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ABSTRACT

The long-term effects of poor maternal oral health are unknown. We determined whether maternal oral health when children were young was a risk indicator for caries experience in adulthood, using oral examination and interview data from age-5 and age-32 assessments in the Dunedin Study, and maternal self-rated oral health data from the age-5 assessment. The main outcome measure was probands' caries status at age 32. Analyses involved 835 individuals (82.3% of the surviving cohort) dentally examined at both ages, whose mothers were interviewed at the age-5 assessment. There was a consistent gradient in age-32 caries experience across the categories of maternal self-rated oral health status (from the age-5 assessment): it was greatest among the probands whose mothers rated their oral health as "poor" or who were edentulous, and lowest among those whose mothers rated their oral health as "excellent". Unfavorable maternal self-rated oral health when children are young should be regarded as a risk indicator for poor oral health among offspring as they reach adulthood.

KEY WORDS: oral health, intergenerational, life-course, risk, family history.

Maternal Oral Health Predicts Their Children's Caries Experience in Adulthood

INTRODUCTION

Severe dental caries experience is now concentrated among a relatively small group whose identification for early preventive intervention is desirable. Currently, inexpensive public health efforts (those not requiring an intra-oral examination or dental personnel) to identify those at highest risk are composed of simple questions about potential risk factors, such as past caries experience, fluoride exposure, and dietary sugar intake. Caries experience, in common with virtually all traits, is likely to be the result of several forms of gene-environment interplay. In this context, it has been suggested that family history reflects the outcome of shared genetic variations and shared non-genetic factors (environmental factors, exposures, and common behaviors) (Khouri, 2003).

In other words, the health of one generation might profoundly affect that of the next (Meigs *et al.*, 2000; Sesso *et al.*, 2001; Jonsson *et al.*, 2004; Lloyd-Jones *et al.*, 2004; Reilly *et al.*, 2005; Harding *et al.*, 2006). In support, there is evidence suggesting that maternal health may have a strong influence on disease risk in offspring (Karter *et al.*, 1999; Meigs *et al.*, 2000; Sesso *et al.*, 2001; Reilly *et al.*, 2005).

Is maternal history a risk indicator for oral disease as well? There are intergenerational processes that can link maternal oral health (and maternal oral health beliefs, attitudes, and behaviors) to oral health and disease risk in offspring (Grytten *et al.*, 1988; Sasahara *et al.*, 1998; Mattila *et al.*, 2000; Bedos *et al.*, 2005; Li *et al.*, 2005; Alm *et al.*, 2008; Saied-Moallemi *et al.*, 2008; Shearer and Thomson, 2010; Weintraub *et al.*, 2010). However, investigations to date have almost exclusively concentrated on oral health outcomes measured in childhood or adolescence rather than in adulthood. Furthermore, some studies are cross-sectional (and so do not offer strong evidence), and many have not controlled confounding. Others have suffered from methodological problems (selection bias, attrition, or insufficient statistical power). Whether the association between maternal and child oral health persists into adulthood is currently unknown. Thus, the current study sought to determine whether the oral health of mothers of young children can predict the caries experience of those same children when they reach adulthood.

METHODS

This study used oral examination and interview data collected from participants ("probands") during the age-32 assessment of the Dunedin Multidisciplinary Health and Development Study (DMHDS), and interview data obtained from their mothers at the age-5 assessment (in 1977/78). The DMHDS is a prospective cohort study of 1037 children born at Queen Mary Hospital, Dunedin, New Zealand, between 1 April 1972 and 31 March 1973. These 1037 children represent 91% of the 1139 eligible children born

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between these dates, and 972 (96% of the surviving 1015) were assessed at age 32. Ethics approval was granted by the Otago Research Ethics Committee; participants gave informed consent.

Some 922 children (88.9%) were orally examined at age 5. The accompanying parent (919 mothers, three fathers) was asked to self-report about dentate status and (if dentate) to rate her/his own oral health (responses: "excellent", "fairly good", "average", "fairly poor", or "very poor"). The three fathers were excluded from further analysis, as were six mothers who rated their oral health as "don't know". The accompanying parent did not have a clinical oral examination at this time.

