

# Ageing, dental caries and periodontal diseases

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## Abstract

**Aim:** To review the burden of caries and periodontitis in the elderly, changes with age that can explain this burden, and the vulnerability to disease of elderly populations.

**Methods:** An assessment of surveys in two populations was conducted. Indicators for caries were identified by updating a systematic review. Secular trends for smoking and type 2 diabetes were discussed.

**Results:** Changes in the susceptibility to periodontitis with age may be explained by exposure to pro-inflammatory conditions and changes in the healing capacity of cells and tissues. Due to accumulated periodontal destruction, the number of surfaces at risk for caries increases. The sequels of restorative treatment contribute to an increased susceptibility for caries development. Population-based surveys in the United States and Germany demonstrate a high caries experience among elderly people. A comparison of surveys demonstrates a relative improvement of periodontal health among elderly during the last few decades. Nevertheless, prevalence estimates for periodontitis remain high. Risk indicators for root caries include caries experience, the number of surfaces at risk and poor oral hygiene. Secular trends of main risk factors for periodontitis and their likely influence on the future periodontitis burden in the elderly are discussed.

**Conclusion:** Caries and periodontitis burden in the elderly remain high.

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The connection between disease and age is complex. For many diseases, oral conditions included, the variation in occurrence explained by age seems larger than the variation explained by other known variables. Age, whether as a confounder or as a direct determinant of disease, is a key variable with regard to diagnostic, aetiological and intervention

research of caries and periodontitis. It involves lifetime changes in exposures to disease determinants, the cumulative sequel of various conditions along life and biological changes due to ageing that make us more susceptible to diseases and less capable of adapting to injury.

For dental caries, the concept of higher susceptibility in young age has not been confirmed. Instead, a relatively constant risk of occurrence of carious lesions has been demonstrated by long-term cohort studies, with risk being greatly modified by the available surfaces (Peres et al. 2007, 2009, Broadbent et al. 2008, 2013). The number of surfaces at risk is in turn affected by periodontal tissue loss. Moreover, the sequels

of caries, like restored teeth or surfaces, generate treatment needs life-long, accumulating in older age (Ettinger & Mulligan 1999). Further factors prone to change with age, like oral hygiene and microbiologic qualitative and quantitative changes, salivary flow and buffering capacity are associated with caries experience (Yildiz et al. 2016).

Earlier assumptions of a very high occurrence of gingivitis at an early age, a considerable prevalence reduction in the late teens and twenties and a steady increase in the occurrence, severity and extent of periodontitis in adulthood (Marshall Day et al. 1955, Ramfjord 1968) have long been abandoned. Similarly, the earlier idea that periodontitis is to a

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great extent a consequence of ageing and poor oral hygiene (Russell 1963) is clearly not commensurate with current understanding on the aetiology of periodontitis. While the cumulative nature of tissue destruction in caries and periodontitis results in higher severity and extent at older ages, it is now clear that other exposures play a critical role in the most severe and generalized cases of dental disease in adulthood (Hujoel et al. 2003, Chavarry et al. 2009).

While the available epidemiological evidence confirms that the occurrence and severity of periodontal destruction increases with age (Baelum et al. 1997a,b, Beck et al. 1997), only a relatively small portion of people experience severe and extensive periodontal destruction (Baelum et al. 1997a). Studies conducted in populations without or with very limited access to dental treatment demonstrate that a considerably large proportion of surfaces (Baelum 1987) or people (Baelum et al. 1997a) only experience mild loss of periodontal attachment even among the oldest study participants in these studies. Recent results on the global burden of severe periodontitis suggest that a sharp increase in the prevalence of severe periodontitis can be expected between the third and fourth decades of life and that this prevalence can remain relatively stable at older ages (Kassebaum et al. 2014).

While the role of age on caries and periodontitis has been historically attributed to accumulated exposure rather than to a biological effect of ageing on the susceptibility to disease, the results of recent studies show that susceptibility to both diseases may also change with ageing (Hajishengalis 2010, Yildiz et al. 2016).

In order to get insight into the relationships between caries, periodontitis and ageing, we need to examine the burden of these conditions in the elderly today and address changes with age that can explain this burden, and the relative increased vulnerability to disease of elderly populations.

