Socioeconomic position and periodontal disease: a life-course approach

by

Helena Silveira Schuch

(B.D.S, M.Sc. Dentistry)

Submitted for the degree of
Doctor of Philosophy (PhD)
Adelaide Dental School

Supervised by:

A/Prof. Loc Giang Do
Adelaide Dental School
The University of Adelaide

Prof. Marco Aurelio Peres
Adelaide Dental School
The University of Adelaide

A/Prof. Karen Glazer Peres
Adelaide Dental School
The University of Adelaide

2018

‘At the end of every scientific paper there is a familiar coda: more research is needed, more research is needed. What, I wondered, if we added a new coda: more action is needed. It need not be discordant with the first.’

Sir Michael Marmot

DEDICATION

to my family, for their unconditional love and support

my grandparents Iêda de Campos Silveira *(in memoriam)* and José Carlos da Silveira

my parents Carla de Campos Silveira and Luiz Schuch Neto

my brother Maurício Silveira Schuch

my godmother Sônia de Campos Balzano

ACKNOWLEDGEMENTS

I am grateful to my supervisors, Associate Professor Loc Do, Professor Marco Aurelio Peres and Associate Professor Karen Peres, for their advice, guidance and friendship during this journey. Their knowledge and support helped me in becoming an independent researcher and epidemiologist.

I wish to express my gratitude and friendship to the ‘ground floor group’: Ankur Singh, Adyya Gupta, Kamal Hannah, Sofia Christofis *(in memoriam)*, Dandara Haag, Ninuk Haryani, Rahul Nair, Mojdeh Sassani, Claudia Lopez, Gustavo Nascimento and Fabio Leite. I feel very lucky and grateful to have each and all of you as my friends. Sharing the days with you was always a pleasure.

To my Brazilian friends in Australia: Gabriel Borges da Silva, Dandara Haag, David Gonzales-Chica, Sabrina Porto Gonzales, Maria Alejandra and Fernando Porto Gonzales, Andrea Cabañas, Augusto Freitas, Fernanda Ewerling, Marco, Karen, Rodrigo, Rafael, Bárbara and Vicente Peres, for all the good moments we spent together and the support when needed. You've been part of my family in Australia and made the homesick days much easier.

To my partner Will Hann, thanks for all your love and care. Thanks for always motivating me to improve and to be a better version of myself.

To my lifelong friends Isadora Hallal, Raíssa Araújo, Rafaella Araújo, Alice Llanos, Caroline Sousa and to my friend and cousin Guilherme Schuch, because there is no distance to a true friendship.

I am also thankful to the Post-Graduate Programs in Dentistry and Epidemiology of the Federal University of Pelotas and to the cohort study participants, who allowed us to enter their houses and collect the data that made this thesis possible.

I wish to express my gratitude to my friends and colleagues from the EpiBucal Research Group from the Federal University of Pelotas, especially Flávio Fernando Demarco, Marcos Britto Corrêa and Marília Leão Goettems, for their friendship and for encouraging me in the research field.

I would also like to express my gratitude to all ARCPOH (Australian Research Centre for Population Oral Health) staff and students, for all the highly valuable scientific and non-scientific moments shared. Especially, thanks to Professor Lisa Jamieson and Associate Professor Jason Armfield for their collaboration and kindness.

I am grateful to Dr Yvonne Miels, who professionally edited and reviewed this thesis.

Finally, I wish to thank the Brazilian agency CAPES (Coordination for the Improvement of Higher Education Personnel) for providing me the scholarship that supported my PhD.

TABLE OF CONTENTS

ABSTRACT

Background: Periodontal disease is a chronic condition affecting more than 537 million people worldwide, with a prevalence of 7.6% for all ages combined. Although important factors associated with periodontal disease are already known, little is known about the influence of the socioeconomic position (SEP) across the life stages on periodontal disease in adults. There is an increasing understanding that the true risk factors to health inequalities lie in social, economic and political circumstances.

Thesis objectives: The overarching purpose of the thesis was to investigate the influence of SEP during the life-course on periodontal disease in adulthood. The specific objectives of the thesis were: to theoretically explore how life-course epidemiology theories can be applied to explain the relationship between SEP and periodontal disease; to systematically review all longitudinal prospective studies that studied the association between SEP and periodontal disease; to quantify the impact of life-course income trajectories on the occurrence of periodontal disease in adulthood, in the inter-relationship of income with other socioeconomic indicators; and to evaluate the direct effect of early life income on periodontal disease occurrence in adulthood that was not mediated by adulthood income and adulthood education attainment, and behavioural risk factors for periodontal disease, namely smoking status and oral hygiene.

Main findings: To address the objectives, four papers were developed. The first paper presented and discussed a theoretical basis for the use of life-course epidemiology theories, namely the critical period model, critical period with modifier effect model, accumulation of risk model and chain-of-risk model, in explaining the relationship between SEP during the life-course and periodontal disease in adulthood. Through a systematic review, it has been observed that individuals who were exposed to lower socioeconomic conditions earlier in life presented with worse periodontal disease in adulthood. This finding was consistent across the broad range of SEP indicators and measures of disease adopted in the primary studies. The first empirical study, using data from a nested oral health study of the 1982 Pelotas Birth Cohort Study in Brazil (n=539), demonstrated the influence of income trajectories during the first 30 years of life on the occurrence of moderate-to-severe periodontal disease at the age of 31 years. Finally, the findings from the last study, also using data from the same study, supported the hypothesis that early life socioeconomic position has a direct effect on periodontal disease at age 31 that is not mediated by socioeconomic position in adult life, smoking status, or oral hygiene. The findings of the thesis have presented a strong case for applying life-course epidemiological research in investigating the development of periodontal disease. The study has contributed evidence to identifying socioeconomic position as an upstream determinant of the disease since early in life.

DECLARATION

DECLARATION

I certify that this work contains no material which has been accepted for the award of any other degree or diploma in my name, in any university or tertiary institution and, to the best of my knowledge and belief, contains no material previously published or written by another person, except where due reference has been made in the text. In addition, I certify that no part of this work will, in the future, be used in a submission in my name, for any other degree or diploma in any university or other tertiary institution without the prior approval of The University of Adelaide.

I give consent to this copy of my thesis, when deposited in the University Library, being made available for loan and photocopying, subject to the provisions of the *Copyright Act 1968*.

The author acknowledges that copyright of published works contained within this thesis resides with the copyright holder(s) of those works.

I also give permission for the digital version of my thesis to be made available on the web, via the University's digital research repository, the Library Search and also through web search engines, unless permission has been granted by the University to restrict access for a period of time.

Signed _____ 3/09/07

Helena Silveira Schuch Date

RESEARCH OUTCOMES FROM WORK ARISING IN THIS THESIS

Publications

This thesis is prepared in the publication format and includes 4 manuscripts:

1. Schuch HS, Peres KG, Do LG, Peres MA. Can socioeconomic trajectories during the life influence periodontal disease occurrence in adulthood? Hypotheses from a life-course perspective. *Med Hypotheses*. 2015 Jun; 84(6):596-600.
2. Schuch HS, Peres KG, Singh A, Peres MA, Do LG. Socioeconomic position during life and periodontal disease in adulthood: a systematic review. *Community Dentistry and Oral Epidemiology* 2016 Dec 29. doi: 10.1111/cdoe.12278.
3. Schuch HS, Peres KG, Demarco FF, Horta BL, Gigante DP, Peres MA, Do LG. Effect of life course family income trajectories on periodontitis. *Journal of Clinical Periodontology* 2017 Nov 27. doi: 10.1111/jcpe.12845.
4. Schuch HS, Nascimento GG, Peres KG, Peres MA, Do LG. Does early life family income affect periodontal disease in adulthood? Findings from the 1982 Pelotas birth cohort study. To be submitted to *Journal of Dental Research.*

Awards

1. 2017 IADR Lion Dental Research Award for Junior Investigators – Behavioral, Epidemiologic and Health Services Research Group of the International Association for Dental Research. San Francisco – USA.
2. 2017 IADR BEHSR Pre-doctoral Award - Behavioral, Epidemiologic and Health Services Research Group (BEHSR), International Association for Dental Research. San Francisco – USA.
3. Healthy Development Adelaide Travel Grant, Round 2017/1. Adelaide – Australia.
4. Award of best presentation, School of Dentistry, Florey International Postgraduate Research Conference 2016. Adelaide – Australia.
5. JL Eustace International Travel Award, 2015. Adelaide – Australia.
6. Award of best presentation, Florey Foundation of Medical Research, Florey International Postgraduate Research Conference 2015. Adelaide – Australia.
7. Winifred E. Preedy Postgraduate Bursary, Australian Federation of University Women – South Australia AFUW-SA Inc. Trust Fund, 2015. Adelaide – Australia.
8. Colgate Senior Travel Award, 2014. Adelaide – Australia.

Conference Presentations

*The abstracts appear in the Appendix sections 9.1 to 9.7

1. Schuch HS, Peres KG, Demarco FF, Peres MA, Do LG Effects of income trajectories on periodontal disease: birth-cohort study findings. 95th IADR/AADR/CADR General Session & Exhibition, 2017, San Francisco – USA. (Oral presentation).
2. Schuch HS, Peres KG, Peres MA, Do LG. Modelling socioeconomic trajectories on oral health outcomes over the life course. South Australia Population Health Conference 2016. Adelaide – Australia (Oral presentation).
3. Schuch HS, Peres KG, Peres MA, Do LG. Effects of income trajectories over the life course on the occurrence of periodontal disease in adulthood: a population-based birth cohort study. 2016 Florey Postgraduate Research Conference – Adelaide - Australia. (Poster presentation).
4. Schuch HS, Peres KG, Peres MA, Do LG. Modelling socioeconomic trajectories on oral health outcomes over the life course. 94th IADR/AADR/CADR General Session and Exhibition, 2016, Seoul – South Korea. (Oral presentation – Epi Forum).
5. Schuch HS, Peres KG, Singh A, Peres MA, Do LG. Effect of socioeconomic position during the life on periodontitis in adulthood: a systematic review. IADR Australian and New Zealand Division Meeting, 2015, Dunedin – New Zealand. (Oral presentation).

6. Schuch HS, Peres KG, Singh A, Peres MA, Do LG. Effect of socioeconomic position during the life on periodontitis in adulthood: a systematic review. 2015 Florey Postgraduate Research Conference – Adelaide – Australia. (Poster presentation).
7. Schuch HS, Peres KG, Singh A, Peres MA, Do LG. Effect of socioeconomic position during the life on periodontitis in adulthood: a systematic review. School of Dentistry Research Day 2015 – The University of Adelaide - Australia. (Oral presentation).

CHAPTER 1 - INTRODUCTION

Periodontal disease is a bacterially mediated inflammation that extends into the infection of the tissues which support the teeth, causing loss of connective tissue and alveolar bone (Pihlstrom, Michalowicz & Johnson 2005; Thomson, Sheiham & Spencer 2012). It negatively impacts on general health (Otomo-Corgel et al. 2012) and the quality of life of the individual and the population (Aslund, Pjetursson & Lang 2008; Buset et al. 2016; Ferreira et al. 2017; Shanbhag, Dahiya & Croucher 2012). In the first Global Burden of Disease (GBD) study, conducted in 1990, the overall prevalence of periodontal disease was 6.05%. This prevalence has been steadily increasing since then, and in the most recent GBD study, in 2015, the overall prevalence of periodontal disease was 7.63% for all ages combined, affecting 538 million people worldwide. When stratified by sex, the prevalence varies from 7.05% in women to 8.20% in men. Among those aged 30 to 34 years old, including both males and females, the global prevalence of periodontal disease was 6.73% (Kassebaum et al. 2017). The fact that life expectancy is increasing worldwide, combined with people retaining their teeth for longer, might be related to the observed increasing prevalence of periodontal disease in the population.

Some of the risk factors for periodontal disease have already been studied, including behavioural and psychological conditions such as smoking, alcohol consumption, inadequate diet and nutrition, stress and psychological problems and poor oral hygiene (Dentino et al. 2013; Genco & Borgnakke 2013; Sheiham & Watt 2000). The scientific literature also indicates a greater risk of periodontal disease among people with less years of education and those reporting lower

income (Albandar 2002; Eke et al. 2012). Most of the evidence on socioeconomic inequalities in periodontal disease has focused on adulthood risk factors, and there are very important gaps related to development of periodontal disease in understanding the relationship between living conditions during the life-course and the occurrence of the disease in adulthood.

Socioeconomic position (SEP) refers to the social and economic factors that influence what positions individuals or groups hold within the structure of a society (Galobardes, Lynch & Davey-Smith 2007). The most frequently used measures of SEP at individual level are income, education attainment and occupation. Although many studies have investigated the effect of SEP during different stages in life on health later on (Hemmingsson, Lundberg & Diderichsen 1999; Kuh & Ben-Shlomo 1997), only a few have evaluated oral health outcomes with clinically evaluated oral health data (Peres et al. 2007; Thomson et al. 2013). Further, SEP is usually studied as an additional risk factor for periodontal disease, with SEP measured at the same time as the outcome being most frequently used. There is also evidence of differential effects of childhood SEP vs adulthood SEP on oral health (Bernabe et al. 2011; Shin et al. 2015). Therefore, there is a need to understand the link between socioeconomic position across the life span and periodontal disease in adulthood. The scarcity of longitudinal prospective studies from early life to adulthood has made it difficult to identify the role of SEP earlier in life on periodontal disease in adulthood.

Life-course epidemiology studies the biological, behavioural and psychosocial processes that operate in the lifespan of an individual, or across generations, that influence the development of disease risk in adulthood. Fundamentally, it aims to integrate the processes of biological and social risk, instead of drawing false dichotomies between them (Kuh et al. 2003). This theory, of a life-course approach, has been more and more discussed within the scientific community, and there is increasing evidence of the influence of life-course experiences in diseases in adulthood. Studies have been pointing out that social and biological risks accumulated during the life-course, especially during critical periods in early life, are the main determinants of health later on (Hertzman 1999; Kawachi & Berkman 2000). Few studies have been using a life-course approach to study periodontal disease (Poulton et al. 2002; Thomson et al. 2013). Considering that the risk factors to systemic chronic diseases are similar to the main risk factors to periodontal disease, it is plausible that the life-course epidemiology approach applied to general health also can be applied to such a disease. Adopting a life-course approach in studying socioeconomic inequalities in a chronic condition such as periodontal disease is more than recommended; it is of paramount importance if one wants to have a comprehensive and complete understanding of the research problem in question.

Studies with a prospective cohort design sustain the perspective of life-course in health, since they imply that the health status at each age is not only a consequence of the current situation, but also of an accumulation of conditions that were incorporated during the lifespan (Nicolau et al. 2007). Considering the

lack of relevant studies, it is of paramount importance to investigate the development of periodontal disease by applying the life-course approach to data collected in longitudinal birth cohort research. Specifically, this approach allows the researchers to evaluate the role of SEP measured by income at different moments during the life-course on the occurrence of disease later on. It also allows to evaluate potential mediating effect of other conditions over the life-course on the SEP-periodontal disease relationship, such as smoking and oral hygiene status.

1.1 Research Hypotheses

- It is hypothesised that socioeconomic position is an upstream determinant of periodontal disease. It is hypothesised that socioeconomic position during the life-course shapes the socio-behavioural risk profile that may influence the occurrence of periodontal disease.
- It is also hypothesised that early life SEP has a long-term effect on periodontal disease in adulthood. The potential pathways of that effect can be plausibly examined in the light of the life-course epidemiology theories.

1.2 Study Aims

The overarching purpose of the thesis was to investigate the influence of SEP during the life-course on periodontal disease in adulthood.

1.2.1 Specific objectives

1. To theoretically explore how life-course epidemiology theories can be applied to explain the relationship between socioeconomic position and periodontal disease.

2. To systematically review evidence of life-course influence of socioeconomic positions on periodontal status in adulthood.

3. To quantify the impact of life-course income trajectories on the occurrence of periodontal disease in adulthood.

4. To evaluate the direct effect of family income at the time of a participant's birth on periodontal disease occurrence in adulthood.

1.3 Thesis structure

This thesis reports the work conducted during my PhD in Dentistry/Public Health at the Australian Research Centre for Population Oral Health (ARCPOH), Adelaide Dental School, The University of Adelaide, from April 2014 to July 2017. This PhD thesis is structured as a thesis by publication format and consists of 8 chapters. Papers published/submitted/in submission format are included in different chapters, preceded by a short statement that links the paper to the body of the work, as well as the highlights of each paper.

In Chapter 1, the researcher presents the background for the research in socioeconomic inequalities in periodontal health, the research hypothesis, study aims and specific objectives.

Chapter 2 covers the literature on the social determinants of health, what is known about the social determinants of oral health and specifically on periodontal disease, the life-course epidemiology approach, and periodontal disease from an epidemiological perspective.

Chapter 3 describes the methods used in the systematic review and the 1982 Pelotas Birth Cohort Study, which data was used in the empirical studies.

Chapter 4 consists of a published paper in the *Medical Hypotheses* journal, a theoretical paper explaining the potential links between socioeconomic position and periodontal disease under several life-course models, which are: the critical period, the critical period with modifier effect, the accumulation of risk, and the chain-of-risk models. Reviewing the previous literature, it discusses the plausibility of using those theories on such a relationship, and the pathways through which SEP can affect periodontal health.

The paper published in *Community Dentistry and Oral Epidemiology* reports on the systematic review and is presented on Chapter 5. The systematic review was conducted in order to establish if socioeconomic position earlier in life affects periodontal disease later on. The review question that guided the study was ‘Does SEP earlier in life influence periodontal status in adulthood?’

Chapters 6 and 7 display the two manuscripts conducted on the empirical component of the thesis. Data from the 1982 Pelotas Birth Cohort Study was used in the empirical studies.

Chapter 6 presents a paper submitted for publication in the *Journal of Clinical Periodontology.* The paper, entitled ‘Life-course family income trajectories on periodontitis: birth cohort study’, evaluates the effects of socioeconomic trajectories from birth to age 30 on periodontal disease in adulthood.

Chapter 7 was developed to identify the effect of early life SEP on periodontal disease in adulthood. This chapter is prepared in submission format and the paper is going to be submitted to the *Journal of Dental Research*.

Chapter 8 consists of a discussion of the main findings, limitations and strengths of the present thesis, future public health and research implications, and general conclusions.

The Appendices Section includes the abstracts of conference presentations performed during the PhD candidature and related to this thesis; the data collection form developed for the systematic review; the template of the letter sent to expert researchers in order to identify potential unpublished studies that could be suitable for inclusion in the systematic review; the ethical approvals for the oral health sub-studies in the 1982 Pelotas Birth Cohort Study. The last document in the Appendices Section is a published paper resulted from a collaboration of the student during the PhD Candidature. This paper is a Scoping Review on area-level social inequalities and population oral health and is published in the *Social Science and Medicine – Population Health.*

1.4 References

Albandar, JM 2002, 'Global risk factors and risk indicators for periodontal diseases', *Periodontol 2000*, vol. 29, pp. 177-206.

Aslund, M, Pjetursson, BE & Lang, NP 2008, 'Measuring oral health-related quality-of-life using OHQoL-GE in periodontal patients presenting at the University of Berne, Switzerland', *Oral Health Prev Dent*, vol. 6, no. 3, pp. 191-197.

Bernabe, E, Suominen, AL, Nordblad, A, Vehkalahti, MM, Hausen, H, Knuuttila, M, Kivimaki, M, Watt, RG, Sheiham, A & Tsakos, G 2011, 'Education level and oral health in Finnish adults: evidence from different lifecourse models', *J Clin Periodontol*, vol. 38, no. 1, Jan, pp. 25-32.

Buset, SL, Walter, C, Friedmann, A, Weiger, R, Borgnakke, WS & Zitzmann, NU 2016, 'Are periodontal diseases really silent? A systematic review of their effect on quality of life', *J Clin Periodontol*, vol. 43, no. 4, Apr, pp. 333-344.

Dentino, A, Lee, S, Mailhot, J & Hefti, AF 2013, 'Principles of periodontology', *Periodontol 2000*, vol. 61, no. 1, Feb, pp. 16-53.

Eke, PI, Page, RC, Wei, L, Thornton-Evans, G & Genco, RJ 2012, 'Update of the case definitions for population-based surveillance of periodontitis', *J Periodontol*, vol. 83, no. 12, Dec, pp. 1449-1454.

Ferreira, MC, Dias-Pereira, AC, Branco-de-Almeida, LS, Martins, CC & Paiva, SM 2017, 'Impact of periodontal disease on quality of life: a systematic review', *J Periodontal Res*, Feb 08.

Galobardes, B, Lynch, J & Smith, GD 2007, 'Measuring socioeconomic position in health research', *Br Med Bull*, vol. 81-82, pp. 21-37.

Genco, RJ & Borgnakke, WS 2013, 'Risk factors for periodontal disease', *Periodontol 2000*, vol. 62, no. 1, Jun, pp. 59-94.

Hemmingsson, T, Lundberg, I & Diderichsen, F 1999, 'The roles of social class of origin, achieved social class and intergenerational social mobility in explaining social-class inequalities in alcoholism among young men', *Soc Sci Med*, vol. 49, no. 8, Oct, pp. 1051-1059.

Hertzman, C 1999, 'The biological embedding of early experience and its effects on health in adulthood', *Ann N Y Acad Sci*, vol. 896, pp. 85-95.

Kassebaum, NJ, Smith, AGC, Bernabé, E, Fleming, TD, Reynolds, AE, Vos, T, Murray, CJL, Marcenes, W & Collaborators, GOH 2017, 'Global, Regional, and National Prevalence, Incidence, and Disability-Adjusted Life Years for Oral Conditions for 195 Countries, 1990–2015: A Systematic Analysis for the Global Burden of Diseases, Injuries, and Risk Factors', *Journal of dental research*, vol. 96, no. 4, pp. 380-387.

Kawachi, I & Berkman, LF 2000, 'A historical framework for social epidemiology', in I Kawachi & LF Berkman (eds), *Social Epidemiology*, Oxford University Press, New York, pp. 3-12.

Kuh, D & Ben-Shlomo, Y 1997, *A Life-course Approach to Chronic Disease Epidemiology*, Oxford University Press.

Kuh, D, Ben-Shlomo, Y, Lynch, J, Hallqvist, J & Power, C 2003, 'Life course epidemiology', *J Epidemiol Community Health*, vol. 57, no. 10, Oct, pp. 778-783.

Nicolau, B, Thomson, WM, Steele, JG & Allison, PJ 2007, 'Life-course epidemiology: concepts and theoretical models and its relevance to chronic oral conditions', *Community Dent Oral Epidemiol*, vol. 35, no. 4, Aug, pp. 241-249.

Otomo-Corgel, J, Pucher, JJ, Rethman, MP & Reynolds, MA 2012, 'State of the science: chronic periodontitis and systemic health', *J Evid Based Dent Pract*, vol. 12, no. 3 Suppl, Sep, pp. 20-28.

Peres, MA, Peres, KG, de Barros, AJ & Victora, CG 2007, 'The relation between family socioeconomic trajectories from childhood to adolescence and dental caries and associated oral behaviours', *J Epidemiol Community Health*, vol. 61, no. 2, Feb, pp. 141-145.

Pihlstrom, BL, Michalowicz, BS & Johnson, NW 2005, 'Periodontal diseases', *Lancet*, vol. 366, no. 9499, Nov 19, pp. 1809-1820.

Poulton, R, Caspi, A, Milne, BJ, Thomson, WM, Taylor, A, Sears, MR & Moffitt, TE 2002, 'Association between children's experience of socioeconomic disadvantage and adult health: a life-course study', *Lancet*, vol. 360, no. 9346, Nov 23, pp. 1640-1645.

Shanbhag, S, Dahiya, M & Croucher, R 2012, 'The impact of periodontal therapy on oral health-related quality of life in adults: a systematic review', *J Clin Periodontol*, vol. 39, no. 8, Aug, pp. 725-735.

Sheiham, A & Watt, RG 2000, 'The common risk factor approach: a rational basis for promoting oral health', *Community Dent Oral Epidemiol*, vol. 28, no. 6, Dec, pp. 399-406.

Shin, BM, Ryu, JI, Sheiham, A, Do, LG & Jung, SH 2015, 'Which life course model better explains the association between socioeconomic position and periodontal health?', *J Clin Periodontol*, vol. 42, no. 3, Mar, pp. 213-220.

Thomson, WM, Shearer, DM, Broadbent, JM, Foster Page, LA & Poulton, R 2013, 'The natural history of periodontal attachment loss during the third and fourth decades of life', *J Clin Periodontol*, vol. 40, no. 7, Jul, pp. 672-680.

Thomson, WM, Sheiham, A & Spencer, AJ 2012, 'Sociobehavioral aspects of periodontal disease', *Periodontol 2000*, vol. 60, no. 1, Oct, pp. 54-63.

CHAPTER 2 – LITERATURE REVIEW

This chapter presents a detailed Literature Review of the social determinants of health, life-course epidemiology and its theories, social inequalities in oral health and an epidemiological approach to periodontal disease.

2.1 Social determinants of health

The occurrence of health conditions varies substantially between and within populations, and genetic and biological predispositions can only partially explain these variations. Underlying such differences are the conditions in which people are born, grow, work, live and age. This set of conditions, known as the social determinants of health, includes family income, maternal and early child development conditions, housing and sanitation, access to education and health services, job-related conditions, and many others (Commission on Social Determinants of Health 2008).

In an upstream level, structural and political conditions such as macroeconomic and social policies may also relate to health. However, there is still no consensus about the health effects of income inequality at population level. As Lynch and colleagues argued, this lack of evidence of direct effect of income inequality in health at population level is not contradictory to the large body of evidence that at the individual level those with higher incomes also are healthier (Lynch et al., 2004).

Since the twentieth century, the prevailing etiological models for adult chronic diseases have emphasised adult risk factors, with a focus on individual lifestyle.

However, the importance of the social determinants of health is not a recent topic. In a paper published in 1985, in contrast to the lifestyle theory, Blane argued convincingly that the causative role of individual behaviours had been exaggerated. According to him, they should be seen '… as indicators of other factors which are more straightforwardly related to the social structure, and which are the true aetiological agents.' (Blane 1985). He was addressing the 'causes of the causes of diseases'. Also in 1985, Geoffrey Rose published a paper that became classic, entitled 'Sick Individuals and Sick Populations' (Rose 1985). The central point of the paper was that individual and population approaches to improving health are fundamentally different and achieve different aims. He postulated that the determinants of individual cases are different to the determinants of the incidence of a disease in a population. According to Rose, in an individual approach, a preventive strategy seeks to identify high-risk, susceptible individuals and to offer them some individual protection. By contrast, the population strategy seeks to control the determinants of incidence in the population as a whole. After 20 years, a group of researchers published a paper reinforcing Rose's ideas, showing that it remains highly relevant to public health (Doyle, Furey & Flowers 2006). Although Rose's paper reverberated in his time, many people were against his ideas. Possibly, it was because of the mistaken idea that population approaches frequently yielded small benefit to individuals (Charlton 1995). However, even though the ideas of 'causes of the causes' are not new, they have been discussed worldwide only in the last few years. In 2005, the Word Health

Organization implemented the Commission on Social Determinants of Health. It drew the attention of governments, civil society, international organisations, and donors to pragmatic ways of creating better social conditions for health, especially for the world's most vulnerable people. In October 2011, the first World Conference on Social Determinants of Health took place in Brazil, and, at its conclusion, the participants, who were Heads of Government, Ministers and government representatives, formulated the Rio Political Declaration on Social Determinants of Health (World Conference on Social Determinants of Health 2011). A short time after they presented the document, the Federation of Medical Students posted a declaration stating that the main problem of the Rio Declaration was that it failed to explicitly explain how the unfair distribution of power, resources and wealth would be addressed, especially by Member States (International Federation of Medical Students' Federation 2011).

The social gradient in health means that health is shaped by the socioeconomic distribution in the population. In general, the poorest of the poor, around the world, have the worst health. Within countries, the evidence shows that, in general, the lower an individual's socioeconomic position, the worse is his/her health. This is a global phenomenon, seen in low, middle, and high income countries (WHO Commission on Social Determinants of Health 2008). The social gradient in health means that health inequalities affect everyone.

Such a gradient can be observed in both general and oral health conditions. As an example, the social gradient has been observed across different health conditions, such as perceived oral and general health, periodontal disease and

ischaemic hearth disease (Sabbah et al 2007). The socioeconomic indicator adopted by such a study was education, categorised as less than 12 years of study, 12 years of study, or more than 12 years.

The social determinants of health include factors such as the local environment one lives in, the level of education one attains, and the amount of money one earns. The conceptual framework proposed by the WHO CSDH (World Health Organization, Solar & Irwin 2010) outlined how the major determinants relate to each other, and the mechanisms involved in generating inequalities in population health. The framework shows how important are the 'structural determinants', the socioeconomic and political contexts that generate the social hierarchy in any society, and the resulting socioeconomic position of its individuals. The intermediary determinants refer to how socioeconomic position then influences health through the circumstances and risks of diseases. Individuals from lower socioeconomic groups are born, live, work and age in less favourable circumstances than those from higher socioeconomic groups. These include: material and social conditions such as housing and working conditions and quality of neighbourhoods; psychosocial factors such as stress and social support; and behavioural and biological factors. The framework also includes health services and highlights the importance of fair access to good quality care (WHO Commission on Social Determinants of Health 2008).

Inequitable distribution of health is present in society when more deprived individuals have poorer health while the richer has a disproportional share of better health. The larger the social gradients in health, the worse it reflects on

this society. The 1998 Nobel Laureate in Economics, Amartya Sen has argued that health is a human right and equity in health is a key indicator for societal wellbeing (Sen 2008). In seeing health as a right, he acknowledged the need for a strong social commitment to good health. The key point of Sen's work is that health equality has different meanings across distinct theories, and these differences lie on what each theory judges as valuable. For example, the income egalitarian will prize an equal distribution of incomes, while the committed democrat will insist on equal political rights for all (Sen 2002). Additionally, the concept of health as a human right incorporates health in a larger understanding of social justice, when we consider the need for equity in the achievement and distribution of health.

Amartya Sen also discussed the distinction between health achievements and the capability to achieve health, with the view that it is unjust that some people will lack the opportunity to achieve good health because of inadequate social arrangements (Sen 2002).The conditions that may contribute to health achievements are not restricted to health care, and they include factors such as genetic propensity, individual income, food habits and lifestyles, the environment, and work conditions. Accordingly, in order to advance health equity, Sen proposes that we need to have broad actions in the political, social, economic, scientific, and cultural arenas (Sen 2008).

2.2 Social inequalities in oral health

Oral diseases affect 3.5 billion people globally, and the most recent estimates from the Global Burden of Disease Study (2015) show that, for the first time

since the first GBD Study in the early 1990s, oral diseases are ranked in the top 10 leading causes of Years Lived with Disability (YLDs) worldwide (Kassebaum et al. 2017). The same social gradient that is usually observed in general health is also evident in aspects of oral health, such as dental caries and the number of teeth (Sabbah et al. 2007; Sanders et al. 2006; Steele et al. 2015).

To illustrate the shape of the socioeconomic-oral health gradient, a study was conducted with Australian adults aged 43 to 57 years. The researchers measured socioeconomic position through two different indicators: as an indicator of objective socioeconomic position – equivalised household income was adopted; as subjective socioeconomic position – the MacArthur Scale of Subjective Social Status (Adler et al. 2000) was the instrument selected. They were able to identify a social gradient in the four outcomes evaluated: number of teeth present and oral health impact profile – assessed by means of the Oral Heath Impact Profile (OHIP-14) instrument, global self-rated oral health, and satisfaction with chewing ability. All measures were self-reported (Sanders et al. 2006).

What is of major concern is that, although a decline in the absolute prevalence of oral diseases has been observed over time, the social gradient in those conditions is persistent. Elani and colleagues used nationally representative data from Canada and the United States to evaluate socioeconomic inequalities in terms of education, income and place of birth in the following outcomes: edentulism, proportion of individuals having at least 1 untreated decayed tooth, and proportion of individuals having at least 1 filled tooth over a 35-year period

(Elani et al. 2012). The authors identified a reduction in absolute figures, but the social gradient in the outcomes persisted over time. Additionally, the problem of social inequalities in oral health is of even more concern if we take into consideration that SEP inequalities are widening in most countries, making the perspective of reducing health inequalities more challenging (Mackenbach et al. 2003; OECD 2011).

Several theories have been proposed in order to explain how social inequalities may affect oral health. The most discussed ones at an individual level of SEP are the materialist, behavioural and psychosocial theories. Basically, the materialist theory links SEP to oral health outcomes through the idea that those of higher SEP would be able to afford goods and services that would lead them to better health, such as better food, hygiene products and access to health care (Mejia, Armfield & Jamieson 2014). The behavioural theory lies in the idea that socioeconomic conditions would shape oral health-related risk factors and behaviours, such as smoking status and oral hygiene practices (Thomson, Sheiham & Spencer 2012). Finally, the psychosocial theory states that SEP would affect health in two potential ways: the first is related to the understanding that someone's perception of his/her social position would impact on his/her health, and the second is that people from lower SEP have higher stress and lower perceived control, and this in turn would affect their health (Singh et al. 2016).

2.3 Social determinants of health and periodontal disease

Studies on the causes or risk factors of periodontal disease have historically focused on individual characteristics, such as behaviours, psychosocial characteristics and systemic conditions (Genco & Borgnakke. 2013). In a recently published review paper on periodontitis causation, the authors systematically review the literature in order to summarise evidence on potential causes of periodontitis. Available evidence was identified in six risk factors: alcohol, diabetes mellitus, frequency of oral hygiene, obesity, putative periodontal pathogens and socioeconomic position (Nascimento et al. 2017). The authors appropriately discuss how an approach focused on individual risk factors overlooks the causes of causes and reduces periodontitis to a collection of independent single factors, ignoring their interactions. Additionally to individual risk factors, social and political contexts also play a role in the causation of periodontal disease and are strongly related to health inequalities.

It is well known that SEP is inversely associated with the development and progression of chronic diseases in adulthood (Galobardes, Lynch & Smith 2007; Mackenbach et al. 2000), which may in turn trigger symptomatic conditions and, potentially, worse self-perceived health. The same patterns observed for general chronic conditions are also plausible for oral health, and there is evidence of inequalities in periodontal disease by individual income and education from almost 50 years ago (Kelly & Engel 1969).

The most frequently SEP indicator used in epidemiological studies is education. Education is relatively stable during adult life, and its effect on health can be

either via increased knowledge and related behaviours or via occupation and income, considering that the higher the education attainment, the higher the likelihood of achieving a better job and earning a higher income (Borrell & Crawford 2012). The meta-analysis conducted by Boillot and colleagues (Boillot et al. 2011) on the association between low education and chronic periodontitis reported a pooled estimate odds ratio (OR) of 2.11 (95% CI 1.22 to 3.63), but since only 2 of the eighteen selected studies were longitudinal in design, the pooled OR was reported based on only 2 studies.

Income is also commonly used as a SEP indicator. It is the SEP indicator that most directly related to material resources and, unlike education, it is not a very stable measure. The fact that income changes over time allows the study of dynamics of change in SEP, for example modelling trajectories of income over the life span. Also, as income usually reflects education attainment, it may combine both behaviours influenced by education and the affordability of hygiene products and health services. Income is also closely linked to occupation. High income earners are expected to have better occupation positions, in the way that better occupations usually have better salaries. Income is usually measured as individual or household income. The use of household income is useful since is gives information of the material resources of all living in the household, and the person being surveyed may or may not be the main earner in the household. Income is arguably the best single indicator of material living standards (Galobardes et al. 2006).

A difficulty when working with income is that individuals are sometimes reluctant to disclose their income in surveys. To the best of this researcher's knowledge, the first systematic review with longitudinal studies addressing the effect of income on periodontal disease and published in the scientific literature is the one developed as part of this thesis and presented in Chapter 5. The aforementioned social gradient observed in general health and other oral health outcomes was also observed for periodontal disease when considering income as the SEP indicator: using nationally representative data from a developed and a developing country, Australia and Vietnam respectively, researchers observed a social gradient on periodontal disease across five social groupings based on household income (Thomson, Sheiham & Spencer 2012). In the United States, income and education were shown to be independently associated with periodontal disease (Borrell & Crawford 2008). The authors also evaluated race/ethnicity as a marker of social inequality, and identified an association of such an indicator with periodontal disease independently of income and education.

Finally, occupation is also a measure of SEP used in several studies, including the longest cohort study worldwide with clinical oral examination – the Dunedin Multidisciplinary Health and Development Study (Poulton et al. 2002). Using data from such a study, reseachers evaluated trajectories of periodontal disease from age 26 to 38 years old. They observed that those of lower SEP in adulthood and childhood, measured by parental occupation at childhood and individual occupation in adulthood respectively, experienced higher membership

of moderately increasing and markedly increasing trajectories of periodontal disease than to those of higher SEP (Thomson et al. 2013). Occupation is directly related to income, and it also captures a broader perspective on the subject's social position in the social hierarchy. Additionally, it can capture stress related to certain occupations, although it is important to note that the meaning of occupation can differ according to the context (place and time) where the measure was collected (Borrell & Crawford 2012).

There is evidence on the association between SEP and periodontal disease. However, it is of paramount importance to emphasise that the vast proportion of evidence in the scientific literature is based on cross-sectional studies, and there is a lack of evidence from long-term longitudinal studies. There is a need to capture SEP experiences during the life span and to explore how these exposures relate to periodontal disease later on. Adopting a life-course approach in studying socioeconomic inequalities in a chronic condition such as periodontal disease is more than recommended; it is of paramount importance if one wants to have a comprehensive and complete understanding of the research problem in question.

2.4 Life-course epidemiology

Life-course epidemiology is a theoretical approach that studies the pathways of health and illness during the lifespan. It aims to elucidate the influence of exposures during gestation, childhood, adolescence, early adulthood and across generations to health later on in time. Applying the life-course approach to understand health inequalities is useful because it allows the investigation of

how social and biological factors operating during the life-course and across generations contribute to the development of inequalities in adult health and disease, and it tests different pathways linking the exposures to the health outcomes. Such an approach emphasises a temporal and social perspective, and how conditions during all phases of life will shape the patterns of health and illness later on. The four most common theories of life-course epidemiology in the scientific literature are: the critical period theory; the critical period with effect modifier; the accumulation of risk theory; and the chain of risk theory.

The first of them proposes that exposures during a critical period of development during early life have lifelong effects on adult health, independently of adulthood circumstances. According to the critical period theory, experiences in early childhood act as a determinant factor in the development of diseases in adult life, and it has been discussed that such a process works as biological programming (Barker 1992, 1994; Wadsworth et al. 1985). The importance of considering early life events to understand later conditions is not new. Back in 1667, the English poet John Milton (1608–1674) wrote in his *Paradise Lost*:

‘The childhood shows the man,
As the morning shows the day.’(144a, lines 220-21)

In the oral health field, however, only recently has this theory been discussed. Specifically addressing periodontal disease, there is a theory that the experience of lower SEP in early life would affect stress hormones during an important period of maturation of the immune system, and this would impact the

immune response in situations of infections, thereby making the individual more susceptible to the development of diseases such as periodontal disease (Nicolau, Netuveli, et al. 2007; Schuch et al. 2015) (Figure 1).

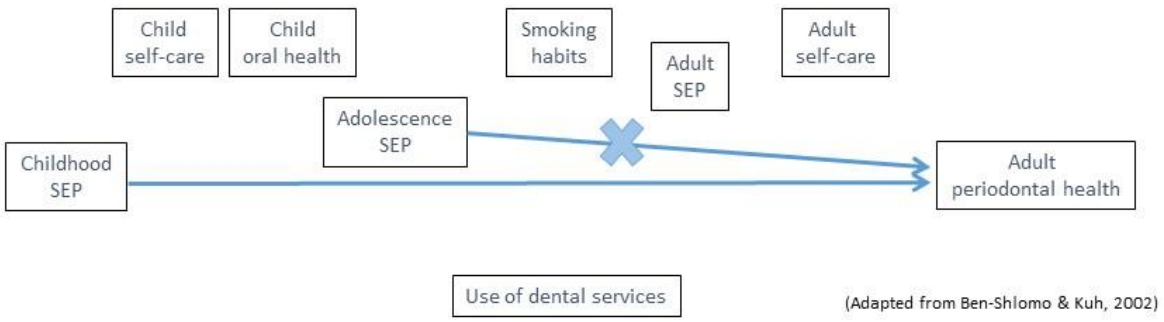

Figure 1. Critical period theory.

The second theory, the theory of critical period with effect modifiers, postulates that the main exposures in early life may interact with subsequent events. In this theory, early life circumstances are identified as the first step towards adult health, although with an indirect effect, influencing adult health outcomes through social mechanisms, such as restricting education opportunities later on, which may lead, in turn, to socioeconomic and health conditions later in life (Kuh et al. 2003). As an example of this theory, one can argue that behaviours such as oral health practices and smoking status are learnt early in life, and that these behaviours would then increase the risk of periodontal disease later on. Although situations later in life could also impact on such behaviours, the strongest influence would come from conditions (and indeed conditioning) experienced early in life (Schuch et al. 2015) (Figure 2).

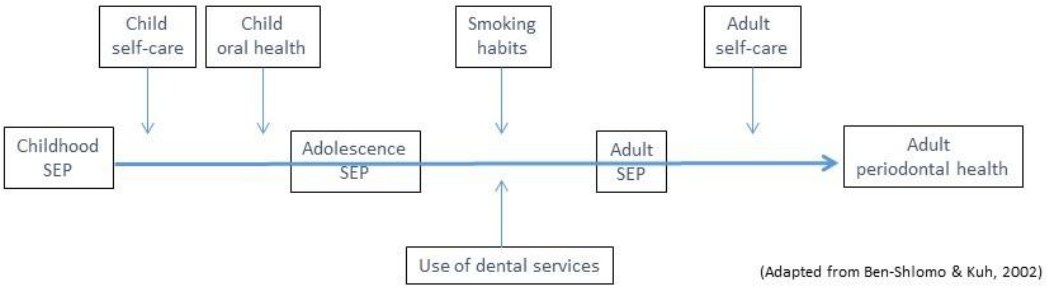

Figure 2. Critical period with effect modifiers theory.

The accumulation of risk theory states that chronic diseases in adults can be caused by an accumulation of risks during the life of the individual, such as social exposures, previous diseases, the context and conditions where the person lives and his/her behaviours. This theory hypothesises that the intensity and duration of exposure to harmful or beneficial social and/or physical contexts during the life-course affects health conditions in a dose-response relationship, in what has been called as the hypothesis of accumulation of risk (Kuh & Ben-Shlomo 1997). This theory has been related to periodontal disease from an understanding that lower SEP is linked to several risk factors for such a disease, such as smoking status, poor oral hygiene and greated susceptibility to infections. The combination of these exposures, accumulating during the life span would therefore increase the risk of periodontal disease later in life (Nicolau, Thomson, et al. 2007) (Figure 3).

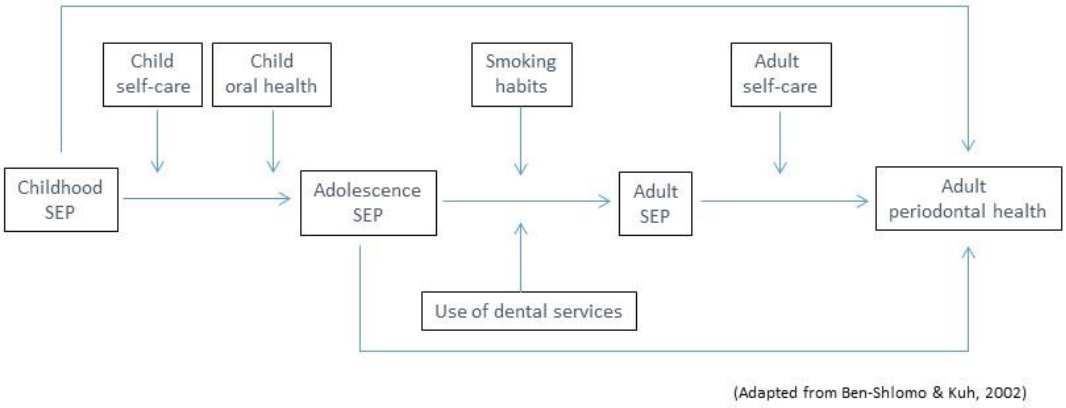

Figure 3. Accumulation of risk theory.