Calibrated examiners carried out dental examinations for caries and missing teeth on 932 of the 972 probands who attended the age-32 assessment (conducted November 2003-June 2005). They estimated accumulated tooth loss by recording the presence or absence of each tooth (excluding third molars), and noting the reason for its absence. Tooth surfaces were examined for caries and restorations. They used longitudinal caries experience data to identify 3 distinct trajectories of dental caries experience from ages 5 to 32 (high, moderate, and low), using a group-based trajectory analysis model (Broadbent *et al.*, 2008). A similar approach determined long-term plaque exposure (Broadbent *et al.*, 2010). The simplified oral hygiene index (OHI-S) was used to quantify plaque accumulation on 6 index teeth (Greene and Vermillion, 1964). OHI-S data from ages 5, 9, 15, 18, 26, and 32 were then used to identify three distinct 'plaque groups': low plaque (group mean OHI-S = 0.59, $N = 320$, 38.6% of this sample); moderate plaque (mean = 0.93, $N = 415$, 50.1%); and high plaque (mean = 1.45, $N = 93$, 11.2%) (Broadbent *et al.*, 2010).

Socio-economic status (SES) at age 32 was determined according to standard NZ indices which apply a six-interval classification according to occupation; for example, a doctor scores 1 and a laborer scores 6 (Irving and Elley, 1977; Elley and Irving, 1985). Those scoring 1 or 2 were allocated to the high-SES group; those with a score of 3 or 4 were medium-SES; and those scoring 5 or 6 were low-SES. Socio-economic status from birth to 15 yrs was calculated as the average of the highest SES level of either parent, assessed at 0, 3, 5, 7, 9, 11, 13, and 15 yrs. Participants were also asked whether they were routine or episodic users of dental care.

Statistical Analysis

Proband age-32 DMFS, DS, FS, MS, and number of missing teeth were computed from age-32 clinical data. Descriptive and bivariate analyses used SPSS version 16.0 (SPSS Inc., Chicago, IL, USA). Multivariate analyses used Stata version 11.0 (StataCorp, College Station, TX, USA). Chi-square tests examined the statistical significance of associations observed between categorical variables. Mann-Whitney U tests (or Kruskal-Wallis tests where appropriate) were used for continuous dependent variables. *Post hoc* comparisons (using the Tukey test) were conducted to determine which groups differed. Statistical tests were two-tailed ($\alpha = 0.05$). Multivariate analysis used the generalized linear model (GLM)

command with modified Poisson regression analysis (using a robust error variance procedure) to estimate the relative risk of having a high caries trajectory at age 32, and to model the age-32 DMFS.

RESULTS

Of the original 1037 children, 919 (88.6%) had an oral examination at age 5, with their mothers reporting on their own oral health at this assessment, and 972 (95.8% of surviving cohort) were assessed at age 32. The bivariate analysis was limited to the 835 (82.3% of surviving cohort) study members who were dentally examined at both ages, and whose mothers had been interviewed (and self-rated their oral health or were edentulous) at the age-5 assessment. The multivariate analysis was further restricted to the 825 study members who had both a caries and a plaque trajectory. An attrition analysis found significant differences between those who were examined at both ages, and those who were examined at age 5 only (data not shown – available on request). The latter were more likely than the former to be low-SES, and to have a mother who self-rated her oral health as poor, or was edentulous, at the age-5 assessment.

Associations were found (see Table 1) between mothers being edentulous or having poor self-rated oral health status at the age-5 assessment and the proband's prevalence of severe caries (DMFS > 20), 1 or more teeth missing (due to caries), and of having a DMFS greater than the median at age 32; and of having a high caries trajectory from ages 5 to 32. Mothers who rated their oral health as poor or who were edentulous at the age-5 assessment had offspring with higher mean DMFS, DS, FS, and MS by age 32 than did mothers who did not rate their oral health as poor, and were dentate.

Unadjusted bivariate associations between probands' caries experience by age 32 and maternal self-rated oral health 27 yrs earlier revealed a gradient of higher prevalence and greater severity of caries experience in the proband at age 32 by poorer maternal self-rated oral health/edentulous status at the age-5 assessment (Table 1).

Multivariate modeling was used to determine the relative risk (RR) of having a high caries trajectory between ages 5 and 32, for each of the categories of maternal self-rated oral health at the age-5 assessment (using the "excellent" category as a referent), while controlling for sex, episodic use of dental services, SES 0-15 yrs, SES at 32, and plaque trajectory. There was a consistent gradient of RR across the categories of maternal self-rated oral health status at the age-5 assessment, greatest among the probands whose mothers rated their oral health as "fairly poor/very poor/edentulous", and lowest among those with an "excellent" rating (Table 2, Fig.).

