midlife and long-term risk of Alzheimer disease and vascular dementia. *Arch Intern Med* 2011;171(4):333-9.
57. Tyas SL, White LR, Petrovitch H, Webster Ross G, Foley DJ, Heimovitz HK, et al. Mid-life smoking and late-life dementia: the Honolulu-Asia Aging Study. *Neurobiol Aging* 2003;24(4):589-96.
58. Nepal B, Brown L, Ranmuthugala G. Modelling the impact of modifying lifestyle risk factors on dementia prevalence in Australian population aged 45 years and over, 2006-2051. *Australas J Ageing* 2010;29(3):111-6.

59. Park DC, Bischof GN. The aging mind: neuroplasticity in response to cognitive training. *Dialogues Clin Neurosci* 2013;15(1):109-19.
60. Sabia S, Elbaz A, Dugravot A, Head J, Shipley M, Hagger-Johnson G, et al. Impact of smoking on cognitive decline in early old age: the Whitehall II cohort study. *Arch Gen Psychiatry* 2012;69(6):627-35.
61. Park B, Park J, Jun JK, Choi KS, Suh M. Gender differences in the association of smoking and drinking with the development of cognitive impairment. *PLoS One* 2013;8(10):e75095.
62. Dregan A, Stewart R, Gulliford MC. Cardiovascular risk factors and cognitive decline in adults aged 50 and over: a population-based cohort study. *Age Ageing* 2013;42(3):338-45.
63. Ott A, Andersen K, Dewey ME, Letenneur L, Brayne C, Copeland JR, et al. Effect of smoking on global cognitive function in nondemented elderly. *Neurology* 2004;62(6):920-4.
64. Richards M, Jarvis MJ, Thompson N, Wadsworth ME. Cigarette smoking and cognitive decline in midlife: evidence from a prospective birth cohort study. *Am J Public Health* 2003;93(6):994-8.
65. Cancer Research UK. Oral Cancer Key Facts [Internet] 2014. Available from <http://www.cancerresearchuk.org/cancer-info/cancerstats/keyfacts/oral-cancer/oral-cancer>. [Accessed October 19 2014].
66. Christen AG. The impact of tobacco use and cessation on oral and dental diseases and conditions. *Am J Med* 1992;93(1A):25S-31S.
67. Gandini S, Botteri E, Iodice S, Boniol M, Lowenfels AB, Maisonneuve P, et al. Tobacco smoking and cancer: a meta-analysis. *Int J Cancer* 2008;122(1):155-64.
68. Parkin DM. 2. Tobacco-attributable cancer burden in the UK in 2010. *Br J Cancer* 2011;105 Suppl 2:S6-S13.
69. Antunes J, Toporcov TN, Biazzevic MG, Boing AF, Bastos JL. Gender and racial inequalities in trends of oral cancer mortality in Sao Paulo, Brazil. *Rev Saude Publica* 2013;47(3):470-8.
70. Johnson N. Tobacco use and oral cancer: a global perspective. *J Dent Educ* 2001;65(4):328-39.
71. Seoane-Mato D, Aragonés N, Ferreras E, Garcia-Perez J, Cervantes-Amat M, Fernandez-Navarro P, et al. Trends in oral cavity, pharyngeal, oesophageal and gastric cancer mortality rates in Spain, 1952-2006: an age-period-cohort analysis. *BMC Cancer* 2014;14:254.
72. Adair T, Hoy D, Dettrick Z, Lopez AD. Trends in oral, pharyngeal and oesophageal cancer mortality in Australia: the comparative importance of tobacco, alcohol and other risk factors. *Aust N Z J Public Health* 2011;35(3):212-9.
73. Samet JM. The 1990 Report of the Surgeon General: The Health Benefits of Smoking Cessation. *Am Rev Respir Dis* 1990;142(5):993-4.
74. Warnakulasuriya S, Dietrich T, Bornstein MM, Casals Peidro E, Preshaw PM, Walter C, et al. Oral health risks of tobacco use and effects of cessation. *Int Dent J* 2010;60(1):7-30.