The fourth theory is known as the chain of risk theory. It proposes that a sequence of linked exposures may increase the risk of diseases, and it would happen due to a harmful exposure or experience leading to another harmful exposure and so on, triggering a chain model (Kuh et al. 2003). These linked exposures can be either biological or social (for example, a child with repeated episodes of disease in childhood, leading to missing days at school and, consequently, poor education attainment, which in turn may lead to harmful behaviours later in life) that can increase the risk of development of diseases (Kuh & Ben-Shlomo 1997). It is expected that SEP early in life will affect socioeconomic achievements later on. For example, children raised in well-off families usually attend better schools and have higher quality parenting and this may lead to higher educational achievement, better occupation and higher income later on. All of these linked SEP exposures would then impact on periodontal disease (Figure 4).

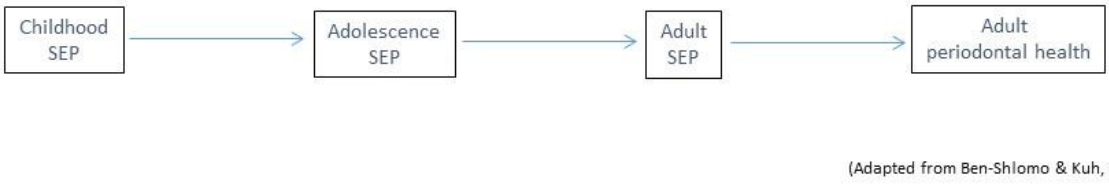

Figure 4. Chain of risk theory.

2.4.1 Life-course epidemiology and the social determinants of health

The research on the social determinants of health, and particularly in adopting a life-course approach, is fundamental if we would like to understand health equity. As quote from the final report of the Early Child Development Knowledge Network of the Commission on Social Determinants of Health states, 'health development during the early years provides the essential building blocks that enable people to lead a flourishing life in many domains, including social, emotional, cognitive, and physical well-being' (Irwin, Siddiqi & Hertzman 2007). In the words of Dr Margaret Chan, Director-General of the WHO, '[…] no one should be denied access to life-saving or health-promoting interventions for unfair reasons, including those with economic or social causes. When health is concerned, equity really is a matter of life and death' (Sheiham 2009).

Unfair and avoidable inequalities in periodontal disease have been pointed out in the scientific literature, with socially disadvantaged individuals having worse

periodontal conditions than those who are better off. Studying SEP and periodontal disease under a life-course epidemiology approach aims to identify which factors are associated with the development and progression of disease later in life, enabling researchers and policy-makers to tackle such a problem and to minimise its effects on those already suffering from periodontal disease. Evidence from studies with a life-course perspective are expected to be useful in shaping the nature, level, purpose and timing of interventions to prevent periodontal disease (Thomson, Sheiham & Spencer 2012).

2.5 Periodontal disease

Periodontal disease is a polymicrobial and polygenic inflammatory disease (Armitage 2002) affecting the supporting tissues of the teeth, leading to pathological detachment of collagen fibres from the cementum and the junctional epithelium to migrate apically (Savage et al. 2009). The majority of periodontal diseases are chronic conditions, but aggressive types such as gingivitis and aggressive periodontitis also exist. Clinical manifestation of chronic periodontal disease is sensitive and may be altered by important environmental and individual conditions, such as oral hygiene, smoking, emotional stress and diabetes (Baelum & Lopez 2013; Petersen & Baehni 2012). Considering the chronic nature of most periodontal diseases, its low rate of development and the fact that conditions in different periods of life can cumulatively increase the risk of disease, the life-course approach is the most appropriate approach to investigate such a disease.

According to the GBD 2015 Study, the global prevalence of periodontal disease for all ages combined is 7.6%, while in Brazil it is 10.4%. Looking for the trends in the prevalence of such disease in the world and in the Brazilian context, it is noteworthy that both estimates increased over time, with a considerably higher increase in Brazil: while the estimates worldwide increased from 6.1% in 1990 to 7.6% in 2015, a prevalence change of +1.5%, in Brazil the prevalence increased by 2.9% (from 7.5% in 1990 to 10.4% in 2015). The GBD Study is based on clinical examination of periodontal conditions, and considered as cases those individuals with a gingival pocket depth equal or more than 6 mm, or a clinical attachment loss (CAL) more than 6 mm (Kassebaum et al. 2017).

Although the disease is often asymptomatic, a recently published systematic review showed that periodontal disease impacts on the quality of life of sufferers in a dose-response relationship, with those with severe periodontal disease experiencing a significantly greater impact than mild to moderate periodontal disease (Ferreira et al. 2017). Periodontal disease can lead to pain, as well as affecting function and causing psychological discomfort. Also, its end point, tooth loss, has clear functional and emotional impacts on individuals' lives.

Defining periodontal disease in epidemiological studies is a challenge. This measurement issue has been extensively discussed in the scientific literature (Baelum & Lopez 2013; Dentino et al. 2013), and even nowadays there is no consensus between researchers about the 'best case' definition. The issue of defining periodontal disease is considered by researchers as one of the most significant factors impacting on data interpretation (Tonetti, Claffey & European

Workshop in Periodontology group 2005). Several indices have been proposed to measure periodontal disease in epidemiological studies. In order to describe and discuss such indexes, I divided the indices into historical and contemporary indexes.

2.5.1.2 Historical indices to measure periodontal disease

Russell's Periodontal Index

One of the first indices proposed to allow quantitative comparisons between or within human populations, and that required minimum use of equipment (in other words, it was suitable for epidemiological studies) was the Russell's Periodontal Index (Russell 1956). Such an index scored each tooth in the mouth progressively according to its clinical condition, and the overall score was calculated giving relatively little weight to soft tissue inflammation and great weight to bone loss. An important limitation of Russell's Periodontal Index was the intrinsic idea that soft tissue inflammation, or gingivitis, inevitably turns into periodontal disease.

Periodontal Treatment Need System

In 1973, the Periodontal Treatment Need System was proposed (Johansen, Gjermo & Bellini 1973). As the name suggests, the index was intended to suggest levels of treatment required based on indicators of pocket depth and gingival inflammation, together with dental calculus and presence of plaque. In the Periodontal Treatment Need System, all surfaces of all teeth were examined, although just the worst conditions of each quadrant were recorded.

Community Periodontal Index and Treatment Needs

Partially based on the Periodontal Treatment Need System, in 1982 the World Health Organization proposed the Community Periodontal Index of Treatment Needs (CPITN) (Ainamo et al. 1982). Although it was not developed as an indicator of disease activity, and it has many limitations, it is still widely used. The CPITN is based on the clinical examination of 6 index teeth representing sextants (the 4 first molars, one maxillary and one mandibular anterior central incisor), and it comprises shallow and deep periodontal pockets (4-5mm and 6+mm, respectively), dental calculus and bleeding on probing. As it can be observed, such an index also relies on the model for the natural progression of periodontal disease that was observed in the previously described indices. The use of CPITN in epidemiological studies may lead to an unrealistic high estimate of periodontal treatments needed that do not reflect the real burden of periodontal disease in the population – since it considers dental calculus and bleeding on probing as indicators of disease activity, while it is known nowadays that these assumptions are not correct. Also, the CPITN only records the worst score of the 6 segments of the mouth, therefore over-simplifying and missing much of the clinical information. Although still used, the use of the CPITN has been the target of much criticism (Baelum, Fejerskov, et al. 1993; Baelum & Lopez 2013; Baelum, Manji, et al. 1993; Baelum & Papapanou 1996; Carlos, Wolfe & Kingman 1986).

As the concepts of periodontal disease occurrence and progression changed over the years, there was also a shift in the way periodontal data were collected

and classified in the epidemiological context. Such a change resulted in a decreased use of indices measured at the individual level and the increased adoption of periodontal indices measured at site-level, combining a range of periodontal disease-related parameters, such as alveolar bone loss, clinical attachment level, probing pocket depth and gingival recession (Baelum & Lopez 2013). It is of paramount importance to note that, although the current measures are examined for each site, these measures are then changed to person-level estimates, since the unit of analysis for investigations of risk factors must be the individual rather than the site (Thomson, Sheiham & Spencer 2012).

2.5.1.3 Contemporary Indices to measure periodontal disease

Extent and Severity Index

In an attempt to capture the maximum amount of information from a clinical examination, and to allow comparison of studies with distinct populations, the Extent and Severity Index (ESI) was formulated (Carlos, Wolfe & Kingman 1986). According to such an index, proposed by Carlos and colleagues in 1986, the extent of periodontal disease is considered as the percentage of examined sites with attachment loss exceeding 1 millimetre. The severity of disease is classified using mean loss of attachment, considering sites with at least 1 millimetre of attachment loss. One of the main limitations of the ESI is the fact that the estimates are derived from only two contralateral quadrants of the person.

Dunedin Multidisciplinary Health and Development Study periodontal disease case definition

The longest cohort study worldwide with clinical oral examination is the Dunedin Multidisciplinary Health and Development Study, started in between April 1st 1972 and March 30th, 1973 (Poulton, Moffitt & Silva 2015). In the Dunedin Study, three sites (mesiobuccal, buccal, and distolingual) per tooth in all teeth except third molars are examined, and information on probing depth and gingival recession is recorded. Attachment loss (AL) for each site is obtained by summing gingival recession and probing depth. As indicators of periodontal disease, reports from the Dunedin Multidisciplinary Health and Development Study frequently adopt the cut-off points of 1 or more sites with 4+ mm of AL or 1 or more sites with 5+ mm AL (Shearer et al. 2011; Thomson et al. 2007).

Centre for Disease Control and Prevention (CDC) and American Academy of Periodontology (AAP) periodontal disease case definition

In 2007, the Disease Control and Prevention (CDC), in partnership with the American Academy of Periodontology (AAP), developed standard definitions to the surveillance of mild, moderate and severe periodontal disease, based on measures of pocket depth and loss of clinical attachment in interproximal sites (Eke et al. 2012; Page & Eke 2007). The CDC-AAP periodontal disease case definition is based on examination of six sites per tooth (mesio-buccal, mid-buccal, disto-buccal, mesio-lingual, mid-lingual, and disto-lingual) in all teeth present, excluding third molars. For epidemiological studies, periodontal disease was classified in a more specific way. Mild periodontal disease was

defined as 2 or more interproximal sites with 3+ mm of attachment loss and 2 or more interproximal sites with 4+ mm of pocket depth, but in a different tooth, or one site with 5+ mm of pocket depth. Moderate periodontal disease was defined as having at least 2 teeth with interproximal clinical attachment loss of 4 or more millimetres, or at least 2 teeth with 5+ mm of pocket depth in interproximal sites. Severe periodontal disease was defined as the presence of at least 2 teeth with 6+ mm of clinical attachment loss in interproximal sites and 1 or more interproximal sites with pocket depth of 5+ mm. The CDC-AAP periodontal disease case definitions are the ones adopted by the American National Health and Nutrition Examination Survey (NHANES). Figures from NHANES 2009 and 2010, using the CDC-AAP case definitions, show a prevalence of moderate-to-severe periodontal disease of 47% among adults. When evaluating the levels of severity separately, the prevalence of mild, moderate and severe periodontal disease was of 8.7%, 30.0%, and 8.5%, respectively. The overall prevalence was calculated by summing the prevalence of each stage, and it ranged from 24.4% among adults aged 30 to 34 years old to over 70% among adults of 65+ years old (Eke et al. 2012).

The 5th European workshop in Periodontology case definition

Following the 5th European Workshop in Periodontology, Tonetti and Claffey, on behalf of one of the workshop's groups, proposed new criteria for defining periodontal disease and disease progression in epidemiological studies of risk factors of periodontal disease (Tonetti, Claffey & European Workshop in Periodontology group 2005). Their initiative aimed to establish a framework that

allows some consistency of data interpretation across global epidemiological studies. Two threshold levels of periodontal disease case definition were proposed: the first level consisted of the presence of proximal attachment loss of 3+ mm in 2 or more non-adjacent teeth; and the second level consisted of the presence of proximal attachment loss of 5+ mm in at least 30% of all present teeth. The authors referred to the first level of periodontal disease as a more sensitive case definition, including incipient cases of disease, and the second as a more specific case definition, aiming to identify only cases with substantial extent and severity. It is important to highlight that these criteria were not designed for the assessment of prevalence of periodontal disease across populations and/or age groups, but to focus on the identification of periodontal disease risk factors.

The way considered to be the most appropriated to measure periodontal disease has been changing and evolving over time. Heterogeneity was observed between the indices in terms of measurement tools, particularly the types of probes used, areas examined (full/part mouth), location of probing and diagnostic thresholds. The methodological inconsistency in the use of disease indicators make large variations in the periodontitis definition inevitable (Savage et al. 2009) and there are implications and consequences of using the different measures.

For example, although part-mouth assessments can be quick and cheaper, they may underestimate periodontal disease prevalence in populations with less

susceptibility or overestimate the prevalence if the teeth selected for examination are first molars and lower incisors (Carlos, Wolfe, & Kingman. 1986). Additionally, attention should be given to which measures are taken into consideration to define periodontal disease and how they are measured (continuous versus categorical measures). Eminent researchers in periodontology (Tonetti & Claffey. 2005) highlighted that periodontitis cannot be reflected by measurements of only a single variable such as attachment loss or bone loss but required additional measurement such as pocket depth.

Currently, recording periodontal attachment loss at six sites per tooth on all teeth is considered to be the gold standard measure (Thomson, Sheiham & Spencer 2012). From such an assessment, different case definitions proposed by the contemporary indices aforementioned can be derived.

2.6 References

Adler, NE, Epel, ES, Castellazzo, G & Ickovics, JR 2000, 'Relationship of subjective and objective social status with psychological and physiological functioning: preliminary data in healthy white women', *Health Psychol*, vol. 19, no. 6, Nov, pp. 586-592.

Armitage, GC 2002, 'Classifying periodontal diseases--a long-standing dilemma', *Periodontol 2000*, vol. 30, pp. 9-23.

Baelum, V & Lopez, R 2013, 'Periodontal disease epidemiology - learned and unlearned?', *Periodontol 2000*, vol. 62, no. 1, Jun, pp. 37-58.

Barker, DJP 1992, *Foetal and infant origin of adult disease.*, BMJ Publishing Group, London.

Barker, DJP 1994, *Mothers, Babies, and Disease in Later Life*, BMJ Publishing Group, London.

Blane, D 1985, 'An assessment of the Black Report's explanations of health inequalities', *Sociology of Health & Illness*, vol. 7, no. 3, pp. 423-445.

Boillot, A, El Halabi, B, Batty, GD, Range, H, Czernichow, S & Bouchard, P 2011, 'Education as a predictor of chronic periodontitis: a systematic review with meta-analysis population-based studies', *PLoS One*, vol. 6, no. 7, p. e21508.

Borrell, LN & Crawford, ND 2008, 'Social disparities in periodontitis among United States adults 1999-2004', *Community Dent Oral Epidemiol*, vol. 36, no. 5, Oct, pp. 383-391.

Borrell, LN & Crawford, ND 2012, 'Socioeconomic position indicators and periodontitis: examining the evidence', *Periodontol 2000*, vol. 58, no. 1, Feb, pp. 69-83.

Carlos, JP, Wolfe, MD & Kingman, A 1986, 'The extent and severity index: a simple method for use in epidemiologic studies of periodontal disease', *J Clin Periodontol*, vol. 13, no. 5, May, pp. 500-505.

Charlton, BG 1995, 'A critique of Geoffrey Rose's 'population strategy' for preventive medicine', *J R Soc Med*, vol. 88, no. 11, Nov, pp. 607-610.

Commission on Social Determinants of Health, WHO 2008, 'Closing the gap in a generation: Health equity through action on the social determinants of health. Final Report of the Commission on Social Determinants of Health', World Health Organization, Geneva.

Doyle, YG, Furey, A & Flowers, J 2006, 'Sick individuals and sick populations: 20 years later', *J Epidemiol Community Health*, vol. 60, no. 5, May, pp. 396-398.

Elani, HW, Harper, S, Allison, PJ, Bedos, C & Kaufman, JS 2012, 'Socio-economic inequalities and oral health in Canada and the United States', *J Dent Res*, vol. 91, no. 9, Sep, pp. 865-870.

Ferreira, MC, Dias-Pereira, AC, Branco-de-Almeida, LS, Martins, CC & Paiva, SM 2017, 'Impact of periodontal disease on quality of life: a systematic review', *J Periodontal Res*, Feb 08.

Galobardes, B, Lynch, J & Smith, GD 2007, 'Measuring socioeconomic position in health research', *Br Med Bull*, vol. 81-82, pp. 21-37.

International Federation of Medical Students' Federation, I 2011, 'Medical students’ perspective on the Rio Declaration', Rio de Janeiro, Brazil.

Irwin, LG, Siddiqi, A & Hertzman, C 2007, 'Early Child Development: A Powerful Equalizer', *Final Report for the World Health Organization's Commission on the Social Determinants of Health*.

Kassebaum, NJ, Smith, AGC, Bernabé, E, Fleming, TD, Reynolds, AE, Vos, T, Murray, CJL, Marcenes, W & Collaborators, GOH 2017, 'Global, Regional, and National Prevalence, Incidence, and Disability-Adjusted Life Years for Oral Conditions for 195 Countries, 1990–2015: A Systematic Analysis for the Global Burden of Diseases, Injuries, and Risk Factors', *Journal of Dental Research*, vol. 96, no. 4, pp. 380-387.

Kelly, JE & Engel, A 1969, 'Selected examination findings related to periodontal disease among adults', *Vital Health Stat 11*, no. 35, Sep, pp. 1-31.

Kuh, D & Ben-Shlomo, Y 1997, *A Life-course Approach to Chronic Disease Epidemiology*, Oxford University Press.

Kuh, D, Ben-Shlomo, Y, Lynch, J, Hallqvist, J & Power, C 2003, 'Life course epidemiology', *J Epidemiol Community Health*, vol. 57, no. 10, Oct, pp. 778-783.

Lynch J, Smith GD, Harper S, Hillemeier M, Ross N, Kaplan GA, et al, 2004. 'Is income inequality a determinant of population health? Part 1. A systematic review.' *Milbank Q.* vol. 82, no. 1, pp. 5–99.

Mackenbach, JP, Bos, V, Andersen, O, Cardano, M, Costa, G, Harding, S, Reid, A, Hemstrom, O, Valkonen, T & Kunst, AE 2003, 'Widening socioeconomic inequalities in mortality in six Western European countries', *Int J Epidemiol*, vol. 32, no. 5, Oct, pp. 830-837.

Mackenbach, JP, Cavelaars, AE, Kunst, AE & Groenhof, F 2000, 'Socioeconomic inequalities in cardiovascular disease mortality; an international study', *Eur Heart J*, vol. 21, no. 14, Jul, pp. 1141-1151.

Mejia, G, Armfield, JM & Jamieson, LM 2014, 'Self-rated oral health and oral health-related factors: the role of social inequality', *Aust Dent J*, vol. 59, no. 2, Jun, pp. 226-233.

Nicolau, B, Netuveli, G, Kim, JW, Sheiham, A & Marcenes, W 2007, 'A life-course approach to assess psychosocial factors and periodontal disease', *J Clin Periodontol*, vol. 34, no. 10, Oct, pp. 844-850.

Nicolau, B, Thomson, WM, Steele, JG & Allison, PJ 2007, 'Life-course epidemiology: concepts and theoretical models and its relevance to chronic oral conditions', *Community Dent Oral Epidemiol*, vol. 35, no. 4, Aug, pp. 241-249.

OECD 2011, *Divided We Stand: Why Inequality Keeps Rising*, OECD Publishing, Paris.

Petersen, PE & Baehni, PC 2012, 'Periodontal health and global public health', *Periodontol 2000*, vol. 60, no. 1, Oct, pp. 7-14.

Poulton, R, Caspi, A, Milne, BJ, Thomson, WM, Taylor, A, Sears, MR & Moffitt, TE 2002, 'Association between children's experience of socioeconomic disadvantage and adult health: a life-course study', *Lancet*, vol. 360, no. 9346, Nov 23, pp. 1640-1645.

Rose, G 1985, 'Sick individuals and sick populations', *Int J Epidemiol*, vol. 14, no. 1, Mar, pp. 32-38.

Sabbah, W, Tsakos, G, Chandola, T, Sheiham, A & Watt, RG 2007, 'Social gradients in oral and general health', *J Dent Res*, vol. 86, no. 10, Oct, pp. 992-996.

Sanders, AE, Slade, GD, Turrell, G, Spencer, AJ & Marcenes, W 2006, 'The shape of the socioeconomic-oral health gradient: implications for theoretical explanations', *Community Dent Oral Epidemiol*, vol. 34, no. 4, Aug, pp. 310-319.

Savage A, Eaton KA, Moles DR, Needleman I 2009, 'A systematic review of definitions of periodontitis and methods that have been used to identify this disease', J Clin Periodontol, vol. 36, pp. 458-467.

Schuch, HS, Peres, KG, Do, LG & Peres, MA 2015, 'Can socioeconomic trajectories during the life influence periodontal disease occurrence in adulthood? Hypotheses from a life-course perspective', *Med Hypotheses*, vol. 84, no. 6, Jun, pp. 596-600.

Sen, A 2002, 'Why health equity?', *Health Econ*, vol. 11, no. 8, Dec, pp. 659-666.

Sen, A 2008, 'Why and how is health a human right?', *Lancet*, vol. 372, no. 9655, Dec 13, p. 2010.

Sheiham, A 2009, 'Editorial - Closing the gap in a generation: Health equity through action on the social determinants of health. A Report of the WHO Commission on Social Determinants of Health (CSDH) 2008.', *Community Dental Health*, vol. 29, no. 1, pp. 2-3.

Singh, A, Harford, J, Schuch, HS, Watt, RG & Peres, MA 2016, 'Theoretical basis and explanation for the relationship between area-level social inequalities and population oral health outcomes – A scoping review', *SSM - Population Health*, vol. 2, 12//, pp. 451-462.

Steele, J, Shen, J, Tsakos, G, Fuller, E, Morris, S, Watt, R, Guarnizo-Herreno, C & Wildman, J 2015, 'The Interplay between socioeconomic inequalities and clinical oral health', *J Dent Res*, vol. 94, no. 1, Jan, pp. 19-26.

Thomson, WM, Shearer, DM, Broadbent, JM, Foster Page, LA & Poulton, R 2013, 'The natural history of periodontal attachment loss during the third and fourth decades of life', *J Clin Periodontol*, vol. 40, no. 7, Jul, pp. 672-680.

Thomson, WM, Sheiham, A & Spencer, AJ 2012, 'Sociobehavioral aspects of periodontal disease', *Periodontol 2000*, vol. 60, no. 1, Oct, pp. 54-63.

Tonetti, MS, Claffey, N & European Workshop in Periodontology group, C 2005, 'Advances in the progression of periodontitis and proposal of definitions of a periodontitis case and disease progression for use in risk factor research. Group C consensus report of the 5th European Workshop in Periodontology', *J Clin Periodontol*, vol. 32 Suppl 6, pp. 210-213.

Wadsworth, ME, Cripps, HA, Midwinter, RE & Colley, JR 1985, 'Blood pressure in a national birth cohort at the age of 36 related to social and familial factors, smoking, and body mass', *Br Med J (Clin Res Ed)*, vol. 291, no. 6508, Nov 30, pp. 1534-1538.

World Conference on Social Determinants of Health, W 2011, 'Rio Political Declaration on Social Determinants of Health', Rio de Janeiro, Brazil.

World Health Organization, W, Solar, O & Irwin, A 2010, 'A conceptual framework for action on the social determinants of health'.

CHAPTER 3 – METHODS

This chapter will present the theoretical framework and methods of the systematic review, and the 1982 Pelotas Birth Cohort Study, the population-based cohort study that comprised the empirical studies.

3.1 Systematic review

In order to identify, appraise, select and synthesise all high quality research evidence evaluating the impact of income trajectories on periodontal disease in adulthood, a systematic review was conducted. The research question that guided this systematic review is: 'Does socioeconomic position (SEP) earlier in life influence periodontal status in adulthood?' Socioeconomic position, in the review, was considered as taking measures of individual-level relative disadvantage into account. Potentially eligible papers were those addressing longitudinal epidemiological studies that included clinically assessed chronic periodontal disease as the outcome, and that measured SEP at least once prior to the outcome. We intended to identify all original studies that used a longitudinal perspective to study the effect of SEP earlier in life on periodontal disease later on, regardless of idiom, geographic location, or period of publication. To write the report of this systematic review, the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines for reporting systematic reviews were followed (Moher et al. 2009). The paper published from the systematic review can be found in Chapter 5 of this thesis.

3.1.1 Information sources and search strategies

Electronic searches were carried out by the PhD student in 6 bibliographic sources. The development of the search queries was previously structured with the assistance of the Research Librarian of The University of Adelaide, Mr Michael Draper, who has expertise in health research. The terms were organised into 3 groups: the first group of terms limited the design of the target studies, aiming to capture only studies with a longitudinal approach. The second group of terms was focused on the exposure adopted, and the socioeconomic position. Finally, the third group was defined based on periodontal disease, the outcome of interest of the systematic review.

3.1.2 Literature search and study selection

Searches were carried out by the PhD student, and the search results in each of the data sources were stored and managed with EndNote, version X6 for Windows. The final search was updated in 17th December 2015. Initially, references in duplicate were identified and removed, and the EndNote database was shared with the second reviewer. The second reviewer of the systematic review was a fellow researcher, Ankur Singh (A.S.). A.S. has a strong interest in socioeconomic inequalities in oral health outcomes. In parallel to the development of the present systematic review, A.S. also conducted a scoping review for his project, where the author of this thesis collaborated as second reviewer. Such a study, published in the *Social Sciences and Medicine – Population Health* Journal in 2016, is entitled ‘Theoretical basis and explanation for the relationship between area-level social inequalities and population oral

health outcomes – A scoping review', and can be found in the Appendices section of this thesis. The reading of the titles and abstracts of the papers was performed independently by two of the authors, aiming to eliminate clearly irrelevant reports. Discussions aiming to reach a consensus were the method adopted in case of disagreement regarding eligibility, followed by reading the article entirely if necessary. In cases where a consensus could not be reached, it was resolved by a third reviewer (Loc Do).

3.1.3 Data collection process

After that, the selected papers were fully read, and the papers that demonstrably matched the aims had their data extracted to a previously prepared and tested spreadsheet (Appendix Figure 1).

The information extracted was cross-checked by the authors and, when necessary, a new consultation was performed by reading the full texts again. To avoid double counting, data from multiple reports of the same study were identified. Aiming to identify and include any relevant grey literature, the literature search was supplemented with hand searches on reference lists of all relevant publications, textbooks and international health organisations' websites. As well, a standard letter was sent to life-course epidemiology researchers, asking for any relevant data to include in the present systematic review. The model of the letter that was sent to researchers can be viewed in the Appendices (Appendix Figure 2).

3.1.4 Quality assessment

The methodological quality assessment of the studies that met the eligibility criteria was performed by the two reviewers (H.S.S. and A.S.), using the Newcastle-Ottawa Quality Assessment Scale (NOS) for cohort studies (Wells et al. 2011). The scale ranged from 1 (poor quality) to 9 (high quality). Disagreements were resolved through discussion.

Table 1. Methodological quality assessment (Newcastle-Ottawa Scale)

Study	Selection	Comparability	Outcome	Total
Buchwald et al, 2013	3	2	3	8
Haas et al, 2012	4	2	3	9
Ismail et al, 1990	3	2	3	8
Lu et al, 2011	4	2	3	9
Machtei et al, 1999	1	0	3	4
Paulander et al, 2004	4	2	3	9
Poulton et al, 2002	4	2	3	9
Thomson et al, 2013	4	2	3	9

*Maximum scores: Selection = 4, Comparability = 2, Outcome = 3, Total = 9

3.1.5 Data analysis

Results from the different studies were combined qualitatively. The heterogeneity in exposure, as well as in periodontal disease examination and case definition, was considered as an impediment to pooling the studies' results in a statistical meta-analysis.

3.2 Population-based birth cohort study

3.2.1 Study population

Studies with prospective cohort design support a life cycle perspective – it assumes that the state of health at any age is the result, not only of current conditions, but also of an accumulation of conditions that have been incorporated throughout life. The 1982 Pelotas Birth Cohort Study was the data source used to conduct the empirical studies and to respond the Thesis Objectives 3 and 4.

Pelotas is a medium-sized city with a population of approximately 350,000 people, located in the southernmost state of Brazil. Brazil is part of the “BRICS countries”, which refers to Brazil, Russia, India, China and South Africa, since they are considered to be at a similar stage of newly advanced economic development. Brazil is the country with the 9th highest Gross Domestic Product (GDP) in the world (International Monetary Fund, 2017). Recent data showed that the unemployment rate in Brazil is of 4.9% of the labour force (2017), and 14.8% of adult population (25-64 year-old) had completed tertiary education (2015) (OECD, 2017A, 2017B).

Pelotas is a city in South Brazil, which is the Brazilian macro region with the relatively highest number of municipalities classified as high human development (65% of cities in South Brazil have a Human Development Index (HDI) between 0.700-0.799). When analysing into quintiles the income component of the HDI at National level, none of the 1191 municipalities in South Brazil is the poorest quintile of income. In fact, almost 80% of South Brazilian cities are in the two most

affluent quintiles of HDI income in the country. The mean wage in Pelotas in 2015 was of 2.8 national minimum wages (IBGE, 2017).

The baseline of this study is a population-based prospective cohort of births. In 1982, all hospital births that occurred in the city of Pelotas, were identified and 5,914 live babies, whose family lived in the urban area of the city, were weighed and their mothers interviewed. This population was followed up several times, and details on the methodology of the project have already been published (Peres et al. 2011; Victora & Barros 2006). Three dental assessments were done in this sample, at 15, 24 years and 31 years (Figure 1). Data from the 24 and 31 years dental assessments were used in the empirical studies. However, the outcome of the thesis, periodontal disease, was collected only in the last dental evaluation, the Oral Health Sub-study 2013 (OHS-2013).

All sub-studies of the 1982 Pelotas Birth Cohort Studies were approved by Ethics Committees. The Oral Health Sub-study 2013 was approved by the Human Ethics Research Committee of the Federal University of Pelotas, under protocol number 384.332.

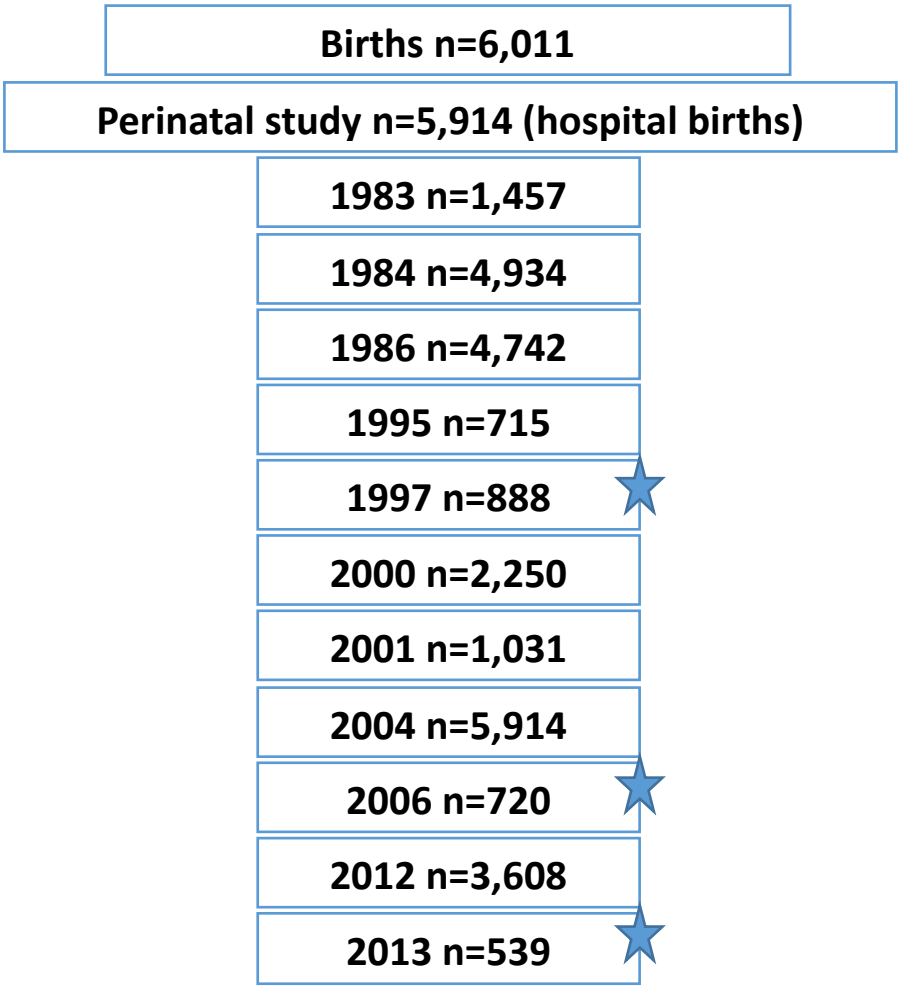

Figure 1. Main phases of the Pelotas birth cohort study.
★ Oral health sub-studies

3.2.2 Oral Health Sub-studies

3.2.2.1 Oral Health Sub-study 1997 (OHS-97)

When study participants were 15 years old, in 1997, the first Oral Health Sub-study was conducted (OHS-97). In order to select a sub-sample from the baseline study, a systematic sample of 20% of the census tracts in the city limits were visited (70 out of 259 census tracts), and 1,076 participants were located. Of these 1,076, 900 adolescents were randomly selected and invited to

participate in the OHS-97. This sample size was sufficient to estimate the prevalence rates for outcomes considered as unknown (P = 50%), with a sample error of 5 percentage points and 95% confidence interval.

The OHS-97 consisted of home visits to all participants by a dentist and an interviewer, with 888 of the 900 invited adolescents participating in the oral health sub-study. Data collection comprised a structured interview and a clinical oral examination. In the OHS-97, clinical information was collected on dental caries, malocclusions and oral lesions. Questionnaires assessed behaviours and oral hygiene habits, as well as use of dental services. Information on eating habits and non-nutritive sucking habits in childhood was also collected. The fieldwork lasted 12 weeks, from February to April 2006.

3.2.2.2 Oral Health Sub-study 2006 (OHS-06)

When participants were 24 years old, in 2006, all those who participated in the OHS-97 were invited to a new oral health sub-study (OHS-06). Following the methods used in the OHS-97, data collection was conducted through home visits and consisted of a structured interview and clinical oral examinations. The researchers were able to contact and to collect data from 720 individuals. Data collection was performed by six trained and calibrated dentists, and four trained interviewers – final year undergraduate students at the Dental School, Federal University of Pelotas.

Among the clinical conditions assessed were periodontal pockets, gingival bleeding and dental calculus. Additional collected information was on:

dental caries, use of and/or need for dental prostheses, quality of restorations in posterior teeth, and soft tissue lesions. Periodontal pockets, dental calculus and gingival bleeding were measured for all teeth present excluding third molars, six sites per tooth (three on the buccal side and three on the lingual or palatal side of each tooth). Periodontal pockets were classified as shallow if equal or greater than 5mm and deep if equal or greater than 6mm. Calculus and periodontal pockets at tooth level were then assessed, and the condition was considered present in that tooth if it was identified in at least one of the surfaces examined. The interviewers collected information on behaviours and oral hygiene habits, toothache experience in the last 4 weeks, and use of dental services.

Detailed methodological information on the OHS-97 and the OHS-06 can be found elsewhere (Peres et al. 2011).

3.2.2.3 Oral Health Sub-study 2013 (OHS-13)

The Oral Health Sub-study 2013 comprised the 888 individuals in the sample selected for the first study of oral health (OHS-97). The research team consisted of 6 PhD students, who performed the clinical oral examinations and 10 interviewers, who were undergraduate or Master Students. Two PhD students supervised fieldwork and a research assistant was hired to contact the participants and to purchase and organise the necessary research materials. The PhD candidate was one of the clinical examiners in the data collection.

The calibration exercises were conducted in individuals aged between 25 and 40 years old and who were not enrolled in the birth cohort study. A gold-

standard examiner was selected by the study co-ordinators. Each examiner was requested to conduct a complete oral clinical examination in 20 individuals, and the results were recorded by an interviewer. The results from the calibration process were combined, and the Kappas and Intraclass correlation coefficients were calculated for the different outcomes of interest. The inter-examiner final values were Intraclass correlation index of 0.85 for periodontal probing depth; Kappa 0.84 for use of and need for dental prosthesis; Kappa 0.65 for Dental Aesthetic Index (DAI); Kappa 0.89 for Decayed, Missing and Filling – Superficies (DMF-S).

Data collection

The data collection consisted of questionnaire and clinical oral examination. Time required for one participant complete data collection (including both questionnaire and examination) was around 25 minutes. The research assistant contacted all participants and organised a time for an interviewer and an examiner to visit the participant in his/her house and perform the data collection. The participants also had the option of coming to the Research Centre to be examined, since it is located in the city centre.

Questionnaire

The data collection included a face-to-face questionnaire, conducted by 10 previously trained interviewers. The questionnaire included questions on oral health services use and behaviours, as well as previous experiences with oral health and treatments, and oral and self-rated oral health.

As a quality control, 10% of the participants were randomly selected and contacted via phone, and 10 questions of the questionnaire were asked to check reliability of answers. The Kappa index for the quality control was over 0.80 for all questions, assuring high reliability of the questionnaire. The participants were also asked how satisfied they were with the data collection. They were asked to rate their experience from 0 (very unsatisfied) to 10 (very satisfied), and the mean score was 9.3.

Clinical oral examination

The clinical oral examinations were conducted following biosafety procedures recommended by the World Health Organization. Examiners used an artificial head-light, dental mirror, sterile gauze and PCP2 periodontal probe with 2-mm banding (Hu-Friedy PCP-2; Rotterdam, the Netherlands). Clinical oral examinations assessed 4 periodontal health indicators: bleeding on probing, supragingival dental calculus, gingival recession and periodontal pocket depth. The outcomes were measured by examining six sites on all teeth present, except third molars, and were collected with continuous measurements, in millimetres. Gingival recession was recorded as the distance from the cemento-enamel junction to the free gingival margin, with this being recorded as negative if there was gingival enlargement. Periodontal pocket depth was measured as the distance from the free gingival margin to the bottom of the pocket and recorded in positive values. Clinical attachment loss was computed as the sum of the gingival recession and the probing pocket depth. Also, the study

evaluated other clinical outcomes, such as dental caries, malocclusion and quality of posterior restorations.

Main exposure

The main exposure measured for this thesis is socioeconomic position, collected by means of household income. Income represented the sum of earnings by all people living in the cohort participant's house in the previous month. Income was categorised in tertiles for analytical purposes, and then dichotomised as relatively poorer (first income teritle) versus middle and higher income tertiles (2^{nd} and 3^{rd} tertiles). Income was collected during the life-course. Income at the time of the participant's birth was referred to as early life income, and income at age 23 was referred as adulthood income. Also, trajectories of income were drawn using income information from birth and ages 15, 19, 23 and 30. Other measures of socioeconomic position were additionally assessed and included in the statistical analysis, such as maternal education and years of formal education completed by the cohort participant. Both education variables were collected in number of years of study. Maternal education was collected at participant's birth and categorised as 0-4 years, 5-8 years and 9 or more years of study. A participant's education in adulthood was collected at age 30 and categorised as 0-8 years of study, 9-11 years and 12 or more years. In Brazil, up to 8 years of study corresponds to primary school, 9 to 11 years corresponds to high school, and 12 or more means more than high school. Information on

socioeconomic characteristics (education and income) was collected during different periods in the life-course, as displayed in Table 2.

Dependent variable

In the empirical studies of this thesis, periodontal disease was defined based on different combinations of measures of periodontal pockets and periodontal attachment loss (Savage et al. 2009). The prevalence of periodontal disease was measured adopting the criteria defined in partnership by the Center for Disease Control and Prevention (CDC) and the American Academy of Periodontology (AAP) (Eke et al. 2012; Page & Eke 2007).

Covariates

Considering that the present study follows a life-course approach, variables included were collected during distinct life periods. As covariates, I included participants' sex, smoking status, dental calculus, bleeding on probing, presence of periodontal pockets and dental flossing. Smoking status at age 23 was dichotomised as smoker (19.4%) and non-smoker (80.6%). Dental calculus was dichotomised as 0/1 tooth with dental calculus (25.6%) versus 2 or more (74.4%). Bleeding on probing was categorised as 0/1 tooth (79.4%) versus 2 or more (20.6%). Periodontal pocketing at age 24 was evaluated as absent (96.5%) or present in 1 or more teeth (3.5%). Finally, the use of dental flossing was categorised as 'yes or sometimes' (51.6%) and 'never' (48.6%).

The Directed Acyclic Graph (DAG) presented in Figure 2 represents the theoretical framework adopted by the researchers to guide the statistical analysis. In this study, SEP was the exposure, and it was collected by means of income in early life and income in adulthood (age 23). The baseline confounders in the causal relationship between early life income and adulthood income and periodontal disease at age 31 included sex and maternal education at participant's birth. Clinical conditions collected in adulthood, namely dental calculus and bleeding on probing were considered to be in the causal pathway between SEP and the outcome and therefore treated as mediators. Similarly, behavioural conditions (smoking status and dental flossing) and education attainment collected in adulthood were also considered to be in the causal pathway and therefore treated as mediators.

3.2.3 Statistical Analysis

Analyses of the empirical studies were conducted using Stata, version 14.0 (Stata Corporation, College Station, TX, USA). Detailed information on specific statistical approaches are presented within each manuscript.

Income trajectories were analysed as the main exposure in the first empirical study. In order to construct these trajectories, several statistical approaches could have been used. The most used are Growth Curve Modelling (GCM), Growth Mixture Modelling (GMM) and Group Based Trajectory Modelling (GBTM). They are all based on distinct technical assumptions about the distribution of trajectories in the population. For instance, GCM assumes

that all individuals in the population follow a similar functional form of development, and it models the population distribution of trajectories based on continuous distributions functions. GMM uses two or more GCMs to model population variability in the developmental trajectories. The basic outputs of the GMM are two or more growth curve models, each of which is interpretable in the same way as a single group GCM, and estimates of the proportion of the population following each such GCM. GBTM takes no stand on the population distribution of trajectories and, instead, uses the trajectory groups as a statistical device for approximating the unknown distribution of trajectories across population members. Group-based trajectory model differs of the Growth mixture model by not consider that the population is composed of distinct groups defined by their developmental trajectories (Nagin 2005; Nagin & Odgers 2010).

Group Based Trajectory Modelling was the technique adopted in this thesis (Nagin 2005; Nagin & Odgers 2010). GBTM aims to identify clusters of individuals with similar trajectories, and the model itself models the trajectories based on maximum likelihood estimation. Trajectories were drawn from 5 points during the life-course of 539 individuals, and the output from the software showed a mean number of income information of 4.8 per individual, confirming the low rate of missing information on the main explanatory variable. To construct the trajectories, the Logit distribution model was adopted; as well, the polynomial cubic, or third order polynomial type was used. To test the effect of income trajectories on periodontal disease, multivariable log-Poisson

Regression analyses with robust variance estimation were conducted. Model 1 included only socio-demographic characteristics, specifically the income trajectories, sex, maternal education at the participant's birth and the participant's education at age 30. Model 2 included all socio-demographic variables, behavioural variables at ages 23 and 24 and clinical variables at age 24. All variables were retained in Model 2, regardless of their P value. We also tested interactions between income trajectories with maternal education at participants' birth and participants' education on the occurrence of periodontal disease at age 31 in each of the models.

To test the critical period model, we used Marginal Structural Models (MSM) with Inverse Probability Weights (IPW). This technique is based on the counterfactual assumption and allows for the estimation of the controlled direct effect (CDE), which is the effect of exposure on outcome that is not mediated by later factors in the causal pathway (Robins, Hernan & Brumback 2000). MSM is a technique to analyse longitudinal data that differentiate between confounders and mediators in the analysis. Instead of conditioning on confounders, it uses measured covariates to give weights to exposed and non-exposed groups. By using IPW, it is possible to simulate randomisation using observational data. The exposures for this study were early life income and adulthood income. As mediators, three variables were included: smoking status at age 24, dental calculus (also collected at age 24 as an indicator of dental hygiene), and participants´ education by age 30. Stabilised weights of exposure were calculated based on the participant's sex. After that, the value of mediators was

fixed, while a comparison of the expected outcome was estimated, conditional on the exposure and the mediators for different values of the exposure (0 or 1). Multinomial Regression Models were then applied to estimate the CDE of early life income on periodontal disease in adulthood.