Based on a negative binomial approach, the probands' DMFS score at age 32 was modeled for each of the five categories of maternal self-rated oral health at the age-5 assessment, while controlling for sex, episodic use of dental services, SES at 0-15 yrs, SES at 32, and plaque trajectory. There was a consistent gradient of age-32 caries experience across the categories of maternal self-rated oral health status at the age-5 assessment, whereby it was greatest among the probands whose mothers

Table 1. Proband Caries and Tooth Loss Prevalence and Severity Due to Caries at Age 32, and High Caries Trajectory between Ages 5 and 32 by Mothers' Self-rated Oral Health Status at the Age-5 Assessment

	Age 32 Caries Experience							
	Severe Caries (DMFS > 20) (%)	One or More Teeth Missing ¹ (%)	DMFS Greater than Median (%)	High Caries Trajectory ² (%)	Mean DMFS (SD)	Mean DS (SD)	Mean MS ¹ (SD)	Mean FS (SD)
Mother self-rated oral health as poor, or was edentulous 27 years earlier ³								
No (N = 593)	156 (26.3) ^a	116 (19.6) ^a	282 (47.6) ^a	63 (10.7) ^a	14.6 (12.9) ^b	2.0 (4.1) ^c	2.0 (5.5) ^b	10.5 (9.7) ^b
Yes (N = 242)	99 (40.9)	82 (33.9)	161 (66.5)	61 (25.4)	22.1 (18.1)	3.1 (5.6)	5.2 (11.2)	13.9 (11.1)
Mothers' self-rated oral health 27 years earlier								
Excellent (N = 58)	4 (6.9) ^a	8 (13.8) ^d	19 (32.8) ^a	3 (5.2) ^a	9.3 (7.2) ^{efg}	0.7 (1.2) ^e	0.9 (2.7) ^e	7.7 (6.5) ^{efg}
Fairly good (N = 215)	58 (27.0)	43 (20.0)	95 (44.2)	18 (8.4)	14.2 (13.5) ^{hi}	2.3 (4.8)	2.3 (5.6) ^f	9.5 (9.2) ^{hi}
Average (N = 320)	94 (29.4)	65 (20.3)	168 (52.5)	42 (13.2)	15.8 (13.1) ^{ejk}	2.0 (3.8) ^f	2.0 (5.8) ^g	11.7 (10.3) ^e
Fairly poor (N = 98)	34 (34.7)	25 (25.5)	61 (62.2)	22 (22.9)	20.4 (18.2) ^{hij}	2.7 (5.6)	4.2 (10.1)	13.6 (12.4) ^{hij}
Very poor or edentulous (N = 144)	65 (45.1)	57 (39.6)	100 (69.4)	39 (27.1)	23.3 (18.0) ^{gik}	3.3 (5.6) ^{ef}	5.9 (11.9) ^{efg}	14.1 (10.2) ^{gij}

¹Due to caries.²N = 830.³Dichotomous variable.^ap < 0.001; chi-square test.^bp < 0.001; Mann-Whitney U test.^cp < 0.05; Mann-Whitney U test.^dp < 0.005; chi-square test.^eKruskal-Wallis test (a trend test confirmed a gradient across the mother's self-rated oral health ordered group); estimates with different symbols are significantly different from each other (by *post hoc* criteria).

rated their oral health as "very poor/edentulous", and lowest among those whose mothers rated their oral health as "excellent" (Table 3, Fig.). The mean age-32 DMFS among those in the "excellent" group was less than half that of the "very poor/edentulous" group.

DISCUSSION

Analysis of these prospective cohort study data supports the hypothesis that maternal oral health when a child is young has an impact on that child's oral health status in adulthood. Children of mothers with poor oral health had themselves (on average) poorer oral health almost three decades later.

These findings are unprecedented. Until now, it has not been possible to examine the nature and extent of such cross-generational associations in oral health, because the requisite data have not been available. The Dunedin Study is distinct in its longevity, sample size, retention rate, oral health data (including intergenerational data), and information on a range of potential risk, ameliorating, exacerbating, and confounding factors. In addition to the unique proband data set, data were also collected from the mothers when their children were young. This provided a rare opportunity to investigate intergenerational associations in oral health, and to broaden understanding of the possible causal associations between the oral health of mothers of young children and the oral health of these children many years later.