75. Gupta PC, Murti PR, Bhonsle RB, Mehta FS, Pindborg JJ. Effect of cessation of tobacco use on the incidence of oral mucosal lesions in a 10-yr follow-up study of 12,212 users. *Oral Dis* 1995;1(1):54-8.
76. Do LG, Spencer AJ, Dost F, Farah CS. Oral mucosal lesions: findings from the Australian National Survey of Adult Oral Health. *Aust Dent J* 2014;59(1):114-20.
77. Pihlstrom BL, Michalowicz BS, Johnson NW. Periodontal diseases. *Lancet* 2005;366(9499):1809-20.

78. Ramirez JH, Arce R, Contreras A. Why must physicians know about oral diseases? *Teach Learn Med* 2010;22(2):148-55.
79. Humphrey LL, Fu R, Buckley DI, Freeman M, Helfand M. Periodontal disease and coronary heart disease incidence: a systematic review and meta-analysis. *J Gen Intern Med* 2008;23(12):2079-86.
80. Sfyroeras GS, Roussas N, Saleptsis VG, Argyriou C, Giannoukas AD. Association between periodontal disease and stroke. *J Vasc Surg* 2012;55(4):1178-84.
81. Bergstrom J. Tobacco smoking and chronic destructive periodontal disease. *Odontology* 2004;92(1):1-8.
82. Johnson GK, Hill M. Cigarette smoking and the periodontal patient. *J Periodontol* 2004;75(2):196-209.
83. Tomar SL, Asma S. Smoking-attributable periodontitis in the United States: findings from NHANES III. National Health and Nutrition Examination Survey. *J Periodontol* 2000;71(5):743-51.
84. Mai X, Wactawski-Wende J, Hovey KM, LaMonte MJ, Chen C, Tezal M, et al. Associations between smoking and tooth loss according to the reason for tooth loss: the Buffalo OsteoPerio Study. *J Am Dent Assoc* 2013;144(3):252-65.
85. Mokeem SA, Vellappally S, Preethanath RS, Hashem MI, Al-Kheraif AA, Anil S. Influence of smoking on clinical parameters and gingival crevicular fluid volume in patients with chronic periodontitis. *Oral Health Dent Manag* 2014;13(2):469-73.
86. Martinez-Canut P, Lorca A, Magan R. Smoking and periodontal disease severity. *J Clin Periodontol* 1995;22(10):743-9.
87. Calsina G, Ramon JM, Echeverria JJ. Effects of smoking on periodontal tissues. *J Clin Periodontol* 2002;29(8):771-6.
88. Morse DE, Avlund K, Christensen LB, Fiehn NE, Molbo D, Holmstrup P, et al. Smoking and drinking as risk indicators for tooth loss in middle-aged Danes. *J Aging Health* 2014;26(1):54-71.
89. Buchwald S, Kocher T, Biffar R, Harb A, Holtfreter B, Meisel P. Tooth loss and periodontitis by socio-economic status and inflammation in a longitudinal population-based study. *J Clin Periodontol* 2013;40(3):203-11.
90. Albandar JM, Streckfus CF, Adesanya MR, Winn DM. Cigar, pipe, and cigarette smoking as risk factors for periodontal disease and tooth loss. *J Periodontol* 2000;71(12):1874-81.
91. Rosa EF, Corraini P, Inoue G, Gomes EF, Guglielmetti MR, Sanda SR, et al. Effect Of Smoking Cessation On Non-Surgical Periodontal Therapy: Results After 24 Months. *J Clin Periodontol* 2014.
92. Beck JD, Sharp T, Koch GG, Offenbacher S. A 5-year study of attachment loss and tooth loss in community-dwelling older adults. *J Periodontol Res* 1997;32(6):516-23.