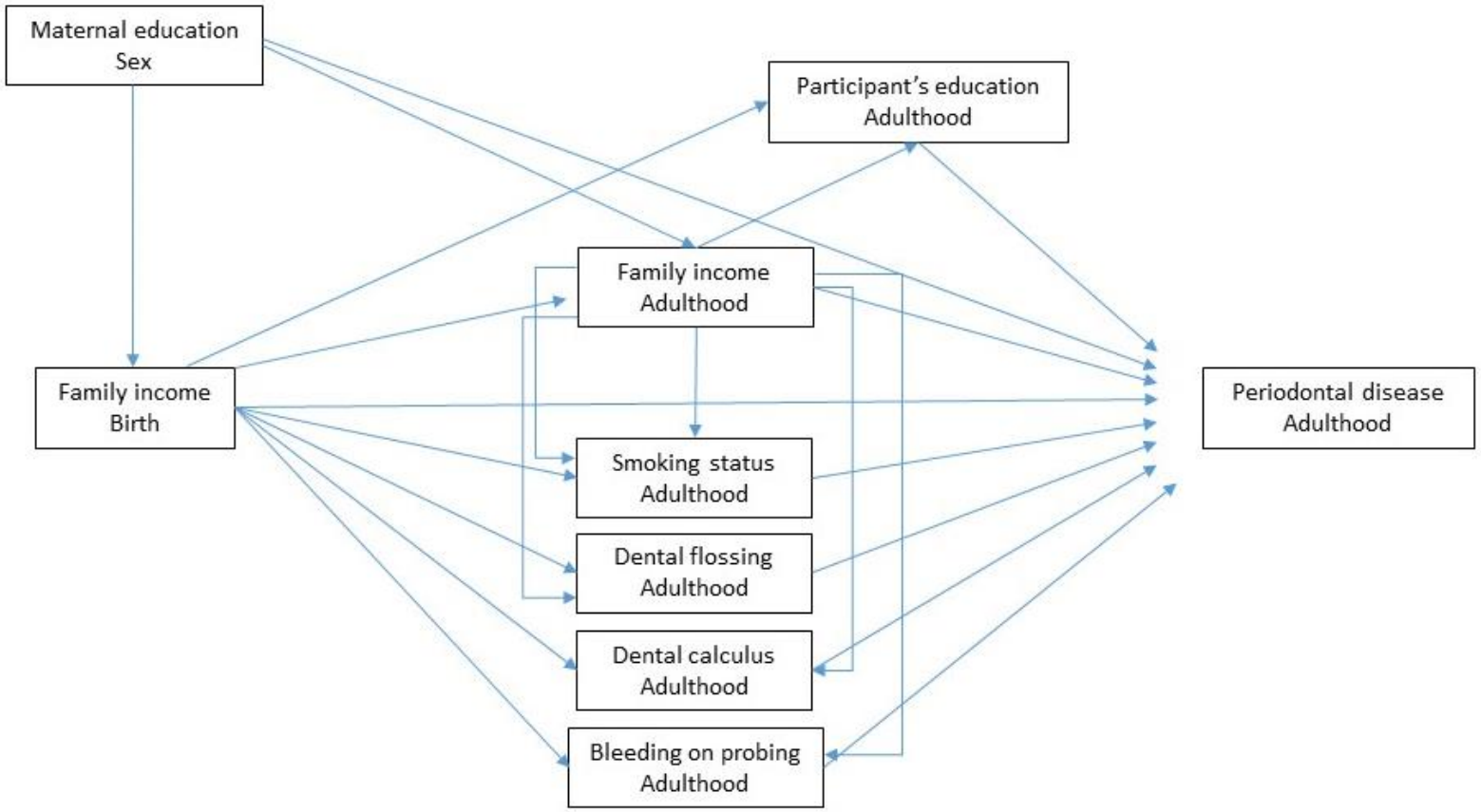

Figure 2. Directed acyclic graph (DAG) showing the conceptual framework of the thesis

Table 2. Main variables of interest collected in the cohort study.

Variables	**Birth**	**15 years**	**19 years**	**23 years**	**24 years**	**30 years**	**31 years**
SEP indicators							
Family income	■	■	■	■		■	
Maternal Education	■						
Participant's education						■	
Covariates							
Sex	■						
Smoking habits					■		
Dental flossing					■		
Bleeding on probing					■		
Dental calculus					■		
Periodontal pockets					■		
Outcome							
Periodontal disease							■

3.3 References

Ainamo, J, Barmes, D, Beagrie, G, Cutress, T, Martin, J & Sardo-Infirri, J 1982, 'Development of the World Health Organization (WHO) community periodontal index of treatment needs (CPITN)', *Int Dent J*, vol. 32, no. 3, Sep, pp. 281-291.

Baelum, V, Fejerskov, O, Manji, F & Wanzala, P 1993, 'Influence of CPITN partial recordings on estimates of prevalence and severity of various periodontal conditions in adults', *Community Dent Oral Epidemiol*, vol. 21, no. 6, Dec, pp. 354-359.

Baelum, V & Lopez, R 2013, 'Periodontal disease epidemiology - learned and unlearned?', *Periodontol 2000*, vol. 62, no. 1, Jun, pp. 37-58.

Baelum, V, Manji, F, Fejerskov, O & Wanzala, P 1993, 'Validity of CPITN's assumptions of hierarchical occurrence of periodontal conditions in a Kenyan population aged 15-65 years', *Community Dent Oral Epidemiol*, vol. 21, no. 6, Dec, pp. 347-353.

Baelum, V & Papapanou, PN 1996, 'CPITN and the epidemiology of periodontal disease', *Community Dent Oral Epidemiol*, vol. 24, no. 6, Dec, pp. 367-368.

Carlos, JP, Wolfe, MD & Kingman, A 1986, 'The extent and severity index: a simple method for use in epidemiologic studies of periodontal disease', *J Clin Periodontol*, vol. 13, no. 5, May, pp. 500-505.

Dentino, A, Lee, S, Mailhot, J & Hefti, AF 2013, 'Principles of periodontology', *Periodontol 2000*, vol. 61, no. 1, Feb, pp. 16-53.

Eke, PI, Page, RC, Wei, L, Thornton-Evans, G & Genco, RJ 2012, 'Update of the case definitions for population-based surveillance of periodontitis', *J Periodontol*, vol. 83, no. 12, Dec, pp. 1449-1454.

Johansen, JR, Gjermo, P & Bellini, HT 1973, 'A system to classify the need for periodontal treatment', *Acta Odontol Scand*, vol. 31, no. 5, pp. 297-305.

Instituto Brasileiro de Geografia e Estatística (IBGE), 2017. https://cidades.ibge.gov.br/brasil/rs/pelotas/panorama Accessed on 4th December 2017.

International Monetary Fund, World Economic Outlook Database. Accessed on 24th October 2017.

Moher, D, Liberati, A, Tetzlaff, J, Altman, DG & Group, P 2009, 'Preferred reporting items for systematic reviews and meta-analyses: the PRISMA Statement', *Open Med*, vol. 3, no. 3, pp. e123-130.

Nagin, DS 2005, *Group-Based Modeling of Development*, Harvard Univ. Press., Cambridge, MA.

Nagin, DS & Odgers, CL 2010, 'Group-based trajectory modeling in clinical research', *Annu Rev Clin Psychol*, vol. 6, pp. 109-138.

OECD 2017A, Unemployment rate (indicator). doi: 10.1787/997c8750-en. Accessed on 4th December 2017. https://data.oecd.org/unemp/unemployment-rate.htm

OECD 2017B, Adult education level (indicator). doi: 10.1787/36bce3fe-en. Accessed on 4th December 2017. https://data.oecd.org/eduatt/adult-education-level.htm

Page, RC & Eke, PI 2007, 'Case definitions for use in population-based surveillance of periodontitis', *J Periodontol*, vol. 78, no. 7 Suppl, Jul, pp. 1387-1399.

Peres, KG, Peres, MA, Demarco, FF, Tarquinio, SB, Horta, BL & Gigante, DP 2011, 'Oral health studies in the 1982 Pelotas (Brazil) birth cohort: methodology and principal results at 15 and 24 years of age', *Cad Saude Publica*, vol. 27, no. 8, Aug, pp. 1569-1580.

Poulton, R, Moffitt, TE & Silva, PA 2015, 'The Dunedin Multidisciplinary Health and Development Study: overview of the first 40 years, with an eye to the future', *Soc Psychiatry Psychiatr Epidemiol*, vol. 50, no. 5, May, pp. 679-693.

Robins, JM, Hernan, MA & Brumback, B 2000, 'Marginal structural models and causal inference in epidemiology', *Epidemiology*, vol. 11, no. 5, Sep, pp. 550-560.

Russell, AL 1956, 'A system of classification and scoring for prevalence surveys of periodontal disease', *J Dent Res*, vol. 35, no. 3, Jun, pp. 350-359.

Savage, A, Eaton, KA, Moles, DR & Needleman, I 2009, 'A systematic review of definitions of periodontitis and methods that have been used to identify this disease', *J Clin Periodontol*, vol. 36, no. 6, Jun, pp. 458-467.

Shearer, DM, Thomson, WM, Caspi, A, Moffitt, TE, Broadbent, JM & Poulton, R 2011, 'Inter-generational continuity in periodontal health: findings from the Dunedin family history study', *J Clin Periodontol*, vol. 38, no. 4, Apr, pp. 301-309.

Thomson, WM, Broadbent, JM, Welch, D, Beck, JD & Poulton, R 2007, 'Cigarette smoking and periodontal disease among 32-year-olds: a prospective study of a representative birth cohort', *J Clin Periodontol*, vol. 34, no. 10, Oct, pp. 828-834.

Thomson, WM, Sheiham, A & Spencer, AJ 2012, 'Sociobehavioral aspects of periodontal disease', *Periodontol 2000*, vol. 60, no. 1, Oct, pp. 54-63.

Tonetti, MS, Claffey, N & European Workshop in Periodontology group, C 2005, 'Advances in the progression of periodontitis and proposal of definitions of a periodontitis case and disease progression for use in risk factor research. Group C consensus report of the 5th European Workshop in Periodontology', *J Clin Periodontol*, vol. 32 Suppl 6, pp. 210-213.

Victora, CG & Barros, FC 2006, 'Cohort profile: the 1982 Pelotas (Brazil) birth cohort study', *Int J Epidemiol*, vol. 35, no. 2, Apr, pp. 237-242.

Wells, GA, Shea, B, D., OC, Peterson, J, Welch, V, Losos, M & Tugwell, P 2011, *The Newcastle-Ottawa Scale (NOS) for assessing the quality of nonrandomised studies in meta-analyses*, viewed 20/04/2015, <http://www.ohri.ca/programs/clinical_epidemiology/oxford.asp>.

CHAPTER 4 (PAPER 1)

Can socioeconomic trajectories during the life influence periodontal disease occurrence in adulthood? Hypotheses from a life-course perspective

Linkage to the body of work

The idea of this paper arose during the first year of PhD candidature, when the student was reviewing the literature on life-course epidemiology and periodontal disease. When reading about the life-course epidemiology theories, the natural exercise was to contextualise the theories to apply them to the research questions in the thesis. From that exercise, the first draft of the paper was developed. The paper starts by giving an overview and defining life-course epidemiology, periodontal disease and life-course epidemiology. The manuscript then focuses on discussing the pathways through which socioeconomic position during the life-course can affect periodontal disease in adulthood, by applying each of the life-course epidemiology theories. The final section of the paper summarises the theories presented, briefly discusses the most suitable life-course epidemiology theory for the research question, and indicates future directions of research on the topic.

Highlights

- The paper addresses theoretical explanations of the relationship between SEP and periodontal disease using each of the life-course theories.
- Although all theories are suitable to study such a relationship, the authors discuss the fact that the accumulation-of-risk theory is apparently the most appropriate life-course theory to explain the effect of life-course SEP on periodontal disease in adulthood.

Statement of authorship

Statement of Authorship

Title of Paper	Can socioeconomic trajectories during the life influence periodontal disease occurrence in adulthood? Hypotheses from a life-course perspective
Publication Status	☑ Published ☐ Accepted for Publication ☐ Submitted for Publication ☐ Unpublished and Unsubmitted work written in manuscript style
Publication Details	Schuch HS, Peres KG, Do LG, Peres MA. Can socioeconomic trajectories during the life influence periodontal disease occurrence in adulthood? Hypotheses from a life-course perspective. *Medical Hypotheses* 2015; 84: 596-600.

Principal Author

Name of Principal Author (Candidate)	Helena Silveira Schuch		
Contribution to the Paper	Performed literature search, analysed and interpreted, wrote the paper and acted as corresponding author.		
Overall percentage (%)	80%		
Certification:	This paper reports on original research I conducted during the period of my Higher Degree by Research candidature and is not subject to any obligations or contractual agreements with a third party that would constrain its inclusion in this thesis. I am the primary author of this paper.		
Signature		Date	03/07/2017

Co-Author Contributions

By signing the Statement of Authorship, each author certifies that:

i. the candidate's stated contribution to the publication is accurate (as detailed above);

ii. permission is granted for the candidate in include the publication in the thesis; and

iii. the sum of all co-author contributions is equal to 100% less the candidate's stated contribution.

Name of Co-Author	Karen Glazer Peres		
Contribution to the Paper	Supervised development of work, helped in interpretation, manuscript evaluation and edition.		
Signature		Date	03/07/2017

Name of Co-Author	Loc Giang Do		
Contribution to the Paper	Supervised development of work, helped in interpretation, manuscript evaluation and edition.		
Signature		Date	3/7/2017

Name of Co-Author	Marco Aurelio Peres		
Contribution to the Paper	Supervised development of work, helped in interpretation, manuscript evaluation and edition.		
Signature		Date	3/7/2017

Medical Hypotheses 84 (2015) 596–600

Contents lists available at ScienceDirect

Medical Hypotheses

journal homepage: www.elsevier.com/locate/mehy

Can socioeconomic trajectories during the life influence periodontal disease occurrence in adulthood? Hypotheses from a life-course perspective

H. S. Schuch *, K. G. Peres, L. G. Do, M. A. Peres

Australian Research Centre for Population Oral Health (ARCPOH), School of Dentistry, The University of Adelaide, Adelaide, SA, Australia

ARTICLE INFO

Article history:
Received 11 December 2014
Accepted 5 March 2015

ABSTRACT

Chronic periodontal disease (CPD) is a highly prevalent, multifactorial, bacterially induced inflammatory disease, characterized by pathologic loss of periodontal attachment and alveolar bone with onset mostly in adulthood. While cross-sectional data have demonstrated significant associations between adverse socioeconomic position (SEP) and poor periodontal conditions, there is a gap in the literature on the understanding of how SEPs in different life stages impact on the occurrence of this disease later on. Life-course epidemiology offers different theoretical models to study the pathway of health and illness during the lifespan, and the hypothesis of the present study is that the relationship between SEP and CPD can be explained based on different life-course epidemiology theories: (a) critical period model; (b) critical period with modifier effect model; (c) accumulation of risk model; (d) chain-of-risk model. Under the first theoretical model, the association between SEP and CPD may be explained by an inflammatory hypothesis, considering that childhood adverse socioeconomic backgrounds alter the immunoinflammatory response that leads to disease in adulthood regardless of conditions later in life. The second model postulates that the early life SEP modifies the host immunoinflammatory response, and the risk of disease will be modified over the life-course by socio-behavioural influences. The third, "accumulation of risk model", may explain such relationship taking into account exposures during different periods of life. However, this model does not consider the moment when the exposure occurred, only taking into consideration the number of episodes during the life cycle. Finally, the potential explanation to the role of socioeconomic position on chronic periodontal disease, using a chain-of-risk model, is that early low SEP may cause social stress related to social hierarchies, what may, in turn, trigger endocrine, neural and immune changes, that reflect on elevated levels of cytokines, consequently turning these individuals more likely to develop periodontal disease. To summarize, this paper suggests potential explanations of the relationship between SEP during the lifespan and the occurrence of chronic periodontal disease in adult life, under a life-course framework. Longitudinal studies focusing on such relationship should be conducted, aiming to provide evidence regarding the hypotheses here called in question.

Introduction

Life-course epidemiology is a theoretical framework that studies the pathway of health and illness during the lifespan. It aims to elucidate the influence of exposures during gestation, childhood, adolescence, early adulthood and across generations on the health later on [1]. A focus of such perspective has been to understand the interaction of biological, behavioural, and psychosocial process with health over time. This framework is especially useful to evaluate chronic diseases, including oral conditions (e.g., periodontal disease, oral cancer), considering that the majority of these diseases requires decades of exposure before it manifests as overt pathology, because the damage to the biological system caused by environmental hazards (e.g., stress) is slow and cumulative in nature [2].

Chronic periodontal disease is a multifactorial, bacterially induced inflammatory disease, characterized by pathologic loss of periodontal attachment and alveolar bone [3–5], which negatively impacts the health [6] and the quality of life in individual and population levels [7,8]. Considering the natural history of chronic periodontal disease, a relatively low ratio of damage to age is observed, resulting in a slow rate of progression [9]. Based on that,

* Corresponding author at: Australian Research Centre for Population Oral Health (ARCPOH), The University of Adelaide, Ground Floor, 122 Frome Street, Adelaide, SA, Australia. Tel.: +61 8 8313 31296; fax: +61 8 8313 34858.
E-mail address: helena.schuch@adelaide.edu.au (H. S. Schuch).

http://dx.doi.org/10.1016/j.mehy.2015.03.011
0306-9877/

it is uncommon to observe clinical manifestations of such disease in young people [10]. According to the Global Burden of Disease 2010 Study [11], severe periodontitis is the 6th most prevalent disease worldwide (global prevalence of 11% for all ages combined). After dental caries, periodontal disease is the leading cause of tooth loss among adults [12,13]. Some of its downstream risk factors are well known, including behaviours-related factors, such as tobacco use, inadequate oral hygiene; and health systemic conditions, such as uncontrolled diabetes and psychosocial stress [3,14]. Also, some factors have been identified as associated to higher prevalence of periodontal disease, such as excessive alcohol consumption [15], obesity and inadequate diet [16]. However, the current evidence is not sufficient to confirm if the latter factors are true risk factors for periodontal disease [17].

Socioeconomic position (SEP) is defined as "the relative position of a family or individual in a hierarchical social structure based on their access to or control over wealth, prestige and power" [18], which is usually measured by variables such as education, occupation, income, wealth and place of residence [19]. The role of SEP in health has been largely studied, focusing on systemic [20,21] and on oral health in general [22,23], and on periodontal health [4,24]. There is a consensus that the socioeconomic position plays a major role in the ill-health process, which individuals from adverse socioeconomic background presenting poorer health conditions than their counterparts. However, it remains unclear the precise role of socioeconomic variables during the life spans on periodontal disease; particularly the importance of the timing and length of exposure, as well as the mechanisms linking socioeconomic trajectories during the life-course and periodontal disease occurrence in adulthood.

In spite of the current knowledge about the relationship between socioeconomic position and periodontal outcomes [4,25], there is a gap in the literature on the understanding of how the socioeconomic position in different moments during the life-course impacts on the occurrence of chronic periodontal disease later on. Based on that, this study aims to explore how life-course epidemiology could be applied to elucidate the relationship between SEP and periodontal diseases. The hypothesis of the present study is that such relationship can be explained based on different life-course epidemiology theories: (a) critical period (programming model); (b) critical period with modifier effect model; (c) accumulation of risk model; (d) chain-of-risk model (see Fig. 1).

Critical period model

The 'critical period model' is when an exposure acting during a specific period has lasting or lifelong effects on the structure or function of organs, tissues, and body systems that are not modified in any dramatic way by later experience [1]. The hypothesis of critical period, in this case, is that children that grown in a low socioeconomic backgrounds are more likely to develop chronic periodontal disease in adulthood, in spite of conditions later in life.

There is growing evidence linking exposures to low socioeconomic position in childhood and occurrence of periodontal disease in adulthood. There is a theory hypothesizing that such an exposure may impact the stress hormones during sensitive periods of immune system maturation, what may in turn impair host immune responses, and afterward changing the response to an infectious challenge [26]. It should be highlighted that both the hypo-responsiveness and the hyper-responsiveness of the host immune response can result in tissue destruction, reinforcing the impact of an unbalanced immune system on periodontal outcomes [27]. In addition, the inflammatory hypothesis has been discussed, arguing that children from adverse socioeconomic backgrounds are more susceptible to inflammatory diseases in adulthood [28–31]. Considering that chronic periodontal disease consists of a low grade local infection associated with a moderate systemic inflammatory response [32], there is plausibility to assume that the elevated inflammatory markers in blood can be related to an unbalanced inflammatory response, which may, in turn, makes those individuals more susceptible to the occurrence of chronic periodontal disease. Likewise, it has been hypothesized that those elevated systemic markers in blood may acerbate other inflammatory processes in the same pathway, increasing the risk for systemic chronic diseases, such as cardiovascular and cerebrovascular diseases [32,33]. The current evidences, however, do not suggest which life stages could be critical in developing periodontal disease in adulthood.

Critical period with modifier effect model

This second model is an extension of the first. It proposes that the effect of exposures in early life may be modified by experiences over the life-course, either to enhance the effects on chronic disease ('synergism') or diminish them ('antagonism') [1]. The hypothesis linked to this model assumes a prominent role for early life exposures on adulthood health, as well as considers the range of potential processes through which exposures acting at different stages of life can, singly or in combination, influence disease risk.

This model may help to elucidate the relationship between socioeconomic position and periodontal disease. Individuals from adverse socioeconomic position in very early life will be more prone to develop periodontal disease later on, and exposures afterward could play a role in the development of the disease. The possibility of social mobility is suggested in this approach, since a downward socioeconomic trajectory may increase the susceptibility of one to periodontal disease, as well as an upward trajectory from poor childhood background to advantaged adult social position will assuage the effect of low socioeconomic background on health, increasing the resistance to periodontal disease. Moreover, in this evaluation, the role played by periodontal disease risk factors also identified as consequences of downward social mobility should be considered. In a population-based study conducted with a sample of 28,198 Swedish adults, a downward social mobility was associated significantly with higher odds of smoking [34], and the relationship between this habit and periodontal disease has been well established [3]. Although social mobility has usually been presented as part of the critical period with modifier effect model, such perspective has sometimes been linked to different main conceptual life course models. Cable [35], for example, pointed it out as incorporated into the accumulation of risk model. On the other hand, some researchers present the social mobility as one of the life course models, and often dividing such model in intra-generational and inter-generational mobility [36].

Based on the proposed hypothesis of the critical period model, the socioeconomic position in early life modifies the host immunoinflammatory response, and the present theoretical model proposes that the risk of disease will be modified over the life-course. It is well known that personal behaviors and characteristics, such as smoking status, oral hygiene and susceptibility to systemic diseases are closely related with socioeconomic position [1,12,37]. The critical period with modifier effect model considers the influence of these socio behavioural characteristics in mediating the relationship between socioeconomic position and periodontal disease. Indeed, according to Genco (2013), the recent understanding of the epidemiology of periodontal disease and the role of risk factors enables the comprehension that the rate of progression, age at onset, and severity of periodontal disease in an individual are often determined by systemic risk factors in the host

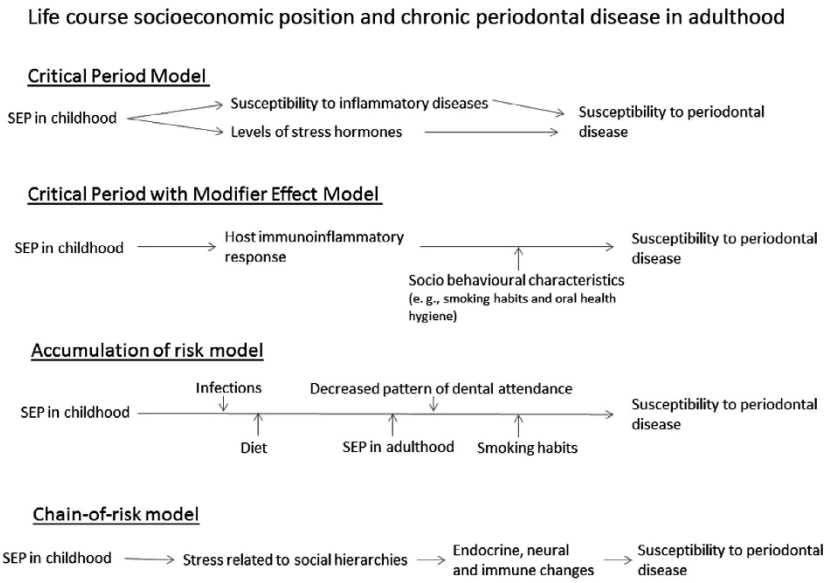

Fig. 1. Potential mechanisms of association between socioeconomic position during the life course and periodontal disease later on, according to 4 life course theories: critical period, critical period with modifier effect, accumulation of risk and chain-of-risk.

[3], and these factors are strongly influenced by the socioeconomic gradient. The current view of periodontitis is that it is a multifactorial disease that also is influenced by genetic and environmental risk factors [38], which is consistent with the critical period with modifier effect model.

Accumulation of risk model

The "accumulation of risk model" postulates that various biological and social factors throughout life independently, cumulatively and interactively influence health and disease in adult life [39]. Considering the association between socioeconomic position and periodontal disease, this model hypothesizes that those who were exposed to adverse socioeconomic situations over longer time will have considerably higher periodontal disease rates than those who had the same exposure for less time during the lifespan.

Indeed, this perspective enables the researcher to adopt a wider and richer pathway to explain the occurrence of periodontal disease. This model takes into account that children who grew up in adverse socioeconomic background are more likely to be exposed to infections and to have poor diet, for example, and these exposures could be related to chronic periodontal disease later on. Other situations have also been pointed out as possible mediators of the relationship between socioeconomic position and periodontal disease. The inverse association between socioeconomic position and smoking habits has been well documented [40], as well as the effects of such habits on periodontal outcomes [3,41]. Likewise, not only those who are lower in the socioeconomic hierarchy have poorer access to dental services, and also they are more likely to use those episodically (in response to crises such as a toothache or a periodontal abscess) rather than in a routine, preventive manner [12], and this decreased pattern of dental attendance on people from adverse socioeconomic backgrounds in turn may lead to a higher burden of periodontal disease.

In fact, in order to understand the socioeconomic differentials of an outcome it should be taken into consideration how a variety of exposures that increase the disease risk are influenced by social circumstances across the life-course and how this social dependence can lead to clustering of those exposures across time [1]. The different exposures during the life-course can lead to chronic periodontal disease later on. However, this model does not consider the moment when the exposure occurred, only the number of episodes during the life cycle. For example, the acquirement of hygiene habits can be impaired by adverse socioeconomic circumstances in childhood [42]; in the same way, there is evidence that smoking initiation, in essence, occurs during adolescence [43], and that adolescents from a low socioeconomic background are more prone to become smokers [38,44]; even more, low socioeconomic position during adulthood can lead to conditions that increase the susceptibility to periodontal disease, such as systemic diseases, i.e. cardiovascular diseases and diabetes mellitus [3,12,45]. Other conditions can also be linked to both socioeconomic position and chronic periodontal disease, although the period of life of its occurrence does not enhance or diminish its effects; this is the case of poor-quality restorative dentistry, for example. The conditions mentioned above are only some of the wide group of exposures that might be distributed in socially patterned ways and that can lead to periodontal disease. We theorise that these risk exposures, accumulated gradually and differentially, may increase the risk of chronic periodontal disease. However, it is not elucidated yet if the period of life when the socioeconomic exposition happens can shape the effect on health or if the major importance should be given to the number of exposures, irrespective of the timing.

Chain-of-risk model

The "chain-of-risk model" is a modified version of the of the accumulation model and is also been described as a 'pathway model' [1]. It argues that one bad experience or exposure tends

to lead to another and then another, in a probabilistic pathway rather than deterministic, creating a sequence of linked exposures that can lead to impaired function and increased risk of disease [39]. Socioeconomic position may be chained to periodontal outcomes, and a potential hypothesis to such relationship is based on the increased likelihood of a set of biological events and social adverse experiences over time presented by those from adverse socioeconomic background, what afterward can result in social inequalities in chronic periodontal disease.

There is a pathway of understanding such relationship that suggests the exposure to low socioeconomic position may cause stress related to social hierarchies, which may, in turn, trigger endocrine, neural and immune changes, that result in elevated levels of cytokines [26,46], consequently making these individuals more likely to develop periodontal disease. However, the influence of stress-related social position on health outcomes only was detected in non-primate human studies, and the authors argue that such generalisation of data from monkey studies to human societies does not appear warranted [47].

The most appropriate explanation to the role of socioeconomic position on chronic periodontal disease, using a chain-of-risk model, seem to be the concept that socioeconomic factors at different life stages may operate either via social chains of risk or by influencing exposures to causal factors at earlier life stages that form part of long term biological or psychological chains of risk [39]. This possible mechanism can be explored using the prospective study design with multiple data collection points and comprehensive pool of data.

Final considerations

The development of chronic periodontal disease usually extends over a long period of time, that is, there are time lags between exposure, disease initiation and clinical recognition [42]. Considering this characteristic of chronicity and the cumulative nature of periodontal disease, as well as of its risk factors, a life-course perspective seems to be appropriate to study this condition. Even though it is possible to identify pathways of the aetiology of periodontal disease in each of the models proposed by life-course epidemiology, the accumulation model has been identified as the most appropriate theoretical approach for life-course epidemiology investigation in periodontal health [12,42]. The present paper suggests potential explanations to the relationship between socioeconomic position during the lifespan and the occurrence of chronic periodontal disease in adult life, under a life-course approach. However, should be pointed out the difficulty in formal testing between the different life course models. Although there are some statistical techniques that allow such comparison [47–50], sometimes it is not possible to strictly separate the life course models, since they may operate together. This situation may be a barrier when independently testing the different hypotheses supported by the models.

It is well-known that studies with prospective cohort design support life cycle perspective, due to the accuracy of reported information and its long-term design. Therefore, longitudinal studies focusing on such relationship should be conducted, aiming to provide evidence regarding the hypotheses here called in question. Once the key mechanisms linking socioeconomic status during early life stages and periodontal disease in adulthood are elucidated, public policies and efforts can be implemented to target the more susceptible population groups at appropriate periods of life. Such programs may lead to better prevention of the disease and more effectively reduce socioeconomic inequalities in periodontal outcomes.

Conflict of interest statement

The authors report no financial or other conflict of interest relevant to the subject of this article.

Acknowledgements

The authors would like to thank the Brazilian government agency CAPES for the PhD scholarship (HSS – Process 13774-13-1).

References

[1] Kuh D, Ben-Shlomo Y. A life course approach to chronic disease epidemiology. New York: Oxford University Press; 2004.
[2] Nicolau B, Marcenes W. How will a life course framework be used to tackle wider social determinants of health? Community Dent Oral Epidemiol 2012;40 Suppl. 2:33–8.
[3] Genco RJ, Borgnakke WS. Risk factors for periodontal disease. Periodontol 2000;2013(62):59–94.
[4] Boillot A, El Halabi B, Batty GD, Range H, Czernichow S, Bouchard P. Education as a predictor of chronic periodontitis: a systematic review with meta-analysis population-based studies. PloS One 2011;6:e21508.
[5] Slots J. Periodontology: past, present, perspectives. Periodontol 2000;2013(62):7–19.
[6] Otomo-Corgel J, Pucher JJ, Rethman MP, Reynolds MA. State of the science: chronic periodontitis and systemic health. J Evid Based Dent Pract 2012;12:20–8.
[7] Aslund M, Pjetursson BE, Lang NP. Measuring oral health-related quality-of-life using OHQoL-GE in periodontal patients presenting at the University of Berne, Switzerland. Oral Health Prev Dent 2008;6:191–7.
[8] Shanbhag S, Dahiya M, Croucher R. The impact of periodontal therapy on oral health-related quality of life in adults: a systematic review. J Clin Periodontol 2012;39:725–35.
[9] Armitage GC, Cullinan MP, Seymour GJ. Comparative biology of chronic and aggressive periodontitis: introduction. Periodontol 2000;2010(53):7–11.
[10] Demmer RT, Papapanou PN. Epidemiologic patterns of chronic and aggressive periodontitis. Periodontol 2000;2010(53):28–44.
[11] Marcenes W, Kassebaum NJ, Bernabe E, et al. Global burden of oral conditions in 1990–2010: a systematic analysis. J Dent Res 2013;92:592–7.
[12] Thomson WM, Sheiham A, Spencer AJ. Sociobehavioral aspects of periodontal disease. Periodontol 2000;2012(60):54–63.
[13] Corbet EF, Leung WK. Epidemiology of periodontitis in the Asia and Oceania regions. Periodontol 2000;2011(56):25–64.
[14] Sheiham A, Watt RG. The common risk factor approach: a rational basis for promoting oral health. Community Dent Oral Epidemiol 2000;28:399–406.
[15] da Amaral C, Vettore S, Leao MV. A. The relationship of alcohol dependence and alcohol consumption with periodontitis: a systematic review. J Dent 2009;37:643–51.
[16] Hatem AE. Epidemiology and risk factors of periodontal disease. In: Manakil J, editor. Periodontal Diseases – A Clinician's Guide ;2012 p. 213–230.
[17] Van Dyke TE, Sheilesh D. Risk factors for periodontitis. J Int Acad Periodontol 2005;7:3–7.
[18] Mueller CW, Parcel TL. Measures of socioeconomic status: alternatives and recommendations. Child Dev 1981;52:13–30.
[19] Calixto OJ, Anaya JM. Socioeconomic status. The relationship with health and autoimmune diseases. Autoimmun Rev 2014;13:641–54.
[20] Braveman P, Gottlieb L. The social determinants of health: it's time to consider the causes of the causes. Public Health Rep 2014;129 Suppl. 2:19–31.
[21] Barros FC, Victora CG, Scherpbier R, Gwatkin D. Socioeconomic inequities in the health and nutrition of children in low/middle income countries. Rev Saude Publica 2010;44:1–16.
[22] Costa SM, Martins CC, Bonfim M de L, et al. A systematic review of socioeconomic indicators and dental caries in adults. Int J Environ Res Public Health 2012;9:3540–74.
[23] Peres MA, Peres KG, de Barros AJ, Victora CG. The relation between family socioeconomic trajectories from childhood to adolescence and dental caries and associated oral behaviours. J Epidemiol Community Health 2007;61:141–5.
[24] Klinge B, Norlund A. A socio-economic perspective on periodontal diseases: a systematic review. J Clin Periodontol 2005;32 Suppl. 6:314–25.
[25] Borrell LN, Crawford ND. Socioeconomic position indicators and periodontitis: examining the evidence. Periodontol 2000;2012(58):69–83.
[26] Nicolau B, Netuveli G, Kim JW, Sheiham A, Marcenes W. A life-course approach to assess psychosocial factors and periodontal disease. J Clin Periodontol 2007;34:844–50.
[27] Pihlstrom BL, Michalowicz BS, Johnson NW. Periodontal diseases. Lancet 2005;366:1809–20.
[28] Galobardes B, Lynch JW, Davey Smith G. Childhood socioeconomic circumstances and cause-specific mortality in adulthood: systematic review and interpretation. Epidemiol Rev 2004;26:7–21.

[29] Galobardes B, Smith GD, Lynch JW. Systematic review of the influence of childhood socioeconomic circumstances on risk for cardiovascular disease in adulthood. Ann Epidemiol 2006;16:91–104.
[30] Camelo LV, Giatti L, Neves JA, et al. Life course socioeconomic position and C-reactive protein: mediating role of health-risk behaviors and metabolic alterations. The Brazilian Longitudinal Study of Adult Health (ELSA-Brasil). PloS One 2014;9:e108426.
[31] Poulton R, Caspi A, Milne BJ, et al. Association between children's experience of socioeconomic disadvantage and adult health: a life-course study. Lancet 2002;360:1640–5.
[32] Kalburgi V, Sravya L, Warad S, Vijayalaxmi K, Sejal P, Hazeil D. Role of systemic markers in periodontal diseases: a possible inflammatory burden and risk factor for cardiovascular diseases? Ann Med Health Sci Res 2014;4:388–92.
[33] Libby P, Ridker PM, Maseri A. Inflammation and atherosclerosis. Circulation 2002;105:1135–43.
[34] Lindstrom M, Moden B, Rosvall M. A life-course perspective on economic stress and tobacco smoking: a population-based study. Addiction 2013;108:1305–14.
[35] Cable N. Life course approach in social epidemiology: an overview application and future implications. J Epidemiol 2014;24(5):347–52.
[36] Niedzwiedz CL, Katikireddi SV, Pell JP, Mitchell R. Life course socio-economic position and quality of life in adulthood: a systematic review of life course models. BMC Public Health 2012 Aug;9(12):628.
[37] Scambler G. Health inequalities. Sociol Health Illn 2012;34:130–46.
[38] Kornman KS. Mapping the pathogenesis of periodontitis: a new look. J Periodontol 2008;79:1560–8.
[39] Kuh D, Ben-Shlomo Y, Lynch J, Hallqvist J, Power C. Life course epidemiology. J Epidemiol Community Health 2003;57:778–83.
[40] Overland S, Tjora T, Hetland J, Aaro LE. Associations between adolescent socioeducational status and use of snus and smoking. Tob Control 2010;19:291–6.
[41] Sakki TK, Knuuttila ML, Vimpari SS, Hartikainen MS. Association of lifestyle with periodontal health. Community Dent Oral Epidemiol 1995;23:155–8.
[42] Nicolau B, Thomson WM, Steele JG, Allison PJ. Life-course epidemiology: concepts and theoretical models and its relevance to chronic oral conditions. Community Dent Oral Epidemiol 2007;35:241–9.
[43] Ajdacic-Gross V, Landolt K, Angst J, et al. Adult versus adolescent onset of smoking: how are mood disorders and other risk factors involved? Addiction 2009;104:1411–9.
[44] Hanson MD, Chen E. Socioeconomic status and health behaviors in adolescence: a review of the literature. J Behav Med 2007;30:263–85.
[45] Jaffiol C, Thomas F, Bean K, Jego B, Danchin N. Impact of socioeconomic status on diabetes and cardiovascular risk factors: results of a large French survey. Diabetes Metab 2013;39:56–62.
[46] Sapolsky RM. The influence of social hierarchy on primate health. Science 2005;308:648–52.
[47] Petticrew M, Davey Smith G. The monkey puzzle: a systematic review of studies of stress, social hierarchies, and heart disease in monkeys. PloS One 2012;7:e27939.
[48] Mishra G, Nitsch D, Black S, De Stavola B, Kuh D, Hardy R. A structured approach to modelling the effects of binary exposure variables over the life course. Int J Epidemiol 2009 Apr;38(2):528–37.
[49] Robins JM, Blevins D, Ritter G, Wulfsohn M. G-estimation of the effect of prophylaxis therapy for Pneumocystis carinii pneumonia on the survival of AIDS patients. Epidemiology 1992 Jul;3(4):319–36.
[50] Robins JM, Hernan MA, Brumback B. Marginal structural models and causal inference in epidemiology. Epidemiology 2000 Sep;11(5):550–60.

CHAPTER 5 (PAPER 2)

Socioeconomic position during life and periodontitis in adulthood: a systematic review

Linkage to the body of work

The systematic review was the first paper proposed for the PhD project. During the first stages of drafting the research proposal, it was recognised that it was necessary to understand what had been done in the literature on the relationship between socioeconomic position and periodontal disease, and so this present systematic review was outlined. Developing the systematic review, the researchers systematically identified all papers published in the scientific literature on the topic. This also allowed the researchers to identify and discuss how different SEP indicators relate to periodontal disease. As a conclusion, it was observed that SEP earlier in life has detrimental effects on periodontal disease later on, despite the SEP measure adopted.

Highlights

- There is a lack of studies with a longitudinal approach studying the effects of SEP on periodontal disease in adulthood.
- The majority of studies with a longitudinal approach have had a short-term follow-up between 5 and 11 years.
- From the 8 papers based on 7 studies found in the literature, it was summarised that lower SEP earlier in life increases the probability of periodontal disease occurrence and progression later on, regardless of the SEP measure adopted by the study.

Statement of authorship

Statement of Authorship

Title of Paper	Socioeconomic position during life and periodontitis in adulthood: a systematic review
Publication Status	☑ Published ☐ Accepted for Publication ☐ Submitted for Publication ☐ Unpublished and Unsubmitted work written in manuscript style
Publication Details	Schuch HS, Peres KG, Singh A, Peres MA, Do LG. Socioeconomic position during life and periodontitis in adulthood: a systematic review. *Community Dentistry and Oral Epidemiology.* 2017 Jun;45(3):201-208.

Principal Author

Name of Principal Author (Candidate)	Helena Silveira Schuch
Contribution to the Paper	Performed literature review, analysed and interpreted data, wrote manuscript and acted as corresponding author.
Overall percentage:	80%
Certification:	This paper reports on original research I conducted during the period of my Higher Degree by Research candidature and is not subject to any obligations or contractual agreements with a third party that would constrain its inclusion in this thesis. I am the primary author of this paper.
Signature	Date 03/07/2017

Co-Author Contributions

By signing the Statement of Authorship, each author certifies that:

i. the candidate's stated contribution to the publication is accurate (as detailed above);
ii. permission is granted for the candidate in include the publication in the thesis; and
iii. the sum of all co-author contributions is equal to 100% less the candidate's stated contribution.

Name of Co-Author	Karen Glazer Peres
Contribution to the Paper	Supervised development of work, helped in interpretation, manuscript evaluation and edition.
Signature	Date 03/07/2017

Name of Co-Author	Ankur Singh
Contribution to the Paper	Analysed and interpreted data, critically evaluated and edited manuscript.
Signature	Date 3/07/2017

Name of Co-Author	Marco Aurelio Peres		
Contribution to the Paper	Supervised development of work, helped in interpretation, manuscript evaluation and edition.		
Signature		Date	3/7/2017

Name of Co-Author	Loc Giang Do		
Contribution to the Paper	Supervised development of work, helped in interpretation, manuscript evaluation and edition.		
Signature		Date	3/7/2017

Received: 27 April 2016 | Accepted: 27 November 2016

DOI: 10.1111/cdoe.12278

UNSOLICITED SYSTEMATIC REVIEW

WILEY Community Dentistry and Oral Epidemiology

Socioeconomic position during life and periodontitis in adulthood: a systematic review

Helena Silveira Schuch | Karen G. Peres | Ankur Singh | Marco A. Peres | Loc G. Do

Australian Research Centre for Population Oral Health (ARCPOH), Adelaide Dental School, The University of Adelaide, Adelaide, South Australia, Australia

Correspondence
Helena Silveira Schuch, Australian Research Centre for Population Oral Health (ARCPOH), The University of Adelaide, Adelaide, South Australia, Australia.
Email: helena.schuch@adelaide.edu.au

Abstract

Socioeconomic position (SEP) is a well-known risk indicator for chronic periodontitis. However, it is still unclear how SEP during the life course influences periodontal outcomes in adulthood. This study aimed to systematically review longitudinal studies investigating the influence of individual-level SEP during the life course on subsequent periodontitis in adulthood. Inclusion criteria were epidemiological longitudinal observational studies, in which indicators of relative SEP were assessed prior to clinical assessment of periodontitis. Six electronic databases (PubMed, EMBASE, Web of Science, Scopus, Latin American and Caribbean Health Sciences Literature (LILACS) and ScieLO) were searched. The methodological quality of the studies was assessed using the Newcastle-Ottawa Quality Assessment Scale (NOS). The search identified 1720 papers. After removal of duplicates (n=697), title and abstract screening (n=996), and full-text review (n=19), eight original manuscripts from seven studies were finally included. Sample sizes ranged from 167 to 2806, and the follow-up time from exposure to outcome ranged from 2 to 28 years. Studies evaluated education, occupation or income as SEP indicators. Prevalence, extent and severity of periodontal attachment loss, probing pocket depth and alveolar bone loss were the studied outcomes. Based on NOS, studies presented low risk of bias. Six of eight papers reported that relatively low SEP earlier in life was associated with poorer periodontal health in adulthood. The available scientific evidence demonstrates potential longitudinal impact of earlier lower SEP on later periodontal health. The findings were consistent despite differences in study methods.

KEYWORDS
income, longitudinal studies, periodontal diseases, review, socioeconomic factors

1 | INTRODUCTION

Severe periodontitis was ranked as the 6th most prevalent condition within the Global Burden of Disease 2010 Study, with an estimated prevalence of 10.8%.[1] Well-recognized risk factors for periodontitis include smoking, poorly controlled diabetes and some periodontopathogens.[2] Moreover, lower socioeconomic position (SEP) is a well-identified risk indicator for periodontitis,[3,4] at both contextual[5] and individual levels.[6]

Evidence suggests that SEP earlier in life negatively impacts on the incidence of chronic diseases such as respiratory and cardiovascular diseases (CVD).[7,8] These chronic diseases share several risk indicators with periodontitis, especially health-related behaviours, such as tobacco use,[9] excessive alcohol consumption,[10] unhealthy diet and lack of physical activity.[11] Therefore, it is plausible that the pathways linking SEP and general health could also be applied to oral health. Several potential explanations for the life-course effects of SEP on periodontitis are hypothesized including both behavioural and biological pathways.[12] However, a significant gap persists in understanding the role of SEP in the onset and progression of periodontitis.