This study had some limitations. Those examined at both ages (included in the analysis) differed in some ways from those examined only at age 5. More of the latter were low-SES and had a mother with poor oral health in 1977/78. This may have led to an under-estimation of the strength of the observed associations. Participants' dietary habits or fluoride exposure could not be controlled for. We relied on maternal self-report data at the age-5 assessment and on proband self-report data on SES, and on dental attendance pattern at the age-32 assessment. The reliability and validity of self-report data have been addressed elsewhere (Pinelli and de Castro Monteiro Loffredo, 2007). In the case of the Dunedin Study, interview/examiner-based assessments are more likely to capture valid data than "self-completed" data; participants are aware of the importance of accurate responses, and there is a long history of mutual trust and respect between participants and researchers.

The use of a birth cohort—and the high retention rate—means that the sample is representative of its source population (New Zealand's South Island). Whether the findings can be generalized to the New Zealand population, and to other populations (particularly the United States), has been addressed in earlier work (Thomson *et al.*, 2006), where it was concluded that oral health findings from the DMHDS can cautiously be generalized to these populations.

While these findings are unique, they are reasonably consistent with the limited data on adults in their fourth decade that are

Table 2. Modified Poisson Regression Model for Membership in the 'High Caries Trajectory' between Ages 5 and 32

	Relative Risk for Being in the High Caries Trajectory (95% CI) ^a	
Male	0.92	(0.76, 1.30)
Episodic user of dental services at age 32	1.46	(1.01, 2.12)
Low SES from birth to age 15	0.96	(0.52, 1.75)
Medium SES from birth to age 15	0.83	(0.50, 1.38)
Low SES at age 32	1.23	(0.72, 2.09)
Medium SES at age 32	0.93	(0.55, 1.56)
Moderate plaque trajectory at age 32	1.67	(1.09, 2.55)
High plaque trajectory at age 32	2.00	(1.15, 3.46)
Maternal self-rated oral health 27 years earlier		
Fairly good	1.50	(0.45, 5.04)
Average	2.45	(0.77, 7.80)
Fairly poor	3.93	(1.21, 12.78)
Very poor or edentulous	3.92	(1.21, 12.64)

^aReference categories: male (female, coded 0), episodic user of dental services at age 32 (routine user of dental services at age 32, coded 0), low or medium SES from birth to age 15 (high SES from birth to age 15, coded 0), low or medium SES at age 32 (high SES, coded 0), moderate or high plaque trajectory (low plaque trajectory, coded 0), maternal self-rated oral health 27 years earlier (maternal self-rated oral health as "excellent", coded 0).

available from other studies, thus increasing confidence in the validity of the longitudinal and intergenerational findings (Beltrán-Aguilar *et al.*, 2005; Hopcraft and Morgan, 2006; Slade *et al.*, 2007; Skudutyte-Rysstad *et al.*, 2009). This is true also of the findings for the mothers at the age-5 assessment (Spratley, 1978; Cutress *et al.*, 1979).

The longitudinal associations between maternal self-rated oral health at the age-5 assessment and proband caries experience at age 32 were particularly striking, with clear gradients in caries experience across the categories of maternal self-rated oral health from 27 years earlier. These were apparent not only with the cumulative exposure measure (DMFS) at age 32, but also with that representing high caries experience through the life-course (membership in the high caries trajectory). These gradients were independent of proband sex, SES, pattern of dental attendance, and plaque trajectory. The impact of maternal oral health on adult offspring is consistent with the life-course model, whereby early influences are linked to adult health (Poulton *et al.*, 2002; Kuh and Ben-Shlomo, 2004), and with the notion that adult oral health is a product of gene-environment interplay (Khoury, 2003).

Research has demonstrated associations between parents' beliefs about diet, oral hygiene, and the inevitability of oral disease and their children's oral health (Poutanen *et al.*, 2007; Alm *et al.*, 2008; Saied-Moallemi *et al.*, 2008; Skeie *et al.*, 2008; Ismail *et al.*, 2009). Our findings provide evidence of a strong association between mothers' self-rated oral health and their adult offspring's oral health many years later. Questioning mothers of young children about their own oral health is likely

Figure. Mothers' self-rated oral health status at age-5 assessment by: (a) proband relative risk (RR) of high caries trajectory between ages 5-2¹; and (b) by proband mean DMFS at age 32².

¹Adjusted – modified Poisson regression model; ²Adjusted – negative binomial regression model.

to assist in identifying those children most at risk for severe caries in adulthood.