93. Dietrich T, Maserejian NN, Joshupura KJ, Krall EA, Garcia RI. Tobacco use and incidence of tooth loss among US male health professionals. *J Dent Res* 2007;86(4):373-7.
94. Arora M, Schwarz E, Sivaneswaran S, Banks E. Cigarette smoking and tooth loss in a cohort of older Australians: the 45 and up study. *J Am Dent Assoc* 2010;141(10):1242-9.
95. Featherstone JD. Prevention and reversal of dental caries: role of low level fluoride. *Community Dent Oral Epidemiol* 1999;27(1):31-40.
96. Du Y, Fryzek J, Sekeres MA, Taioli E. Smoking and alcohol intake as risk factors for myelodysplastic syndromes (MDS). *Leuk Res* 2010;34(1):1-5.
97. Dye BA, Fisher MA, Yellowitz JA, Fryar CD, Vargas CM. Receipt of dental care, dental status and workforce in U.S. nursing homes: 1997 National Nursing Home Survey. *Spec Care Dentist* 2007;27(5):177-86.
98. Iida H, Kumar JV, Kopycka-Kedzierawski DT, Billings RJ. Effect of tobacco smoke on the oral health of U.S. women of childbearing age. *J Public Health Dent* 2009;69(4):231-41.

99. Ojima M, Hanioka T, Tanaka K, Aoyama H. Cigarette smoking and tooth loss experience among young adults: a national record linkage study. *BMC Public Health* 2007;7:313.
100. Skudutyte-Rysstad R, Sandvik L, Aleksejuniene J, Eriksen HM. Dental health and disease determinants among 35-year-olds in Oslo, Norway. *Acta Odontol Scand* 2009;67(1):50-6.
101. Bruno-Ambrosius K, Swanholm G, Twetman S. Eating habits, smoking and toothbrushing in relation to dental caries: a 3-year study in Swedish female teenagers. *Int J Paediatr Dent* 2005;15(3):190-6.
102. Ditmyer M, Dounis G, Mobley C, Schwarz E. A case-control study of determinants for high and low dental caries prevalence in Nevada youth. *BMC Oral Health* 2010;10:24.
103. Axelsson P, Paulander J, Lindhe J. Relationship between smoking and dental status in 35-, 50-, 65-, and 75-year-old individuals. *J Clin Periodontol* 1998;25(4):297-305.
104. Zitterbart PA, Matranga LF, Christen AG, Park KK, Potter RH. Association between cigarette smoking and the prevalence of dental caries in adult males. *Gen Dent* 1990;38(6):426-31.
105. Csikar J, Wyborn C, Dyer T, Godson J, Marshman Z. The self-reported oral health status and dental attendance of smokers and non-smokers. *Community Dent Health* 2013;30(1):26-9.
106. Bernabe E, Delgado-Angulo EK, Vehkalahti MM, Aromaa A, Suominen AL. Daily smoking and 4-year caries increment in Finnish adults. *Community Dent Oral Epidemiol* 2014;42(5):428-34.
107. National Health Service. Macular Degeneration [Internet] 2013. Available at: <http://www.nhs.uk/conditions/Macular-degeneration/Pages/Introduction.aspx>, [Accessed October 17 2014]
108. Chakravarthy U, Wong TY, Fletcher A, Piau E, Evans C, Zlateva G, et al. Clinical risk factors for age-related macular degeneration: a systematic review and meta-analysis. *BMC Ophthalmol* 2010;10:31.
109. Mitchell P, Wang JJ, Smith W, Leeder SR. Smoking and the 5-year incidence of age-related maculopathy: the Blue Mountains Eye Study. *Arch Ophthalmol* 2002;120(10):1357-63.
110. Myers CE, Klein BE, Gangnon R, Sivakumaran TA, Iyengar SK, Klein R. Cigarette smoking and the natural history of age-related macular degeneration: the beaver dam eye study. *Ophthalmology* 2014;121(10):1949-55.