Studies on the associations between SEP and periodontitis have most commonly investigated the role of education, occupation,

income, wealth and place of residence, as indicators of SEP.[5,13-15] These studies consistently report social gradients between SEP and periodontal health: the higher the relative SEP, the better the health.[16] Consistently, the literature clearly indicates that SEP is an important risk indicator for periodontitis.

Three systematic reviews have assessed the association between SEP and periodontitis and discussed possible causal relationships.[17-19] Although causality criteria proposed by Bradford Hill[20] have important limitations,[21] it remains in popular usage among most health scientists.[22] Among these criteria, previous systematic reviews have provided evidence for strength of association, consistency of association and dose-response relationship. However, as these systematic reviews mostly reported findings from cross-sectional studies, they cannot provide evidence from a longitudinal perspective. Therefore, evidence related to the temporal relationship is a key to fill the current gap in causal inference. Consequently, this study aimed to systematically review longitudinal studies that investigated influence of individual-level SEP during the life on subsequent periodontitis in adulthood.

2 | METHODS

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) statement and the Cochrane Handbook.[23,24] The review question that guided this systematic review was as follows: "Does SEP earlier in life influence periodontal status in adulthood?"

Potentially eligible papers comprised of all epidemiological observational longitudinal studies that included clinically assessed indicators of periodontitis as outcomes and measured indicators of relative individual-level SEP. Both prospective and retrospective longitudinal studies were considered eligible for inclusion. This review considered studies that clinically assessed periodontitis in adulthood, including at least one of the following measures: clinical attachment loss, periodontal pocket deep and alveolar bone loss, irrespective of periodontitis case definitions. Studies that used only self-reported measures of periodontitis were excluded. The exposure (SEP) should have been collected at least once prior to the outcome by means of education, income and/or occupation. The search did not have any language restriction. Publication date was not considered as a reason for papers' exclusion in this review.

The search was performed in six electronic databases (PubMed, EMBASE, Web of Science, Scopus, LILACS and Scientific Electronic Library Online (SciELO) until 17th December 2015.

Additionally, aiming to search for potentially unidentified publications, hand searches were conducted on the reference lists of papers included in the systematic review. A standard letter seeking new relevant data was also sent to the authors of the included studies to capture any unpublished data.

Search strategy for each database can be found in the supporting information section (A).

Step 1: Electronic searches were performed by the first author.

Step 2: Results from the six databases were combined using the EndNote (×6) software, and duplicate references were identified and removed.

Step 3: Titles and abstracts were first screened independently by two authors (H.S.S. and A.S.). If the necessary information for inclusion or exclusion of the article was unavailable in the title and/or abstract, the full text of the report was read. In case of disagreements regarding eligibility, the two reviewers attempted to reach a consensus through discussions. Persistent disagreements were resolved by the third reviewer (L.G.D.).

Step 4: Full texts of the selected abstracts in Step 3 were retrieved. The authors were contacted if the document was not available online. The papers were read by two reviewers, checking whether they matched the inclusion criteria for this systematic review. In case of exclusion after full-text review, the reasons were recorded by the reviewers (Table S1).

Step 5: Data extraction from the included papers was performed independently by the two reviewers.

2.1 | Data collection process

The selected papers underwent data extraction using a pretested spreadsheet. Data from multiple reports of the same study were identified to avoid duplication. Data regarding study identification and characteristics were defined by author and journal information, participants' characteristics, location and recruitment method. Detailed information on the exposure and the outcome was also recorded. The findings of each study were collected as stated within the paper, along with the information on covariates, confounding factors and effect modifiers. Finally, the study limitations and the key conclusions of studies were collected verbatim.

The methodological quality assessment of the selected studies was performed independently by two reviewers (H.S.S. and A.S.) using the Newcastle-Ottawa Quality Assessment Scale (NOS) for cohort studies.[25] The assessment of bias included criteria on Selection, Comparability and Outcome features. A study was awarded a maximum of one point for each item within the Selection and Outcome categories (totalizing a maximum of four and three points in each category, respectively).[26] A maximum of two points were given for Comparability item. Disagreements were resolved through discussion.

3 | RESULTS

The combined search across the six databases identified 1720 studies. After duplicate removal, screening of titles and abstracts and full-text examinations, eight original papers involving seven primary studies were included in this review and underwent data extraction. The stepwise process of study selection is summarized in Figure 1.

The characteristics of the selected studies are described in Table 1. Five of the eight selected studies were from high-income

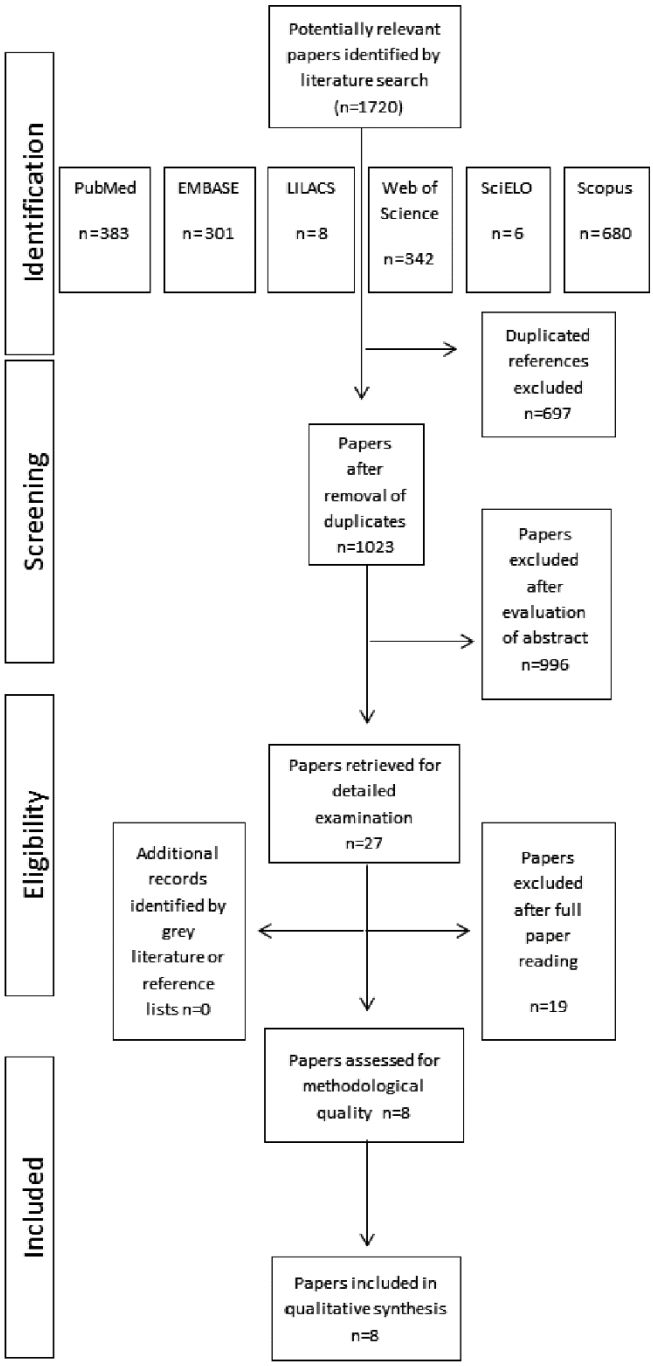

FIGURE 1 Flow chart of studies selection for the systematic review

TABLE 1 Main characteristics of the included papers

First author, (y)	Country	Journal	Sampling characteristics	n (followed up)	Time frame (from exposure to follow-up; y)
Buchwald, (2013)[16]	Germany	J Clin Periodontol	20- to 79-y-old in the north-east of Germany	2806	5
Haas, (2012)[33]	Brazil	J Clin Periodontol	14-103 y (baseline) from Porto Alegre, Brazil	697	5
Ismail, (1990)[30]	USA	J Dent Res	Noninstitutionalized individuals, 5 y of age or older at baseline, residing within an 80-km radius of Tecumseh (Michigan, US).	167	28
Lu, (2011)[32]	China	Community Dent Oral Epidemiol	Follow-up of children who had participated in the Hong Kong Oral Health Survey in 2001 (18 y old during follow-up)	221	6
Machtei, (1999)[31]	USA	J Clin Periodontol	Age range: 25-75 y at baseline	415	2-4
Paulander, (2004)[27]	Sweden	J Clin Periodontol	60 y old, living in the County of Varmland, Sweden	295	10
Poulton, (2002)[28]	New Zealand	The Lancet	Birth to 26 y	931	11
Thomson, (2013)[29]	New Zealand	J Clin Periodontol	Birth to 38 y old during the last evaluation	831	23

countries (one each from Germany, Sweden, New Zealand and two from USA), compared to two from middle-/low-income countries (Brazil and China). The combined sample size of this systematic review was 6363, and sample sizes of the individual studies ranged from 167 to 2806. The follow-up time varied from 2 to 28 years, with the majority between 5 and 11 years.

Details of the socioeconomic indicators adopted by each study and the periodontitis case definitions are presented in Table 2. Education as the sole measure of SEP was adopted by one paper[27] and occupation by another two papers, both from the same cohort study.[28,29] Four studies used both education and household income,[16,30-32] and one study defined SEP by combining information about family income and individual educational level.[33] Measures of periodontitis varied considerably among the studies. The assessed outcomes included periodontal attachment loss (PAL), probing pocket depth and alveolar bone loss. One study assessed periodontal health status using the Community Periodontal Index (CPI),[32] which is a measure including gingival bleeding, dental calculus and periodontal pockets.[34]

Table 3 summarizes the findings of the selected studies on SEP and periodontal outcomes. Two papers reported odds ratios,[28,30] three reported β-coefficient,[16,31,32] one reported relative risk,[27] one showed the mean annual proximal PAL progression[33] and another reported crude associations between childhood SEP and trajectories of periodontitis.[29] Due to the observed heterogeneity in the choice of exposure and outcome variables, and the summary measures between the included studies, a meta-analysis was not possible. Thus, a narrative synthesis was adopted for summarizing the findings, based on the guidelines by Mulrow et al.[35]

Six of the eight selected studies reported that relatively lower SEP earlier in life was associated with poorer periodontal health. Significant associations between SEP and periodontitis were not reported in two studies, which did not account for potential confounding.[27,32] Five of the eight papers assessed SEP during adulthood, before the outcome. Of the three papers that assessed SEP during childhood, one did not find a significant association.[32] The other two papers were from the same study, and reported inverse associations between SEP and periodontitis.[28,29] Half of the included studies reported unadjusted associations between SEP and periodontal outcomes. The four studies which reported adjusted estimates analysed the data using multivariable regression models.[16,27,28,31] The study conducted by Paulander and colleagues did not report a statistically significant association between SEP and periodontitis.[27] However, after stratification by smoking status and adjustment for confounders, their analysis found an inverse association between education attainment and alveolar bone level loss among individuals who had never smoked.

According to NOS, most of the selected studies were considered to be of high quality, while only one study was of low methodological quality (Figure S1). Most studies failed to comply with the NOS criterion related to sample selection procedures.

4 | DISCUSSION

The findings of this systematic review showed that lower SEP during life increased the probability of future occurrence and progression of periodontitis, regardless of the SEP measure. These findings are in accordance with previous systematic reviews.[17-19] However, to the best of the authors' knowledge, this is the first systematic review using evidence entirely from longitudinal studies.

Some strengths of this systematic review should be emphasized. First, the search strategy was broadly developed. The electronic search was performed in six databases of interest in the dental field, and all the steps of this review were performed in duplicate, to avoid individual bias. Second, the quality assessment confirmed the high quality of the included studies, reinforcing the overall finding of the review. Finally, the included studies presented a broad range of socioeconomic measures as well as different assessment of periodontal outcomes, and our findings were consistent despite such variations.

TABLE 2 Descriptive information on SEP and periodontal outcome in the selected papers

	Socioeconomic exposure		Periodontal outcomes			
First author, (y)	Type of exposure	Exposure details	Primary outcome	Case definition	Summary association measures	Comment
Buchwald, (2013)[16]	Education and income	Education: according to the final school grade; Income: monthly household income	Clinical Attachment Loss (CAL)	Distance between the cement-enamel junction (CEJ) and the pocket base rounded to whole millimetres	ß-coefficients	Worked with disease progression
Haas, (2012)[33]	SEP	SEP defined by combining information about family income and individual's educational level	PAL	The distance from the CEJ to the bottom of the pocket/sulcus	Mean annual proximal PAL progression (mm) for all sites	Worked with disease progression
Ismail, (1990)[30]	Education and income	Not described	Pocket depth and periodontal attachment level	Those with a mean loss of periodontal attachment (LPA) difference of less than 2 mm over the 28 y were classified as "low LPA", while those with a mean LPA difference of 2 mm or more were classified as the "high-LPA" group	Odds ratios	Worked with disease progression (low- and high-LPA differences between the follow-up and baseline examinations)
Lu, (2011)[32]	Education and income	Education: Parent's educational attainment; Income: monthly household income	Periodontal health status was assessed by the CPI	Highest CPI score	ß-coefficients	
Machtei, (1999)[31]	Education and income	Education: High School/College; Income: Maximum annual household income	Probing pocket depth (PPD)	The distance from the free gingival margin (FGM) to the clinical base of the pocket in a line parallel to the long axis of the tooth using a Florida Probe System (FPS) with a 10-mm pocket probe	Coefficient/ Correlation	The regression assessed annual change in pocket depth
Paulander, (2004)[27]	Education	Educational level: (i) low-educated individuals (only compulsory school training), or (ii) high-educated individuals (more than compulsory school training)	Alveolar bone loss	Not well defined	Relative Risk (RR)	Worked with Relative Risk (RR) for a mean 10-y ABL loss
Poulton, (2002)[28]	Occupation	Childhood socioeconomic status is the average of the highest socioeconomic status level of either parent, assessed repeatedly at the study member's birth and at ages 3, 5, 7, 8, 11, 13 and 15 y	Periodontal disease	Periodontal disease was judged present if there was more than 4 mm loss of periodontal attachment for at least one site	Odds ratios	Presented only adjusted OR
Thomson, (2013)[29]	Occupation	Childhood socioeconomic status is the average of the highest socioeconomic status level of either parent, assessed repeatedly at the study member's birth and at ages 3, 5, 7, 8, 11, 13 and 15 y	Changes in the occurrence of PAL	The CAL for each site was computed by summing the GR and PD measurements. The changes in CAL between 26 and 32 (and between 32 and 38) were determined for each site by subtracting the later age's CAL from that of the earlier one	Crude association between childhood SES and trajectories of periodontal disease up to age 38 y	Same study of Poulton 2002; Worked with periodontal disease trajectory

CPI, Community Periodontal Index; GR, Gingival recession; PD, Pocket depth; PAL, periodontal attachment loss; SEP; Socioeconomic position.

TABLE 3 Summary of the findings of the included papers

First author, (y)	Main findings
Buchwald, (2013)[16]	Low-education and low-income levels were associated with progression of mean clinical attachment level ($P<.010$ and $P=.046$, respectively)
Haas, (2012)[33]	Subjects of low SES showed higher rates of disease progression than subjects of high SES, respectively
Ismail, (1990)[30]	On the basis of bivariate analyses, the individuals with high-LPA increase presented lower education level compared to those with low-LPA increase
Lu, (2011)[32]	Both the household income level and parental education level for children aged 12 y had no significant effects on highest CPI score at ages 15 and 18 y old ($P>.05$)
Machtei, (1999)[31]	In the stepwise multiple regression analysis, income was predictive of annual change in pocket depth after controlling for baseline probing pocket depth, smoking, thyroid disorder and presence of Capnocytophaga species
Paulander, (2004)[27]	In a model considering all the samples, educational level was not associated with bone loss. However, a predictive mobl including only subjects who had never been smokers revealed that educational level had a statistically significant influence on the 10-y ABL change In a model considering all the samples, educational level was not associated with bone loss. However, a predictive model including only participants who had never been smokers revealed that educational level had a statistically significant influence on the 10-y ABL change
Poulton, (2002)[28]	All dental health measures at age 26 y showed a graded relation with childhood SES. As SES increased, the proportion of individuals with periodontal disease decreased
Thomson, (2013)[29]	On the basis of bivariate analysis, individuals from the lower childhood SES had a higher prevalence of periodontal experience

SES, Socioeconomic status; LPA, Loss of periodontal attachment; CPI, Community Periodontal Index; ABL, Alveolar bone loss.

However, this review is not free of limitations. There was considerable heterogeneity across studies included in this systematic review. For instance, the SEP indicators adopted by the studies included annual or monthly household income, parental or individual educational attainment and parental occupation. Additionally, some studies assessed SEP indicators in childhood. Periodontitis case definitions also varied considerably. Periodontitis was assessed using periodontal pocket depth, clinical attachment level and alveolar bone loss at a time point or progression during the study periods. Analytical methods also varied, with some studies reporting crude associations between the exposure and outcome, and others reporting estimates adjusted for confounding factors. Despite these variations in measures of exposure, case definitions and methodological approaches, the studies consistently reported an inverse association between SEP earlier in life and periodontitis later in life. While it is acceptable to integrate heterogeneous studies in a systematic review, the conclusion should be inferred cautiously given the heterogeneities highlighted earlier in the body of evidence.[35] Additionally, although systematic reviews with meta-analysis are able to provide a summary effect estimate of the included studies, if the primary studies are clinically, methodologically and/or statistically diverse, a pooled effect estimate can be misleading. Therefore, in this systematic review, lack of uniformity in exposure and outcome measures did not allow for a meta-analysis to be conducted.

The review also included studies that reported crude associations between SEP and periodontitis. As the relationship between SEP and chronic diseases is complex and mediators in this association are not well defined, adjusting for potential intermediary factors in a regression analysis can underestimate the association between SEP and periodontitis. Smoking is a potential intermediary factor for such a relationship. The literature clearly demonstrates the SEP gradient in tobacco smoking,[36] and it is also well known that smoking is strongly chained in the web of causation of periodontitis.[37-39] Therefore, it is critical to test the role of smoking as a mediator in the association between SEP and periodontitis. However, there appears to be a lack of tests accounting for the role of smoking in the relationship SEP-periodontitis in the primary studies included in this systematic review.

Two included studies reported nonsignificant associations between SEP indicators and periodontal measures.[27,32] The assessment of periodontitis at a young age (18 years) might explain this inconsistent observation in one of the study, as periodontitis has a low prevalence among that age group.[32] Additionally, this study assessed periodontal status using CPI, whose limitations in outcome detection is well established.[40,41] One study[27] observed that lower educational attainment at age 50 years was not associated with alveolar bone loss in a 10-year follow-up. However, a stratified analysis revealed that educational level had a significant effect on the 10-year alveolar bone level. The authors argued that in addition to the direct effect on periodontal health, smoking might be a marker for low SEP.

The literature points towards the need for a better understanding of potential causal pathways linking SEP and periodontitis.[12] In this systematic review, only the study by Lu et al.[32] aimed to test for mediation between SEP and periodontitis. The stated hypothesis was that socioeconomic childhood characteristics (parental educational attainment and household income) and oral health status in adulthood (dental caries experiences and periodontal health status) were mediated by the utilization of dental services during adolescence. Nevertheless, this hypothesis was rejected. Buchwald et al[16] did not consider covariates, such as smoking status and levels of C-reactive protein, as potential mediators between SEP and

periodontitis because the inclusion of such variables in their multivariable models did not attenuate the association between exposure and outcome. For instance, as smoking status was not associated with both the exposure and outcome, the authors decided not to include it as a potential mediator in the analysis. It should be highlighted that the authors' decision was purely statistically driven, that is, based on their data set and not theoretically driven or based on the scientific literature. None of the remaining studies assessed or discussed potential mediating pathways.

Socioeconomic position can be assessed by a range of measures. The studies included in this systematic review assessed the exposition by objective individual measures, namely household income, father's occupation and participant's or parent's education. Theoretically, there is not a best indicator of SEP[6] and although many SEP indicators are partially correlated, distinct indicators have focused on different social dimensions. It is important to understand what each SEP indicator measures when assessing its influence on health, aiming to better understand the potential mechanisms of such influence. For example, measures of housing crowding and number of siblings may specifically reflect hygiene conditions and act as a marker for infectious disease risk.[6] The ideal SEP indicator varies according to the health condition and the moment of life it is being evaluated.[6,42] It is appropriate to use different indicators of SEP, capturing a range of SEP dimensions. The diverse range of SEP indicators observed in the present study is substantiated in other systematic reviews assessing the association of SEP with general chronic health outcomes.[7,42,43]

This systematic review contributed evidence that low SEP earlier in life may be a risk factor for periodontitis in adulthood. This finding can be explained as theoretically plausible in the light of the current understanding of periodontitis and its pathological pathways. Individuals at relatively lower SEP may be at a higher risk of diseases, through stress-induced ill behaviours and physiological effects of chronic stress.[44,45] The majority of studies included in this review did not evaluate SEP exposure during childhood, as only one of the included studies used a life-course approach that accounted for SEP at childhood.[28,29] According to the life-course theories, childhood adverse socioeconomic background alters the host immuno-inflammatory response.[11] The hypothetical pathways for the effect of SEP on periodontitis are yet to be confirmed. The best way to test such theories is through prospectively designed studies that evaluate participants from childhood until the onset of periodontitis and beyond, given it is a chronic cumulative condition. This approach allows researchers to account for a range of variables over time that could confound the relationship, which may, in turn, introduce bias to the estimates. However, residual confounding may occur due to unmeasured/unadjusted variables, regardless of the study design.

The overall methodological quality of the selected articles in this review was considered high. However, the study by Machtei and colleagues had important methodological limitations[31] such as the use of two different indicators of SEP without detailed explanation of how these indicators were measured and categorized. Although the study was well conducted, it did not report important characteristics, such as the representativeness of the sample. Half of the included studies achieved the highest score in the NOS (Table S2). But, none of the included studies approached the data using specific longitudinal data analysis procedures. Longitudinal studies following an appropriate analytical plan and using measures of incidence of periodontitis still need to be conducted.

5 | CONCLUSION

Our systematic review showed that individuals who were exposed to relative lower socioeconomic conditions earlier in life presented worse periodontal outcomes in adulthood. This finding was consistent across the broad range of SEP indicators and measures of disease adopted in the primary studies. This finding provided evidence for a temporal relationship, a requisite to confirm causal nature of the association between SEP and periodontitis. The review also indicated a need for prospective analytical cohort studies with long-term follow-up testing multiple life-course theories to determine the role SEP during different life periods has on periodontitis in adulthood. It is paramount to understand such a relationship to orientate public policy strategies, aiming to focus the effort on the population at highest risk, implementing such strategies which may then reduce social inequalities in periodontal outcomes.

ACKNOWLEDGEMENTS

The authors would like to thank Mr. Michael Draper, Research Librarian at The University of Adelaide, for his support with the search strategies. The authors are also grateful to the Brazilian government agency (CAPES Coordination for the Improvement of Higher Education Personnel) for the PhD scholarship (HSS - Process 13774-13-1).

REFERENCES

1. Marcenes W, Kassebaum NJ, Bernabe E, et al. Global burden of oral conditions in 1990-2010: a systematic analysis. *J Dent Res*. 2013;92:592–597.
2. Genco RJ, Borgnakke WS. Risk factors for periodontal disease. *Periodontology*. 2000;2013:59 94.
3. Kelly JE, Engel A. Selected examination findings related to periodontal disease among adults, United States - 1960-1962. *Vital Health Stat*. 1974;11:1–38.
4. Albandar JM. Global risk factors and risk indicators for periodontal diseases. *Periodontology*. 2000;2002:177–206.
5. Borrell LN, Burt BA, Warren RC, Neighbors HW. The role of individual and neighborhood social factors on periodontitis: the third National Health and Nutrition Examination Survey. *J Periodontol*. 2006;77:444 453.
6. Susin C, Dalla Vecchia CF, Oppermann RV, Haugejorden O, Albandar JM. Periodontal attachment loss in an urban population of Brazilian adults: effect of demographic, behavioral, and environmental risk indicators. *J Periodontol*. 2004;75:1033–1041.
7. Galobardes B, Lynch JW, Davey Smith G. Childhood socioeconomic circumstances and cause-specific mortality in adulthood: systematic review and interpretation. *Epidemiol Rev*. 2004;26:7 21.

8. Pollitt RA, Rose KM, Kaufman JS. Evaluating the evidence for models of life course socioeconomic factors and cardiovascular outcomes: a systematic review. *BMC Public Health*. 2005;5:7.
9. Zeng J, Williams SM, Fletcher DJ, et al. Reexamining the association between smoking and periodontitis in the dunedin study with an enhanced analytical approach. *J Periodontol*. 2014;85:1390–1397.
10. Tezal M, Grossi SG, Ho AW, Genco RJ. Alcohol consumption and periodontal disease. The Third National Health and Nutrition Examination Survey. *J Clin Periodontol*. 2004;31:484–488.
11. Petersen PE, Ogawa H. The global burden of periodontal disease: towards integration with chronic disease prevention and control. *Periodontology*. 2000;2012:15–39.
12. Schuch HS, Peres KG, Do LG, Peres MA. Can socioeconomic trajectories during the life influence periodontal disease occurrence in adulthood? Hypotheses from a life-course perspective. *Med Hypotheses*. 2015;84:596–600.
13. Lynch J, Kaplan G. Socioeconomic position. In: Berkman LF, Kawachi I, eds. *Social Epidemiology*. USA: Oxford University Press; 2000: 13–35.
14. Susin C, Albandar JM. Aggressive periodontitis in an urban population in southern Brazil. *J Periodontol*. 2005;76:468–475.
15. Sabbah W, Tsakos G, Chandola T, Sheiham A, Watt RG. Social gradients in oral and general health. *J Dent Res*. 2007;86:992–996.
16. 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;40:203–211.
17. Klinge B, Norlund A. A socio-economic perspective on periodontal diseases: a systematic review. *J Clin Periodontol*. 2005;32(Suppl 6):314–325.
18. Boillot A, El Halabi B, Batty GD, Range H, Czernichow S, Bouchard P. Education as a predictor of chronic periodontitis: a systematic review with meta-analysis population-based studies. *PLoS ONE*. 2011;6:e21508.
19. Borrell LN, Crawford ND. Socioeconomic position indicators and periodontitis: examining the evidence. *Periodontology*. 2000;2012:69–83.
20. Hill AB. The environment and disease: association or causation? *Proc R Soc Med*, 1965;58:295–300.
21. Rothman KJ, Greenlan S. Hill's criteria for causality. *Encyclopedia of Biostatistics*. doi: 10.1002/0470011815.b2a03072.
22. Chiolero A. Counterfactual and interventionist approach to cure risk factor epidemiology. *Int J Epidemiol*. 2016. pii: dyw159. [Epub ahead of print].
23. Moher D, Liberati A, Tetzlaff J, Altman DG; Group P. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. *PLoS Med*. 2009;6:e1000097.
24. Higgins JPT, Green S, editors. *Cochrane Handbook for Systematic Reviews of Interventions Version 5.1.0*. The Cochrane Collaboration; 2011. www.cochrane-handbook.org. Accessed 10th November, 2015.
25. Wells GA, Shea B, O'Connell D, et al. The Newcastle-Ottawa Scale (NOS) for assessing the quality of nonrandomised studies in meta-analyses. The Ottawa Hospital; 2011. http://www.ohri.ca/programs/clinical_epidemiology/oxford.asp. Accessed 10th November, 2015.
26. Zangrando MS, Damante CA, Sant' Ana AC, Rubo de Rezende ML, Greghi SL, Chambrone L. Long-term evaluation of periodontal parameters and implant outcomes in periodontally compromised patients: a systematic review. *J Periodontol*. 2015;86:201–221.
27. Paulander J, Wennstrom JL, Axelsson P, Lindhe J. Some risk factors for periodontal bone loss in 50-year-old individuals. A 10-year cohort study. *J Clin Periodontol*. 2004;31:489–496.
28. Poulton R, Caspi A, Milne BJ, et al. Association between children's experience of socioeconomic disadvantage and adult health: a life-course study. *Lancet*. 2002;360:1640–1645.
29. Thomson WM, Shearer DM, Broadbent JM, Page LAF, Poulton R. The natural history of periodontal attachment loss during the third and fourth decades of life. *J Clin Periodontol*. 2013;40:672–680.
30. Ismail AI, Morrison EC, Burt BA, Caffesse RG, Kavanagh MT. Natural history of periodontal disease in adults: findings from the Tecumseh Periodontal Disease Study, 1959-87. *J Dent Res*. 1990;69:430–435.
31. Machtei EE, Hausmann E, Dunford R, et al. Longitudinal study of predictive factors for periodontal disease and tooth loss. *J Clin Periodontol*. 1999;26:374–380.
32. Lu HX, Wong MC, Lo EC, McGrath C. Trends in oral health from childhood to early adulthood: a life course approach. *Community Dent Oral Epidemiol*. 2011;39:352–360.
33. Haas AN, Gaio EJ, Oppermann RV, Rosing CK, Albandar JM, Susin C. Pattern and rate of progression of periodontal attachment loss in an urban population of South Brazil: a 5-years population-based prospective study. *J Clin Periodontol*. 2012;39:1–9.
34. Ainamo J, Bay I. Problems and proposals for recording gingivitis and plaque. *Int Dent J*. 1975;25:229–235.
35. Mulrow C, Langhorne P, Grimshaw J. Integrating heterogeneous pieces of evidence in systematic reviews. *Ann Intern Med*. 1997;127:989–995.
36. Hiscock R, Bauld L, Amos A, Fidler JA, Munafò M. Socioeconomic status and smoking: a review. *Ann N Y Acad Sci*. 2012;1248:107–123.
37. Bergstrom J. Smoking rate and periodontal disease prevalence: 40-year trends in Sweden 1970–2010. *J Clin Periodontol*. 2014;41:952–957.
38. Thomson WM, Poulton R, Broadbent JM, et al. Cannabis smoking and periodontal disease among young adults. *JAMA*. 2008;299:525–531.
39. Thomson WM, Broadbent JM, Welch D, Beck JD, Poulton R. Cigarette smoking and periodontal disease among 32-year-olds: a prospective study of a representative birth cohort. *J Clin Periodontol*. 2007;34:828–834.
40. Baelum V, Papapanou PN. CPITN and the epidemiology of periodontal disease. *Community Dent Oral Epidemiol*. 1996;24:367–368.
41. Leroy R, Eaton KA, Savage A. Methodological issues in epidemiological studies of periodontitis–how can it be improved? *BMC Oral Health*. 2010;10:8.
42. Galobardes B, Lynch J, Smith GD. Measuring socioeconomic position in health research. *Br Med Bull*. 2007;81–82:21–37.
43. Gershon AS, Dolmage TE, Stephenson A, Jackson B. Chronic obstructive pulmonary disease and socioeconomic status: a systematic review. *COPD*. 2012;9:216–226.
44. Wilkinson RG. Socioeconomic determinants of health. Health inequalities: relative or absolute material standards? *BMJ*. 1997;314:591–595.
45. Celeste RK, Nadanovsky P, Ponce de Leon A, Fritzell J. The individual and contextual pathways between oral health and income inequality in Brazilian adolescents and adults. *Soc Sci Med*. 2009;69:1468–1475.

SUPPORTING INFORMATION

Additional Supporting Information may be found online in the supporting information tab for this article.

How to cite this article: Schuch HS, Peres KG, Singh A, Peres MA, and Do LG. Socioeconomic position during life and periodontitis in adulthood: a systematic review. *Community Dent Oral Epidemiol*. 2016;00:1-8. doi:10.1111/cdoe.12278.

Supplementary Material A:

Search strategy for each database:

PubMed: (((((longitudinal studies[mh] OR longitudinal analys*[all] OR longitudinal design*[all] OR longitudinal evaluation*[all] OR longitudinal research[all] OR longitudinal studies[tw] OR longitudinal study[tw] OR longitudinal survey*[all] OR follow up evaluation*[all] OR followup evaluation*[all] OR followup stud*[all] OR follow up stud*[all] OR followup survey*[all] OR follow up survey* [all] OR prospective analys*[all] OR prospective evaluation*[all] OR prospective studies[tw] OR prospective study[tw] OR prospective survey*[all] OR retrospective studies[mh] OR retrospective analys*[all] OR retrospective evaluation*[all] OR retrospective stud*[all] OR retrospective survey*[all])) AND (“Income*” [ALL] OR "Occupation" [ALL] OR "Socioeconomic " [ALL] OR "Education" [ALL] OR “Remuneration” [ALL] OR “Earning*” [ALL] OR “Salar*” [ALL] OR social class* [ALL])) AND ("Periodontitis” [ALL] OR "Periodon*" [ALL] OR “Periodontal Pocket*” [ALL] OR "pocket depth" OR "loss of attachment" OR "gingival bleeding" OR "bleeding after probing" OR “Alveolar Bone Loss” [ALL] OR “Attachment Loss” [ALL] OR “Gingival Recession” [ALL] OR “Bleeding on Probing” [ALL])))

Embase: ('income'/exp OR income* OR remuneration OR salar* OR earning* OR occupation* OR social NEXT/1 class* OR socioeconomic OR education) AND (periodontitis/syn OR periodontal NEXT/2 pocket OR “Alveolar Bone Loss” OR “Attachment Loss” OR “Gingival Recession” OR “Bleeding on Probing”) AND (longitudinal NEXT/5 (analys* OR evaluation* OR research OR stud* OR

survey*) OR prospective NEXT/1 (analys* OR evaluation* OR method OR stud* OR survey*) OR ‘retrospective study’/syn OR retrospective NEXT/2 (analys* OR evaluation* OR research OR survey* OR trial*))

Web of Science: TOPIC: (Income* OR Remuneration OR Salar* OR Earning* OR Occupation OR Socioeconomic OR “social class*” OR education) AND TOPIC: ("Periodon*" OR “Alveolar Bone Loss” OR “Attachment Loss” OR “Gingival Recession” OR “Bleeding on Probing”) AND TOPIC: ("longitudinal analys*" OR "longitudinal evaluation*" OR "longitudinal research" OR "longitudinal stud*" OR "longitudinal survey*" OR “Followup evaluation” OR “Followup stud*” OR “Followup survey*” OR “Follow up survey*” OR "Prospective analys*" OR "Prospective evaluation*" OR "Prospective studies" OR "Prospective study" OR "Prospective survey" OR "Retrospective analys*" OR "Retrospective evaluation*" OR "Retrospective studies" OR "Retrospective study" OR "Retrospective survey")

Scopus: TITLE-ABS-KEY ("Income*" OR "Remuneration" OR "Salar*" OR "Earning*" OR "Occupation" OR "Socioeconomic position" OR "Socioeconomic status" OR "Education" OR “Social class*”) AND TITLE-ABS-KEY ("Periodon*" OR "Alveolar Bone Loss" OR "Attachment Loss" OR "Gingival Recession" OR "Bleeding on Probing") AND TITLE-ABS-KEY ("longitudinal studies" OR "longitudinal analys*" OR "longitudinal design*" OR "longitudinal evaluation*" OR "longitudinal research" OR "longitudinal studies" OR "longitudinal study" OR "longitudinal survey*" OR "follow up evaluation*" OR "followup evaluation*" OR "followup stud*" OR "follow up

stud*" OR "followup survey*" OR "follow up survey*" OR "prospective analys*" OR "prospective evaluation*" OR "prospective studies" OR "prospective study" OR "prospective survey*" OR "Retrospective analys*" OR "Retrospective evaluation*" OR "Retrospective studies" OR "Retrospective study" OR "Retrospective survey")

LILACS: tw:(salário) OR tw:(renda) OR tw:(income) OR tw:(remuneração) OR tw:(remuneration) OR tw:(earning) OR tw:(salar$) OR tw:("Socioeconomic") OR tw:(“Social class$”) OR tw:(Education) OR tw:(ocupação) OR tw:(profissão) OR tw:(“posição socioeconômica”) OR tw:(occupation) AND (tw:(Periodont$)) OR (tw:(recessão gengival)) OR (tw:(“perda de inserção”)) OR (tw:(“Alveolar Bone Loss”)) OR (tw:(“Attachment Loss”)) OR (tw:(“Gingival Recession”)) OR (tw:(“Bleeding on Probing”)) OR (tw:(“sangramento a sondagem”)) OR (tw:(“perda de osso alveolar”)) AND (tw:(longitudinal)) OR (tw:(“follow up”)) OR (tw:(followup))

SciElO: (tw:(salário)) OR (tw:(renda)) OR (tw:(income)) OR (tw:(remuneração)) OR (tw:(remuneration)) OR (tw:(earning)) OR (tw:(salar$)) OR (tw:("Socioeconomic position")) OR (tw:("Socioeconomic status")) OR (tw:(Education)) OR (tw:(ocupação)) OR (tw:(profissão)) OR (tw:(“posição socioeconômica”)) OR (tw:(occupation)) AND (tw:(Periodontitis OR periodontite)) OR (tw:(recessão gengival)) OR (tw:(perda de inserção)) OR (tw:(Periodontal Pocket$)) OR (tw:(Alveolar Bone Loss)) OR (tw:(Attachment Loss)) OR (tw:(Periodon$)) OR (tw:(Gingival Recession)) OR (tw:(Bleeding on Probing)) OR (tw:(doença periodontal)) OR (tw:(sangramento a sondagem)) OR

(tw:(perda de osso alveolar)) AND (tw:(longitudinal)) OR (tw:(follow up)) OR (tw:(followup)) OR Periodontitis OR periodontite OR recessão gengival OR perda de inserção OR Periodontal Pocket$ OR Alveolar Bone Loss OR Attachment Loss OR Periodon$ OR Gingival Recession OR Bleeding on Probing OR doença periodontal OR sangramento a sondagem OR perda de osso alveolar OR longitudinal or follow up or followup

Supplementary Material B:

Table S1. Excluded studies and main reason for exclusion

Study	Country	Reason for exclusion
Bower et al., 2007	Scotland	Cross-sectional design
Crossner et al., 2007	Sweden	Assessed both SEP and CPD at the follow up
Gaetke et al., 2012	Pomerania	Some sample of a previous included study in this review
Grbic et al., 1991	United States of America	Not population-based
Gugushe et al., 1998	Republic of South Africa	Cross-sectional design
Haas et al., 2014	Brazil	Assessed both SEP and CPD at the follow up
Halling et al., 1987	Sweden	No clear definition of case disease
Holst et al., 2012	Norway	Did not evaluate CPD
Hugoson et al., 2000	Sweden	Did not evaluate SEP
Jamieson et al., 2013	Australia	Assessed both SEP and CPD at the follow up
Linden et al., 1996	Ireland	Not population-based
Lucaciu et al., 2014	Romania	Cross-sectional design
Norderyd, O., 1998	Sweden	CPD assessed before SEP
Norderyd et al., 1999	Sweden	CPD assessed before SEP
Oleary, T., 1968	United States of America	Insufficient available information
Opeodu et al., 2007	Nigeria	Not population-based
Peres et al., 2011	New Zealand and Brazil	Did not assess the effect of SEP on CPD
Shearer et al., 2011	New Zealand	Assessed both SEP and CPD at the follow up
Yalcin et al., 2002	Turkey	Not population-based

Table S2. Methodological quality assessment (Newcastle-Ottawa Scale)

Study	Selection	Comparability	Outcome	Total
Buchwald et al, 2013	3	2	3	8
Haas et al, 2012	4	2	3	9
Ismail et al, 1990	3	2	3	8
Lu et al, 2011	4	2	3	9

Machtei et al, 1999	1	0	3	4
Paulander et al, 2004	4	2	3	9
Poulton et al, 2002	4	2	3	9
Thomson et al, 2013	4	2	3	9

*Maximum scores: Selection = 4, Comparability = 2, Outcome = 3, Total = 9

Table S1 Reference List

1. Bower E, Gulliford M, Steele J, Newton T. Area deprivation and oral health in Scottish adults: a multilevel study. *Community Dent Oral Epidemiol* 2007; 35: 118–129.

2. Crossner CG, Unell L. A longitudinal study of dental health from the age of 14 to 41. *Swed Dent J.* 2007;31(2):65-74.

3. Gaetke D, Holtfreter B, Biffar R, Kocher T. Five-year change of periodontal diseases in the Study of Health in Pomerania (SHIP). *J Clin Periodontol* 2012; 39: 357–367.

4. Grbic JT, Lamster IB, Celenti RS, Fine JB. Risk indicators for future clinical attachment loss in adult periodontitis. Patient variables. *J Periodontol.* 1991 May;62(5):322-9.

5. Gugushe TS. The influence of socio-economic variables on the prevalence of periodontal disease in South Africa. *SADJ*. 1998 Feb;53(2):41-6.

6. Haas AN, Wagner MC, Oppermann RV, Rosing CK, Albandar JM, Susin C. Risk factors for the progression of periodontal attachment loss: a 5-year population-based study in south Brazil. *J Clin Periodontol* 2014; 41: 215–223.

7. Halling A, Björn AL. Periodontal status in relation to education and dental attendance. A 12 year longitudinal and a cross-sectional study of a random sample of dentate middle-aged women in Gothenburg. *Swed Dent J.* 1987;11(4):135-45.

8. Holst D, & Schuller AA. Oral health in a life-course: Birth-cohorts from 1929 to 2006 in Norway. *Community Dental Health* (2012) 29, 134–143.

9. Hugoson A, Laurell L: A prospective longitudinal study on periodontal bone height changes in a Swedish population. *J Clin Periodontol* 2000; 27: 665–674.

10. Jamieson LM, Sayers SM & Roberts-Thomson KF. Associations between oral health and height in an Indigenous Australian birth cohort. *Community Dental Health* (2013) 30, 58–64.

11. Linden GJ, Mullally BH, Freeman R. Stress and the progression of periodontal disease. *J Clin Periodontol.* 1996 Jul;23(7):675-80.

12. Lucaciu O, Soare R, Buhatel D, Ionel A, Moga M, Sarbu A, Tiriac M, Campian RS. The impact of socioeconomic inequalities in the distribution of oral pathology in a disadvantaged group in Cluj Napoca, Romania. *HVM Bioflux* 2014;6(4):159-164.

13. Norderyd O. Risk for periodontal disease in a Swedish adult population. Cross-sectional and longitudinal studies over two decades. *Swed Dent J Suppl.* 1998;132:1-67.

14. Norderyd O, Hugoson A, Grusovin G: Risk of severe periodontal disease in a Swedish adult population. A longitudinal study. *J Clin Periodontol* 1999; 26: 608–615.

15. O'Leary TJ. Designs and results of a longitudinal study on periodontal health. *J Periodontol.* 1968 Jan;39(1):47.

16. Opeodu OI & Arowojolu MO. Effect of Social Class on the Prevalence and Severity of Periodontal Disease. *Ann Ib Postgrad Med*. 2007 Jun;5(1):9-11.

17. Peres MA, Thomson WM, Peres KG, Gigante DP, Horta BL, Broadbent JM, Poulton R. Challenges in comparing the methods and findings of cohort studies of oral health: the Dunedin (New Zealand) and Pelotas (Brazil) studies. *Aust N Z J Public Health.* 2011 Dec;35(6):549-56.

18. Shearer DM, Thomson WM, Caspi A, Moffitt TE, Broadbent JM, Poulton R. Intergenerational continuity in periodontal health: findings from the Dunedin Family History Study. *J Clin Peridontol* 2011; 38: 301–309.

19. Yalcin F, Eskinazi E, Soydinc M, Basegmez C, Issever H, Isik G, Berber L, Has R, Sabuncu H, Onan U. The effect of sociocultural status on periodontal conditions in pregnancy. *J Periodontol.* 2002 Feb;73(2):178-82.