#### **Burden of Dental Caries and Periodontitis in the Elderly**

We will describe trends in the burden of both diseases for older people

using population-based surveys with data from the United States and Germany as examples.

For dental caries, national data from the United States recorded by National Health and Nutrition Examination Surveys (NHANES) between 1999 and 2004 can be used for evaluation. To detect changes in oral health condition in the USA, these data have been compared with data from 1988 to 1994 (Dye et al. 2007). More recent NHANES data from 2010 have been published in parts, but not full detail, and cannot be used for full analysis at present. In 2004, caries experience (i.e. decayed, missing or filled teeth – DMFT) was high (17.96) but had been decreasing (–1.16) over the last decades (Dye et al. 2007). Untreated dental caries was only a small fraction of caries experience (0.43), indicating a high restorative index in the elderly. Around half of all caries experience corresponded to missing teeth (MT 8.81). It is relevant to note that the decrease in DMFT may be attributed to a decrease in missing teeth while the number of untreated or filled teeth had not decreased but increased (+0.05) in NHANES 1999–2004. When comparing different age groups, DT and MT were higher in those 75 years or above (DT 0.47 and MT 9.41) compared with those 65–74 years (0.39, 8.32), while FT was lower in the older population (8.42 *versus* 8.96), indicating possible differences in disease patterns and/or dental service provision in younger (65–74 years) and older elderly (75 years or above). Caries experience differed between ethnicities, with higher DMFT in white Americans, mainly due to a significantly higher FT. It is noteworthy that in contrast, Black or Mexican Americans showed a significantly higher MT. While the DMFT was only limitedly graded along different socio-economic groups (as defined by income), the DT and MT component increased with decreasing income, while the FT component decreased. The same applied to educational status. While the caries decline between 1994 and 2004 was shared by nearly all demographic or socio-economic groups of elderly, the DFT component decreased mainly in populations with low income and increased in those

aged 75 years or older. Data from the most recent NHANES 2012 confirmed the decreasing caries experience as well as the high restorative index (Dye et al. 2015). In summary, DMFT in elderly Americans remains high, but has been moderately decreasing between 1994 and 2004, mainly as fewer teeth are missing, while the number of DFT has increased.

In Germany, data from the Fifth German Oral Health Survey can be used (Jordan & Micheelis 2015). Caries experience in the elderly aged 65–74 years (recorded in 2014) (Schiffner 2015) was as high (DMFT 17.7) as in the United States, with a higher number of missing teeth (MT 11.1). The restorative index was high (DT was only 0.5). Again, caries experience was graded along socio-economic groups, with MT (and as a result, DMFT) decreasing with increasing social status, while FT increased. As in the United States, caries experience decreased between 1997 and 2014 (–5.9), largely due to decreasing MT; FT had increased. When separately analysing data for the very old (75 years or above), caries experience was very high (DMFT 21.6), mainly due to a high number of missing teeth (17.8). The caries experience was graded along socio-economic groups (22.2 in those with low status and 18.8 in those with high status), largely as the number of missing teeth was severely graded (MT was 19.0 and 13.1 in those with low and high status). Again, only very few teeth had untreated decay (0.6). If we analyse separately data for root caries, which could be assumed to occur mainly in adults and/or elderly, root caries prevalence was high in those aged 65–74 years (28%) but largely stable when compared with 1997 (29.9%). Prevalence was higher for patients with high social status, defined via education (29%) compared with those with low social status (25.5%) mainly as more surfaces at risk were present (as indicated by a reverse gradient in the root caries index, i.e. the number of carious or filled per all root surfaces). Similar numbers were reported for the very old (75 years or above) (Schiffner 2015): prevalence was 26%, and again higher in individual with high socio-economic status due to a larger number of

teeth being present. A very relevant finding from the latest German survey (Nitschke & Micheelis 2015) was that those subjects receiving support for care ("Pfleigestufe", i.e. governmentally funded assistance) showed significantly higher caries experience (+2.9 teeth), mainly due to more untreated carious teeth, as well as more root caries per available surface (root caries prevalence was lower due to fewer teeth being present compared to those without such assistance). In summary, German data confirm the trends and gradients observed in the United States. Root caries prevalence seems to be largely dependent on the available number of surfaces at risk, which needs to be considered when discussing an ever increasing number of retained teeth.