Future research should consider how disease risk is transmitted from one generation to the next. Genetically determined traits may present in similar ways across generations. Environmental risk factors (such as SES, smoking, mothers' poor oral-health-related knowledge, attitudes, beliefs, and behaviors, early acquisition of *Streptococcus mutans*, suboptimal fluoride exposure, and episodic use of dental health services) may persist across generations. A genetic predisposition coupled with exposure to environmental risk factors forms the basis for gene-environment interplay—that is, the situation where both genetic and environmental factors interact to influence health in individuals and populations.

This study provides strong evidence that the children of mothers with poor self-rated oral health are more likely to grow up to have poorer oral health than those of mothers with good self-rated oral health. Maternal self-rated oral health when children are young appears to be a valid representation of the intricacies of the shared genetic and environmental factors that contribute to oral health throughout the life-course. Unfavorable maternal self-rated oral health should be regarded as a risk indicator for poor oral health among offspring later in adulthood. Simple questions about maternal oral health should form part of a preliminary and inexpensive assessment of a child's future oral disease risk (on both clinical and public health grounds). In addition, it is important that

Table 3. Adjusted* Estimates for Proband Mean DMFS at Age 32, by Maternal Self-rated Oral Health 27 Years Earlier (negative binomial regression model)

	Proband Mean DMFS by Age 32 (95% CI)	
Maternal self-rated oral health 27 years earlier		
Excellent	9.7	(7.7, 12.1)
Fairly good	14.0	(12.4, 15.7)
Average	15.6	(14.2, 17.1)
Fairly poor	19.2	(16.2, 22.8)
Very poor or edentulous	20.0	(17.3, 23.1)
Mother self-rated oral health as poor/ edentulous 27 years earlier ^a		
Yes	19.6	(17.5, 22.0)
No	14.4	(13.4, 15.5)

*Controlling for sex, episodic use of dental services, SES from birth to age 15, SES at age 32, and plaque trajectory.

^aDichotomous variable.

mothers are told that their oral health can have an impact on their child's oral health, and dentists should encourage mothers of young children to receive dental care.

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REFERENCES

Alm A, Wendt L, Koch G, Birkhed D (2008). Oral hygiene and parent-related factors during early childhood in relation to approximal caries at 15 years of age. *Caries Res* 42:28-36.

Bedos C, Brodeur JM, Arpin S, Nicolau B (2005). Dental caries experience: a two-generation study. *J Dent Res* 84:931-936.

Beltrán-Aguilar E, Barker L, Canto M, Dye B, Gooch B, Griffin S, *et al.* (2005). Surveillance for dental caries, dental sealants, tooth retention, edentulism, and enamel fluorosis — United States, 1988-1994 and 1999-2002: National Center for Chronic Disease Prevention and Health Promotion. *MMWR Surveill Summ* 54:1-43.

Broadbent JM, Thomson WM, Poulton R (2008). Trajectory patterns of dental caries experience in the permanent dentition to the fourth decade of life. *J Dent Res* 87:69-72.

Broadbent JM, Thomson WM, Boyens JV, Poulton R (2010). Dental plaque and oral health over the first 30 years of life. *J Am Dent Assoc* (in press).

Cutress T, Hunter P, Davis P, Beck D, Croxson L (1979). Adult oral health and attitudes to dentistry in New Zealand 1976. Wellington: Medical Research Council of New Zealand.

Elley W, Irving J (1985). The Elley-Irving socio-economic index 1981 Census revision. *NZ J Educ Stud* 20:115-128.

Greene JC, Vermillion JR (1964). The Simplified Oral Hygiene Index. *J Am Dent Assoc* 68:7-13.

Grytten J, Rossow I, Holst D, Steele L (1988). Longitudinal study of dental health behaviors and other caries predictors in early childhood. *Community Dent Oral Epidemiol* 16:356-359.

Harding AH, Griffin SJ, Wareham NJ (2006). Population impact of strategies for identifying groups at high risk of type 2 diabetes. *Prev Med* 42:364-368.

Hopcraft M, Morgan M (2006). Pattern of dental caries experience on tooth surfaces in an adult population. *Community Dent Oral Epidemiol* 34:174-183.

Irving J, Elley W (1977). A socio-economic index for the female labour force in New Zealand. *NZ J Educ Stud* 12:154-163.

Ismail A, Sohn W, Lim S, Willem JM (2009). Predictors of dental caries progression in primary teeth. *J Dent Res* 88:270-275.

Jonsson S, Thorsteinsdottir U, Gudbjartsson DF, Jonsson HH, Kristjansson K, Arnason S, *et al.* (2004). Familial risk of lung carcinoma in the Icelandic population. *J Am Med Assoc* 292:2977-2983; *erratum in J Am Med Assoc* 293:163, 2005.