Table S3. Summary of the findings of the included papers

First author/Year	Summary measures	Results	Crude or Adjusted
Buchwald, 2013	ß-coefficients and 95% Confidence Interval for progression of mean attachment loss in mm	Education (ref. > 10th grade) 10th grade -0.11 (-0.21; 0.00) Less than 10th grade -0.27 (-0.40; -0.14) Income/person (ref > 960 €) 600 –959/person-0.08 (-0.18; 0.01) < 600/person -0.11 (-0.22; -0.00)	Adjusted for adjusted for smoking (never, quit, current), obesity (BMI quartiles, 16.79–23.79, 23.80–26.82, 26.83–30.02, 30.03–58.43) and for CRP categories (< 1, 1 to < 3, 3 mg/l).
Haas, 2012	Mean annual proximal PAL progression (mm) for all sites	Socioeconomic status (SES) High, Mean (SE): 0.09 (0.01) Medium, Mean (SE): 0.09 (0.01) Low, Mean (SE): 0.13 (0.01)** Total, Mean (SE): 0.10 (0.01)	Crude
	Mean annual proximal PAL progression (mm) for the worst site	Socioeconomic status (SES) High, Mean (SE): 0.27 (0.01) Medium, Mean (SE): 0.28 (0.01) Low, Mean (SE): 0.36 (0.02)** Total, Mean (SE): 0.31 (0.01)	Crude
	Number of teeth affected (mm) with different threshold of proximal PAL progression	≥3 teeth affected (mm)* High SES, Mean (SE): 3.7 (0.3) Medium SES, Mean (SE): 3.3 (0.3) Low SES, Mean (SE): 4.3 (0.3) Overall, Mean (SE): 3.8 (0.2) High SES, Mean (SE): 3.7 (0.3) Medium SES, Mean (SE): 3.3 (0.3) Low SES, Mean (SE): 4.3 (0.3)	Crude
	Number of teeth affected (mm) with different threshold of proximal PAL progression	≥4 teeth affected (mm)* High SES, Mean (SE): 1.3 (0.2) Medium SES, Mean (SE): 1.4 (0.2) Low SES, Mean (SE): 1.9 (0.2) Overall, Mean (SE): 1.5 (0.1)	Crude
	Number of teeth affected (mm) with different threshold of proximal PAL progression	≥5 teeth affected (mm) High SES, Mean (SE): 0.5 (0.1) Medium SES, Mean (SE): 0.5 (0.1) Low SES, Mean (SE): 0.7 (0.1) Overall, Mean (SE): 0.6 (0.1)	Crude

	Number of sites affected (mm) with different threshold of proximal PAL progression	≥3 sites affected (mm)* High SES, Mean (SE): 5.4 (0.5) Medium SES, Mean (SE): 4.84 (0.5) Low SES, Mean (SE): 6.8 (0.6) Overall, Mean (SE): 5.7 (0.3)	Crude
	Number of sites affected (mm) with different threshold of proximal PAL progression	≥4 sites affected (mm) High SES, Mean (SE): 1.9 (0.3) Medium SES, Mean (SE): 1.8 (0.3) Low SES, Mean (SE): 2.6 (0.3) Overall, Mean (SE): 2.1 (0.2)	Crude
	Number of sites affected (mm) with different threshold of proximal PAL progression	≥5 sites affected (mm) High SES, Mean (SE): 0.7 (0.2) Medium SES, Mean (SE): 0.6 (0.1) Low SES, Mean (SE): 0.9 (0.2) Overall, Mean (SE): 0.7 (0.1)	Crude
Ismail, 1990	Odds Ratios (95% Confidence Intervals) for loss of periodontal attachment (LPA)	Education (Low) 3.05 (1.10, 8.43) Income 1.01 (0.41, 2.49)	Crude, SES indicators not included in the multivariate logistic regression analysis
Lu, 2011	Path Analysis	There was no relationship between the income level and the periodontal status (P > 0.05), and the parental education level also had no significant direct and indirect effects on any of the variables at the subsequent parts in the path model (P > 0.05).	A Path Analysis was adopted to investigate a possible chain of risk, linking socioeconomic characteristics (parental education attainment and household income) to periodontal health status (CPI score), mediated by the utilization of dental services during adolescence. No direct nor indirect relationship could be found between socioeconomic variables and the CPI score.
Machtei, 1999	Coefficient/Correlation for annual change in pocket depth	Income Coefficient -0.054 S.E. 0.015 p-value 0.0000 Correlation 0.596	Adjusted for baseline pocket depth, presence of Cs4, smoking and thyroid disorder
Paulander, 2004	Relative Risk (95% Confidence Intervals) for a mean 10-year alveolar bone level loss (ABL) >0.5 mm.	Considering the whole sample: Educational level (low comparing to high) 0.94 (0.68, 1.28)	Crude
	Relative Risk (95% Confidence Intervals) for a mean 10-year alveolar bone level loss (Westhoff et al.) >0.5 mm.	Stratified by smoking status (never-smokers): Education level* (low comparing to high) Coefficient 0.12 SE 0.041 Standardized coefficient 0.205	Adjusted for no. of teeth, mean ABL and % healthy sites (dependent variable: mean 10-year ABL loss)
Poulton, 2002	Odds Ratios (95% Confidence Intervals) for proportion with periodontal disease	Low vs high socioeconomic status (SES)** Childhood socioeconomic status 2.88 (1.53–5.39) Medium vs high SES	Adjusted for sex, infant health, and adult socioeconomic status.

		Childhood socioeconomic status 1.39 (0.80–2.44)	
Thomson, 2013	Periodontitis experience trajectory group according to childhood SES	High SES* Very low (VL) 84 (58.3%) Low 44 (30.6%) Moderately increasing (MDI) 15 (10.4%) Markedly increasing (MKI) 1 (0.7%) Medium SES VL 319 (60.0%) Low 158 (29.7%) MDI 44 (8.3%) MKI 11 (2.1%) Low SES VL 54 (35.8%) Low 59 (39.1%) MDI 29 (19.2%) MKI 9 (6.0%)	Crude

*p<0.05
**p<0.01

CHAPTER 6 (PAPER 3)

Effect of life-course family income trajectories on periodontitis

Linkage to the body of work

This paper was developed with the aim of testing the suitability to explain the relationship between life-course socioeconomic position and periodontal disease in adulthood. For that, we modelled family income trajectories from birth to adulthood and tested whether these trajectories were impacting on the development of periodontal disease later on. We also tested whether income would interact with other SEP indicators, namely maternal education at the participant's birth and the participant's own education in adulthood, in affecting periodontal disease. Using the Group-based Trajectory Modelling (GBTM), three income trajectories were identified. The main finding of the study was that individuals in the "low and variable" income trajectory had a higher prevalence of periodontal disease by age 31, after adjusting for covariates.

Highlights

- Three income trajectories were identified in the sample, using the Group-based Trajectory Modelling (GBTM): relatively stable high income, relatively stable middle income, and low and variable income trajectory.
- Participants from the income trajectory 3 (low and variable income) had a 2.1 (95% CI: 1.1; 4.1) times higher prevalence of moderate-to-severe periodontal disease at age 31 than participants in the stable high income trajectory after adjustments for covariates.

- Interactions between income trajectories and other SEP indicators were not significant in any of the models tested.

Statement of authorship form

Statement of Authorship

Title of Paper	Life course family income trajectories on periodontitis: birth cohort study
Publication Status	☐ Published ☐ Accepted for Publication ☑ Submitted for Publication ☐ Unpublished and Unsubmitted work written in manuscript style
Publication Details	Schuch HS, Peres KG, Demarco FF, Horta BL, Gigante DP, Peres MA, Do LG. Life course family income trajectories on periodontitis: birth cohort study. *Journal of Clinical Periodontology*. Submitted 2017.

Principal Author

Name of Principal Author (Candidate)	Helena Silveira Schuch		
Contribution to the Paper	Contributed to data collection, analysed and interpreted data, wrote manuscript and acted as corresponding author.		
Overall percentage (%)	80%		
Certification:	This paper reports on original research I conducted during the period of my Higher Degree by Research candidature and is not subject to any obligations or contractual agreements with a third party that would constrain its inclusion in this thesis. I am the primary author of this paper.		
Signature		Date	031 07 2017

Co-Author Contributions

By signing the Statement of Authorship, each author certifies that:

i. the candidate's stated contribution to the publication is accurate (as detailed above);
ii. permission is granted for the candidate in include the publication in the thesis; and
iii. the sum of all co-author contributions is equal to 100% less the candidate's stated contribution.

Name of Co-Author	Karen Glazer Peres		
Contribution to the Paper	Supervised development of work, helped in interpretation, manuscript evaluation and edition.		
Signature		Date	03/07/2017

Name of Co-Author	Flavio Fernando Demarco		
Contribution to the Paper	Contributed to data collection, critically evaluated and edited manuscript.		
Signature		Date	03/07/2017

Name of Co Author	Bernardo Lessa Horta
Contribution to the Paper	Contributed to data collection, critically evaluated and edited manuscript.
Signature	Date 03/07/2017

Name of Co-Author	Denise Petrucci Gigante
Contribution to the Paper	Contributed to data collection, critically evaluated and edited manuscript.
Signature	Date 03/07/2017

Name of Co-Author	Marco Aurelio Peres
Contribution to the Paper	Supervised development of work, helped in interpretation, manuscript evaluation and edition.
Signature	Date 3/7/2017

Name of Co-Author	Loc Giang Do
Contribution to the Paper	Supervised development of work, helped in interpretation, manuscript evaluation and edition.
Signature	Date 3/7/17

Accepted: 21 November 2017
DOI: 10.1111/jcpe.12845

EPIDEMIOLOGY (COHORT STUDY OR CASE-CONTROL STUDY)

WILEY Journal of Clinical Periodontology

Effect of life-course family income trajectories on periodontitis: Birth cohort study

Helena S. Schuch[1] | Karen G. Peres[1] | Flavio F. Demarco[2,3] | Bernardo L. Horta[3] | Denise P. Gigante[3] | Marco A. Peres[1] | Loc G. Do[1]

[1]Australian Research Centre for Population Oral Health (ARCPOH), Adelaide Dental School, The University of Adelaide, Adelaide, SA, Australia

[2]Postgraduate Program in Dentistry, Federal University of Pelotas, Pelotas, Brazil

[3]Postgraduate Program in Epidemiology, Federal University of Pelotas, Pelotas, Brazil

Correspondence
Helena Silveira Schuch, Adelaide Health & Medical School (AHMS) Building, Adelaide, SA, Australia.
Email: helena.schuch@adelaide.edu.au

Funding information
National Counsel of Technological and Scientific Development, Grant/Award Number: #403257/2012-3 and #475979/2013-3

Abstract

Aims: To quantify the impact of life course income trajectories on periodontitis in adulthood.

Materials and Methods: Data from the 1982 Pelotas Birth Cohort Study, Brazil, were used. Information on family income was collected at birth and ages 15, 19, 23 and 30 years. Group-based trajectory modelling was used to identify income trajectories. Periodontal measures were assessed through clinical examination at age 31. Log-Poisson regression models were used to estimate prevalence ratios (PRs) of any and moderate/severe periodontitis, as outcomes.

Results: Prevalence of any periodontitis and moderate/severe periodontitis was 37.3% and 14.3% (n = 539). Income trajectories were associated with prevalence of moderate/severe periodontitis. Adjusted PR in participants in low and variable income trajectory was 2.1 times higher than in participants in stable high-income trajectory. The unadjusted association between income trajectories and prevalence of any periodontitis was explained by the inclusion of behavioural and clinical variables in the model.

Conclusions: Low and variable life course income increased the prevalence of moderate/severe periodontitis at age 31 years. The findings may inform programmes in identifying and targeting potentially at-risk groups during the life course to prevent periodontitis.

KEYWORDS
cohort studies, disparities, health status, periodontal disease, social mobility, socioeconomic factors

1 | INTRODUCTION

It is widely debated within social epidemiology that social conditions are more than distal causes of disease. Acting through several pathways, they have a fundamental role in disease causation (Link & Phelan, 1995). Socio-economic positions are changeable during the life course, and the effect of socio-economic position (SEP) on health outcomes differs according to the extent and period of exposure to relative social disadvantage. For example, it has been suggested that the experience of short and long periods of poverty is fundamentally different and, therefore, should be studied and categorized separately (McDonough, Sacker, & Wiggins, 2005; Walker, 1998). Within such a context, it is plausible to assume that the effect of life course SEP trajectories in shaping adult health conditions may be different of a single-time SEP information (Chen, Martin, & Matthews, 2007). It is important to note that a minimum of three assessments is required to form a trajectory.

There is a substantial gap of studies evaluating the effect of SEP trajectories on oral health. Chronic periodontitis, together with

untreated dental decay and severe tooth loss, was indicated as a key oral health condition in the 2010 Global Burden of Disease study (Marcenes et al., 2013), and it remains a relevant public health problem. Studies that demonstrate the association between earlier SEP and periodontitis later in life neglect the dynamic processes through which social factors may affect health during the life course (Link & Phelan, 1995). A literature review conducted on PubMed database in June, 2016, found only four papers evaluating the influence of socio-economic trajectories on oral health outcomes, and none of these evaluated periodontitis (Delgado-Angulo & Bernabe, 2015a,b; Peres, Peres, Demarco et al., 2011; Peres, Peres, Thomson et al., 2011). Additionally, it is critical to understand the simultaneous and individual roles of different SEP measures, such as income and education, and their trajectories when studying the effects of SEP on health status (Hallal et al., 2012).

Although there is evidence pointing towards the association of SEP and a series of health outcomes, the majority of these studies have a cross-sectional design. Studies with a longitudinal design allow for constructing SEP trajectories over the life span and estimating its effects on health outcomes later in life (Chen et al., 2007). A temporal relationship on the association between relative lower socio-economic position and periodontitis has been evidenced in a systematic review, with individuals exposed to relative lower socio-economic position earlier in life presenting worse periodontal outcomes in adulthood (Schuch, Peres, Singh, Peres, & Do, 2017). However, to the best of the authors' knowledge, there is only one population-based prospective cohort study to date that investigated the association between childhood SEP and periodontitis in adulthood (Poulton et al., 2002). This study, however, did not capture the perspective of income trajectories over the life course, and it only evaluated the influence of exposure to socio-economic disadvantage in early life with periodontal outcomes in adulthood. Additionally, this study was conducted in a high-income country (New Zealand).

Therefore, the aims of the study were as follows: (i) to construct income trajectories over the life course of a birth cohort sample; (ii) to quantify its impact on the occurrence of periodontitis in adulthood controlling for covariates; and (iii) to analyse the interaction between income trajectories and participant's education and maternal education at participants' birth on the occurrence of periodontitis.

Clinical Relevance

Scientific rationale for study: There is a lack of studies evaluating income trajectories over the life course and how it affects periodontitis onset and progression in adulthood.

Principal findings: Individuals from low and variable income trajectories have double the risk of presenting moderate-to-severe periodontitis in adulthood than those in relatively stable high-income trajectories.

Practical implications: Special attention should be given to those patients at lower socio-economic trajectory groups over the life course, as they may be at a higher risk of developing periodontitis in adulthood.

2 | METHODS

2.1 | Study population

This study used data from the 1982 Pelotas Birth Cohort Study and is reported according to the STROBE (STrengthening the Reporting of OBservational studies in Epidemiology) Statement. Pelotas is a medium-sized city, with around 300,000 inhabitants, located in the southernmost state of Brazil. The prospective population-based birth cohort study started in 1982, when all hospital births that occurred in the city of Pelotas, Brazil, were identified. 5,914 live births, whose families lived in the urban area of the city, were included on the cohort study. This population has been followed eleven times (waves) since 1982. Oral health substudies were conducted when participants were aged 15 (1997), 24 (2006) and 31 years (2013). The Ethics Committee of the Federal University of Pelotas approved all substudies, and written informed consent was obtained from participants. Methodological details of the cohort study and previous results of the oral health substudies have been published elsewhere (Horta et al., 2015; Peres, Peres, Demarco et al., 2011).

2.2 | Variables—main exposure

The main exposure variable was trajectories of relative family income over the life course. To estimate the income trajectories, family income data from participants at birth and at ages 15, 19, 23 and 30 years were used. Income at each assessment was collected through face-to-face interviews. Young adulthood is usually a phase where individuals may become financially more independent and constitute new families. Family income referred to the total amount of household earnings in the month before the interview. Income information at each time point was categorized into tertiles, and for analytical purposes, the 2nd and 3rd tertiles were combined. Thus, the dichotomized income at each age comprised the poorest group (1st tertile) versus middle and highest income groups (2nd and 3rd tertiles). This categorization was adopted based on the evidence that middle and upper income groups in Brazil were comparable, while the poor lagged well behind (Victora, Fenn, Bryce, & Kirkwood, 2005).

Group-based trajectory analysis (GBTM; Nagin, 2005; Nagin & Odgers, 2010) was the statistical technique adopted to construct the income trajectories. This is a statistical method derived from a finite mixture modelling for approximating unknown trajectories across population members. GBTM aims to identify clusters of individuals with similar trajectories, and the model itself forms the trajectories based on maximum-likelihood estimation. A plug-in in the Software Stata, version 14.0 (Stata Corporation, College Station, TX, USA), was used for forming the trajectories (Jones & Nagin, 2013). When fitting the data using GBTM, the number of groups, the model distribution and the polynomial type were chosen. To decide the number of groups that best represented the heterogeneity in developmental trajectories

in our sample, Bayesian information criteria (BIC; Raftery, 1995) was used as a parameter. The logit distribution was the model adopted in our analyses, considering the dichotomous distribution of the income at each time point. Finally, a cubic model was used, which represents the highest polynomial order allowed with the Stata procedure 'TRAJ'. Trajectories were formed from five points during the life course of 539 individuals, and the output from the software showed a mean of 4.8 information on income per individual, confirming the low rate of missing information on the income variables.

2.3 | Variables—outcomes

The outcome of this study was periodontitis assessed at age 31 years. The research team consisted of six dentists as oral epidemiological examiners and six interviewers. The oral health substudy at age 31 was conducted in 2013 and comprised the 888 individuals in the sample selected from the oral health substudy at age 15 (1997). In 1997, a systematic sample of 70 (27%) of 259 census tracks in the city was selected and dwellings within those limits were visited. A total of 1,076 adolescents were interviewed. From those, a random sample of 900 adolescents was selected to participate in the oral health substudy at age 15.

The clinical oral examinations were conducted at participants' houses, following biosafety procedures recommended by the World Health Organization. Examiners used headlight, dental mirror and PCP2 periodontal probe with 2-mm banding (Hu-Friedy PCP-2; Rotterdam, the Netherlands). Prior to the clinical examination, the research team was trained and calibrated and 30 volunteers were clinically examined. The lowest intra-examiner reliability measure for periodontal outcomes was 0.85 (intra-class correlation coefficient). Clinical oral examination assessed four periodontal health indicators: bleeding on probing, supragingival dental calculus, gingival recession and periodontal pocket depth. Gingival recession and pocket depth were measured at six sites (mesiobuccal, midbuccal, distobuccal, mesiolingual, midlingual and distolingual) on all teeth present, except third molars, and were recorded in millimetres. Dental implants were excluded in the examination, as well as teeth when the measurement was not possible for reasons such as large amount of dental calculus, orthodontic band or non-cooperation of the participant. Attachment loss (AL) was estimated as sum in millimetres of gingival recession and pocket depth at each site.

The criteria to define periodontitis were that proposed in partnership by the Centers for Disease Control and Prevention (CDC) and the American Academy of Periodontology (AAP; Eke, Page, Wei, Thornton-Evans, & Genco, 2012; Page & Eke, 2007). Two combinations of the CDC-AAP criteria were adopted as outcomes of this study: prevalence of any periodontitis and prevalence of moderate-to-severe periodontitis. Cases of any periodontitis were defined as having 2+ interproximal sites with 3+ mm of AL and 2+ interproximal sites with 4+ mm of periodontal pocket depth. Moderate-to-severe periodontitis was defined as having 2+ interproximal sites (not on same tooth) with AL of 4+ mm, or 2+ interproximal sites (not on same tooth) with 5+ mm of pocket depth.

2.4 | Covariates

Covariates included sex and maternal education at participant's birth, smoking status at age 23, dental flossing, bleeding on probing, dental calculus and presence of periodontal pocket at age 24 and participant's level of education at age 30. Detailed description of time of collection and categories of each variable is displayed in Table 2.

2.5 | Statistical analyses

Analyses were conducted using Stata, version 14.0. Descriptive analyses evaluated the representativeness of the oral health substudy at age 31 compared to the original cohort and the baseline of the oral health study. We also computed the distribution of the variables on our sample and the prevalence of the outcomes according to each independent variable. Additionally, we cross-tabulated the income trajectories against the outcome and covariates. Multivariable log-Poisson regression analyses with robust variance estimation were conducted for the two definitions of the outcome to estimate the effect of income trajectories on periodontitis. Model 1 included only sociodemographic characteristics, specifically the income trajectories, sex, maternal education at participant's birth and participant's education at age 30. Model 2 included all sociodemographic variables, behavioural variables at ages 23 and 24 and clinical variables at age 24. All variables were retained in Model 2, regardless of their p value. Interactions between income trajectories with maternal education at participants' birth and participants' education on the occurrence of periodontitis at age 31 were also tested separately in Model 2 for the two outcomes.

3 | RESULTS

A total of 539 individuals participated in the oral health substudy at age 31. Socio-economic and demographic characteristics of the followed-up sample were similar to the original 1982 Pelotas Birth Cohort study and to the baseline of the oral health study (age 15; Table 1). The prevalence of any periodontitis and moderate-to-severe periodontal disease was 37.3% and 14.3%, respectively (Table 2).

The GBTM identified three distinct income trajectories: trajectory 1 (Stable high income) comprised of 31.6% of the sample; trajectory 2 (Stable middle income) accounted for 46.0%; and trajectory 3 (Low and variable income) comprising 22.4% of the total sample. The latter trajectory is characterized by a relatively low income from birth to age 15 after which a steady increase in income was observed (Figure 1). The BIC value associated with this analysis was −1,514.99. As a comparison, the BIC value associated with four trajectory groups was −1,526.56 and with five trajectory groups was −1,541.89.

Distribution of the sample characteristics by income trajectories is presented in Table 3. A clear gradient was observed in distribution of the outcomes by income trajectories. Individuals in the low and variable income trajectory had higher prevalence of periodontitis

TABLE 1 Comparison of demographic and socio-economic characteristics at birth between the original sample and those located in the follow-up study at age 31. 1982 Pelotas Birth Cohort Study, Brazil

Variables	Pelotas birth cohort, age 0, 1982 *n* (%)	Baseline of oral health study, age 15, 1997 *n* (%)	Follow-up study, age 31, 2013 *n* (%)
Sex			
Male	3,037 (51.4)	480 (54.1)	273 (50.6)
Female	2,876 (48.6)	408 (45.9)	266 (49.3)
Total	5,913 (100.0)	888 (100.0)	539 (100.0)
Maternal skin colour			
White	4,851 (82.1)	743 (83.8)	454 (84.2)
Black	1,060 (17.9)	144 (16.2)	85 (15.8)
Total	5,911 (100.0)	887 (100.0)	539 (100.0)
Family income at birth			
≤1 MW	1,288 (21.9)	161 (18.2)	93 (17.3)
1.1–3 MW	2,789 (47.4)	457 (51.7)	282 (52.4)
>3 MW	1,808 (30.7)	266 (30.1)	163 (30.3)
Total	5,885 (100.0)	884 (100.0)	538 (100.0)
Maternal education at birth (years)			
0–4	1,960 (33.2)	285 (32.2)	162 (30.1)
5–8	2,454 (41.5)	393 (44.4)	254 (47.2)
9+	1,493 (25.3)	208 (23.5)	122 (22.7)
Total	5,907 (100.0)	886 (100.0)	538 (100.0)

defined by the two case definitions. Women were about 53% of the sample in stable high-income and stable middle-income trajectories, while almost 58% in low and variable income trajectory. Presence of periodontal pocketing at age 24 also showed a gradient, with 8.8% of individuals from trajectory 3 having 1+ teeth with 4 mm+ periodontal pocket, compared to 1.5% in trajectory 1. Table 3 also presents distribution of income tertiles at each age on income trajectories over the life course.

Unadjusted estimates showed that participants in income trajectory 3 had 1.5 (95% CI: 1.0; 1.9) times higher prevalence of any periodontitis than those in income trajectory 1 (Table 4). The inclusion of sociodemographic characteristics in Model 1 slightly attenuated the difference in the prevalence ratio between the two extreme trajectories, but it remained significant (trajectory 3 PR 1.4 [95% CI: 1.0; 2.0]). The unadjusted association between income trajectories and prevalence of any periodontitis lost its statistical significance after the inclusion of health behaviours and clinical oral variables in Model 2 (trajectory 3 PR 1.4 [95% CI: 1.0; 2.0]). Sex, dental calculus and presence of periodontal pocketing at age 24 were associated with prevalence of any periodontitis in the final model, with being male and those with higher number of teeth with calculus and periodontal pocket presenting higher adjusted PRs.

Table 5 displays log-Poisson regression models of the association of moderate-to-severe periodontitis by income trajectory during the life course. After adjustment for covariates, income trajectories were significantly associated with moderate-to-severe periodontitis, with participants in trajectory 3 having 2.1 (95% CI: 1.1; 4.1) times higher prevalence than participants in trajectory 1. Sex and presence of periodontal pocketing at age 24 were also associated with moderate-to-severe periodontitis at age 31.

The final models for both outcomes do not include interaction terms, as interactions between trajectories and participants' education at age 30 and trajectories and maternal education at birth were not statistically significant in any model.

4 | DISCUSSION

Income trajectories from birth to age 30 influenced the occurrence of any periodontitis and of moderate-to-severe periodontitis at age 31 in a middle-income country. This association remained significant even after adjustment for sociodemographic, behavioural and clinical variables. To the best of the authors' knowledge, this is the first paper in the health literature evaluating the influence of income trajectories on periodontal health outcomes.

Some strengths of the present study are noteworthy. Firstly, the 1982 Pelotas Birth Cohort Study is one of the longest and largest running birth cohorts and the only with clinical oral health data in low- and middle-income countries (Horta et al., 2015). Secondly, trained and calibrated dentists conducted the data collection, and the inter- and intra-examiner's reliability was considerably high. Thirdly, appropriate analytical techniques were applied to model income trajectories. With the use of the GBTM, the researchers were able to capture and combine changes of income levels during the life course from birth to the age of 30 years. Additionally, the participants followed up in the OH-13 presented sociodemographic characteristics comparable

TABLE 2 Description of the sample. 1982 Pelotas Birth Cohort Study, Brazil

Variables	The 2013 follow-up *n* (%)	Any periodontitis[a] *n* (%)	Moderate or severe periodontitis[b] *n* (%)
Income trajectory	*n* = 539	*n* = 201	*n* = 77
1 (Stable high income)	38.4	31.4	23 (11.1)
2 (Stable middle income)	40.4	38.5	32 (14.7)
3 (Low and variable income)	21.2	45.6	22 (19.3)
Sex	*n* = 539	*n* = 201	*n* = 77
Male	50.6	42.9	50 (18.3)
Female	49.3	31.6	27 (10.1)
Education (years of study—age 30)	*n* = 492	*n* = 185	*n* = 73
12+	45.5	34.8	27 (12.1)
9–11	31.5	37.4	21 (13.5)
0–8	23.0	43.4	25 (22.1)
Maternal education at birth (years)	*n* = 538	*n* = 200	*n* = 77
9+	22.7	36.9	17 (13.9)
5–8	47.2	35.4	34 (13.4)
0–4	30.1	40.1	26 (16.1)
Smoking status	*n* = 511	*n* = 189	*n* = 72
Never	70.8	37.3	50 (13.8)
Former	9.8	24.0	4 (8.0)
Current	19.4	42.4	18 (18.2)
Dental flossing (age 24)	*n* = 477	*n* = 177	*n* = 65
Yes or sometimes	51.6	83 (33.7)	30 (12.2)
Never	48.4	94 (40.7)	35 (15.1)
Bleeding on probing (age 24)	*n* = 539	*n* = 201	*n* = 77
0 or 1 tooth	79.4	149 (34.8)	56 (13.1)
2 or more teeth	20.6	52 (46.8)	21 (18.9)
Dental calculus (age 24)	*n* = 539	*n* = 201	*n* = 77
0 or 1 tooth	25.6	34.1	22 (15.9)
2 or more teeth	74.4	38.4	55 (13.7)
Presence of 4 mm+ periodontal pocket (age 24)	*n* = 539	*n* = 201	*n* = 77
0 tooth	96.5	36.2	72 (13.8)
1 or more teeth	3.5	68.4	5 (26.3)
Periodontitis (CDC-AAP) age 31	*n* = 539		
Health	62.7	–	–
Mild	23.0	–	–
Moderate/severe	14.3	–	–

[a]Any periodontitis according to the CDC-AAP case definition (Eke et al., 2012).
[b]Health/mild versus moderate/severe periodontitis, according to the CDC-AAP case definition (Eke et al., 2012).

to those in the overall cohort study. The young age of our sample might also be a strength, allowing for early investigation of periodontitis. Finally, a comprehensive periodontal clinical examination was performed, allowing the researchers to use internationally recommended periodontitis case definitions (CDC-AAP case definitions).

A possible limitation of the study was that the covariates included in the models represented the situation in a specific time point; that is, they were not time-varying covariates. It might have limited the capacity of capturing all potential covariates, changes in covariates and their potential interactions. For example, smoking at different periods of life and changes in smoking status could affect periodontitis differently (Thomson, Broadbent, Welch, Beck, & Poulton, 2007). However, we were not able to capture such peculiarities.

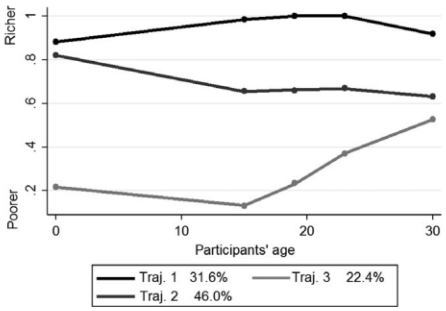

FIGURE 1 Income trajectories from birth to age 30. 1982 Pelotas Birth Cohort Study, Brazil

There are different pathways that can explain the association between income trajectories and periodontitis (Schuch, Peres, Do, & Peres, 2015). The most commonly discussed one is through behavioural variables, with smoking status being the most theoretically and empirically recognized predecessor condition of periodontitis (Thomson et al., 2007). In fact, it is well known that smoking status follows a socio-economic gradient in the population, with individuals standing lower in the socio-economic hierarchy presenting higher prevalence of smoking habits that their richer counterparts (Hiscock, Bauld, Amos, Fidler, & Munafo, 2012) and this is true also for individuals living in Brazil (Barros, Cascaes, Wehrmeister, Martinez-Mesa, & Menezes, 2011). Additionally, as aforementioned, smoking status is a risk factor for periodontitis, and it is plausible to assume it is in the causal pathway between income trajectories and periodontitis. Having said that, after accounting for smoking status, our results remained significant, suggesting that a direct effect of income trajectories on periodontal outcomes may exist. Other important conditions, such as bleeding on probing and presence of dental calculus, as indicators of oral hygiene, and dental pattern flossing, were also accounted for in our analysis. Our results showed that income trajectories during the first 30 years of life influenced the occurrence of moderate-to-severe periodontitis at age 31, regardless of these behavioural factors.

Another potential link between income trajectories and periodontitis is a stress pathway. According to such a pathway, psychosocial stressors over the life course may affect the immune response of the individual, leading to an increased susceptibility of periodontitis (Schuch et al., 2015). It has been argued that such an exposure may have an exacerbated effect if happening in early childhood, as it is the period of the immune system maturation (Nicolau, Netuveli, Kim, Sheiham, & Marcenes, 2007). This reinforces the need of considering the physical and emotional environment of the population when promoting health, as these circumstances potentially impact many health outcomes, including periodontitis. It is also paramount to understand the potential impact children's life circumstances can have for their future periodontal health, as well as triggered factors form childhood that have lifelong effects.

TABLE 3 Description of the sample according to socio-economic trajectories. 1982 Pelotas Birth Cohort Study, Brazil

	Trajectory 1		Trajectory 2		Trajectory 3	
	Stable high income		Stable middle income		Low and variable income	
Variables	*n*	%	*n*	%	*n*	%
Periodontitis (CDC-AAP; *n* = 539)						
No periodontitis	142	42.0	134	39.6	62	18.3
Mild	42	20.3	52	23.8	30	26.3
Moderate/severe	23	11.1	32	14.7	22	19.3
Sex (*n* = 539)						
Male	111	53.6	114	52.3	48	42.1
Female	96	46.4	104	47.7	66	57.9
Education (years of study—age 30; *n* = 492)						
12+	126	70.0	83	39.9	15	14.4
9–11	38	21.1	78	37.5	39	37.5
0–8	16	8.9	47	22.6	50	48.1
Maternal education at birth (years; *n* = 538)						
9+	78	37.7	36	16.5	8	7.1
5–8	96	46.4	120	55.1	38	33.6
0–4	33	15.9	62	28.4	67	59.3
Smoking status (*n* = 511)						
Never	160	80.8	131	64.5	71	64.5
Former	17	8.6	20	9.9	13	11.8
Current	21	10.6	52	25.6	26	23.6
Dental flossing (age 24; *n* = 477)						
Yes or sometimes	113	61.4	99	51.8	34	33.3
Never	71	38.6	92	48.2	68	66.7
Bleeding on probing (age 24; *n* = 539)						
0 Tooth	171	82.6	174	79.8	83	72.8
1 or more teeth	36	17.4	44	20.2	31	27.2
Dental calculus (age 24; *n* = 539)						
0 Tooth	63	30.4	47	21.6	28	24.6
1 or more teeth	144	69.6	171	78.4	86	74.4
Presence of 4 mm+ periodontal pocket (age 24; *n* = 539)						
0 Tooth	204	8.5	212	97.2	104	91.2
1 or more teeth	3	1.5	6	2.8	10	8.8
Income birth (*n* = 539)						
1st tertile	27	13.0	34	15.6	98	86.0
2nd and 3rd tertiles	180	87.0	184	84.4	16	14.0
Income age 15 (*n* = 534)						
1st tertile	0	—	86	39.8	105	92.9
2nd and 3rd tertiles	205	100.0	130	60.2	8	7.1
Income age 19 (*n* = 530)						
1st tertile	0	—	84	39.3	89	80.2
2nd and 3rd tertiles	205	100.0	130	60.7	22	19.8

(Continues)

TABLE 3 (Continued)

Variables	Trajectory 1 Stable high income		Trajectory 2 Stable middle income		Trajectory 3 Low and variable income	
	n	%	*n*	%	*n*	%
Income age 23 (*n* = 511)						
1st tertile	0	–	69	34.0	80	72.7
2nd and 3rd tertiles	198	100.0	134	66.0	30	27.3
Income age 30 (*n* = 470)						
1st tertile	0	–	96	47.1	47	48.5
2nd and 3rd tertiles	169	100.0	108	52.9	50	51.5

A third potential pathway for the association between income trajectories and periodontitis may be related to a neo-materialist explanation. Additionally to individual exposures related to lack of resources, the neo-material interpretation considers the impact of societal factors on health outcomes. Under this explanation, contextual conditions related to the place where people live, such as a how resources are invested on human, physical, health and social infrastructure, are expected to have an effect on health outcomes, with individuals from more unequal societies presenting a higher burden of disease (Lynch, Smith, Kaplan, & House, 2000).

The evidence of the present study is in line with previous research in social epidemiology, showing that long-term and persistent exposure to relative socio-economic disadvantage earlier in life is associated with periodontitis later on (Poulton et al., 2002; Schuch et al.,

TABLE 4 Multivariable analyses between any periodontitis (CDC-AAP case definitions) and demographic, socio-economic, behavioural and clinical characteristics. Log-Poisson regression analysis. 1982 Pelotas Birth Cohort Study, Brazil

Variables	Crude analysis (*n* = 539) PR (95% CI)	*p*	Model 1 (*n* = 491) PR (95% CI)	*p*	Model 2 (*n* = 433) PR (95% CI)	*p*
SEP trajectory						
1 (Stable high income)	1.0	<.01	1.0	.05	1.0	.11
2 (Stable middle income)	1.2 (0.9;1.6)		1.2 (0.9;1.5)		1.1 (0.8;1.4)	
3 (Low and variable income)	1.5 (1.0;1.9)		1.4 (1.0;2.0)		1.4 (1.0;2.0)	
Sex						
Male	1.0	<.01	1.0	.01	1.0	.03
Female	0.7 (0.6;0.9)		0.8 (0.6;0.9)		0.8 (0.6;1.0)	
Education (years—age 30)						
12+	1.0	.13	1.0	.56	1.0	.67
9–11	1.1 (0.8;1.4)		1.0 (0.8;1.3)		1.0 (0.7;1.3)	
0–8	1.2 (0.9;1.6)		1.1 (0.8;1.5)		1.1 (0.8;1.6)	
Maternal education at birth						
9+	1.0	.53	1.0	.45	1.0	.43
5–8	1.0 (0.7;1.3)		0.8 (0.6;1.0)		0.8 (0.6;1.1)	
0–4	1.1 (0.8;1.5)		0.9 (0.6;1.2)		0.8 (0.6;1.2)	
Smoking status (age 23)						
Never	1.0	.62	–	–	1.0	.83
Former	0.6 (0.4;1.1)		–		0.7 (0.4;1.1)	
Current	1.1 (0.9;1.5)		–		1.0 (0.8;1.4)	
Dental flossing (age 24)						
Yes or sometimes	1.0	.12	–	–	1.0	.55
Never	1.2 (0.9;1.5)		–		0.9 (0.7;1.2)	
Bleeding on probing (age 24)	1.0 (1.0;1.1)	<.01	–	–	1.0 (1.0;1.1)	.09
Dental calculus (age 24)	1.0 (1.0;1.0)	<.01	–	–	1.0 (1.0;1.0)	.04
Periodontal pocket (age 24)	1.2 (1.1;1.3)	<.01	–	–	1.1 (1.1;1.2)	<.01
Number of teeth (age 24)	1.0 (0.8;1.1)	.73			1.0 (0.8;1.1)	.79
SEP traj × Education age 30[a]		.06				
SEP traj × Maternal education[a]		.25				

Model 1: Sociodemographic variables. Model 2: Full model.
[a]Interactions were included separately in Model 2 and not statistically significant in the final model.

TABLE 5 Multivariable analyses between moderate or severe periodontitis (CDC-AAP case definitions) and demographic, socio-economic, behavioural and clinical characteristics. Log-Poisson regression analysis. 1982 Pelotas Birth Cohort Study, Brazil

	Crude analysis (n = 539)		Model 1 (n = 491)		Model 2 (n = 433)	
Variables	PR (95% CI)	p	PR (95% CI)	p	PR (95% CI)	p
SEP trajectory						
1	1.0	.04	1.0	.058	1.0	<.05
2	1.3 (0.8;2.2)		1.2 (0.7;1.9)		1.1 (0.6;2.1)	
3	1.7 (1.0;3.0)		1.8 (1.0;3.2)		2.1 (1.1;4.1)	
Sex						
Male	1.0	<.01	1.0	.01	1.0	.02
Female	0.6 (0.4;0.9)		0.6 (0.4;0.9)		0.6 (0.4;0.9)	
Education (years—age 30)						
12+	1.0	.02	1.0	.17	1.0	.60
9–11	1.1 (0.7;1.9)		1.0 (0.6;1.7)		0.9 (0.5;1.6)	
0–8	1.8 (1.1;3.0)		1.5 (0.9;2.4)		1.2 (0.6;2.2)	
Maternal education (years)						
9+	1.0	.59	1.0	.51	1.0	.55
5–8	1.0 (0.6;1.7)		0.7 (0.4;1.3)		0.8 (0.4;1.6)	
0–4	1.2 (0.7;2.0)		0.8 (0.4;1.4)		0.8 (0.4;1.6)	
Smoking status (age 23)						
Never	1.0	.43	–	–	1.0	.93
Former	0.6 (0.2;1.5)		–		0.7 (0.3;1.9)	
Current	1.3 (0.8;2.2)		–		1.1 (0.6;1.9)	
Dental flossing (age 24)						
Yes or sometimes	1.0	.35	–	–	1.0	.52
Never	1.2 (0.8;2.0)		–		0.8 (0.5;1.4)	
Bleeding on probing (age 24)		.25		–		.34
Continuous	1.0 (1.0;1.1)		–		1.0 (1.0;1.1)	
Dental calculus (age 24)						
Continuous	1.0 (1.0;1.1)	.23	–	–	1.0 (1.0;1.1)	.44
Presence of periodontal pocket (age 24)						
Continuous	1.2 (1.1;1.4)	<.01	–	–	1.2 (1.0;1.3)	<.05
Number of teeth (age 24)						
Continuous	1.1 (0.8;1.4)	.73			1.1 (0.9;1.5)	.40
SEP traj × Education age 30[a]		<.01				
SEP traj × Maternal education[a]		.06				

Model 1: Sociodemographic variables. Model 2: Full model.
[a]Interactions were included separately in Model 2 and not statistically significant in the final model.

2017). Although there have been studies evaluating the effect of early childhood exposures and social mobility in oral health, few studies analysed an association using a life course approach. The current study is the first one using the perspective of income trajectories over the lifespan.

Some questions on the association between socio-economic position during the life course and periodontitis in adulthood remained unclear. As it has been suggested, the fact that cumulative family income is clearly important for the occurrence of disease does not preclude the idea that income at a specific point may have a stronger effect than in other moments of life (Chen et al., 2007). It is important to know exactly what is most critical period of life, that is when the socio-economic disadvantage will have a greater impact on occurrence of disease later on. Such evidence would inform a timely appropriate

intervention in the group of risk. It is theoretically plausible to assume that some behavioural characteristics over the life course, such as smoking status and flossing pattern, would mediate such an association. This role, however, is still not clear in the literature, and further studies are needed to deeply understand this question.

In conclusion, income trajectories from birth to age 30 years affected moderate-to-severe periodontitis measured in the fourth decade of life, even after controlling for demographic, behavioural and clinical variables. The findings contribute to identifying income as a structural determinant of this chronic oral condition and inform population-based measures to prevent periodontitis.

ACKNOWLEDGEMENTS

This article is based on data from the study "Pelotas Birth Cohort, 1982" conducted by Postgraduate Program in Epidemiology at Universidade Federal de Pelotas with the collaboration of the Brazilian Public Health Association (ABRASCO). From 2004 to 2013, the Wellcome Trust supported the 1982 birth cohort study. The International Development Research Center, World Health Organization, Overseas Development Administration, European Union, National Support Program for Centers of Excellence (PRONEX), the Brazilian National Research Council (CNPq) and the Brazilian Ministry of Health supported previous phases of the study. The Oral Health Study-2013 (OHS-13) was supported by the National Counsel of Technological and Scientific Development (CNPq; #403257/2012-3: FFP and #475979/2013-3: MBC). The authors are also grateful to the Brazilian government agency (CAPES—Coordination for the Improvement of Higher Education Personnel) for the PhD scholarship (HSS—Process 13774-13-1).

CONFLICT OF INTEREST

The authors declare no conflict of interest.

ORCID

Helena S. Schuch http://orcid.org/0000-0001-9932-9698

Flavio F. Demarco http://orcid.org/0000-0003-2276-491X

REFERENCES

Barros, A. J., Cascaes, A. M., Wehrmeister, F. C., Martinez-Mesa, J., & Menezes, A. M. (2011). Tobacco smoking in Brazil: Regional inequalities and prevalence according to occupational characteristics. *Ciencia e Saude Coletiva*, *16*(9), 3707–3716. https://doi.org/10.1590/S1413-81232011001000008

Chen, E., Martin, A. D., & Matthews, K. A. (2007). Trajectories of socioeconomic status across children's lifetime predict health. *Pediatrics*, *120*(2), e297–e303. https://doi.org/10.1542/peds.2006-3098

Delgado-Angulo, E. K., & Bernabe, E. (2015a). Comparing lifecourse models of social class and adult oral health using the 1958 National Child Development Study. *Community Dental Health*, *32*(1), 20–25.

Delgado-Angulo, E. K., & Bernabe, E. (2015b). Intergenerational mobility and adult oral health in a British cohort. *Community Dentistry and Oral Epidemiology*, *43*(3), 255–261. https://doi.org/10.1111/cdoe.12149

Eke, P. I., Page, R. C., Wei, L., Thornton-Evans, G., & Genco, R. J. (2012). Update of the case definitions for population-based surveillance of periodontitis. *Journal of Periodontology*, *83*(12), 1449–1454. https://doi.org/10.1902/jop.2012.110664

Hallal, P. C., Clark, V. L., Assuncao, M. C., Araujo, C. L., Goncalves, H., Menezes, A. M., & Barros, F. C. (2012). Socioeconomic trajectories from birth to adolescence and risk factors for noncommunicable disease: Prospective analyses. *Journal of Adolescent Health*, *51*(6 Suppl), S32–S37. https://doi.org/10.1016/j.jadohealth.2012.06.022

Hiscock, R., Bauld, L., Amos, A., Fidler, J. A., & Munafo, M. (2012). Socioeconomic status and smoking: A review. *Annals of the New York Academy of Sciences*, *1248*, 107–123. https://doi.org/10.1111/j.1749-6632.2011.06202.x

Horta, B. L., Gigante, D. P., Goncalves, H., dos Santos, Motta J., Loret de Mola, C., Oliveira, I. O., … Victora, C. G. (2015). Cohort profile update: The 1982 Pelotas (Brazil) Birth Cohort Study. *International Journal of Epidemiology*, *44*(2), 441, 441a-441e. https://doi.org/10.1093/ije/dyv017

Jones, B. L., & Nagin, D. S. (2013). A note on a stata plugin for estimating group-based trajectory models. *Sociological Methods & Research*, *42*(4), 608–613. https://doi.org/10.1177/0049124113503141

Link, B. G., & Phelan, J. (1995). Social conditions as fundamental causes of disease. *Journal of Health and Social Behavior, Special No*, 80–94. https://doi.org/10.2307/2626958

Lynch, J. W., Smith, G. D., Kaplan, G. A., & House, J. S. (2000). Income inequality and mortality: Importance to health of individual income, psychosocial environment, or material conditions. *British Medical Journal*, *320*(7243), 1200–1204. https://doi.org/10.1136/bmj.320.7243.1200

Marcenes, W., Kassebaum, N. J., Bernabé, E., Flaxman, A., Naghavi, M., Lopez, A., & Murray, C. J. (2013). Global burden of oral conditions in 1990-2010: a systematic analysis. *J Dent Res.*, *92*(7), 592–597. https://doi.org/10.1177/0022034513490168.