111. Thornton J, Edwards R, Mitchell P, Harrison RA, Buchan I, Kelly SP. Smoking and age-related macular degeneration: a review of association. *Eye (Lond)* 2005;19(9):935-44.
112. Zerbib J, Delcourt C, Puche N, Querques G, Cohen SY, Sahel J, et al. Risk factors for exudative age-related macular degeneration in a large French case-control study. *Graefes Arch Clin Exp Ophthalmol* 2014;52(6):899-907.
113. Evans JR, Fletcher AE, Wormald RP. 28,000 Cases of age related macular degeneration causing visual loss in people aged 75 years and above in the United Kingdom may be attributable to smoking. *Br J Ophthalmol* 2005;89(5):550-3.
114. National Health Service. Age-related cataracts. 2014. Available at: <http://www.nhs.uk/Conditions/Cataracts-age-related/Pages/Introduction.aspx>. [Accessed October 17 2014]
115. Ye J, He J, Wang C, Wu H, Shi X, Zhang H, et al. Smoking and risk of age-related cataract: a meta-analysis. *Invest Ophthalmol Vis Sci* 2012;53(7):3885-95.
116. Richter GM, Torres M, Choudhury F, Azen SP, Varma R. Risk factors for cortical, nuclear, posterior subcapsular, and mixed lens opacities: the Los Angeles Latino Eye Study. *Ophthalmology* 2012;119(3):547-54.

117. Lindblad BE, Hakansson N, Wolk A. Smoking cessation and the risk of cataract: a prospective cohort study of cataract extraction among men. *JAMA Ophthalmol* 2014;132(3):253-7.
118. Weintraub JM, Willett WC, Rosner B, Colditz GA, Seddon JM, Hankinson SE. Smoking cessation and risk of cataract extraction among US women and men. *Am J Epidemiol* 2002;155(1):72-9.
119. Lindblad BE, Hakansson N, Svensson H, Philipson B, Wolk A. Intensity of smoking and smoking cessation in relation to risk of cataract extraction: a prospective study of women. *Am J Epidemiol* 2005;162(1):73-9.
120. Christen WG, Glynn RJ, Ajani UA, Schaumberg DA, Buring JE, Hennekens CH, et al. Smoking cessation and risk of age-related cataract in men. *JAMA* 2000;284(6):713-6.
121. National Health Service. Glaucoma. 2014. Available at: <http://www.nhs.uk/conditions/glaucoma/pages/introduction.aspx>. [Accessed October 17 2014]
122. Klein BE, Klein R, Ritter LL. Relationship of drinking alcohol and smoking to prevalence of open-angle glaucoma. The Beaver Dam Eye Study. *Ophthalmology* 1993;100(11):1609-13.
123. Leske MC, Connell AM, Wu SY, Hyman LG, Schachat AP. Risk factors for open-angle glaucoma. The Barbados Eye Study. *Arch Ophthalmol* 1995;113(7):918-24.
124. Ponte F, Giuffre G, Giammanco R, Dardanoni G. Risk factors of ocular hypertension and glaucoma. The Casteldaccia Eye Study. *Doc Ophthalmol* 1994;85(3):203-10.
125. Wang D, Huang Y, Huang C, Wu P, Lin J, Zheng Y, et al. Association analysis of cigarette smoking with onset of primary open-angle glaucoma and glaucoma-related biometric parameters. *BMC Ophthalmol* 2012;12:59.
126. Kolar P. Risk factors for central and branch retinal vein occlusion: a meta-analysis of published clinical data. *J Ophthalmol* 2014;2014:724780.
127. Ali A, Ku JH, Suhler EB, Choi D, Rosenbaum JT. The course of retinal vasculitis. *Br J Ophthalmol* 2014;98(6):785-9.
128. Yanagi M, Misumi M, Kawasaki R, Takahashi I, Itakura K, Fujiwara S, et al. Is the association between smoking and the retinal venular diameter reversible following smoking cessation? *Invest Ophthalmol Vis Sci* 2014;55(1):405-11.