With corresponding reservations due to differences in the sampling strategy and examinations used in the surveys considered, a comparison of periodontal health for seniors between the National Health and Nutrition Examination Surveys (NHANES) III (1989–1994) and NHANES 1999–2004 shows improvement in periodontal health for seniors 65 years of age and older (Dye et al. 2007). The prevalence of probing depth (PD), attachment loss (AL) and gingival recession decreased. The observed periodontal improvement was also apparent independently of whether cases of periodontitis were identified as having  $\geq 1$  periodontal site with  $\geq 3$  mm of attachment loss and  $\geq 4$  mm probing depth at the same site, or using the CDC-AAP definitions for periodontitis surveillance in populations (Page & Eke 2007).

While the observed mean attachment loss and mean probing depth also decreased among elderly between both examination surveys, mean gingival recession increased, both for people 65–74 years and for seniors 75 years and older (Dye et al. 2007). Previously identified gradients for exposure to smoking and social inequalities remained apparent in the second survey.

Even though the observed trends for improvement of periodontal conditions among seniors 65 years of age and older since 1988–1994 have been recently confirmed with a comparison of observations from

NHANES 2009–2010 and NHANES 2011–2012 (Eke et al. 2015), the most recent description of the burden of periodontitis among elderly people demonstrates a worryingly high burden of periodontitis, with 40.7% of people 65 years and older experiencing  $AL \geq 6$  mm and 22.7% presenting with  $PD \geq 5$  mm, respectively (Eke et al. 2015). While the total prevalence of periodontitis for people 65 years and older was estimated to be 66%, severe periodontitis defined using the CDC/AAP case definition (Eke et al. 2012b) or the EFP case definition (Tonetti & Clafey 2005) was estimated to range between 11% and 20.6%, respectively (Eke et al. 2015).

In Germany, Schutzhold et al. (2015) evaluated changes in periodontal health during the last two decades using two population-based studies conducted during 1997–2001 and 2008–2012 in north-east Germany and the German Oral Health Studies III and IV, which are population-based surveys conducted in 1997 and 2005. Their main finding for seniors 65 years and older was a significant overall decrease in the prevalence and extent of attachment loss with a relatively stable mean probing depth. The prevalence of severe periodontitis (Page & Eke 2007) for people 65 years of age and older decreased in all studies, whereas the number of retained teeth increased (Schutzhold et al. 2015).

Results of the recent German Oral Health Study V (Kocher & Hoffmann 2015), which included a comparatively smaller subgroup of older participants 85–100 years, revealed no differences in the prevalence of mild, moderate or severe periodontitis according to CDC/AAP criteria between subjects 75–84 years and subjects 85–100 years, whereas the prevalence of edentulousness was higher in the oldest group of participants, as expected. The overall prevalence of attachment loss  $\geq 5$  mm reported was significantly lower in the 5th German survey (67.4%) when compared to the 4th survey (89.1%), whereas the difference in the prevalence of attachment loss  $\geq 3$  mm between both surveys was minor (3%).

Despite the prevalence estimates for periodontitis decrease, the larger

number of teeth retained (mean 19.3) can result in a sustained need for periodontal care.

The comparison of the findings of the 4th and 5th German Oral Health studies reveals a significant fall in the prevalence of severe periodontitis (24%) according to CDC/AAP criteria among subjects 65–74 years, whereas the prevalence of moderate periodontitis has remained stable, thus resulting in a higher occurrence of healthy subjects and subjects with mild periodontitis.

### **The Complex Relationship Between Periodontal Tissue Destruction and Age**

As previously discussed, a common finding from epidemiological studies is the observation of loss of attachment associated with increasing age (Papapanou et al. 1991, Mack et al. 2004, Eke et al. 2012a, Renvert et al. 2013, Thomson et al. 2013b). Different hypotheses have been proposed to explain the relationship between periodontal tissue destruction and advancing age. The "cumulative" hypothesis indicates that increased periodontal tissue destruction can be explained by the chronic exposure to the effects of periodontitis. On the other hand, the "age-related susceptibility" hypothesis poses that advancing age increases the risk to periodontitis by a dysregulation of the immune system or "immunosenescence" (Hajishengallis 2010). Several mechanistic studies have evaluated the role of immunosenescence as a progressive change in the defensive mechanisms against pathogens. These changes may involve both innate and acquired immunity as well (Hajishengallis 2010). This is clearly an emerging field, and several immune functions seem to be altered in association with ageing (Gibson et al. 2016). However, we will not extend in this topic as it will be reviewed extensively in a related article within this series (Preshaw and Conrads, *Journal of Clinical Periodontology*, 2017).