McDonough, P., Sacker, A., & Wiggins, R. D. (2005). Time on my side? Life course trajectories of poverty and health. *Social Science and Medicine*, *61*(8), 1795–1808. https://doi.org/10.1016/j.socscimed.2005.03.021

Nagin, D. S. (2005). *Group-based modeling of development*. Cambridge, MA: Harvard Univ. Press. https://doi.org/10.4159/9780674041318

Nagin, D. S., & Odgers, C. L. (2010). Group-based trajectory modeling in clinical research. *Annual Review of Clinical Psychology*, *6*, 109–138. https://doi.org/10.1146/annurev.clinpsy.121208.131413

Nicolau, B., Netuveli, G., Kim, J. W., Sheiham, A., & Marcenes, W. (2007). A life-course approach to assess psychosocial factors and periodontal disease. *Journal of Clinical Periodontology*, *34*(10), 844–850. https://doi.org/10.1111/j.1600-051X.2007.01123.x

Page, R. C., & Eke, P. I. (2007). Case definitions for use in population-based surveillance of periodontitis. *Journal of Periodontology*, *78*(7 Suppl), 1387–1399. https://doi.org/10.1902/jop.2007.060264

Peres, K. G., Peres, M. A., Demarco, F. F., Tarquinio, S. B., Horta, B. L., & Gigante, D. P. (2011). Oral health studies in the 1982 Pelotas (Brazil) birth cohort: Methodology and principal results at 15 and 24 years of age. *Cadernos de Saude Publica*, *27*(8), 1569–1580. https://doi.org/10.1590/S0102-311X2011000800012

Peres, M. A., Peres, K. G., Thomson, W. M., Broadbent, J. M., Gigante, D. P., & Horta, B. L. (2011). The influence of family income trajectories from birth to adulthood on adult oral health: Findings from the 1982 Pelotas birth cohort. *American Journal of Public Health*, *101*(4), 730–736. https://doi.org/10.2105/AJPH.2009.184044

Poulton, R., Caspi, A., Milne, B. J., Thomson, W. M., Taylor, A., Sears, M. R., & Moffitt, T. E. (2002). Association between children's experience of socioeconomic disadvantage and adult health: A life-course study. *Lancet*, *360*(9346), 1640–1645. https://doi.org/10.1016/S0140-6736(02)11602-3

Raftery, A. E. (1995). Bayesian model selection in social research. *Sociological Methodology*, *25*, 111–163. https://doi.org/10.2307/271063

Schuch, H. S., Peres, K. G., Do, L. G., & Peres, M. A. (2015). Can socioeconomic trajectories during the life influence periodontal disease occurrence in adulthood? Hypotheses from a life-course perspective. *Medical Hypotheses*, *84*(6), 596–600. https://doi.org/10.1016/j.mehy.2015.03.011

Schuch, H. S., Peres, K. G., Singh, A., Peres, M. A., & Do, L. G. (2017). Socioeconomic position during life and periodontal disease in adulthood: A systematic review. *Community Dentistry and Oral Epidemiology*, *45*(3), 201–208. https://doi.org/10.1111/cdoe.12278

Thomson, W. M., Broadbent, J. M., Welch, D., Beck, J. D., & Poulton, R. (2007). Cigarette smoking and periodontal disease among 32-year-olds: A prospective study of a representative birth cohort. *Journal of Clinical Periodontology*, *34*(10), 828–834. https://doi.org/10.1111/j.1600-051X.2007.01131.x

Victora, C. G., Fenn, B., Bryce, J., & Kirkwood, B. R. (2005). Co-coverage of preventive interventions and implications for child-survival strategies: Evidence from national surveys. *Lancet*, *366*(9495), 1460–1466. https://doi.org/10.1016/S0140-6736(05)67599-X

Walker, R. (1998). Rethinking poverty in a dynamic perspective. In H. J. Andress (Ed.), *Empirical poverty research in a comparative perspective* (pp. 29–50). Brookfield, VT: Avebury.

How to cite this article: S Schuch H, G Peres K, Demarco FF, et al. Effect of life-course family income trajectories on periodontitis: Birth cohort study. *J Clin Periodontol*. 2017;00:1–10. https://doi.org/10.1111/jcpe.12845

CHAPTER 7 (PAPER 4)

The direct effect of early life income on periodontitis in a birth cohort

Linkage to the body of work

The final paper of the thesis was conducted to test the life-course epidemiology theory of the critical period on the relationship between SEP assessed by income and periodontal disease in adulthood. Using a novel statistical technique, the researchers tested the effect of early life SEP on periodontal disease, controlling the effect for SEP in young adulthood and for well-known risk factors for periodontal disease, namely smoking status and oral hygiene status, measured by dental calculus. The analyses adjusted for two potential confounding factors of the association between SEP and periodontal disease: sex and maternal education at the time of the participant´s birth. The findings confirmed the hypothesis that early life SEP has had a direct effect on periodontal disease in adulthood that is not mediated by SEP in adulthood, smoking or oral hygiene status.

Highlights

- Early life SEP measured by family income at a participant's birth has long-term effects on periodontal disease.
- There was a direct effect of early life SEP on periodontal disease in adult individuals from families with low income at birth; they have more than double the risk of having moderate-to-severe periodontal disease in adulthood after conditioning on adulthood income and education.
- Early life SEP has effects on periodontal disease at age 31that are not mediated by smoking and oral hygiene status in young adulthood.

Statement of authorship form

Statement of Authorship

80%Title of Paper	The direct effect of early life income on periodontitis in a birth cohort
Publication Status	☐ Published ☐ Accepted for Publication ☐ Submitted for Publication ☑ Unpublished and Unsubmitted work written in manuscript style
Publication Details	Schuch HS, Nascimento GG, Peres KG, Demarco FF, Correa MB, Horta BL, Peres MA, Do LG. The direct effect of early life income on periodontitis in a birth cohort.

Principal Author

Name of Principal Author (Candidate)	Helena Silveira Schuch
Contribution to the Paper	Helped in data collection, analysed and interpreted data, wrote manuscript and acted as corresponding author.
Overall percentage (%)	80%
Certification:	This paper reports on original research I conducted during the period of my Higher Degree by Research candidature and is not subject to any obligations or contractual agreements with a third party that would constrain its inclusion in this thesis. I am the primary author of this paper.
Signature	Date 05.02.2018

Co-Author Contributions

By signing the Statement of Authorship, each author certifies that:

i. the candidate's stated contribution to the publication is accurate (as detailed above);

ii. permission is granted for the candidate in include the publication in the thesis; and

iii. the sum of all co-author contributions is equal to 100% less the candidate's stated contribution.

Name of Co-Author	Gustavo Giacomelli Nascimento
Contribution to the Paper	Contributed to data collection, assisted in statistical analyses, critically evaluated and edited manuscript.
Signature	Date 05/02/2018

Name of Co-Author	Karen Glazer Peres
Contribution to the Paper	Supervised development of work, helped in interpretation, manuscript evaluation and edition.
Signature	Date 05/02/2018

Name of Co-Author	Flavio Fernando Demarco		
Contribution to the Paper	Contributed to data collection, critically evaluated and edited manuscript.		
Signature		Date	05/02/2018

Name of Co-Author	Marcos Britto Correa		
Contribution to the Paper	Contributed to data collection, critically evaluated and edited manuscript.		
Signature		Date	05/02/2018

Name of Co-Author	Bernardo Lessa Horta		
Contribution to the Paper	Contributed to data collection, critically evaluated and edited manuscript.		
Signature		Date	05 / 02 / 2018

Name of Co-Author	Marco Aurelio Peres		
Contribution to the Paper	Supervised development of work, helped in interpretation, manuscript evaluation and edition.		
Signature		Date	05/02/2018

Name of Co-Author	Loc Giang Do		
Contribution to the Paper	Supervised development of work, helped in interpretation, manuscript evaluation and edition.		
Signature		Date	05/02/2018

The direct effect of early life income on periodontitis in a birth cohort

Schuch HS[1], Nascimento GG[2,3], Peres KG[1], Peres MA[1], Do LG[1]

[1]Australian Research Centre for Population Oral Health (ARCPOH), Adelaide Dental School, The University of Adelaide, Australia.

[2]Postgraduate Program in Dentistry, Federal University of Pelotas, Brazil.

[3]Section of Periodontology, Department of Dentistry and Oral Health, Aarhus University, Aarhus, Denmark.

.

Corresponding author:

Helena Silveira Schuch

Adelaide Health & Medical School (AHMS) Building

Cnr of North Terrace and George Street, Level 9, South Australia 5005, Australia

Ph +61 8 8313 0612

E-mail: helena.schuch@adelaide.edu.au

Manuscript to be submitted to the Journal of Dental Research

ABSTRACT

The aim of this study was to determine the controlled direct effect of early life socioeconomic position (SEP) measured by household income at a participant's birth on periodontitis at the age of 31 years, controlling for adulthood income and education attainment, smoking, and dental calculus. Data from the 1982 Pelotas Birth Cohort Study, Brazil were used. Early life SEP was measured by family income at the participant's birth and at the age of 23 years and categorised as relatively low (1st tertile) and relatively middle and high income groups (2nd and 3rd tertiles). Periodontal measures were assessed through clinical examination in the 2013 Oral Health sub-study at age 31 (OHS-13) and categorised as healthy, mild or moderate-to-severe disease. Three variables were included as mediators: smoking status at age 24; dental calculus as an indicator of dental hygiene, also at age 24; and the participant´s education level by age 30. Confounding variables included sex and maternal education at the participant's birth. Marginal structural models with inverse probability weights were used to assess the controlled direct effect (CDE) of early life SEP on periodontitis. Multinomial regression models were used to estimate risk ratios (RRs) and its 95% Confidence Intervals (CI). The prevalence of mild and moderate-to-severe periodontitis was 37.3% and 14.3%, respectively (n= 539). The CDE of SEP on periodontitis showed that individuals from the lowest income tertile had at least double the risk of moderate-to-severe periodontitis that was not mediated by different mediators (RR of 2.4 (95%CI 1.4;4.1) for smoking status; RR of 2.1 (95%CI 1.2;3.7) for dental calculus, RR of 2.2 (95%CI 1.2;4.1) for a participant's education at age 30. Conditioning on adulthood income, the RR was 2.4

(95%CI 1.4;4.1). Early life appears to be a critical period in the development of periodontitis in adulthood.

Introduction

Half of the world's population suffer from oral diseases (Kassebaum et al. 2017). Periodontitis is an infectious disease resulting in inflammation within periodontal tissues and alveolar bones (American Academy of Periodontology Task Force Report on the Update to the 1999 Classification of Periodontal Diseases and Conditions 2015). It affects more than 537 million people, with a prevalence of 7.6% for all ages combined. Some health-related behaviours are known risk factors for periodontitis, such as smoking and inadequate oral hygiene (Watt and Petersen 2012). The vast majority of known risk factors for periodontitis are influenced by social conditions (Thomson, Sheiham and Spencer 2012). The 'causes behind the causes' of health are known as social determinants of health (Link and Phelan 1995).

The social determinants of health include (but are not restricted to) social and economic experiences at a certain time during life, such as income, education opportunities and attainment, employment and the working environment. Life-course epidemiology postulates that health conditions are influenced by dynamic changes of the circumstances in which people are born, grow, live, work and age (Ben-Shlomo and Kuh 2002). Understanding those influences can shed light on the onset and progression of chronic health conditions in order to inform effective and timely interventions. Despite there is increasing evidence on the association between socioeconomic position (SEP) and periodontitis (Boillot et al. 2011; Borrell and Crawford 2012; Klinge and Norlund 2005, Schuch et al. 2017) there is still a gap in the literature on the influence of the circumstances in which

people are born and grow, on their periodontal health later in life based on longitudinal studies.

The critical period theory of the life-course epidemiology postulates that conditions in a specific development period in life, usually in early life, determine the occurrence of disease later on (Ben-Shlomo and Kuh 2002). This model has proven to be accurate for several chronic conditions (Hayman et al. 2011). In the oral health field, there is evidence that relatively low SEP at birth is associated with the number of unsound teeth in young adulthood, regardless of family income in adolescence and young adulthood (Peres et al. 2011). A birth cohort study in Dunedin, NZ, identified association between early life SEP, measured by parental occupation, and periodontitis in the third and fourth decades of life. The findings showed an independent association between parental occupation with periodontitis (Poulton et al. 2002). However, some specific statistical analysis techniques not included in Dunedin study may help to deal with time-dependent confounding and covariates. For example, by using Marginal Structural Models (MSM) it is possible to simulate a hypothetical experiment with observational data based on counter-factual assumption. The effect of confounding factors and mediators can be controlled for in the analysis. Using MSM it is possible to estimate the controlled direct effect (CDE) of exposure on outcome that is not mediated by later factors in the causal pathway (Robins, Hernan and Brumback 2000).

Living conditions such as income can have lifelong effects on periodontal health. However, assessing the effect of early life income is difficult because of the mediating effect of adulthood income (Milanovic 2011). The use of

MSM with inverse probability weighting can properly address the CDE of early life income on periodontitis by setting a mediator (e.g., adulthood income) to a specific value for all individuals in a specific population. There is a gap in the literature on the effects of early life SEP on periodontitis, and there is absolutely no evidence of this effect in middle and low income countries. Therefore, this study aimed to estimate the CDE of family income at a participant's birth, controlling for adulthood income and educational attainment, on periodontitis in young adulthood. Additionally, the same effect, controlling for behavioural risk factors was also estimated.

Methods

1982 Pelotas birth cohort study

Data from the 1982 Pelotas Birth Cohort Study was used. In 1982, the three maternity hospitals in the city of Pelotas, South Brazil, were visited daily, and all 5,914 children born in that year were invited to be part of the prospective population-based birth cohort study. This population has been followed up several times. Oral health sub-studies were conducted at years 1997 (OHS-97), 2006 (OHS-06) and 2013 (OHS-13). For the present study information gathered at years 1982, at the participants' birth, 2005, at age 23, 2006, at age 24, and in 2012, at 31 years old were used. All sub-studies were approved by the Ethics Committee of the Federal University of Pelotas, and a signed consent form was collected from all participants. Detailed methodological information can be assessed elsewhere (Horta et al. 2015; Peres et al. 2011).

Oral Health sub-study 2013

The first Oral Health Sub-Study was carried out in 1997 (OHS-97), when participants were 15 years-old. At that time, a random sample of 900 individuals was invited to take part in the OHS-97. A total of 888 individuals completed the OHS-97. In 2013 (OHS-13), the researchers intended to follow up the 888 participants of the OHS-97.

A team of 6 examiners and 6 interviewers conducted the OHS-13. The examiners were trained dentists with experience in epidemiological studies. Interviews and clinical oral examinations were conducted through home visits. Prior to the data collection, examiners were trained and calibrated and 30 volunteers were clinically examined. The lowest intra-class correlation coefficient for periodontal examination data was 0.85. The questionnaire included socio-demographic and behavioural information. Clinical oral examination was conducted following the biosafety procedures recommended by the World Health Organization for epidemiological surveys, and a head-light, dental mirror, and PCP2 periodontal probe with 2-mm banding were used (Hu-Friedy PCP-2; Rotterdam, the Netherlands). Gingival recession and periodontal pocket depth were examined in six sites (mesiobuccal, midbuccal, distobuccal, mesiolingual, midlingual and distolingual) per tooth of all teeth, excluding 3rd molars. Teeth that could not be assessed due to physical barriers such as large amount of calculus and an orthodontic band were excluded from the oral examination. Attachment loss (CAL) was estimated as a sum in millimetres of gingival recession and pocket depth at each site.

Exposures

Early life income at birth and at age 23 years were the exposures. We categorised income information at each time into tertiles, and then dichotomised into relatively poorer (1st tertile) versus relatively middle and richer groups (2nd and 3rd tertiles). There is evidence that middle and upper income groups in Brazil are comparable, while the poor lag well behind (Victora et al. 2005).

Family income at both ages was collected through face-to-face questionnaires, referring to the sum of the earnings in the previous month from all people living in the house. At age 23, income was collected as a continuous variable, therefore the categorisation into tertiles was straightforward. At birth, however, family income was collected in 5 pre-specified categories. In order to obtain three income categories with a comparable number of individuals, a principal components analysis was conducted with 4 variables: health services payment mode (out of pocket, public free, or private health insurance), maternal education, height, and skin colour. A score was derived from the first component that was used to rank individuals within family income groups. After that, cut-off points within each category were identified, in order to form 3 groups with approximately the same number of individuals. (Peres et al. 2011).

Outcome

The outcome was periodontitis, assessed through clinical examination at age 31. In the present study, periodontitis was defined based on the case definitions proposed in partnership between the Centers for Disease Control and Prevention (CDC) and the American Academy of Periodontology (Eke et

al. 2012). For the present study, an ordinal variable was created: (i) healthy/no periodontitis, (ii) mild, and (iii) moderate-to-severe periodontitis.

Mediators

As mediators, three variables were included: smoking status, dental calculus as an indicator of dental hygiene, and participants´ education at age 30. Smoking status and dental calculus were collected at age 24. Smoking status was dichotomised as current or former smoker versus non-smoker. Dental calculus was categorised in tertiles, and the second and third tertiles were then combined. The final variable for dental calculus was thus comparing the worst tertile of dental calculus versus the first and second tertiles combined. Education was collected in years of study when participants were 30 years old, and categorised as 0-8 years of study (corresponding to primary school in Brazil), 9-11 (high school), and 12 or more years of study.

Confounding factors

We considered as confounders factors sex and maternal education at the participant's birth. Maternal education was collected in years of study and categorised as 0-4 years, 5-8 and 9 or more years. (Figure 1 - DAG).

Statistical analysis

Figure 1 represents the Directed Acyclic Graph (DAG). The pathway of interest in this study was the direct path from early life income to periodontitis in adulthood. We estimated the CDE of family income at a participant's birth on periodontitis occurrence at age 31 not mediated by smoking, oral hygiene and participant's education at age 30. CDE is the

direct effect of exposure on outcome; in other words, the effect that is not mediated by any other condition (VanderWeele 2011). For this purpose, the values of the mediators were fixed, while a comparison of the expected outcome was estimated, conditional on the exposure and the mediators for different values of the exposure (0 or 1). Stabilised weights were calculated for the exposure and each mediator. The final stabilised weight was obtained by multiplying the weight of the exposure by the weight of mediators.

The stabilised inverse probability weight of early life income was estimated based on the following formula:

$$W_i^X = \frac{P(X = x_i)}{P(X = x_i | C = c_i)}$$

Where x_i, and c_i are respectively the actual values of exposure, and confounders for individual *i*.

Stabilised weights of mediators were estimated as follows:

$$W_i^M = \frac{P(M = m_i | X = x_i)}{P(M = m_i | X = x_i, C = c_i)}$$

where $x_i(t)$, $m_i(t)$ are the actual values of the exposure and the mediator; and ci represents the baseline confounders for individual *i*. Multinomial regression models were used to evaluate the effect of early life income on periodontitis, controlling for adult income and different mediators. All analyses were conducted using the software Stata 14.1 (StataCorp; College Station; TX; USA).

Results

From 888 in OHS-07 a total of 539 (61.0%) individuals participated in the OHS-13. Socioeconomic and demographic indicators of those participants who were followed-up in the OHS-13 were comparable with the original 1982 Pelotas Birth Cohort study (Table 1). Table 2 shows the characteristics of the sample stratified by sex. The overall prevalence of mild periodontitis was 23.0% and moderate-to-severe periodontitis 14.3%. Women tended to have less moderate-to-severe periodontitis than males. Apart from dental calculus at age 31, all other variables had a similar distribution among men and women.

Table 3 displays a cross-tabulation of covariates and the two levels of periodontitis. Crude estimates from Multinomial Regression analyses of covariates and periodontitis can be also observed in Table 2. Individuals in the lowest tertile of income at birth had a prevalence of mild periodontitis of 21.8%, while it was only 12.6% among those in middle or highest income categories. The lower income group had 1.7 higher risk of presenting such level of disease than those in the middle or higher income groups. Higher risk of moderate-to-severe periodontitis in the crude analysis was observed among males and those in the worst tertile of dental calculus. An association was also observed between the more severe level of disease and being a smoker at age 23, as well as having less than 12 years of study at age 30.

Adjusted estimates from the MSMs are presented in Table 4. Participant's sex and maternal education at the participant's birth were included as confounders. However, when maternal education was included in the model, the mean weight of the exposure was 0.93, violating one of the requirements of MSM that the mean stabilised weight should be close to 1.00, After

removing this variable, the stabilised weight was 1.00, indicating a consistent and stable model. CDE of family income at birth on periodontitis in adulthood showed that individuals from the lowest income tertile had at least double the risk of moderate-to-severe periodontitis that was not mediated by different mediators (Risk Ratio – RR - 2.4 (95%CI 1.4;4.1)) for smoking status; RR 2.1 (95%CI 1.2;3.7) for dental calculus, RR 2.2 (95%CI 1.2;4.1) for participant's education. Additionally, results from MSM showed that individuals in the lowest income tertile at birth had 2.4 (95%CI 1.4;4.1) times the risk of moderate-to-severe periodontitis at age 31 than those from middle and high income groups, conditioning on family income at age 23. Sensitivity analyses using different case definitions is presented in Supplementary Tables S1-S4.

Discussion

In this study, early life income had a direct effect on periodontitis in adulthood, corroborating the life-course epidemiology theory of a critical period. Although this theory has proven to be accurate for other chronic health conditions, to the best of the authors' knowledge there is only one study testing this theory on periodontal conditions using a prospective study, and it was conducted in a high income country and used occupation as the indicator of SEP (Poulton et al. 2002). Additionally, is the first using a robust statistical technique (MSM) to account for confounders and meditators to test critical period hypothesis in assessing periodontitis. The study findings

contribute a proof of principle of cause and effect between early life income and periodontitis in adulthood.

The robust statistical technique and with the study design are among the major strengths of the present study. The use of MSM allowed both the correct control of mediators and confounding factors in the analysis and the consistent estimate of the effect of SEP on periodontitis, controlled by mediators and weighted on confounding factors (Robins, Hernan and Brumback 2000). Another point that deserves attention is the quality of the data. The 1982 Pelotas Birth Cohort Study is the largest and longest birth cohort study in any middle or low income country, and one of the only with oral health data clinically collected. For the present study, a comprehensive periodontal examination was adopted, allowing the adoption of internationally recognised case definitions. The examination of six sites per tooth is an advantage if compared to the Dunedin Multidisciplinary Health and Development Study, which collects information on three sites per tooth. Additionally, the response rate was over 60%, acceptable for a longitudinal study with such long follow-up. The cross-comparison of the followed-up sample and the original birth cohort demonstrated the representativeness of the followed-up sample and reinforced the internal consistency of our study, showing that the likelihood of selection bias was low.

The young age of the sample might have limited the statistical power of our analysis, since periodontitis has relatively low prevalence at this age. Nevertheless, significant effect of early life income on the prevalence of periodontitis has been observed. Another limitation is that income at birth

was collected in pre-specified categories, restricting the use of this information.

Our findings are in line with those from previous studies that showed that early life socioeconomic circumstances were important determinants for future disease experience. Indeed, it has been discussed that time is significant for shaping the experience of SEP disadvantage on health (McDonough, Sacker and Wiggins 2005). There are different hypotheses for such a lifelong effect on health outcomes, and the two most frequently explored mechanisms are based on behavioural and psychosocial explanations. Additionally, a neo-materialist mechanism has been emphasised.

The behavioural pathway involves socially patterned behaviours such as inadequate hygiene, infrequent use of oral health care and smoking status. It is argued that these behaviours are learnt in early life, and that early life social conditions would influence and shape behaviours later on. These exposures would then increase the risk of periodontitis development and progression. It is increasing the understanding of the impact of systemic risk factors shaped by SEP conditions on the onset, rate of progression, and severity of periodontitis (Bergstrom, Eliasson and Dock 2000; Genco and Borgnakke 2013; Schuch et al. 2015; Thomson et al. 2007).

The psychosocial pathway identifies conditions such as social capital and stress as linking factors between socioeconomic disadvantage and poorer health. In fact, perceived social disadvantage and financial hardship may lead to increased stress levels. Increased stress may predispose to periodontitis through elevated levels of cytokines in the host, due to changes

in the immune, endocrinal, and neural systems that may occur as a consequence of stress. Stress can also increase harmful behaviours that, in turn, may lead to impaired periodontal health, such as smoking status and poor oral hygiene (Genco and Borgnakke 2013). From an understanding that poor oral hygiene can lead to periodontal health, researchers intended to include a proxy for oral hygiene in our models. The most straightforward conditions are dental calculus, flossing and brushing. Over half of the sample reported flossing at least once a day, and 95.6% reported brushing 2+ times a day (data not shown). Since these variables are self-reported and this may be subject to information bias, we used of dental calculus as a proxy of dental hygiene, since it was clinically and therefore objectively measured.

The neo-materialist explanation lies in the idea that socioeconomic inequalities in health may be due to differential affordability of food, housing, hygiene products and access to health care. This explanation also includes upstream determinants of health, from an understanding of the way that societies are organised and resources are invested on human, physical, health and social structure and how this impacts on the health of individuals (Lynch, Kaplan and Salonen 1997). Taking the perspective of periodontitis, societal characteristics such as income distribution, access to health services and the quality of health services may influence the disease occurrence. Public policies at different levels may also affect periodontitis, and tobacco taxation is a clear example due to the well-established effect of tobacco use on periodontitis.

Our study suggests that relatively low family income in early life increased the risk of moderate-to-severe periodontitis in adulthood, and this may

challenge the assumption that oral health in adulthood would be more influenced by immediate rather than past socioeconomic circumstances (Lee and Han 2016). Although there is evidence of the relationship between SEP early in life and SEP in adulthood, our analysis showed a direct effect of early life SEP on moderate-to-severe periodontitis, controlling for income or education in adult life, as well as some potential behavioural pathways. The study findings support the hypothesis that early life SEP has a direct effect on periodontitis in adulthood. Being a well-designed longitudinal cohort, our study contributes towards a better understanding of the social determinants of periodontitis. Such knowledge is paramount in informing public policies about the timely interventions, in order to reduce oral health inequalities. Our findings corroborate with previous evidence on life-course effects of social conditions on chronic diseases, indicating that reducing income inequalities in early life may have lifelong benefits on oral health.

References

American Academy of Periodontology Task Force Report on the Update to the 1999 Classification of Periodontal Diseases and Conditions, (2015). *J Periodontol*, vol. 86, no. 7, Jul, pp. 835-838.

Ben-Shlomo, Y & Kuh, D (2002). A life course approach to chronic disease epidemiology: conceptual models, empirical challenges and interdisciplinary perspectives, *Int J Epidemiol*, vol. 31, no. 2, Apr, pp. 285-293.

Bergstrom, J, Eliasson, S & Dock, J (2000). A 10-year prospective study of tobacco smoking and periodontal health', *J Periodontol*, vol. 71, no. 8, Aug, pp. 1338-1347.

Boillot, A, El Halabi, B, Batty, GD, Range, H, Czernichow, S & Bouchard, P (2011). Education as a predictor of chronic periodontitis: a systematic review with meta-analysis population-based studies, *PLoS One*, vol. 6, no. 7, p. e21508.

Borrell, LN & Crawford, ND (2012). Socioeconomic position indicators and periodontitis: examining the evidence, *Periodontol 2000*, vol. 58, no. 1, Feb, pp. 69-83.

Eke, PI, Page, RC, Wei, L, Thornton-Evans, G & Genco, RJ (2012). Update of the case definitions for population-based surveillance of periodontitis, *J Periodontol*, vol. 83, no. 12, Dec, pp. 1449-1454.

Genco, RJ & Borgnakke, WS (2013). Risk factors for periodontal disease, *Periodontol 2000*, vol. 62, no. 1, Jun, pp. 59-94.

Hayman, LL, Helden, L, Chyun, DA & Braun, LT (2011). A life course approach to cardiovascular disease prevention, *Eur J Cardiovasc Nurs*, vol. 10 Suppl 2, Jul, pp. S20-31.

Horta, BL, Gigante, DP, Goncalves, H, dos Santos Motta, J, Loret de Mola, C, Oliveira, IO, Barros, FC & Victora, CG (2015). Cohort Profile Update: The 1982 Pelotas (Brazil) Birth Cohort Study, *Int J Epidemiol*, vol. 44, no. 2, Apr, pp. 441, 441a-441e.

Kassebaum, NJ, Smith, AGC, Bernabé, E, Fleming, TD, Reynolds, AE, Vos, T, Murray, CJL, Marcenes, W & Collaborators, GOH (2017). Global, Regional, and National Prevalence, Incidence, and Disability-Adjusted Life Years for Oral Conditions for 195 Countries, 1990–2015: A Systematic

Analysis for the Global Burden of Diseases, Injuries, and Risk Factors, *Journal of dental research*, vol. 96, no. 4, pp. 380-387.

Klinge, B & Norlund, A (2005). A socio-economic perspective on periodontal diseases: a systematic review, *J Clin Periodontol*, vol. 32 Suppl 6, pp. 314-325.

Lee, HJ & Han, DH (2016) Early-life socioeconomic position and periodontal status in Korean adults, *Community Dent Oral Epidemiol*, vol. 44, no. 1, Feb, pp. 11-23.

Link, BG & Phelan, J (1995). Social conditions as fundamental causes of disease, *J Health Soc Behav*, vol. Spec No, pp. 80-94.

Lynch, JW, Kaplan, GA & Salonen, JT (1997). 'Why do poor people behave poorly? Variation in adult health behaviours and psychosocial characteristics by stages of the socioeconomic lifecourse, *Soc Sci Med*, vol. 44, no. 6, Mar, pp. 809-819.

McDonough, P, Sacker, A & Wiggins, RD (2005). Time on my side? Life course trajectories of poverty and health, *Soc Sci Med*, vol. 61, no. 8, Oct, pp. 1795-1808.

Milanovic, B (2011). *The haves and the have-nots: a short and idiosyncratic history of global inequality*, Basic Books, New York.

Peres, KG, Peres, MA, Demarco, FF, Tarquinio, SB, Horta, BL & Gigante, DP 2011, 'Oral health studies in the 1982 Pelotas (Brazil) birth cohort: methodology and principal results at 15 and 24 years of age', *Cad Saude Publica*, vol. 27, no. 8, Aug, pp. 1569-1580.

Peres, MA, Peres, KG, Thomson, WM, Broadbent, JM, Gigante, DP & Horta, BL (2011). The influence of family income trajectories from birth to adulthood on adult oral health: findings from the 1982 Pelotas birth cohort, *Am J Public Health*, vol. 101, no. 4, Apr, pp. 730-736.

Poulton, R, Caspi, A, Milne, BJ, Thomson, WM, Taylor, A, Sears, MR & Moffitt, TE (2002). Association between children's experience of socioeconomic disadvantage and adult health: a life-course study, *Lancet*, vol. 360, no. 9346, Nov 23, pp. 1640-1645.

Robins, JM, Hernan, MA & Brumback, B (2000) Marginal structural models and causal inference in epidemiology, *Epidemiology*, vol. 11, no. 5, Sep, pp. 550-560.

Schuch, HS, Peres, KG, Do, LG & Peres, MA (2015). Can socioeconomic trajectories during the life influence periodontal disease occurrence in adulthood? Hypotheses from a life-course perspective, *Med Hypotheses*, vol. 84, no. 6, Jun, pp. 596-600.

Schuch, HS, Peres, KG, Singh, A, Peres, MA & Do, LG (2017) Socioeconomic position during life and periodontitis in adulthood: a systematic review, *Community Dent Oral Epidemiol*, vol. 45, no. 3, Jun, pp. 201-208.

Thomson, WM, Broadbent, JM, Welch, D, Beck, JD & Poulton, R (2007). Cigarette smoking and periodontal disease among 32-year-olds: a prospective study of a representative birth cohort, *J Clin Periodontol*, vol. 34, no. 10, Oct, pp. 828-834.

Thomson, WM, Sheiham, A & Spencer, AJ (2012) Sociobehavioral aspects of periodontal disease, *Periodontol 2000*, vol. 60, no. 1, Oct, pp. 54-63.

VanderWeele, TJ (2011) Controlled direct and mediated effects: definition, identification and bounds, *Scand Stat Theory Appl*, vol. 38, no. 3, Sep, pp. 551-563.

Victora, CG, Fenn, B, Bryce, J & Kirkwood, BR (2005) Co-coverage of preventive interventions and implications for child-survival strategies: evidence from national surveys, *Lancet*, vol. 366, no. 9495, Oct 22-28, pp. 1460-1466.

Watt, RG & Petersen, PE (2012). Periodontal health through public health--the case for oral health promotion, *Periodontol 2000*, vol. 60, no. 1, Oct, pp. 147-155.

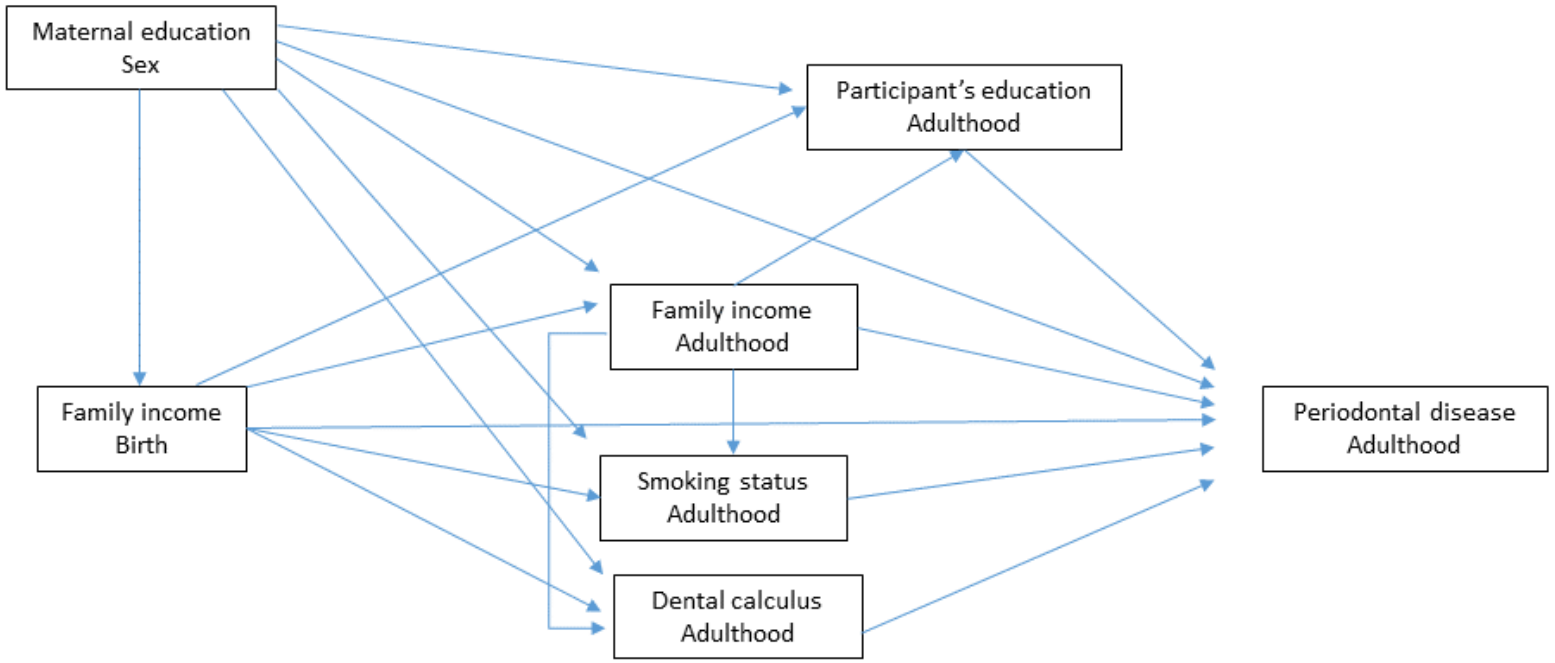

Figure 1. Directed acyclic graph (DAG).

Table 1. Comparison of demographic and socioeconomic characteristics at birth between the original sample and those located in the OHS-2013. 1982 Pelotas Birth Cohort Study, Brazil.

	The Pelotas birth cohort		Baseline of the nested oral health study (age 15)		The 2013 follow-up (age 31)	
Variables	n(%)	95% CI	n(%)	95% CI	n(%)	95% CI
Sex						
Male	3,037 (51.4)	(50.1;52.6)	480 (54.1)	(50.8;57.3)	273 (50.6)	(46.4;54.9)
Female	2,876 (48.6)	(47.4;49.9)	408 (45.9)	(42.7;49.2)	266 (49.3)	(45.1;53.6)
Maternal skin colour						
White	4,851 (82.1)	(81.1;83.0)	743 (83.8)	(81.2;86.1)	454 (84.2)	(80.9;87.1)
Black	1,060 (17.9)	(17.0;18.9)	144 (16.2)	(13.9;18.8)	85 (15.8)	(12.9;19.1)
Family Income at birth						
≤1 MW	1,288 (21.9)	(20.8;23.0)	161 (18.2)	(15.8;20.9)	93 (17.3)	(14.3;20.7)
1.1-3 MW	2,789 (47.4)	(46.1;48.7)	457 (51.7)	(48.4;55.0)	282 (52.4)	(48.2;56.6)
>3 MW	1,808 (30.7)	(29.6;31.9)	266 (30.1)	(27.2;33.2)	163 (30.3)	(26.5;34.3)
Maternal education at birth (years)						
0-4	1,960 (33.2)	(32.0;34.4)	285 (32.2)	(29.2;35.3)	162 (30.1)	(26.4;34.1)
5-8	2,454 (41.5)	(40.3;42.8)	393 (44.4)	(41.1;47.7)	254 (47.2)	(43.0;51.4)
9+	1,493 (25.3)	(24.2;26.4)	208 (23.5)	(20.8;26.4)	122 (22.7)	(19.3;26.4)

MW: Minimum wage.

Table 2. Sample characteristics. 1982 Pelotas Birth Cohort Study/Brazil.

Characteristics	n	%	Male			Female		
Sex			n	%	95% CI	n	%	95% CI
Male	273	50.6	-	-		-	-	
Female	266	49.4	-	-		-	-	
Maternal education								
9+ years of study	122	22.7	58	21.3	16.8;26.6	64	24.1	19.3;29.6
0-8 years of study	416	77.3	214	78.7	73.4;83.2	202	75.9	70.4;80.7
Family income at birth								
High (Middle and highest tertiles)	380	70.5	203	74.4	68.8;79.2	177	66.5	60.6;72.0
Low (Lowest tertile)	159	29.5	70	25.6	20.8;31.2	89	33.5	28.0;39.4
Family income at age 23								
High (Middle and highest tertiles)	362	70.8	192	75.6	69.9;80.5	170	66.1	60.1;71.7
Low (Lowest tertile)	149	29.2	62	24.4	19.5;30.1	87	33.9	28.3;39.9
Smoking status at age 24								
Non smoker	412	80.6	201	79.1	73.7;83.7	211	82.1	75.9;86.3
Smoker	99	19.4	53	20.9	16.3;26.3	46	17.9	13.7;23.1
Dental calculus at age 24								
2nd and 3rd tertiles	324	67.9	153	64.3	57.9;70.2	171	71.5	65.5;76.9
1st tertile (worst)	153	32.1	85	35.7	29.8;42.1	68	28.5	23.1;34.5
Participant's education at age 30								
12+ years of study	224	45.5	103	42.2	36.1;48.5	121	48.8	42.6;55.0
0-11 years of study	268	54.5	141	57.8	51.5;63.9	127	51.2	45.0;57.4
Periodontal condition at age 31								
Healthy	338	62.7	156	57.1	51.2;62.9	182	68.4	62.5;73.8
Mild disease	124	23.0	67	24.5	19.8;30.0	57	21.4	16.9;26.8
Moderate-to-severe disease	77	14.3	50	18.3	14.1;23.4	27	10.2	7.0;14.4

Table 3: Frequency of periodontitis by covariates and crude estimates from Multinomial Logistic regression models. 1982 Pelotas Birth Cohort Study/Brazil.

	Mild periodontitis			Moderate to severe periodontitis			Crude Mild		Crude Moderate-to-Severe	
Characteristics	n	%	(95% CI)	n	%	(95% CI)	RR	(95% CI)	RR	(95% CI)
Sex										
Male	67	24.5	(19.8;30.0)	50	18.3	(14.1;23.4)	1.0		1.0	
Female	57	21.4	(16.9;26.8)	27	10.2	(7.0;14.4)	0.7	(0.5;1.1)	0.5	(0.3;0.8)
Maternal education										
9+ years of study	28	23.0	(16.3;31.4)	17	13.9	(8.8;21.4)	1.0		1.0	
0-8 years of study	95	22.8	(19.0;27.1)	60	14.4	(11.4;18.2)	1.0	(0.6;1.6)	1.04	(0.6;1.9)
Family income at birth										
High (Middle and highest tertiles)	83	21.8	(18.0;26.3)	48	25.8	(19.5;33.2)	1.0		1.0	
Low (Lowest tertile)	41	12.6	(9.6;16.4)	29	18.2	(12.9;25.1)	1.4	(0.9;2.2)	1.7	(1.0;2.9)
Family income at age 23										
High (Middle and highest tertiles)	82	22.7	(18.6;27.3)	52	14.4	(11.1;18.4)	1.0		1.0	
Low (Lowest tertile)	35	23.5	(17.3;31.1)	20	13.4	(8.8;20.0)	1.0	(0.7;1.7)	0.9	(0.5;1.7)
Smoking status at age 24										
Non smoker	93	22.6	(18.8;26.9)	54	13.1	(10.2;16.7)	1.0		1.0	
Smoker	24	24.2	(16.7;33.8)	18	18.2	(11.7;27.2)	1.2	(0.7;2.0)	1.6	(0.9;2.8)
Dental calculus at age 24										
2nd and 3rd tertiles	67	20.7	(16.6;25.5)	37	11.4	(8.4;15.4)	1.0		1.0	
1st tertile (worst)	45	29.4	(22.7;37.2)	28	18.3	(12.9;25.3)	1.8	(1.2;3.9)	2.1	(1.2;3.6)
Participant's education at age 30										
12+ years of study	51	22.8	(17.7;28.8)	27	12.1	(8.4;17.1)	1.0		1.0	
0-11 years of study	61	22.8	(18.1;28.2)	46	17.2	(13.1;22.2)	1.1	(0.7;1.7)	1.5	(0.9;2.6)

RR: Risk ratio

Table 4. Controlled direct effect from marginal structural models of family income at participant's birth on periodontitis age 31. Multinomial logistic regression. 1982 Pelotas Birth Cohort Study/Brazil.

	Risk Ratio Mild PD	Risk Ratio Moderate-to-Severe PD
Direct effect controlled for smoking		
Family income at birth		
Middle and high income	1.0	1.0
Low income	1.5 (0.9;2.4)	2.4 (1.4;4.1)
Direct effect controlled for dental calculus		
Family income at birth		
Middle and high income	1.0	1.0
Low income	1.3 (0.8;2.2)	2.1 (1.2;3.7)
Direct effect controlled for participant's education at age 30		
Family income at birth		
Middle and high income	1.0	1.0
Low income	1.3 (0.7;2.4)	2.2 (1.2;4.1)
Direct effect controlled for income at age 23		
Family income at birth		
Middle and high income	1.0	1.0
Low income	1.6 (0.9;2.6)	2.4 (1.4;4.1)

All models used stabilized weights accounting for sex.