Karter AJ, Rowell SE, Ackerson LM, Mitchell BD, Ferrara A, Selby JV, *et al.* (1999). Excess maternal transmission of type 2 diabetes. The Northern California Kaiser Permanente Diabetes Registry. *Diabetes Care* 22:938-943.

Khoury MJ (2003). Genetics and genomics in practice: the continuum from genetic disease to genetic information in health and disease. *Genet Med* 5:261-268.

Kuh D, Ben-Shlomo Y, editors (2004). A life course approach to chronic disease epidemiology. 2nd ed. Oxford, UK: Oxford University Press.

Li Y, Caulfield PW, Dasanayake AP, Wiener HW, Vermund SH (2005). Mode of delivery and other maternal factors influence the acquisition of *Streptococcus mutans* in infants. *J Dent Res* 84:806-811.

Lloyd-Jones DM, Nam BH, D'Agostino RB Sr, Levy D, Murabito JM, Wang TJ, *et al.* (2004). Parental cardiovascular disease as a risk factor for cardiovascular disease in middle-aged adults: a prospective study of parents and offspring. *J Am Med Assoc* 291:2204-2211.

Mattila ML, Rautava P, Sillanpää M, Paunio P (2000). Caries in five-year-old children and associations with family-related factors. *J Dent Res* 79:875-881.

Meigs JB, Cupples LA, Wilson PW (2000). Parental transmission of type 2 diabetes: the Framingham Offspring Study. *Diabetes* 49:2201-2207.

Pinelli C, de Castro Monteiro Loffredo L (2007). Reproducibility and validity of self-perceived oral health conditions. *Clin Oral Investig* 11:431-437.

Poulton R, Caspi A, Milne BJ, Thomson WM, Taylor A, Sears MR, *et al.* (2002). Association between children's experience of socioeconomic disadvantage and adult health: a life-course study. *Lancet* 360:1640-1645.

Poutanen R, Lahti S, Seppä L, Tolvanen M, Hausen H (2007). Oral health-related knowledge, attitudes, behavior, and family characteristics among Finnish schoolchildren with and without active initial caries lesions. *Acta Odontol Scand* 65:87-96.

Reilly J, Armstrong J, Dorosty A, Emmett P, Ness A, Rogers I, *et al.* (2005). Early life risk factors for obesity in childhood: cohort study. *BMJ* 330:1357.

Saied-Moallemi Z, Virtanen JI, Ghofranipour F, Murtomaa H (2008). Influence of mothers' oral health knowledge and attitudes on their children's dental health. *Eur Arch Paediatr Dent* 9:79-83.

Sasahara H, Kawamura M, Kawabata K, Iwamoto Y (1998). Relationship between mothers' gingival condition and caries experience of their 3-year-old children. *Int J Paediatr Dent* 8:261-267.

Sesso H, Lee I, Gaziano J, Rexrode K, Glynn R, Buring J (2001). Maternal and paternal history of myocardial infarction and risk of cardiovascular disease in men and women. *Circulation* 104:393-398.

- Shearer DM, Thomson WM (2010). Intergenerational continuity in oral health – a review. *Community Dent Oral Epidemiol* 38:479-486.
- Skeie MS, Espelid I, Riordan PJ, Klock KS (2008). Caries increment in children aged 3-5 years in relation to parents' dental attitudes: Oslo, Norway 2002 to 2004. *Community Dent Oral Epidemiol* 36:441-450.
- Skudutyte-Rysstad R, Sandvic J, Aleksejuniene J, Eriksen H (2009). Dental health and disease determinants among 35-year-olds in Oslo, Norway. *Acta Odontol Scand* 67:50-56.
- Slade G, Spencer AJ, Roberts-Thomson KF, editors (2007). Australia's dental generations: The Australian National Survey of Adult Oral Health 2004-2006. Canberra: Australian Institute of Health and Welfare (Dental Statistics and Research series No. 34).
- Spratley MH (1978). Supply and demand for prosthetic treatment in Victoria, Australia. *Community Dent Oral Epidemiol* 6:151-158.
- Thomson WM, Broadbent JM, Poulton R, Beck J (2006). Changes in periodontal disease experience from 26 to 32 years of age in a birth cohort. *J Periodontol* 77:947-954.
- Weintraub JA, Prakash P, Shain SG, Laccabue M, Gansky SA (2010). Mothers' caries increases odds of children's caries. *J Dent Res* 89: 954-958.