In this section, we will consider alternative sources of periodontal tissue deterioration not related to the dysfunction of the immune system or immunosenescence. In this regard, an emerging hypothesis relates to cellular senescence. Cellular

senescence refers to a complex cellular programme characterized by an arrest in the proliferative capabilities of cells (Childs et al. 2015). This is an important factor for the development of organismal ageing (Jeyapalan & Sedivy 2008, Tchkonja et al. 2013). As mentioned before, cellular senescence is characterized by an irreversible growth arrest condition (Campisi 2001) and may be induced by diverse stimuli including telomere-dependent senescence, DNA-damaging agents like reactive oxygen species and X rays, chromatin perturbation, oncogenic stimuli among others (Campisi & d'Adda di Fagagna 2007). Senescent cells have important features that include, among several changes, the up-regulation of genes that block the cell cycle (p16 and p21) and the development of the senescence-associated secretory phenotype (SASP) (Rodier & Campisi 2011). This latter response is characterized by an extensive reprogramming of genes that code for cytokines, growth factors and matrix-degrading enzymes (Rodier & Campisi 2011). Therefore, it has been proposed that senescent cells contribute with the release of the SASP mediators that undermine tissue structure and function in aged tissues. Of note, this source of potential inflammation is independent of the presence of pathogens or invading microorganisms. These studies had led to the notion of a new concept known as “inflammaging” that depicts an inflammatory state associated with ageing not necessarily associated with infection (Freund et al. 2010).

Another potential explanation for the deterioration of periodontal tissues relates to type I collagen. This is the most abundant protein in the periodontium and maintains the attachment of teeth with the alveolar bone and soft tissues (Bartold et al. 2000). It has been observed that type I collagen decreases in the periodontium with increasing age (Ohi et al. 2006, Lim et al. 2014). This phenomenon was recently reproduced by a study that identified a progressive atrophy of the tooth supporting structures associated with ageing in rats (Leong et al. 2012). Another non-immune source of periodontal tissue deterioration associated with ageing relates to deficiencies in the

wound healing response. In fact, distinct studies have identified important deficiencies in the wound healing response in aged periodontal tissues (Benatti et al. 2006, Guo & Dipietro 2010, Caceres et al. 2014). Therefore, it is possible that an inadequate wound healing response may not restore the damaged tissues necessary to compensate the continuous exposure to the inflammatory insult. Future studies should confront these hypotheses using different experimental approaches. We may conclude that tissue destruction and loss of periodontal attachment in aged individuals may have different origins that include the progression of periodontitis and the atrophy of the periodontal tissues due to ageing. These effects may leave extensive root surfaces exposed to the dental biofilms increasing the risk of root caries in elderly subjects.

#### Indicators for (Root) Caries in the Elderly

As discussed, it can be assumed that the risk of experiencing dental caries seems to be relatively constant within specific trajectories, with these trajectories being characterized by various life course parameters including elements of socio-economic status and oral hygiene (Broadbent et al. 2006, 2008, 2013, Peres et al. 2011, Crocombe et al. 2012, Thomson et al. 2013a). Nevertheless, this risk seems to be changing in higher age, with a number of age-related factors being possible contributors

to this change. In the following section, we aim to systematically assess which parameters are associated with dental caries in the elderly. To do so, we focused on risk indicators for root caries development, as we assume root caries to occur mainly in adults.