Table S1. Controlled direct effect from marginal structural models of family income at participant's birth on periodontitis at age 31. Multinomial logistic regression.1982 Pelotas Birth Cohort Study/Brazil. Outcome: Dunedin Study case definition 1

	Risk Ratio Dunedin 1*
Direct effect controlled for smoking	
Family income at birth	
Middle and Highest income	1.0
Lowest income	1.2 (0.8;1.8)
Direct effect controlled for dental calculus	
Family income at birth	
Middle and Highest income	1.0
Lowest income	1.1 (0.7;1.7)
Direct effect controlled for participant's education at age 30	
Family income at birth	
Middle and Highest income	1.0
Lowest income	1.2 (0.7;1.9)
Direct effect controlled for income at age 23	
Family income at birth	
Middle and Highest income	1.0
Lowest income	1.2 (0.8;1.8)
All models used stabilized weights accounting for sex.	
*Case definition 1: one or more sites with 4+mm of clinical attachment loss	

Table S2. Controlled direct effect from marginal structural models of family income at participant's birth on periodontitis at age 31. Multinomial logistic regression.1982 Pelotas Birth Cohort Study/Brazil. Outcome: Dunedin Study case definition 2

	Risk Ratio Dunedin 2*
Direct effect controlled for smoking	
Family income at birth	
Middle and Highest income	1.0
Lowest income	1.6 (1.0;2.5)
Direct effect controlled for dental calculus	
Family income at birth	
Middle and Highest income	1.0
Lowest income	1.5 (0.9;2.4)
Direct effect controlled for participant's education at age 30	
Family income at birth	
Middle and Highest income	1.0
Lowest income	1.6 (0.9;2.7)
Direct effect controlled for income at age 23	
Family income at birth	
Middle and Highest income	1.0
Lowest income	1.5 (1.0;2.4)
All models used stabilized weights accounting for sex.	
*Case definition 2: two or more sites with 4+mm of clinical attachment loss	

Table S3. Controlled direct effect from marginal structural models of family income at participant's birth on periodontitis at age 31. Multinomial logistic regression.1982 Pelotas Birth Cohort Study/Brazil. Outcome: Dunedin Study case definition 3

	Risk Ratio Dunedin 3*
Direct effect controlled for smoking	
Family income at birth	
Middle and Highest income	1.0
Lowest income	1.5 (0.9;2.3)
Direct effect controlled for dental calculus	
Family income at birth	
Middle and Highest income	1.0
Lowest income	1.4 (0.9;2.3)
Direct effect controlled for participant's education a t age 30	
Family income at birth	
Middle and Highest income	1.0
Lowest income	1.5 (0.9;2.5)
Direct effect controlled for income at age 23	
Family income at birth	
Middle and Highest income	1.0
Lowest income	1.3 (0.9;2.1)

All models used stabilized weights accounting for sex.

*Case definition 3: one or more sites with 5+mm of clinical attachment loss

Table S4. Controlled direct effect from marginal structural models of family income at participant's birth on periodontitis at age 31. Multinomial logistic regression.1982 Pelotas Birth Cohort Study/Brazil. Outcome: 5th European Workshop (Tonetti and Claffey)

	Risk Ratio Tonetti and Claffey Sensitive
Direct effect controlled for smoking	
Family income at birth	
Middle and Highest income	1.0
Lowest income	1.9 (1.2;3.0)
Direct effect controlled for dental calculus	
Family income at birth	
Middle and Highest income	1.0
Lowest income	1.8 (1.2;2.7)
Direct effect controlled for participant's education at age 30	
Family income at birth	
Middle and Highest income	1.0
Lowest income	2.0 (1.3;3.0)
Direct effect controlled for income at age 23	
Family income at birth	
Middle and Highest income	1.0
Lowest income	2.0 (1.3;3.1)

All models used stabilized weights accounting for sex.

* Tonetti and Claffey specific case definition (presence of proximal attachment loss of 5+ mm in at least 30% of all present teeth) not evaluated as outcome due to low prevalence (0.7%, 4 individuals).

CHAPTER 8 - DISCUSSION AND CONCLUSIONS

8. Discussion

This chapter presents a summary of the thesis' main findings, overall discussion of the whole project, strengths and limitations, implications of the findings for public health and research, and the overall conclusions.

8.1 Summary of findings

This study used theoretical and empirical methods to explore the effects of socioeconomic position during the life-course on periodontal disease occurrence in adulthood. The project has identified that theoretical pathways can be drawn from each of the life-course epidemiology models in relation to how SEP may affect periodontal disease later on. The first paper of the thesis suggests that the most appropriate model to explain this relationship appears to be the accumulation of risk model. However, it is possible to identify causal pathways for the effect of SEP on periodontal disease in the light of each of the presented life-course epidemiology models. We also systematically reviewed evidence from longitudinal prospective studies on the temporal relationship between SEP and periodontal disease. We identified that exposure to relatively lower SEP earlier in life increased the risk of having periodontal disease later on, irrespective of the SEP indicator adopted or the case definition of periodontal disease in the primary studies.

Empirical evidence was presented to support the life-course epidemiology models of critical period and accumulation of risk on such a relationship. Evidence for the accumulation of risk model was observed through influence of

income trajectories from birth to age 30 on the occurrence of moderate-to-severe periodontal disease at age 31, after adjusting for covariates. Interactions between income trajectories and other SEP indicators were tested but none were significant in the final models. The underlying hypothesis addressed in the final paper of the thesis was that early life income has long-term effects on periodontal disease, and that some of this effect is independent of adulthood SEP and behavioural characteristics of the individual. We found evidence to support the hypothesis and, even after controlling for adulthood income, those from the lowest early life income tertile had a 2.4 times higher risk of developing moderate-to-severe periodontal disease at age 31 than those from middle and high income tertiles.

8.2 Strengths of this study

The present thesis has a number of strengths, including the high quality of the primary data, the sophisticated statistical techniques, and the familiarity of the researcher with the study and fieldwork.

The 1982 Pelotas Birth Cohort Study has high methodological quality. It is considered one of the largest and longest-running birth cohorts in low- and middle-income countries (Horta et al. 2015). Further, it is the longest and largest birth cohort with oral health clinically examined in low and middle-income countries. The baseline study captured more than 99% of all births in the city in the year of 1982, meaning that this population-based study is representative of the city's population.

The study design is among the main strengths of the thesis. The study collected data at multiple points from birth to age 31 years, allowing the exploration of the research question under a life-course approach. Covariates included in the study were collected during different moments over the life-course, capturing conditions that could affect the outcome through distinct pathways. The prospective study design also minimised the risk of information bias, since participants' information was collected using a short recall period.

The longitudinal study design of the empirical papers allowed the establishment of a causal connection between exposure and outcome. A temporal relationship was also captured in the systematic review, where one of the inclusion criteria was that information on SEP was collected at least once prior to periodontal disease evaluation. This temporal characteristic is an innovative approach in systematic reviews and it allowed the researchers to infer causality and reinforce the role of SEP as a risk factor for periodontal disease in adulthood.

Another strength is the comparability of the followed-up sample in the OHS-13 with the original sample and the baseline oral health sample. The similarity in demographic and socioeconomic characteristics, such as sex, maternal skin colour, family income and maternal education shows the unlikelihood of attrition bias in the sub-sample. Additionally, we were able to contact and examine more than 60% of our target sample, which was comparable to most other cohort studies conducted in high income countries at a similar age (66% in the 1970 British birth cohort study at age 30 and 70% in the 1958 British birth cohort study at age 33) (Elliott & Shepherd 2006; Power & Elliott 2006). Our attrition

rate is fairly similar to other well-known cohort studies in middle and low income countries (Richter et al. 2012).

Periodontal status was rigorously assessed, with the examination of 6 sites per tooth of all present teeth excluding third molars. Gingival recession and probing pocket depth in millimetres were collected using the acceptable protocol and instrument. This allowed the researchers to classify periodontal disease using one of the most recommended case definitions described in recent periodontal literature, the case definition proposed in partnership between the American Academy of Periodontology and the Centers for Disease Control and Prevention (Eke et al. 2012; Page & Eke 2007). Additionally, examiners were trained and calibrated dentists with high levels of reproducibility, confirmed by the high intra-class correlation coefficient achieved in the calibration process.

The statistical techniques applied in Chapters 6 and 7 are significant strengths of the thesis. The use of group-based trajectory modelling and Marginal Structural Models is innovative in the oral health literature. Searches conducted on the scientific literature confirmed the innovative character of using such approaches: the researcher identified only 4 relevant papers that actually evaluated SEP trajectories (Delgado-Angulo & Bernabe 2015a, 2015b; Peres et al. 2011; Peres et al. 2011), and only 4 studies were identified that used Marginal Structural Models in oral health research (Chaffee, Feldens & Vitolo 2014; Ju, Jamieson & Mejia 2016; Krishna Rao et al. 2015; Nascimento et al. 2017).

In addition to being innovative, the use of these methods is theoretically appropriate for addressing the research questions. Using GBTM, we were able to test the accumulation of risk model, by capturing income changes over time and testing its impact on periodontal health. Group-based trajectory modelling provides an output that is easily understandable, due to its visual representation, which is an advantage for the dissemination of the findings. Besides that, GBTM identifies development trajectories solely based on the data, without relying on any prior assumptions, which is an advantage over other trajectory techniques (Shearer et al. 2017).

Marginal structural models represent an analytical technique used for causal inference in epidemiology. Their main advantage is their capacity to reduce bias produced by confounding factors and mediators in the statistical analysis, which is a limitation of standard regression models of observational studies. In our models, to test the critical period effect, we controlled for several conditions known to mediate the relationship between family income in childhood and periodontal disease, namely family income in young adulthood, education in young adulthood, smoking and dental calculus, assessed as a proxy of dental hygiene. In order to handle potential confounders, we used inverse probability weights, which is a technique used in MSM to simulate a randomised clinical trial. Based on confounders, we estimated the probability weight of being assigned to the treatment or control group (exposed or unexposed to relatively lower family income). The assumption of the correct model specification was

confirmed based on exposure weights, which varied in our study between 0.99 and 1.00.

Finally, the involvement of the researcher with planning the most recent oral health sub-study and the fieldwork is an additional strength. Prior to commencing PhD study, at The University of Adelaide, the researcher did a Masters course in Dentistry at The Federal University of Pelotas, Brazil, where the birth cohort study is conducted. During that time, the researcher contributed to planning the OHS-13, including project grant writing, submission to Ethics Committee, training and calibration, as well as undertaking the fieldwork. The researcher worked as a clinical examiner in the data collection.

8.3 Limitations of this study

Some limitations of this study deserve attention. The slow progression rate of periodontal disease, and the fact that it has a cumulative effect during life may have limited our statistical power, taking into account the age of the sample. However, the prevalence of periodontal disease observed in our sample was comparable to other studies conducted with populations at a similar age (Vettore, Marques & Peres 2013).

Additionally, the sample size of 539 individuals is relatively small. Cohort studies with clinical examination, especially with examinations conducted at participants' houses, are expensive to conduct, and require human resources. Attrition was inevitable after 31 years despite initiatives to maximise the follow-up rate. Extensive attempts were made to contact participants, via telephone,

social media, or visiting the address in the study records, if other approaches did not work. Researchers also went to different cities within the state to interview and examine participants who had moved to other cities. However, the study had sufficient statistical power to achieve its objectives.

Using income as the indicator of SEP has some limitations. The main problem associated with its use is the possibility of measurement bias, based on the idea that the rich tend to under-report their income, while the poor do the opposite. Income is also less stable than other indicators, such as education, since income questions usually refer to the month before the data collection, and the earnings of individuals may vary over time for some employment categories. Having acknowledged this limitation, collecting household income is a common practice in Brazilian surveys and Censuses. Additionally, there is evidence from Brazilian data based on studies relying on self-reporting of income showing significant social inequalities in health (Victora 2016). Also, because education is cumulative and more stable than income in adulthood, it is not possible to capture the dynamics of change in SEP during the life span if assessing SEP by means of education. Income trajectories, as adopted in Chapter 6, have the ability to capture such changes.

The way family income was collected at birth is another limitation of our data. Family income at birth was collected in 5 categories and, in order to explore this variable into tertiles, further analyses were applied. Combining the family income variable with the other four variables, namely health services payment mode (out of pocket, public free, or private health insurance), maternal

education, height, and skin colour, researchers from the 1982 Pelotas Birth Cohort Study conducted a principal components analysis to obtain a variable with three categories, and this derived variable was the one used in the analysis within this thesis (Peres et al. 2011). It is important to highlight that the quantification of socioeconomic position at individual or household level was uncommon in Brazil during the time of the first data collection. Thus, collecting such information was novel for that period (Victora 2016).

Another limitation related to the exposure in this study, family income, was the difficulty to capture familial structure and life events that might have affected household income. Information on familial structure and life events would be important especially when evaluating income at age 23 years. Different scenarios can be expected at that age. A young adult could still live with his/her parents; could be a single parent with dependent children; a single adult living by himself/herself; or a young adult in a relationship with or without children. The same level of household income could have different impact on young adults in different household types. Information was not available to estimate equivalised household income, which could have taken into account different scenarios.

Familial structure and key life events, such as having kids, are also closely related to income trajectories. For example, a study using data from the first ten waves of the British Household Panel Survey (BHPS) from1991 to 2000 reported that 24% of single parents experienced upward trajectories over time, compared to 13% of all individuals in the sample. Among adults in childless

relationship, only 4% experience an upward trajectory, whilst a quarter experience a downward trajectory. This might be related to the fact that these life events were collected at the beginning of the period when adults in childless couples were more likely to have children at the subsequent waves. Hence, their equivalised income decreased, leading to downward income trajectories (Rigg & Sefton 2004). Other life events (such as formation of a new partnership and leaving the family home) can also impact measurable individual income and income trajectories after that event. In our study, we were not able to capture such characteristics of the participants and to observe how they influenced income trajectories and the relationship of income and income trajectories with the outcome. The main reason for the difficulty of evaluating the impact of life events on income trajectories in our study was the relatively small sample size. In paper 3, largest the trajectory was trajectory 2, stable middle income, with 218 individuals. Of those 218 participants, 84 presented any type of periodontal disease and 32 presented moderate-to-severe periodontal disease. Therefore, creating sub-groups in each income trajectory was not possible because of expected low statistical power of such small sub-groups. An additional aspect that would limit our analytical capacity is that information on familial structure and life events was only partially collected in the Pelotas Birth Cohort Study.

In the final manuscript of the thesis, the researcher assessed several mediators of the SEP-periodontal disease relationship. All available mediators in the study were used. However, it was not possible to estimate the controlled direct effect of exposure on outcome after controlling for the effect of all mediators

combined, only each mediator at a time. The development of a statistical approach able to combine several mediators at the same time has been discussed by eminent statisticians (Lin et al. 2017), and a novel technique was proposed in 2017 (VanderWeele & Tchetgen Tchetgen 2017).That publication addresses the theoretical and mathematical basis of the technique, but (to date) it has not yet been applied in epidemiological research.

Care should be taken in applying our research findings to other contexts. Even within Brazil, the generalisability of the present study findings is limited, since South Brazil has relatively better social indicators than other regions. However, our empirical findings may be discussed as a 'proof of principle' that early life income inequality affects periodontal disease later on. Our findings confirm the high quality data and sophisticated analytical technique, and the causal effect that was suggested by the systematic review of the current scientific literature (Chapter 5).

8.4 General discussion

A broader understanding of initiation and progression of periodontal disease is needed in order to prevent its occurrence. It means going beyond the mouth and more immediate oral health behaviours. It also recognises that factors acting during different stages of life can shape and modify people's health. Social determinants strongly influence the health profile of the population, shape healthy or harmful behaviors, as well as impacting on the way the healthcare systems are organised.

Several systematic reviews have demonstrated the association between social conditions at the individual level and periodontal disease (Boillot et al. 2011; Borrell & Crawford 2012; Klinge & Norlund 2005). There is also evidence of the association between contextual conditions and periodontal disease (Susin et al. 2004). This evidence is, however, mainly based on cross-sectional studies. Longitudinal studies with a long follow-up period are important in establishing a temporal link between exposure and outcome, and identifying in which period of life the exposure to relatively lower SEP will result in a greater impact on periodontal disease.

The life-course epidemiology approach is suitable for studying the long-term effect of socioeconomic inequalities on periodontal health because it captures effects of conditions where the individual was born, grewup, and lived, on the onset and progression of a chronic disease such as periodontal disease. The ideal study design to be used in life-course epidemiology is the prospective longitudinal study, particularly where information is collected from birth to adulthood, because it captures the time sequence of events and is suitable to test causal life-course theories (Nicolau et al. 2007). Findings from this thesis have contributed evidence to support two life-course epidemiological theories: the accumulation of risk and the critical period.

Chapter 6 shows evidence on how trajectories of income from birth to age 30 impact on periodontal disease measured at age 31. This finding is in line with the accumulation of risk theory, which proposes that various conditions acting cumulatively during a life period will lead to health or illness later on (Kuh &

Ben-Shlomo 1997). Although this model is focused on the number of episodes of exposure to relatively lower income, rather than the moment of the exposure, in our approach of trajectories we were able to identify that, even if those who were poor at birth and during childhood improved their socioeconomic condition later in life, their periodontal health would still be worse than those in consistently high or consistently middle income groups. Taking that into account, we can also consider that Chapter 6 provides some evidence of the life-course epidemiology theory of critical period; in our study, the critical period was childhood and young adolescence (from birth to age 15).

Chapter 7 was conducted to test the critical period theory on the relationship between income and periodontal disease. The hypothesis has been confirmed with evidence of the controlled direct effect (CDE) of income at birth on moderate-to-severe periodontal disease in adulthood. Such effect was not mediated by adulthood income or education. While it is possible that current social conditions would affect oral health in adulthood more than past circumstances (Lee & Han 2016), our evidence demonstrated the role of the early life circumstances.

It has been stablished that early life is a critical period for health development (Barker 1992; Tickell 2011). There is strong evidence that children's life chances are most heavily predicated on their development in the first 5 years of life (Allen 2011). These first years of life, from pregnancy to age 5 years, have been referred to as the "Foundation Years" (Field 2010). The Foundation Years are of paramount importance because they provide a physical, neurocognitive,

and social-emotional substrate for healthy development through childhood and into adulthood(Sawyer et al. 2014). Additionally, social and developmental conditions experienced in early life strongly influence SEP in adulthood. For example, a child's development score at 22 months can serve as an accurate predictor of educational outcomes at 26 years (Feinstein 2003). Also, around 80 per cent of an adult income is determined by where the person was born, along with the income of his/her parents (Milanovic 2011).

One of the limitations of the primary studies included in the systematic review was that some of them used only bivariate analyses, not accounting for confounding factors and mediators. The systematic review identified a need for evaluations accounting for the role of variables known to be mediators of the SEP-periodontal disease relationship, such as smoking. The estimates from the final paper of this thesis showed that people born in low income families had double the prevalence of moderate-to-severe periodontal disease seen in their well-off counterparts, even after controlling for smoking or dental hygiene (assessed by means of dental calculus). That our estimates accounted for important mediators later in life reinforces that early life is a critical period for periodontal disease. Additionally, our findings corroborate current evidence that reveals that inequalities in health are substantial across different population groups in most health-related behaviours and outcomes (Braveman et al. 2010; de Azevedo Barros et al. 2016).

It is known that socioeconomic position shapes behavioural practices, and a substantial part of the social differences in morbidity and mortality results from

uneven patterns of health-related behaviours (de Azevedo Barros et al. 2016). A special focus should be made on behaviours that act as main risk factors for a range of chronic diseases, including periodontal disease (Sheiham & Watt 2000). These risk factors include, but are not limited to, some of the conditions evaluated in the empirical papers of the thesis, such as smoking and inadequate hygiene.

Even though the life-course approach suggests a perspective of analysing past experiences in life, it also leads to a forward-looking approach. It is expected that the knowledge generated using the life-course epidemiology approach will be useful in identifying the nature, level, purpose and timing of interventions to prevent periodontal disease in new generations, and to minimise the impact of periodontal disease on those who have already developed the disease (Thomson, Sheiham & Spencer 2012).

8.5 Study implications

8.5.1 Implications for future research

The key mechanisms linking socioeconomic position during early life stages, with periodontal disease in adulthood, still need to be identified. It is paramount to explore and understand each pathway, as well as the role of each mediator in the relationship between SEP and periodontal disease.

This thesis provides evidence to support the accumulation of risk and critical period life-course epidemiology theories on the aforementioned relationship. The data used in the empirical studies is from a cohort study in a middle-sized

city in South Brazil. Although the findings may be interpreted as a ‘proof of principle’, other high quality prospective studies are recommended, in order to confirm our findings in other contexts and its generalisability. Studies with a similar perspective, but with older populations would also be useful to identify if the influence of SEP on periodontal disease in young adulthood is also detectable in later life. These studies would be also important to discover if the pathways linking exposure and outcome in later adulthood are the same as identified within this thesis.

With the development of new analytical techniques, it is expected that in the near future researchers will be able to assess several mediators at the same time in their analysis (VanderWeele & Tchetgen Tchetgen 2017). This approach would be highly recommended for further investigation of socioeconomic inequalities in periodontal disease, considering the different mediators identified in the scientific literature and how they may be related to each other. A statistical analysis robust enough to include different socioeconomic and behavioural mediators will provide a more comprehensive picture of the effect of SEP on periodontal disease, and better inform public policies to prevent and minimise the burden of periodontal disease at a population level.

8.5.2 Implications for public health

From a public health perspective, interventions at different levels can be promoted in order to achieve a timely prevention of periodontal disease.

Considering the social conditions as the ‘causes of the causes’ of disease (Blane 1985), efforts to reduce social inequalities may also reduce inequalities in periodontal health, as well as in other chronic conditions that are socially determined. Moreover, if class and status are to become a less powerful influence both on individual lives and on whole societies, it will be necessary to reduce the material differences which so often constitute the cultural markers of social differentiation (Pickett & Wilkinson 2015).

The findings from this thesis reveal that early life lower family income has detrimental effects on periodontal conditions. Although the mechanisms behind this link are not completely clear, the findings indicate the need for focusing health policy attention on childhood social circumstances. In fact, there is evidence that greater family financial resources are one of the key factors enabling parental investment in the health and development of children (Maika et al. 2017). It is important to identify those individuals at risk in childhood in order to deliver targeted early life interventions, aiming to reduce inequalities in health.

A population strategy to reduce the burden of periodontal disease is to target behavioural practices that may increase the susceptibility to the condition. Strategies to promote healthy behaviours, especially related to smoking and oral health self-care, are expected to also reduce periodontal disease prevalence, as these are well-known risk factors for periodontal disease (Thomson, Sheiham & Spencer 2012). In addition, combining oral health with general health promotion, through the adoption of the Common Risk Factor

Approach (Sheiham & Watt 2000), may be more effective and lead to better outcomes not only in periodontal health but also in other chronic general conditions that share risk factors with periodontal disease, such as cardiovascular and respiratory diseases.

The Commission on the Social Determinants of Health (CSDH) has emphasised the need to incorporate the issue of health inequality into governments' political agendas (WHO Commission on the Social Determinants of Health 2008). A strategy that has been implemented in developed countries that may help in reducing periodontal disease levels at the population level is related to tobacco taxation. The increased taxation is expected to make cigarettes less affordable and to decrease the demand of smokers, what in the long-term may lead to a lower burden of periodontal disease at population level. Additionally, it can be easily observed that smoking is becoming less and less socially acceptable, with several policies being implemented to reinforce this idea. In Australia, for example, smoking has been banned since the early 2000s in all enclosed public places, workplaces and shared areas, with fines applying for those who smoke in these areas. These initiatives to promote healthy behaviours at population level through healthy environments should be reinforced and supported by governments, in order to achieve healthier societies.

8.6 Conclusions

This thesis examined socioeconomic inequalities in periodontal disease in the light of life-course epidemiology. It has provided high quality evidence that, from

both theoretical and empirical perspectives, socioeconomic position is an upstream determinant of periodontal disease. It has been identified that the critical period and the accumulation of risk life-course epidemiology models are suitable to study the relationship between SEP and periodontal disease. The specific conclusions are:

1. The effects of socioeconomic position on periodontal disease can be theoretically explained according to the life-course theories.

2. Socioeconomic position has long-term effects on periodontal disease, and this relationship seems to be consistent despite differences in study methods.

3. Under the accumulation of risk theory, a low and variable income trajectory from birth to age 30 has a detrimental effect on moderate-to-severe periodontal disease at age 31.

4. Under the critical period theory, early life socioeconomic position assessed by means of income has a controlled direct effect on moderate-to-severe periodontal disease in adulthood that is not completely mediated by adulthood socioeconomic indicators or behavioural conditions.

8.7 References

Allen, G 2011, 'Early intervention: the next steps. An independent report to Her Majesty's Government. ', Cabinet Office, London.

Barker, DJP 1992, *Foetal and infant origin of adult disease.*, BMJ Publishing Group, London.

Blane, D 1985, 'An assessment of the Black Report's explanations of health inequalities', *Sociology of Health & Illness*, vol. 7, no. 3, pp. 423-445.

Boillot, A, El Halabi, B, Batty, GD, Range, H, Czernichow, S & Bouchard, P 2011, 'Education as a predictor of chronic periodontitis: a systematic review with meta-analysis population-based studies', *PLoS One*, vol. 6, no. 7, p. e21508.

Borrell, LN & Crawford, ND 2012, 'Socioeconomic position indicators and periodontitis: examining the evidence', *Periodontol 2000*, vol. 58, no. 1, Feb, pp. 69-83.

Braveman, PA, Cubbin, C, Egerter, S, Williams, DR & Pamuk, E 2010, 'Socioeconomic disparities in health in the United States: what the patterns tell us', *Am J Public Health*, vol. 100 Suppl 1, Apr 01, pp. S186-196.

Chaffee, BW, Feldens, CA & Vitolo, MR 2014, 'Association of long-duration breastfeeding and dental caries estimated with marginal structural models', *Ann Epidemiol*, vol. 24, no. 6, Jun, pp. 448-454.

Commission on Social Determinants of Health, WHO 2008, 'Closing the gap in a generation: Health equity through action on the social determinants of health. Final Report of the Commission on Social Determinants of Health', World Health Organization, Geneva.

de Azevedo Barros, MB, Lima, MG, Medina, LP, Szwarcwald, CL & Malta, DC 2016, 'Social inequalities in health behaviors among Brazilian adults: National Health Survey, 2013', *Int J Equity Health*, vol. 15, no. 1, Nov 17, p. 148.

Delgado-Angulo, EK & Bernabe, E 2015a, 'Comparing lifecourse models of social class and adult oral health using the 1958 National Child Development Study', *Community Dent Health*, vol. 32, no. 1, Mar, pp. 20-25.

Delgado-Angulo, EK & Bernabe, E 2015b, 'Intergenerational mobility and adult oral health in a British cohort', *Community Dent Oral Epidemiol*, vol. 43, no. 3, Jun, pp. 255-261.

Eke, PI, Page, RC, Wei, L, Thornton-Evans, G & Genco, RJ 2012, 'Update of the case definitions for population-based surveillance of periodontitis', *J Periodontol*, vol. 83, no. 12, Dec, pp. 1449-1454.

Elliott, J & Shepherd, P 2006, 'Cohort profile: 1970 British Birth Cohort (BCS70)', *Int J Epidemiol*, vol. 35, no. 4, Aug, pp. 836-843.

Feinstein, L 2003, 'Inequality in the Early Cognitive Development of Children in the Early 1970 Cohort. ', *Economica*, vol. 70, pp. 73-97.

Field, F 2010, 'The Foundation Years: Preventing Poor Children Becoming Poor Adults. The report of the Independent Review on Poverty and Life Chances.', HM Government, London.

Horta, BL, Gigante, DP, Goncalves, H, dos Santos Motta, J, Loret de Mola, C, Oliveira, IO, Barros, FC & Victora, CG 2015, 'Cohort Profile Update: The 1982 Pelotas (Brazil) Birth Cohort Study', *Int J Epidemiol*, vol. 44, no. 2, Apr, pp. 441, 441a-441e.

Ju, X, Jamieson, LM & Mejia, GC 2016, 'Estimating the effects of maternal education on child dental caries using marginal structural models: The Longitudinal Study of Indigenous Australian Children', *Community Dent Oral Epidemiol*, vol. 44, no. 6, Dec, pp. 602-610.

Klinge, B & Norlund, A 2005, 'A socio-economic perspective on periodontal diseases: a systematic review', *J Clin Periodontol*, vol. 32 Suppl 6, pp. 314-325.

Krishna Rao, S, Mejia, GC, Roberts-Thomson, K, Logan, RM, Kamath, V, Kulkarni, M & Mittinty, MN 2015, 'Estimating the effect of childhood socioeconomic disadvantage on oral cancer in India using marginal structural models', *Epidemiology*, vol. 26, no. 4, Jul, pp. 509-517.

Kuh, D & Ben-Shlomo, Y 1997, *A Life-course Approach to Chronic Disease Epidemiology*, Oxford University Press.

Lee, HJ & Han, DH 2016, 'Early-life socioeconomic position and periodontal status in Korean adults', *Community Dent Oral Epidemiol*, vol. 44, no. 1, Feb, pp. 11-23.

Lin, SH, Young, J, Logan, R, Tchetgen Tchetgen, EJ & VanderWeele, TJ 2017, 'Parametric Mediational g-Formula Approach to Mediation Analysis with Time-varying Exposures, Mediators, and Confounders', *Epidemiology*, vol. 28, no. 2, Mar, pp. 266-274.

Maika, A, Mittinty, MN, Brinkman, S & Lynch, J 2017, 'Associations of Early- and Later-Childhood Poverty With Child Cognitive Function in Indonesia: Effect Decomposition in the Presence of Exposure-Induced Mediator-Outcome Confounding', *Am J Epidemiol*, vol. 185, no. 10, May 15, pp. 879-887.

Milanovic, B 2011, *The haves and the have-nots: a short and idiosyncratic history of global inequality*, Basic Books, New York.

Nascimento, GG, Peres, KG, Mittinty, MN, Mejia, GC, Silva, DA, Gonzalez-Chica, D & Peres, MA 2017, 'Obesity and Periodontal Outcomes: A Population-Based Cohort Study in Brazil', *J Periodontol*, vol. 88, no. 1, Jan, pp. 50-58.

Nicolau, B, Thomson, WM, Steele, JG & Allison, PJ 2007, 'Life-course epidemiology: concepts and theoretical models and its relevance to chronic oral conditions', *Community Dent Oral Epidemiol*, vol. 35, no. 4, Aug, pp. 241-249.

Page, RC & Eke, PI 2007, 'Case definitions for use in population-based surveillance of periodontitis', *J Periodontol*, vol. 78, no. 7 Suppl, Jul, pp. 1387-1399.

Peres, KG, Peres, MA, Demarco, FF, Tarquinio, SB, Horta, BL & Gigante, DP 2011, 'Oral health studies in the 1982 Pelotas (Brazil) birth cohort: methodology and principal results at 15 and 24 years of age', *Cad Saude Publica*, vol. 27, no. 8, Aug, pp. 1569-1580.

Peres, MA, Peres, KG, Thomson, WM, Broadbent, JM, Gigante, DP & Horta, BL 2011, 'The influence of family income trajectories from birth to adulthood on adult oral health: findings from the 1982 Pelotas birth cohort', *Am J Public Health*, vol. 101, no. 4, Apr, pp. 730-736.

Pickett, KE & Wilkinson, RG 2015, 'Income inequality and health: a causal review', *Soc Sci Med*, vol. 128, Mar, pp. 316-326.

Power, C & Elliott, J 2006, 'Cohort profile: 1958 British birth cohort (National Child Development Study)', *Int J Epidemiol*, vol. 35, no. 1, Feb, pp. 34-41.

Richter, LM, Victora, CG, Hallal, PC, Adair, LS, Bhargava, SK, Fall, CH, Lee, N, Martorell, R, Norris, SA, Sachdev, HS, Stein, AD & Group, C 2012, 'Cohort profile: the consortium of health-orientated research in transitioning societies', *Int J Epidemiol*, vol. 41, no. 3, Jun, pp. 621-626.

Rigg, JA & Sefton, T 2004, ' Income dynamics and the life cycle. ', CASEpaper, CASE/81. Centre for Analysis of Social Exclusion, London School of Economics and Political Science, London, UK. .

Sawyer, A, Gialamas, A, Pearce, A, Sawyer, MG & Lynch, J 2014, 'Five by Five: A Supporting Systems Framework for Child Health and Development.', Better Start Child Health and Development Research Group, School of Public Health, University of Adelaide.

Shearer, DM, Thomson, WM, Broadbent, JM, Mann, J & Poulton, R 2017, 'Periodontitis is not associated with metabolic risk during the fourth decade of life', *J Clin Periodontol*, vol. 44, no. 1, Jan, pp. 22-30.

Sheiham, A & Watt, RG 2000, 'The common risk factor approach: a rational basis for promoting oral health', *Community Dent Oral Epidemiol*, vol. 28, no. 6, Dec, pp. 399-406.

Susin, C, Dalla Vecchia, CF, Oppermann, RV, Haugejorden, O & Albandar, JM 2004, 'Periodontal attachment loss in an urban population of Brazilian adults: effect of demographic, behavioral, and environmental risk indicators', *J Periodontol*, vol. 75, no. 7, Jul, pp. 1033-1041.

Thomson, WM, Sheiham, A & Spencer, AJ 2012, 'Sociobehavioral aspects of periodontal disease', *Periodontol 2000*, vol. 60, no. 1, Oct, pp. 54-63.

Tickell, C 2011, 'The Early Years: Foundations for life, health and learning: an independent report on the early years foundation stage to Her Majesty's government.', vol. Report No.: Ref: DFE-00177-2011, Department for Education, London.

VanderWeele, TJ & Tchetgen Tchetgen, EJ 2017, 'Mediation analysis with time varying exposures and mediators', *Journal of the Royal Statistical Society: Series B (Statistical Methodology)*, vol. 79, no. 3, pp. 917-938.

Vettore, MV, Marques, RA & Peres, MA 2013, '[Social inequalities and periodontal disease: multilevel approach in SBBrasil 2010 survey]', *Rev Saude Publica*, vol. 47 Suppl 3, Dec, pp. 29-39.

Victora, C 2016, 'Socioeconomic inequalities in Health: Reflections on the academic production from Brazil', *Int J Equity Health*, vol. 15, no. 1, Nov 17, p. 164.

9. APPENDICES

9.1 Abstracts of Conference Presentations - 1

Schuch HS, Peres KG, Demarco FF, Peres MA, Do LG Effects of income trajectories on periodontal disease: birth-cohort study findings. 95th IADR/AADR/CADR General Session & Exhibition, 2017, San Francisco – USA. (Oral presentation).

- This abstract was awarded the IADR BEHSR Predoctoral Award from the Behavioral, Epidemiologic and Health Services Research Group of the International Association for Dental Research. San Francisco – USA.
- At the same conference, the researcher was awarded the 2017 IADR Lion Dental Research Award for Junior Investigators from the Behavioral, Epidemiologic and Health Services Research Group of the International Association for Dental Research. San Francisco – USA.
- The attendance of the IADR conference was supported by a travel grant from the Healthy Development Adelaide Travel Grant, Round 2017/1. Adelaide - Australia

Scientific Group/Network: Behavioral, Epidemiologic and Health Services Research

Effects of income trajectories on periodontal disease: birth-cohort study findings

Schuch H.S. [1*], Peres K.G.[1], Demarco, F.F.[2], Peres M.A. [1], Do L.G. [1]

[1]Australian Research for Population Oral Health (ARCPOH), School of Dentistry, The University of Adelaide.

[2]Postgraduate Programs in Dentistry and Epidemiology, School of Dentistry, Federal University of Pelotas.

*Presenter.

Objectives: To quantify impact of income trajectories over the life course on the occurrence of periodontal disease in adulthood. Methods: Data from the 1982 Pelotas Birth Cohort Study, Brazil was used. Family income was collected at birth and ages 15, 19, 23 and 30 years. Group-based trajectory modelling was used to identify 3 income trajectories based on Bayesian Information Criteria. Periodontal measures were collected through clinical examination in the 2013 Oral Health sub-study at age 31 (OHS-13). Covariates included sociodemographic, behavioural and clinical variables collected over the life course. Log-Poisson regression models were conducted for two outcomes, prevalence of any periodontitis and moderate/severe periodontitis (CDC-AAP case definitions), to estimate prevalence ratios (PRs) and its 95%CIs. Interactions between income trajectories with maternal and participant's education in the models were also tested. Results: 539 participants were followed-up in the OHS-13. The prevalence of any periodontitis and moderate/severe periodontitis were 37.3% and 14.3%, respectively. Some 31.6% of the individuals were in trajectory 1 (relatively stable high income), 46.0% in trajectory 2 (relatively stable middle income) and 22.4% in trajectory 3 (relatively low and variable income). After adjustment for potential confounders, income trajectories were significantly associated with prevalence of

moderate/severe periodontitis. Adjusted PR in participants in trajectory 3 was 2.10 (95%CI:1.1-4.1) against participants in trajectory 1. The crude association between income trajectories and prevalence of any periodontitis was explained by the inclusion of health behaviors and clinical oral variables in the model. Tested interactions were not significant in any model. Conclusions: Low and variable income trajectories from birth to age 30 increased the prevalence of moderate/severe periodontitis later in life. The findings may inform programs in identifying and targeting potentially at-risk individuals early in life in order to prevent periodontitis.

Keywords: Cohort Studies, Chronic Periodontitis, Socioeconomic Factors, Income

9.2 Abstracts of Conference Presentations - 2

Schuch HS, Peres KG, Peres MA, Do LG. Modelling socioeconomic trajectories on oral health outcomes over the life course. South Australia Population Health Conference 2016. Adelaide – Australia (Oral presentation).

Please indicate your preference: [x] Oral [ ] Poster

Title: Modelling socioeconomic trajectories on oral health outcomes over the life course

Helena Schuch, Australian Research Centre for Population Oral Health (ARCPOH), School of Dentistry, The University of Adelaide, A/Prof Karen Peres, Australian Research Centre for Population Oral Health (ARCPOH), School of Dentistry, The University of Adelaide, Prof Marco Peres, Australian Research Centre for Population Oral Health (ARCPOH), School of Dentistry, The University of Adelaide, A/Prof Loc Do, Australian Research Centre for Population Oral Health (ARCPOH), School of Dentistry, The University of Adelaide.

PRESENTER DETAILS

Name: Helena Schuch

Institution: Australian Research Centre for Population Oral Health (ARCPOH), School of Dentistry, The University of Adelaide

Email: helena.schuch@adelaide.edu.au

Twitter: -

Abstract

Introduction: Despite being critical to understand the roles of different socioeconomic position (SEP) trajectories over time when studying the effects of SEP on health status, there is still a gap in the literature on this topic. Such understanding will allow capturing the dynamic processes through which socioeconomic factors may affect health during the life course.
Aims: The presentation aims to discuss different approaches of modelling socioeconomic trajectories over the life course.
Methods: Data from the 1982 Pelotas Birth Cohort (Brazil) will be used to illustrate the methodological discussion. All 5,914 hospital births that occurred in Pelotas, in 1982, were

identified and this population was followed up several times since then. Oral health assessments were performed in a sample from this population at 15 (n=888), 24 years (n=720) and 31 years (n=539 individuals). Socioeconomic position data (income and education) at birth and ages 15, 19, 23 and 30 allow the researchers to draw a socioeconomic profile of the individual over the life course. Periodontal data was collected through clinical examination of 6 sites per tooth on the 28 teeth, and the CDC/AAP criteria were used for periodontal case definition.
Results: Only eight studies have investigated the effect of socioeconomic trajectories in oral health outcomes, with five of them assessing SEP only twice over the life course. A number of statistical methods of modelling trajectories are available. The presentation will discuss some of the most frequently used methods to model socioeconomic trajectories: growth curve, growth mixture and group-based trajectory.
Conclusions: It is not well-defined in the literature the best approach of modelling socioeconomic trajectories over the life course, and specific assumptions of each model should be considered when selecting the most appropriate method for the research question.

Implications for policy and/ or practice: The current available research on the association between SEP and periodontal outcomes is neglecting the dynamic processes through which social factors may affect health during the life course. The use of socioeconomic trajectories over the life course can allow the researchers capture such processes and better explore and understand the effects of SEP on periodontal outcomes, so to intervene in the most critical period.

Keywords (up to 4): Epidemiology, Socioeconomic conditions, Periodontal disease, Methodology

9.3 Abstracts of Conference Presentations - 3

Schuch HS, Peres KG, Peres MA, Do LG. Effects of income trajectories over the life course on the occurrence of periodontal disease in adulthood: a population-based birth cohort study. 2016 Florey Postgraduate Research Conference – Adelaide - Australia. (Poster presentation).

- This presentation was awarded as the best presentation within the School of Dentistry at the Florey International Postgraduate Research Conference 2016. Adelaide – Australia

Effects of socioeconomic trajectories on periodontal disease in adulthood: a population-based birth cohort study

Schuch H.S. *, Peres K.G., Peres M.A., Do L.G.

Australian Research for Population Oral Health (ARCPOH), School of Dentistry, The University of Adelaide. *Poster presenter.

Background and Aims: There is a substantial gap in evidence of the effect of socioeconomic positions (SEP) and its trajectories on oral health, particularly related to periodontal disease, one of the most prevalent chronic conditions in adults. The aim of this study was to test the impact of socioeconomic trajectories over the life course on the occurrence of periodontal disease in

young adults. Methodology: This study evaluated data from the 1982 Pelotas Birth Cohort Study, Brazil. Family income collected at birth and ages 15, 19, 23 and 30 years was used to rank participants' SEP at each time. Group-based trajectory modelling was used to identify SEP trajectories based on Bayesian Information Criteria (BIC). Periodontal disease measures were collected through clinical examination in the Oral Health sub-study at age 31 years conducted in 2012 (OHS-12). The US Centers for Disease Control and Prevention and American Association of Periodontology (CDC-AAP) case definitions were adopted. Covariates included sex and maternal education at participant's birth, smoking status at age 23, dental flossing, bleeding on probing, dental calculus and presence of periodontal pocket at age 24 and participant's education at age 30. Multivariable Log-Poisson Regression models were conducted to evaluate the effect of socioeconomic trajectories on two outcomes: any periodontal disease and moderate or severe periodontal disease. Interactions between socioeconomic trajectories with participant's education and maternal education on the outcomes were also tested. Results: A total of 539 participants were followed-up in the OHS-13 and therefore were included in the present study. The prevalence of any periodontal disease and moderate or severe periodontal disease was 37.3% and 14.3%, respectively. From our sample, there were 57.1% of the individuals in trajectory 1 (relatively stable rich over time), 19.7% in trajectory 2 (relatively stable middle income over time) and 23.2% in trajectory 3 (relatively poor from birth to age 15, slightly and consistently increasing to middle income in young adulthood). After adjustment

for covariates, socioeconomic trajectories affected the prevalence of periodontal disease later on. Compared to the trajectory 1, the trajectory 3 had an adjusted Prevalence Ratio of 1.5 for both outcomes (95% Confidence Intervals 1.0-2.1 for both outcomes). Women had lower prevalence of periodontal disease than men. Having dental calculus and periodontal pocket at age 24 were associated with higher prevalence of any periodontal disease at age 31, but not with moderate or severe periodontal disease. Tested interactions were not significant in any of the models. Conclusions: Socioeconomic trajectories from birth to age 30 affect periodontal disease later in life. The findings may inform programs in identifying and targeting potentially at-risk individuals early in life in order to prevent periodontal disease.

9.4 Abstracts of Conference Presentations - 4

Schuch HS, Peres KG, Peres MA, Do LG. Modelling socioeconomic trajectories on oral health outcomes over the life course. 94th IADR/AADR/CADR General Session and Exhibition, 2016, Seoul – South Korea. (Oral presentation – Epi Forum).

- The attendance of the IADR conference was supported by the following Bursary: Winifred E. Preedy Postgraduate Bursary, Australian Federation of University Women – South Australia AFUW-SA Inc. Trust Fund, 2015. Adelaide – Australia.