We first performed a rapid search of reviews, including systematic reviews which had assessed risk indicators (Fig. 1). One reviewer (FS) searched one database (PubMed via Medline) for the search terms “root caries” combined with “review” (without restrictions of search terms) via the Boolean operator AND, yielded 398 entries. We aimed to include systematic reviews, which assessed risk indicators for root caries in adult and/or elderly populations, with no further restrictions. From identified entries, 30 were screened full text by one reviewer (FS) (Table S1), but only one systematic review identified (Ritter et al. 2010). Given this paucity of data, we decided to instead update the single identified systematic review (Ritter et al. 2010). Again, a rapid systematic review was performed. Original longitudinal observational studies identifying risk indicators of root caries incidence or increment in adults and/or elderly were included. Reviews, cross-sectional studies and studies reporting on risk indicators for overall (coronal and root) caries incidence or increment were excluded. No further restrictions were made. We applied the following search strategy: ((((((risk) OR

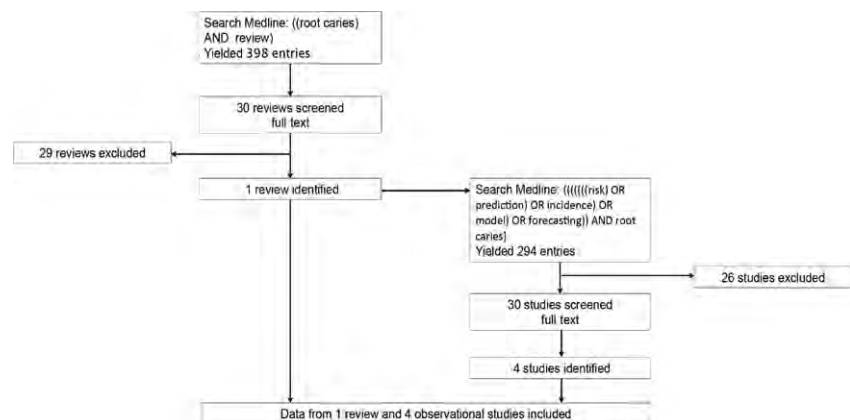


Fig. 1. Flow of search for publications on risk indicators for root caries. One review (Ritter et al. 2010) was identified by a rapid systematic search of reviews. To update this review with more recent data, a second search for observational studies was performed. Eventually, data from one review and four studies were included into our review.

prediction) OR incidence) OR model) OR forecasting)) AND root caries), limiting the publication date to 2009/01/01 to the present to identify only studies not included in the original review by Ritter et al. published in 2010. One reviewer searched PubMed, yielding 294 entries. From these, one reviewer (FS) screened 30 full texts (Table S2), with four being eventually included in the update. Two reviewers (FS, GG) extracted the following data: study author, year, setting, cohort age, sample size, follow-up period in months, root caries, incidence or increment (as given), identified risk indicators. Only risk indicators which were significantly associated with incidence or increment when applying a multivariable regression analysis were included. Indicators were counted, but no further quantitative synthesis applied. The updated review included 18 studies (Table 1).

Overall, 5361 patients had been followed (21–961 per study), over a mean period of 68 months (range: 12–456 months). The majority of studies had assessed patients aged 60 years or above. Root caries incidence ranged between 12% and 77%. A large number (22) of possible risk indicators had been assessed (Table 2). Of these, root caries incidence or increment was most often found to be associated with past root caries experience (Table 2), followed by the number of surfaces at risk, poor oral hygiene, gender (while it remained ambiguous which gender was at higher risk), age and patients having periodontal disease. As the threshold values for risk indicators varied within studies and different analytic approaches were used to assess the effect size of these variables, no meta-analysis could be performed.

### Indicators for Periodontitis in the Elderly

Exposures to main factors known to be positively associated with risk of periodontitis vary in a lifetime. In order to better comprehend periodontitis in an ageing population with higher tooth retention, it is important to understand the burden of these factors and identify eventual secular trends for these exposures.

Table 1. Included studies reporting on risk indicators for root caries incidence or increment ( $N$  = sample size)

Study	Place	Age (years)	$N$	Follow-up (month)	Incidence or increment (%)
Powell et al. (1991)	n/a	≥65	21	12	62
Ravald & Birkhed (1992)	Linköping, Sweden	Mean 51	99	24	50
Joshi et al. (1993)	Boston, USA	≥45	130	16	51
Ravald et al. (1993)	Linköping, Sweden	≥46	27	144	89
Scheinin et al. (1994)	Turku, Finland	Mean 62	96	36	51
Lawrence et al. (1996)	North Carolina, USA	≥65	352	36	n/a
Locker (1996)	Ontario, Canada	≥50	493	36	27
Powell et al. (1998)	Seattle, USA	≥60	256	36	77
Narhi et al. (1999)	Helsinki, Finland	Mean 78	110	60	n/a
Gilbert et al. (2001)	Florida, USA	≥45	723	24	36
Chalmers et al. (2002)	Adelaide, Australia	<79	216	12	n/a
Takano et al. (2003)	Niigata, Japan	72	373	24	36
Fure (2004)	Gothenburg, Sweden	55–75	102	120	12
Phelan et al. (2004)	Various US cities, USA	18–64	308	60	n/a
Yoshihara et al. (2009)	Niigata, Japan	70	261	72	n/a
Sanchez-Garcia et al. (2011)	Mexico city, Mexico	≥60	698	12	44
Thomson et al. (2013a)	Dunedin, New Zealand	38	961	456	23
Ritter et al. (2016)	North Carolina, USA	21–80	155	36	1.2 (range 0–17) surfaces

We selected tobacco smoking and type 2 diabetes because they have been consistently documented to be associated with increased risk of experiencing periodontitis in prospective longitudinal studies conducted in different study populations and because both tobacco smoking and type 2 diabetes can be considered modifiable risk factors.

### Smoking

Tobacco smoking is a leading cause of periodontitis and many other conditions. The prevalence of smoking among subjects 15 years and older has significantly decreased in most countries during the last decade and current projections indicate that further decreases can be expected for 2025 for most countries in almost all regions (Bilano et al. 2015). While these smoking prevalence declines are promising, significant region disparities still exist with particularly low-income and middle-income countries being at risk of worsening smoking tobacco epidemics if current trends do not change (Bilano et al. 2015). Current predictions suggest that at least 1.1 billion people

will be “current tobacco smokers” in 2025 and a rapid increase in smoking exposure is probable in regions like Africa and the eastern Mediterranean (Bilano et al. 2015).

Unfortunately, available literature on smoking exposure among elderly people reveals lack of standardized age categorization across surveys and for many regions only scant information on smoking exposure is available and usually dates back 10 years or more. A recent study including data from 17 European countries revealed an overall smoking prevalence estimate of 13.4% among Europeans aged 65–74 years, which was significantly higher than for people aged ≥ 75 years (8.2%). This study revealed considerable gender and social differences in the distribution of smoking among elderly Europeans with usually lowest prevalence estimates for women and people with a high level of education (Lugo et al. 2013). Very similar findings have been reported for elderly people in several Asian countries (Kim et al. 2013, Yang et al. 2015, Lim et al. 2016), whereas prevalence estimates for persons aged ≥65 years in the United States

Table 2. Number of studies reporting a significant association between root caries incidence or increment and different risk indicators

Risk indicator	References
Root caries experience	Powell et al. (1991), Ravald & Birkhed (1992), Joshi et al. (1993), Scheinin et al. (1994), Lawrence et al. (1996), Locker (1996), Gilbert et al. (2001), Takano et al. (2003), Sanchez-Garcia et al. (2011), Ritter et al. (2016)
Number of surfaces at risk	Joshi et al. (1993), Gilbert et al. (2001), Phelan et al. (2004), Fure (2004), Sanchez-Garcia et al. (2011), Thomson et al. (2013a), Ritter et al. (2016)
Oral hygiene indicators or behaviour	Ravald & Birkhed (1992), Joshi et al. (1993), Scheinin et al. (1994), Takano et al. (2003), Sanchez-Garcia et al. (2011), Thomson et al. (2013a)
Gender	Powell et al. (1991), Chalmers et al. (2002), Yoshihara et al. (2009), Thomson et al. (2013a)
Higher age	Ravald et al. (1993), Phelan et al. (2004)
Periodontal disease	Ravald et al. (1993), Gilbert et al. (2001), Takano et al. (2003)
Bacteria counts	Lawrence et al. (1996), Powell et al. (1998)
Lactobacilli numbers	Scheinin et al. (1994), Fure (2004)
Systemic conditions	Chalmers et al. (2002), Sanchez-Garcia et al. (2011)
Restorations present	Gilbert et al. (2001), Takano et al. (2003)
Ethnicity	Lawrence et al. (1996), Powell et al. (1998)
Xerostomia	Phelan et al. (2004)
Coronal caries experience	Thomson et al. (2013a)
Candida numbers	Scheinin et al. (1994)
Mutans streptococci numbers	Sanchez-Garcia et al. (2011)
Smoker	Phelan et al. (2004), Sanchez-Garcia et al. (2011)
Milk consumption	Yoshihara et al. (2009)
Low socio-economic status	Thomson et al. (2013a)
Irregular dental visiting pattern	Locker (1996)
Being underweight	Yoshihara et al. (2009)
Number of consumed drugs	Fure (2004)
Perceived impact on appearance	Lawrence et al. (1996)

are somehow lower (8.5%) (Jamal et al. 2015, Hu et al. 2016). Gender and social differences are apparent across all these studies (Hu et al. 2016). Findings from a recent study addressing changes in smoking exposure among elderly Chinese between 2001 and 2010 (Yang et al. 2015) suggest that the decrease in the tobacco smoking exposure for the overall population (Bilano et al. 2015) can also be found in some elderly populations.

#### Type 2 diabetes

Improved living conditions and access to health care have increased well-being and life expectancy, from about 50 years in the early 1900s to over 80 years today. Nevertheless, ageing is a major determinant for incapacitating and life-threatening diseases, including type 2 diabetes and cardiovascular disease, several of which are consequently increasing in occurrence in developed ageing populations (Curtis et al. 2005, Niccoli & Partridge 2012).

With ageing, most people experience a fall in the ratio of lean mass to fat mass, particularly in muscle tissue, and a consecutive redistribution of fat that can result in

decreased insulin sensitivity (Bouchard et al. 1993). In many cases, this process can lead to metabolic syndrome and type 2 diabetes (Bouchard et al. 1993, Curtis et al. 2005, Niccoli & Partridge 2012), which then can increase the risk of experiencing or worsening periodontitis (Chavarry et al. 2009). Results from longitudinal studies show that the degree of obesity and the localization of fat contribute to an increased risk for experiencing type 2 diabetes (Ohlson et al. 1985). Interestingly, hyperinsulinemia and insulin resistance are rare findings in people living up to or beyond 100 years, and their insulin resistance is remarkably low compared with that of healthy younger adults (Barbieri et al. 2001). This suggests that while a small group of centenarian survivors may be less susceptible to diabetes type 2, a considerably larger group of elderly can be exposed to an increased risk of periodontal destruction associated with worsening glucose control.

Changes in the number of diabetics due to population growth and ageing, and a secular trend for diabetes prevalence rising, mainly due to changes in lifestyle and nutrition with “excess energy intake and decreased energy expenditure”

(Nguyen & El-Serag 2010), have resulted in an explosive increase, “near quadrupling”, in the number of adults with diabetes globally (N.C.D. Risk Factor Collaboration, 2016). If current trends continue, it is projected that the number of diabetic adults will exceed 700 million by 2025 (N.C.D. Risk Factor Collaboration, 2016), conceivably influencing periodontal care needs among the elderly.

#### Conclusions

The oral health of the elderly has improved considerably during the last few decades, with lower prevalence of caries, periodontitis and edentulism. Nevertheless, a substantial burden of caries and periodontitis is still apparent for this group. Changes in the distribution of caries and periodontitis led to a significantly higher number of retained teeth among elderly, which has resulted in an increased number of surfaces becoming exposed to root caries and in need of supportive therapy. Ongoing secular trends in main determinants of caries and periodontitis in elderly populations, and changes in the susceptibility to caries and periodontitis due to

ageing warrant effective strategies for disease control among the elderly.

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## Supporting Information

Additional Supporting Information may be found in the online version of this article:

**Table S1.** Studies identified in systematic review of reviews on root caries, with reasons for exclusion.

**Table S2.** Studies identified during update of systematic review on root caries, with reasons for exclusion.

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## Clinical Relevance

*Principal findings:* Even though the oral health of the elderly has improved during the last few

decades, a considerable burden of caries and periodontitis is still evident for this group.

*Practical implications:* Seniors 65 years of age and older still experience a considerable burden of caries and periodontitis.