Abstract for 2016 IADR Oral Epi Forum

Presenter's contact information:
Presenter: Helena Silveira Schuch
Training program: PhD in Population Oral Health
Presenter's supervisors: A/Prof Loc Do, Prof Marco Peres, A/Prof Karen Peres
Affiliation: Australian Research Centre for Population Oral Health (ARCPOH), School of Dentistry, The University of Adelaide
Address: 122 Frome Street, Adelaide CBD, 5000, Adelaide, SA, Australia.
E-mail address: helena.schuch@adelaide.edu.au

Title: Modelling socioeconomic trajectories on oral health outcomes over the life course

(Focus: Methodological issues)

Background: Despite being critical to understand the roles of different socioeconomic position (SEP) trajectories over time when studying the effects of SEP on health status, there is still a gap in the literature on this topic. Such

understanding will allow capturing the dynamic processes through which socioeconomic factors may affect health during the life course.
Aims: The presentation aims to discuss different approaches of modelling socioeconomic trajectories over the life course.
Methods: Data from the 1982 Pelotas Birth Cohort (Brazil) will be used to illustrate the methodological discussion. All 5,914 hospital births that occurred in Pelotas, in 1982, were identified and this population was followed up several times since then. Oral health assessments were performed in a sample from this population at 15 (n=888), 24 years (n=720) and 31 years (n=539 individuals). Socioeconomic position data (income and education) at birth and ages 15, 19, 23 and 30 allow the researchers to draw a socioeconomic profile of the individual over the life course. Periodontal data was collected through clinical examination of 6 sites per tooth on the 28 teeth, and the CDC/AAP criteria were used for periodontal case definition.
Results: Only eight studies have investigated the effect of socioeconomic trajectories in oral health outcomes, with five of them assessing SEP only twice over the life course. A number of statistical methods of modelling trajectories are available. The presentation will discuss some of the most frequently used methods to model socioeconomic trajectories: growth curve, growth mixture and group-based trajectory.
Conclusions: It is not well-defined in the literature the best approach of modelling socioeconomic trajectories over the life course, and specific assumptions of each model should be considered when selecting the most appropriate method for the research question.

Questions to be discussed:
1. What are the different approaches of modelling socioeconomic trajectories over the life course?
2. How to define the most appropriate method of modelling socioeconomic trajectories?

I confirm that this work has not been presented or published previously.

9.5 Abstracts of Conference Presentations - 5

Schuch HS, Peres KG, Singh A, Peres MA, Do LG. Effect of socioeconomic position during the life on periodontitis in adulthood: a systematic review. IADR Australian and New Zealand Division Meeting, 2015, Dunedin – New Zealand. (Oral presentation).

- The attendance on the IADR ANZ was supported by the JL Eustace International Travel Award, 2015. Adelaide – Australia.

Abstract for IADR ANZ

Title (Abstract titles are limited to 10 words or less):

Longitudinal effect of socioeconomic status on periodontitis: a systematic review

Authors:

Helena Silveira Schuch[1*], Karen Glazer Peres[1], Ankur Singh[1], Marco A Peres[1] & Loc Giang Do[1]

1. Australian Research Centre for Population Oral Health (ARCPOH), School of Dentistry, The University of Adelaide, Adelaide, South Australia, Australia.

* Presenter

Contact: helena.schuch@adelaide.edu.au

Tel: +61 8 8313 31296

Abstract Text (300 or less):

Aims: To systematically review all longitudinal studies that investigate the effect of socioeconomic status (SES) during the life on the development of periodontal diseases (PD) in adulthood. **Methods**: Potentially eligible papers were those addressing epidemiological observational longitudinal studies that included clinically assessed indicators of PD as outcome and that measured indicators of relative individual level SES. A search was performed in six electronic databases (PubMed, EMBASE, Web of Science, Scopus, LILACS (Latin American and Caribbean Health Sciences Literature) and ScieLO (Scientific Electronic Library Online). The review process was performed independently by 2 authors. First, titles and abstracts were screened, followed by full-texts evaluation and data extraction. The methodological quality assessment of the studies was assessed by the Newcastle–Ottawa Quality Assessment Scale for cohort studies. **Results:** The combined search from all the 6 databases provided 1,507 papers. After removals of duplicates (n=685), title and abstract screening (n=795) and full text review (n=19), 8 original manuscripts involving 7 studies were included in this review. The sample sizes ranged from 167 to 2806, and the time frame (from exposure to follow-up) varied from 2 to 38 years, with the majority of the studies presenting a time frame between 5 to 11 years. SES indicators included education, occupation and family income and outcomes assessed by the studies were mainly periodontal attachment loss, probing pocket depth and alveolar bone loss. In general, studies presented low risk of bias. Between all the papers included, 6 have found that a position of socioeconomic deprivation negatively impacts on periodontal health. **Conclusions**: The scientific evidence demonstrates that socioeconomic indicators

during the life negatively impact the occurrence of PD later on. Further prospective cohort studies are needed to confirm such association and to test in which life period the SES deprivation is more critical to predict PD.

Keywords: Inequality, Socioeconomic Status, Periodontal Diseases, Systematic Review, Longitudinal Studies

Scientific group/network: Behavioral, Epidemiologic and Health Services Research

9.6 Abstracts of Conference Presentations - 6

Schuch HS, Peres KG, Singh A, Peres MA, Do LG. Effect of socioeconomic position during the life on periodontitis in adulthood: a systematic review. 2015 Florey Postgraduate Research Conference – Adelaide – Australia. (Poster presentation).

- This presentation was awarded as the best presentation by the Florey Foundation of Medical Research at the Florey International Postgraduate Research Conference 2016. Adelaide – Australia

Effects of socioeconomic trajectories on periodontal disease in adulthood: a population-based birth cohort study

Schuch H.S. *, Peres K.G., Peres M.A., Do L.G.

Australian Research for Population Oral Health (ARCPOH), School of Dentistry, The University of Adelaide. *Poster presenter.

Background and Aims: There is a substantial gap in evidence of the effect of socioeconomic positions (SEP) and its trajectories on oral health, particularly related to periodontal disease, one of the most prevalent chronic conditions in adults. The aim of this study was to test the impact of socioeconomic trajectories over the life course on the occurrence of periodontal disease in young adults. Methodology: This study evaluated data from the 1982 Pelotas

Birth Cohort Study, Brazil. Family income collected at birth and ages 15, 19, 23 and 30 years was used to rank participants' SEP at each time. Group-based trajectory modelling was used to identify SEP trajectories based on Bayesian Information Criteria (BIC). Periodontal disease measures were collected through clinical examination in the Oral Health sub-study at age 31 years conducted in 2012 (OHS-12). The US Centers for Disease Control and Prevention and American Association of Periodontology (CDC-AAP) case definitions were adopted. Covariates included sex and maternal education at participant's birth, smoking status at age 23, dental flossing, bleeding on probing, dental calculus and presence of periodontal pocket at age 24 and participant's education at age 30. Multivariable log-Poisson Regression models were conducted to evaluate the effect of socioeconomic trajectories on two outcomes: any periodontal disease and moderate or severe periodontal disease. Interactions between socioeconomic trajectories with participant's education and maternal education on the outcomes were also tested. Results: A total of 539 participants were followed-up in the OHS-13 and therefore were included in the present study. The prevalence of any periodontal disease and moderate or severe periodontal disease was 37.3% and 14.3%, respectively. From our sample, there were 57.1% of the individuals in trajectory 1 (relatively stable rich over time), 19.7% in trajectory 2 (relatively stable middle income over time) and 23.2% in trajectory 3 (relatively poor from birth to age 15, slightly and consistently increasing to middle income in young adulthood). After adjustment for covariates, socioeconomic trajectories affected the prevalence of periodontal

disease later on. Compared to the trajectory 1, the trajectory 3 had an adjusted Prevalence Ratio of 1.5 for both outcomes (95% Confidence Intervals 1.0-2.1 for both outcomes). Women had lower prevalence of periodontal disease than men. Having dental calculus and periodontal pockets at age 24 were associated with higher prevalence of any periodontal disease at age 31, but not with moderate or severe periodontal disease. Tested interactions were not significant in any of the models. Conclusions: Socioeconomic trajectories from birth to age 30 affect periodontal disease later in life. These findings may inform programs in identifying and targeting potentially at-risk individuals early in life in order to prevent periodontal disease.

9.7 Abstracts of Conference Presentations - 7

Schuch HS, Peres KG, Singh A, Peres MA, Do LG. Effect of socioeconomic position during the life on periodontitis in adulthood: a systematic review. School of Dentistry Research Day 2015 – The University of Adelaide - Australia. (Oral presentation).

Title:

Longitudinal effect of socioeconomic status on periodontitis: a systematic review

Authors:

Helena Silveira Schuch[1*], Karen Glazer Peres[1], Ankur Singh[1], Marco A Peres[1] & Loc Giang Do[1]

1. Australian Research Centre for Population Oral Health (ARCPOH), School of Dentistry, The University of Adelaide, Adelaide, South Australia, Australia.
   * Presenter

Contact: helena.schuch@adelaide.edu.au

Tel: +61 8 8313 31296

Abstract:

Aims: To systematically review all longitudinal studies that investigate the effect of socioeconomic status (SES) during the life on the

development of periodontal diseases (PD) in adulthood. **Methods**: Potentially eligible papers were those addressing epidemiological observational longitudinal studies that included clinically assessed indicators of PD as outcome and that measured indicators of relative individual level SES. A search was performed in six electronic databases (PubMed, EMBASE, Web of Science, Scopus, LILACS (Latin American and Caribbean Health Sciences Literature) and ScieLO (Scientific Electronic Library Online). The review process was performed independently by 2 authors. First, titles and abstracts were screened, followed by full-texts evaluation and data extraction. The methodological quality assessment of the studies was assessed by the Newcastle–Ottawa Quality Assessment Scale for cohort studies. **Results:** The combined search from all the 6 databases provided 1,507 papers. After removals of duplicates (n=685), title and abstract screening (n=795) and full text review (n=19), 8 original manuscripts involving 7 studies were included in this review. The sample sizes ranged from 167 to 2806, and the time frame (from exposure to follow-up) varied from 2 to 38 years, with the majority of the studies presenting a time frame between 5 to 11 years. SES indicators included education, occupation and family income and outcomes assessed by the studies were mainly periodontal attachment loss, probing pocket depth and alveolar bone loss. In general, studies presented low risk of bias. Between all the papers included, 6 have found that a position of socioeconomic deprivation

negatively impacts on periodontal health. **Conclusions**: The scientific evidence demonstrates that socioeconomic indicators during the life negatively impact the occurrence of PD later on. Further prospective cohort studies are needed to confirm such association and to test in which life period the SES deprivation is more critical to predict PD.

9.8 Appendix Figure 1 – Systematic review data collection form

Data collection form

Note:

- **Record any missing information as unclear or not described, to make it clear that the information was not found in the study report(s), not that you forgot to extract it.**

Review title	**Life course effect of socioeconomic position on periodontal disease in adulthood: a systematic review**
Paper ID *(surname of first author and year of publication e.g. Smith 2001)*	
Notes	

General Information

Date form completed *(dd/mm/yyyy)*	
Name of reviewer	
Paper title	
Journal published	
Reference citation	
Study author contact details	
Notes:	

Characteristics of studies

Study Characteristics		**Yes**	**No**	**Unclear**	**Location in text or source** *(pg & ¶/fig/table)*
Aim of study					
Study design according to the	**Cohort prospective**	☐	☐	☐	
	Cohort retrospective	☐	☐	☐	

	Case-control	☐ ☐ ☐	
Place	Identify the country		
Ethically approved		☐ ☐ ☐	
Informed consent obtained		☐ ☐ ☐	
Participants and setting			
Method of recruitment of participants	☐ Randomly ☐ Convenience		
Total no. of subjects investigated			
Inclusion criteria			
Exclusion criteria			
Type of exposure	Education	☐ ☐ ☐	
	Occupation	☐ ☐ ☐	
	Income	☐ ☐ ☐	
	Other (specify)	☐ ☐ ☐	
Exposure details	Describe how exposure was defined (categories)		
Time points	Record when exposure was collected		
Time frame (from exposure to follow-up)	Record time between the assessment of exposure at last time and the occurrence of PD		
Outcome measures	Define primary outcome		
Outcome details	Record outcome case definition		

Outcome protocol	Record how outcome was measured (e.g. full mouth, partial-mouth)		
Secondary outcome	Record any secondary outcome		
Covariates analysed	List covariates included		
Confounding factors/ effect modifiers accounted for			
Results Occupation (specify, e.g. OR, RR, PR, IRR) (ref. > 10th grade)			
Authors' reported limitations of study's methods/results			
Key conclusions of study authors			
Notes:			

9.9 Appendix Figure 2 – Systematic review letter to researchers

Australian Research Centre for Population Oral Health (ARCPOH)

School of Dentistry
Faculty of Health Sciences

Helena Silveira Schuch
PhD Student

Ground floor, 122 Frome Street

The University of Adelaide
SA 5005
Australia
Telephone +61 8 8313 31296
Facsimile +61 8 8313 34858
helena.schuch@adelaide.edu.au

CRICOS provider number 00123M

19th May, 2015

Prof. Aubrey Sheiham
University College London, UK

Dear Prof. Aubrey,

My name is Helena Schuch and I am a PhD student at the Australian Research Centre for Population Oral Health (ARCPOH), The University of Adelaide. I am conducting a systematic review regarding potential longitudinal effect of socioeconomic position on periodontal disease in adulthood.

The research question that will guide this systematic review is: "Does socioeconomic status early in life influence development of periodontal diseases in adulthood?". We seek to identify all original papers that studied the association between socioeconomic positions and periodontal diseases later in adulthood, regardless of geography or publication date. Potential eligible papers will be those addressing epidemiological observational longitudinal studies that included clinically assessed indicators of periodontal disease as outcomes and that measured indicators of relative individual level socioeconomic position. Publication language will be restricted to English.

We have completed literature search using PubMed, EMBASE, Web of Science, Scopus, LILACS (Latin American and Caribbean Health Sciences Literature) and ScieLO (Scientific Electronic Library Online). We now plan to conduct searching the grey literature in order to capture most of the relevant studies.

Considering your expertise in such topic, I would like to ask if you are aware of any currently unpublished studies that could fit the systematic review aim.

Thank you for your valuable contribution.

Yours sincerely,
Helena Silveira Schuch, DDS, MDS
Australian Research Centre for Population Oral Health (ARCPOH)
School of Dentistry – The University of Adelaide – Australia
Email: helena.schuch@adelaide.edu.au

9.10 Ethical approval – Oral Health Sub-Study 2006

MINISTÉRIO DA EDUCAÇÃO
UNIVERSIDADE FEDERAL DE PELOTAS
PRÓ-REITORIA DE PESQUISA E PÓS-GRADUAÇÃO
DEPARTAMENTO DE PESQUISA

concluído

Pelotas, 15 de agosto de 2005.

Unidade: Fac.Medicina
Departamento: Medicina Social
Prof(a): Aluísio J.D.de Barros

Senhor(a) Professor(a):

De ordem do Senhor Diretor do Departamento de Pesquisa da Pró-Reitoria de Pesquisa e Pós-Graduação, informo a Vossa Senhoria que em reunião realizada no dia 10.08.2005. o Conselho Coordenador do Ensino, da Pesquisa e da Extensão – COCEPE, aprovou o Projeto de Pesquisa:

"Condições socioeconômicas, de comportamento e acesso a serviços e seus impactos na saúde bucal e qualidade de vida: um estudo longitudinal em uma coorte de nascidos vivos no sul do Brasil"

que será incluído na Programação Anual de Pesquisa da UFPel, com o código do projeto número "**4.06.01.124**"

Atenciosamente

Ana Lúcia Koga
CHEFE DA DIVISÃO DE PESQUISA E INICIAÇÃO CIENTÍFICA
PRPPG-UFPel

UNIVERSIDADE FEDERAL DE PELOTAS
FACULDADE DE MEDICINA
COMITÊ DE ÉTICA E PESQUISA

OF. 017/05

Pelotas, 18 de Abril de 2005.

Ilmo. Sr
Prof. Aluisio Jardim D. de Barros

Prezado Pesquisador;

Vimos através deste informá-lo que seu projeto de pesquisa intitulado "Condições socioeconômicas, de comportamento e acesso a serviços e seus impactos na saúde bucal e qualidade de vida: um estudo longitudinal em uma corte de nascidos vivos no sul do Brasil" foi aprovado por este comitê após cumprir as exigências solicitadas.

Prof. José Augusto A. Crespo Ribeiro
Coordenador do CEP/FM/UFPEL

9.11 Ethical approval – Oral Health Sub-Study 2013

FACULDADE DE MEDICINA DA UNIVERSIDADE FEDERAL DE PELOTAS

PARECER CONSUBSTANCIADO DO CEP

DADOS DO PROJETO DE PESQUISA

Título da Pesquisa: Condições de saúde geral, socioeconômicas, comportamentais, clínicas e de acesso a serviços ao longo do ciclo vital: associação com saúde bucal em uma coorte de nascidos vivos no Sul do Brasil

Pesquisador: Flávio Fernando Demarco

Área Temática:

Versão: 1

CAAE: 19551713.9.0000.5317

Instituição Proponente: Faculdade de Medicina da Universidade Federal de Pelotas

Patrocinador Principal: CONS NAC DE DESENVOLVIMENTO CIENTIFICO E TECNOLOGICO

DADOS DO PARECER

Número do Parecer: 384.332

Data da Relatoria: 29/08/2013

Apresentação do Projeto:

As condições bucais mais prevalentes e importantes são cumulativas e crônicas na sua natureza, sendo necessário um longo período para a sua ocorrência. Os estudos com delineamento de coorte prospectiva suportam a perspectiva do ciclo vital ¿ parte do pressuposto de que o estado de saúde em qualquer idade é o resultado não só de condições atuais, mas também de um acúmulo de condições que foram incorporadas ao longo da vida. No entanto, os estudos de coorte de nascidos vivos são escassos no mundo e no Brasil, a coorte de Pelotas, RS, é a única localizada em países de renda média, avalia desfechos bucais e são essenciais para a verificação de etiologia do processo saúde-doença. O presente trabalho tem como objetivo estudar a influência da trajetória socioeconômica ao longo da vida na saúde bucal na vida adulta e a associação entre condições de saúde bucal com condições de saúde geral em adultos. Serão reavaliados todos os indivíduos nascidos em 1982 aos 30 anos (sub-amostra N=720). Eles foram avaliados anteriormente aos 15 anos e aos 24 anos de idade, respectivamente, em 1997 e 2006. As variáveis do exame clínico incluem a presença de cárie dentária coronária; edentulismo, dentição funcional e arco dentário reduzido; uso e necessidade de próteses dentárias; sangramento gengival; doença periodontal; qualidade das restaurações e lesões de tecido mole. Os exames serão realizados nos domicílios dos participantes, com uso de luz artificial (fotóforos acoplados à cabeça), material de exame

Endereço: Rua Prof Araujo, 465 sala 301
Bairro: Centro **CEP:** 96.020-360
UF: RS **Município:** PELOTAS
Telefone:

(espelho plano, sondas periodontais, espátulas de madeira e gaze) devidamente esterilizado. Todos os examinadores, cirurgiões dentistas, pós-graduandos em Odontologia ou Epidemiologia, estarão devidamente paramentados respeitando as normas de biossegurança preconizadas pela Organização Mundial da Saúde. Outras variáveis do estudo, como as perinatais, demográficas, socioeconômicas, demográficas, comportamentais, de saúde bucal (higiene bucal, dor de origem dentária, dificuldades de alimentação em razão de condições bucais, xerostomia, o impacto dos desfechos de saúde bucal na qualidade de vida dos indivíduos e a utilização de serviços) serão coletadas pela aplicação de questionário padronizado e pré-testado previamente em outros estudos epidemiológicos. As condições de saúde geral, como peso, altura, circunferência abdominal, pressão arterial, densidade óssea, espessura da carótida medial, uso de medicamentos, morbidades auto-referidas, uso de serviços de saúde e auto-avaliação de saúde serão obtidas do levantamento de saúde geral em andamento no ano de 2012. Os entrevistadores serão alunos de graduação da Faculdade de Odontologia (UFPel), também com experiência neste tipo de atividade. A equipe de trabalho de campo será composta por 8 examinadores e 8 entrevistadores, além dos supervisores do trabalho de campo e auxiliares para digitação e arquivamento de material. Será elaborado um manual de instruções para a equipe de campo. Estima-se a realização de uma média de 50-60 entrevistas e exames completos por semana, o que totaliza aproximadamente quatro meses de trabalho de campo, incluindo o treinamento, pré-teste e estudo piloto. Estão previstas reuniões semanais de avaliação entre a equipe de campo e os supervisores e coordenadores do estudo. Todos os dados serão avaliados pelo software Stata versão 11.0 ¿ análises descritivas (frequências absolutas e relativas); univariada (teste Qui-quadrado para variáveis categóricas nominais e Qui-quadrado de tendência linear para variáveis ordinais) e multivariável (adoção de modelos hierárquicos onde as variáveis independentes foram ordenadas em blocos que determinarão a entrada das mesmas na análise estatística. Estes modelos devem descrever a relação hierárquica existente entre os possíveis fatores de risco aos desfechos estudados. Somente as variáveis que na análise bivariada apresentarem valor $p<0,25$ serão incluídas nos modelos e as finais com $p<0,05$. Em síntese, os estudos de coorte de saúde bucal são raros, mas oferecem valiosas contribuições para a compreensão dos antecedentes e da história natural de desfechos de saúde bucal e do processo saúde-doença. Além disso, auxiliam na tomada de decisões no campo da Saúde Pública, pois permitem a avaliação da interrelação entre Saúde Bucal e Sistêmica, buscando otimizar tanto recursos humanos quanto materiais e estão de acordo com a teoria do ciclo vital, pois a saúde bucal é resultante de interação de fatores socioeconômicos, biológicos e psicológicas.

Objetivo da Pesquisa:

Objetivos

Geral

- Estudar a condição de saúde bucal em adultos e seus desfechos ao longo da vida, assim como a associação entre a saúde bucal e condições de saúde geral nesta população.

Específicos

- Estimar a incidência e a trajetória de ocorrência dos principais agravos à saúde bucal em adultos, como a cárie dentária e a doença periodontal;
- Estimar o impacto das condições de saúde bucal sobre a qualidade de vida;
- Estimar a prevalência de medo frente a tratamentos odontológicos e seu impacto na qualidade de vida;
- Avaliar longitudinalmente a longevidade e a qualidade das restaurações;
- Verificar o uso e a necessidade de próteses dentárias;
- Estimar o acesso aos serviços de saúde geral e odontológico ao longo da vida;
- Estimar a prevalência das lesões em tecidos moles;
- Estudar a relação entre doença periodontal e sinais sub-clínicos de aterosclerose;
- Estudar a relação entre doença periodontal, perdas dentárias e pressão arterial;
- Verificar a associação entre níveis sanguíneos de proteína C reativa, colesterol e doença periodontal e perda dentária;
- Investigar a associação entre edentulismo e sobrepeso, obesidade central, abdominal e consumo de alimentos ultraprocessados;
- Relacionar a prevalência de lesões cervicais não-cariosas e características oclusais, como presença de facetas de desgaste;
- Avaliar se a experiência de lesão cariosa coronária ao longo da vida predispõe a ocorrência de lesão cariosa de raiz aos 31 anos de idade;
- Estudar a relação entre traumatismos dentários e traumatismos gerais ao longo da vida;
- Estimar a prevalência de desgaste dentário nesta população.
- Avaliar se a perda dentária está associada a trajetória socioeconômica dos indivíduos da coorte.

Avaliação dos Riscos e Benefícios:

A participação no estudo prevê um exame bucal que será realizado por dentistas e não oferece nenhum risco, não causa dor alguma e todos os instrumentos utilizados estarão esterilizados ou serão descartáveis.

Benefícios: conhecer a realidade da saúde dos moradores de Pelotas, a qual poderá melhorar os serviços de saúde nas comunidades. Além disso, se for identificada alguma necessidade de tratamento dentário, ele será realizado na Faculdade de Odontologia da UFPel, sem custo algum ao participante.

Comentários e Considerações sobre a Pesquisa:

O delineamento deste estudo será de uma coorte prospectiva de nascimentos. Em 1982, todos os nascimentos hospitalares que ocorreram na cidade de Pelotas, RS, foram identificados e os 5.914 nascidos vivos, cuja família residia na área urbana da cidade, foram pesados e as mães entrevistadas.

O estudo de saúde bucal de 2013 (ESB-13) compreenderá os 900 indivíduos da amostra selecionada para o primeiro estudo de saúde bucal de 1997. Assim como os levantamentos anteriores, este estudo constará de aplicação de questionário com questões relacionadas à saúde bucal e uso de serviços e exame clínico, onde além da avaliação das restaurações serão avaliadas outras condições bucais. As entrevistas dos participantes e exame clínico de saúde bucal serão realizadas nas casas dos indivíduos. Uma secretária agendará o dia de visita da equipe à residência. A coleta de dados será realizada por equipes compostas por examinadores (cirurgiões-dentistas), anotadores e entrevistadores (acadêmicos de Odontologia – UFPel).

Considerações sobre os Termos de apresentação obrigatória:

OK

Recomendações:

OK

Conclusões ou Pendências e Lista de Inadequações:

OK

Situação do Parecer:

Aprovado

Necessita Apreciação da CONEP:

Não

PELOTAS, 04 de Setembro de 2013

Assinador por:
Patricia Abrantes Duval
(Coordenador)

Endereço: Rua Prof Araujo, 465 sala 301
Bairro: Centro **CEP:** 96.020-360
UF: RS **Município:** PELOTAS
Telefone:

9.12 Manuscripts relevant to thesis

Singh A, Harford J, Schuch HS, Watt RG, Peres MA. Theoretical basis and explanation for the relationship between area-level social inequalities and population oral health outcomes – A scoping review. SSM – Population Health 2 (2016) 451-462.

This paper was developed during the PhD candidature and is part of the thesis of the colleague Ankur Singh. The scoping review was conducted aimed to review the literature on the association between area-level social inequalities and population oral health. The paper is related to the present thesis since it discusses the evidence and the theories of social inequalities. Although the focus of the scoping review was on area-level inequalities, some of the theories also apply to individual level inequalities, and by analyzing and discussing the evidence the researcher increased her knowledge and understanding of the socioeconomic inequalities theories and its application in oral health.

SSM -Population Health 2 (2016) 451–462

Contents lists available at ScienceDirect

SSM -Population Health

journal homepage: www.elsevier.com/locate/ssmph

Review Article

Theoretical basis and explanation for the relationship between area-level social inequalities and population oral health outcomes – A scoping review

Ankur Singh [a,*], Jane Harford [a], Helena S. Schuch [a], Richard G. Watt [b], Marco A. Peres [a]

[a] Australian Research Centre for Population Oral Health (ARCPOH), School of Dentistry, The University of Adelaide, Adelaide, Australia
[b] Research Department of Epidemiology and Population Health, University College London, London, United Kingdom

ARTICLE INFO

Article history:
Received 6 January 2016
Received in revised form
3 May 2016
Accepted 1 June 2016

Keywords:
Inequality
Theories
Oral health
Social determinants
Review

ABSTRACT

This study was conducted to review the evidence on the association between area-level social inequalities and population oral health according to type and extent of social theories. A scoping review was conducted of studies, which assessed the association between area-level social inequality measures, and population oral health outcomes including self-rated oral health, number of teeth, dental caries, periodontal disease, tooth loss, oral health-related quality of life (OHRQoL) and dental pain. A search strategy was applied to identify evidence on PubMed, MEDLINE (Ovid), EMBASE, Web of Science, ERIC, Sociological Abstracts, Social Services Abstracts, references of selected studies, and further grey literature. A qualitative content analysis of the selected studies was conducted to identify theories and categorize studies according to their theoretical basis. A total of 2892 studies were identified with 16 included in the review. Seven types of social theories were used on 48 occasions within the selected studies including: psychosocial ($n=13$), behavioural ($n=10$), neo-material ($n=10$), social capital ($n=6$), social cohesion ($n=4$), material ($n=3$) and social support ($n=2$). Of the selected studies, four explicitly tested social theories as pathways from inequalities to population oral health outcomes, three used a theoretical construct, seven used theories for post-hoc explanation and two did not have any use of theory. In conclusion, psychosocial theories were used most frequently. Although theories were often mentioned, majority of these studies did not test a social theory.

Contents

* Correspondence to: Australian Research Centre for Population Oral Health (ARCPOH), School of Dentistry, The University of Adelaide, Ground Floor, 122 Frome Street, Adelaide, SA 5000, Australia.
E-mail address: ankur.singh@adelaide.edu.au (A. Singh).

http://dx.doi.org/10.1016/j.ssmph.2016.06.001

1. Introduction

> 'He who loves practice without theory is like the sailor who boards ship without a rudder and compass and never knows where he may cast' – *(Leonardo da Vinci* 1452–1519*)*.

Theory is essential to understanding patterns in ideas and observations, and to develop causal explanations (Krieger, 2011, 2014). It has a paramount role in the field of social epidemiology (Bartley, 2004; Krieger, 2014) as the discipline is not only limited to study effects of socio-structural factors on health (Honjo, 2004; Kawachi & Berkman, 2000) but also to understand the causal explanations and to intervene in order to effect change. Oral diseases affect 3.9 billion people and untreated dental caries (tooth decay) is the most prevalent condition globally (Marcenes et al., 2013). Oral diseases significantly affect quality of life (Marcenes et al., 2013) and are associated with significant health care costs (Listl, Galloway, Mossey & Marcenes, 2015). Baker and Gibson (2014) have argued that routine testing of theoretical pathways is not generally evident in the field of social oral epidemiology. This scoping review assesses the extent to which theory is used in any capacity in studies of social inequality and oral health.

1.1. Theory, social ecology and health

A curvilinear association between average national income and overall health has been observed since the late 1970s (Rodgers, 1979). These observations gave rise to the 'income inequality hypothesis' (IIH), which states that beyond a certain threshold of average income within a society, the distribution of income has a greater effect on average population health than average income (Wagstaff & Doorslaer, 2000). This hypothesis has given rise to studies of 'social ecology' to test the association between inequality and overall health. At least 300 studies of social ecology with various health outcomes have been published (Pickett & Wilkinson, 2015a), and, the importance of income inequality as a 'social pollutant' (Subramanian & Kawachi, 2006) has been widely debated over the past three decades (Pickett & Wilkinson, 2015b). While earlier reviews expressed scepticism with regards to the evidence on this relationship (Lynch et al., 2004; Wagstaff & Doorslaer, 2000), more recent reviews have supported this association. These later reviews concluded that detrimental effects of area-level social inequality, primarily income inequality, are universally evident (Kondo et al., 2009; Wilkinson & Pickett, 2006), causally related and affect the majority of the population (Pickett & Wilkinson, 2015a). They are not simply the result of higher rates of poverty in more unequal societies (Pickett & Wilkinson, 2015b).

Several theories/theoretical models have been proposed to explain how area inequalities may influence societal levels of health and disease (Bartley, 2004; Coburn, 2000; Kawachi & Kennedy, 1999; Lynch et al., 2004; Lynch, Smith, Kaplan & House, 2000; Marmot & Wilkinson, 2000; Navarro, 2002; Wilkinson & Pickett, 2006). Six distinct theories are identified that can be tested in studies of the association between social inequality and oral health (Bartley, 2004). The first two represents ecological counterparts to explanations for the association between individual socioeconomic position and health within the Black Report (Townsend, Davidson & Black, 1982), while the remainder were developed specifically to explain differences between populations:

i) Materialist: materialist explanations emphasize the role of the external environment on health; these vary with the level of inequality. Exposure to risks to health, and to protective factors varies with social position. Macroeconomic variables such as levels of production and unemployment affect health. Attention is paid to the roles of stress associated with material factors and with the hazardous nature of work. At an ecological level, more unequal societies have more people exposed to these risks (Townsend et al., 1982; Macintyre, 1997).
ii) Behavioural: behavioural explanations state that unequal societies generate higher levels of unhealthy behaviours. There are two versions of this explanation (Macintyre, 1997). One (hard) version of behavioural explanations identifies individual inadequacy as the main source of this behaviour. A second (soft) version is that behaviours have social gradients and contribute to observed gradients in health status.
iii) Psychosocial: psychosocial was developed to explain individual-level inequalities. At an individual level, psychosocial explanations claim that social position affects health in one of two ways. First, people's perception of their social position affects health. Second, there is an inverse association between levels of control, and resulting chronic stress and social position that affects health. Whether through perception or control/stress, the subsequent effect on health is either through direct physiological changes or through health damaging behaviours (Bartley, 2004). Within unequal societies, due to constant social evaluative threats, it is likely that people who are less well-off tend to compare themselves to those who are relatively better. Such comparisons lead to a constant perception of belonging to a low status group, along with lack of control and coping strategies consequently leads to chronic stress. This stress through either health compromising behaviours or through directly affecting physiological health, may lead to higher levels of disease (Wilkinson, 1997). The more unequal a society, the greater the decrement in power and control and the more damaging the perception and lack of psychosocial assets, thus the greater the impact on health. Because the social gradient is steeper within unequal societies, these effects may be more evident higher up the social gradient compared to more equal societies (Marmot & Wilkinson, 2000).
iv) Social capital: social capital explanations are often described as a subset of psychosocial explanations. These explanations state that unequal distribution of income undermines trust and damages social relationships. This can manifest in low levels of social support or civic participation, or in high levels of antisocial behaviour, particularly crime. This has been accepted as a potential pathway since Kawachi, Kennedy, Lochner, and Prothrow-Stith (1997) demonstrated that the association between inequality and mortality in the United States was mediated by social capital (Kawachi & Kennedy, 1999; Subramanian & Kawachi, 2004).
v) Neo-material: neo-material explanations arise from criticism that the psychosocial and social capital explanations ignore upstream factors that affect health and may be associated with greater inequality. Specifically, they ignore the role of uneven distribution of power and class relations, and labour market dynamics in sustaining and driving inequalities (Muntaner, Lynch & Oates, 1999; Navarro, 2002; Coburn, 2000). This results from a systematic underinvestment in human, physical, health, and social infrastructure that support health (Lynch et al., 2000, 2004).
vi) Structural: the structural explanation states that it is likely that the income inequality results in greater residential segregation leading to spatial concentrations of race and poverty, which in turn influences individual health. This may consequently lead to worse population health (Subramanian & Kawachi, 2004).

Many of these pathways are linked (Lynch & Kaplan, 1997) and some are treated as a subset of others in the literature. These pathways are unlikely to be mutually exclusive with more than

one operating at any time or place, but the role of each may vary according to context and health outcome. But, depending on the different sociological origins of each theory, the policy implications of each theory will be accordingly different. Muntaner and Lynch (1999) argue that 'IIH' and psychosocial interpretation treat income as a resource for purchasing social goods rather than as a product of production relations. At an area level this argument relates to whether inequality is conceptualized on a stratificational (gradational) scale or as a relational product (Muntaner & Lynch, 1999), as also shown at an individual level (Muntaner et al., 2010). Ignoring the relational property of inequality ignores underlying class relations, power dynamics and consequent exploitation that may affect health separately to income. So, a more psychosocial and social capital emphasis may deviate the attention of policy-makers from addressing more relevant structural factors related to social inequalities which impact population health (Muntaner & Lynch, 1999). On the contrary, the psychosocial theorists argue that ignoring the psychosocial mechanisms may ignore the negative impacts of relative deprivation and social comparisons on the physiological and psychological health and social fabric (Marmot and Wilkinson, 2000).

The need to test theoretical pathways between social inequalities and overall health is well established (Bartley, 2004; Campbell et al., 2014; Krieger, 2011, 2014). But, the evidence regarding the use of theory in explaining area level social inequalities and population oral health has not been reviewed. Evidence on the role of pathways between area-level social inequalities and population oral health outcomes clarifies the basis for specific policies in order to reduce the health effects of social inequalities. In order to address the significant gap regarding the use of theory in studies of social ecology in oral health, this scoping review was performed with four objectives: (i) to assess the availability of evidence on the association between area-level social inequality and population oral health according to type of social theories, (ii) to assess the extent to which the literature on this association is theoretically based, (iii) to identify and categorize conceptual and measurement alternatives used in the evidence to measure social class or socioeconomic inequalities according to either stratification or relational approach, and (iv) to identify and highlight any gaps in the literature.

2. Methods

A scoping review determines the extent, range and nature of any research activity, making it a more suitable approach than a systematic review for this research question (Arksey & O'Malley, 2005; Levac, Colquhoun, & O'Brien, 2010). Given the complexity of the review design, a detailed protocol for this scoping review was published which also elaborates this justification (Singh, Harford, Watt, & Peres, 2015). A methodological framework for this review is based on the existing literature and has five steps (Arksey & O'Malley, 2005):

(1) *Identifying the research question*: the research question framed was, 'What is the nature and extent of social theories/theoretical models being used as a basis to explain the associations between area-level social inequalities and population oral health in the existing literature?'
(2) *Identifying relevant studies*: a search strategy was formulated to identify both published studies and grey literature. A three-step search strategy was developed for this review. An initial limited search of MEDLINE was undertaken followed by analysis of the text contained in the title and abstract, and of the index terms used to describe the articles. Following this the next step involved using all identified keywords and index terms to search across all selected databases: PubMed, MEDLINE (Ovid), EMBASE, Web of Science, ERIC (Education Resources Information Center), Sociological Abstracts, Social Services Abstracts. A detailed search strategy including the relevant keywords and MeSH terms was constructed specifically for each selected database. Each data source was individually checked for availability and usage of controlled vocabulary for indexation through the use of hierarchically defined and periodically updated thesauruses (Appendix 1). The search was first conducted on 14th January, 2015 and further updated to identify recent studies on 7th March, 2016. The reference list of all identified reports and articles was searched to identify any additional studies. Finally, the search for unpublished studies included reference lists, book chapters, Thesis (Proquest) and conference abstracts. Furthermore, eight experts were identified and contacted for relevant grey literature based on the criterion that within the literature search they should have published at least twice on this research topic.
(3) *Study selection*: pre-defined inclusion and exclusion criteria were developed to identify relevant studies (Arksey & O'Malley, 2005). Studies were excluded if they were published in a language other than English, or did not include a measure of inequality, or focussed on individual-level inequalities in health outcomes, or had outcomes of interest other than dental caries, periodontal disease, self-rated oral health, number of teeth, tooth loss, oral health-related quality of life (OHRQoL) and dental pain. The detailed inclusion and exclusion criteria are reported elsewhere (Singh et al., 2015).
(4) *Charting the data*: a data charting guide and recording proforma were developed by the reviewers and piloted independently by two investigators (AS and HSS) on five studies who cross-checked extracted information and revised the guide and proforma to address discrepancies. The information charted included study details (author, publication type, study design, locations, population focus, sample size, statistical modelling, geographical unit of aggregation and population oral health outcomes), details on theory (mention of theory, number of use and type of theories) and measure of social inequality (type of inequality and area based quantitative measure of inequality). Based on emerging information from studies this form was constantly updated in consultation with the reviewers. Two (AS and HSS) reviewers independently charted all the extracted information and crosschecked the information to reduce individual bias (Riva, Gauvin, & Barnett, 2007). Any disagreements were resolved firstly by discussion then by intervention of a third reviewer (JH).
(5) *Collating, summarizing and reporting the results*: Extracted data was summarised using narrative synthesis (Arksey & O'Malley, 2005). The proposed *a-priori* approach to data mapping was to categorize included studies both by the type of social theory used and the extent to which social theory as drawn upon by the authors. Selected papers were entered into NVivo v10 software, which was used to identify theories and categorise studies according to extent of their theoretical use. A deductive content analysis using pre-defined categories was performed by analysing extracts on theories from the primary studies (Fig. 1). This process involved analysing elements such as naming the theory, context in which theories are introduced, and, application of theory based on their emphasis within objectives and use as variables within the analysis strategy. Information reflecting these aspects was extracted from the papers under following categories, 'comment on theoretical pathways', 'direct mention of theory', 'inferred theory', 'variables for theory', and 'objectives' (Fig. 1). Based on the analysis of extracted information under these

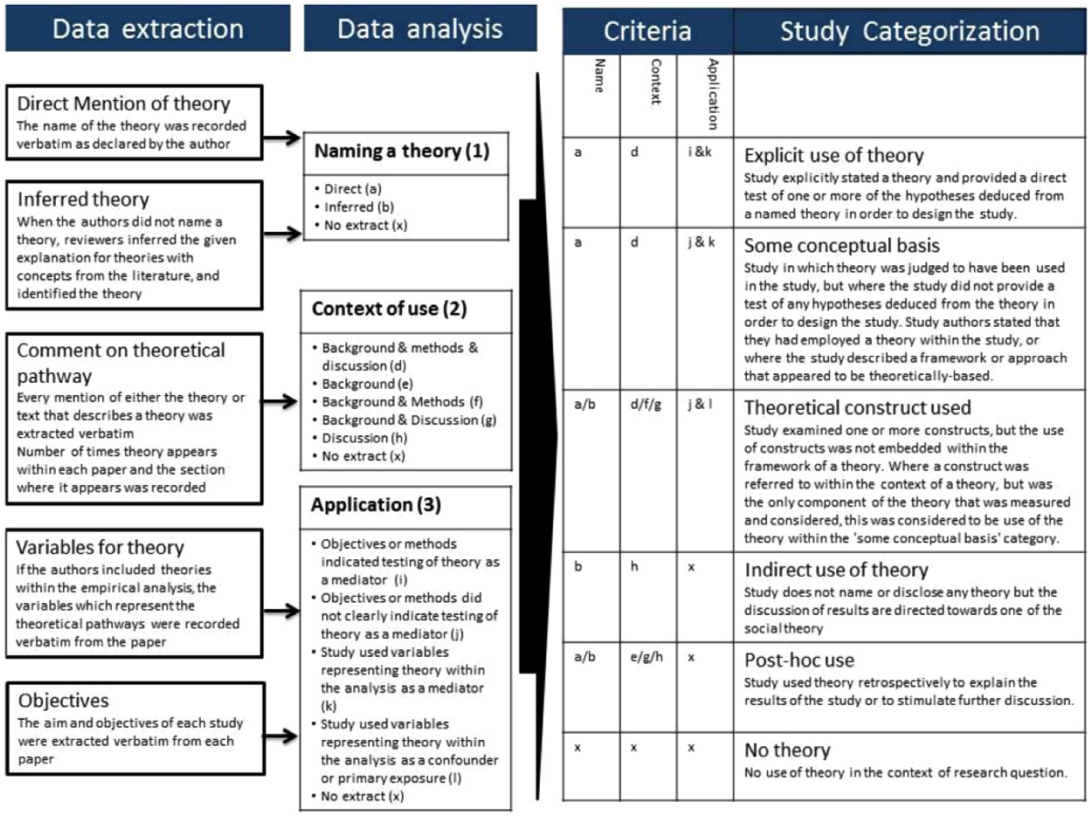

Fig. 1. Deductive content analysis to categorize studies according to their extent of use of theory.

categories, each study was then categorized exclusively into one of the following pre-defined categories for use of theory: 'explicit use of theory', 'some conceptual basis', 'theoretical construct used', 'post-hoc use', 'indirect use of theory' and 'no theory' (Singh et al., 2015). For instance, the study by Aida et al. (2011) had 6 unique mentions of theories, within the introduction, methods and discussion section. The study's objectives clearly mentioned that it aimed to test whether social capital explained the associations between income inequality and dental status. The study explicitly named the 'social capital' theory and used the variables representing theory within the analytical strategy (Appendix 1). Based on this information, the study was categorized under the 'explicit use of theory'. The unit of analysis for this activity was a study rather than a theory, therefore any study with multiple theories was classified according to the highest level use of theory as indicated by this ordering of categories. Additionally, a narrative synthesis was added to highlight the between study differences identified within categories.

The criteria for each of these categories were derived from a systematic review for a similar research question, but applied in a different field (Davies, Walker, & Grimshaw, 2010). Furthermore, the extracted information under 'direct mention of theory' and 'inferred theory' assisted in identifying all types of social theories and the frequency of their use within the studies selected. In order to be inclusive of depletion of social capital pathway as an independent theoretical pathway (Kawachi & Kennedy, 1999), apart from those theories summarized by Bartley (2004); material, neo-material, behavioural/cultural and psychosocial; all extracts (implicit and inferred) that made reference to any dimension of social capital were also identified.

To ensure a reliable process and to reduce individual bias, two reviewers participated in both the data extraction exercise and data categorization exercises. The initial data extraction exercise was performed by AS with HSS crosschecking the decisions regarding number and relevance of extracts. A pilot exercise on two selected studies compared the consistency in data extraction. Both AS and JH conducted the study categorization exercise independently with disagreements resolved through discussion.

A sub-analysis focussed on the choice of measurement variables for area-level social inequality. Studies were categorized according to the measure of inequality used and how it was quantified. A quality assessment of the selected studies was not conducted, as a scoping review does not aim to synthesize evidence according to methodological quality (Arksey & O'Malley, 2005).

3. Results

Overall, 2892 studies were identified by a systematic search on all selected databases, and 1188 duplicates were removed. Some

1600 records were excluded where it was clear from title and abstract they were out of scope based on the inclusion/exclusion criteria leaving 105 relevant titles. Upon full text review another 89 studies were excluded leaving 16 relevant studies for data charting. A flowchart of this process is shown in Fig. 2.

3.1. Summary characteristics of the identified studies

The majority of studies examined the impact on health of inequalities within countries (IDs B,D,F,H,J,K,L,M,N,O&P in Table 1). Five studies examined multiple high income countries (IDs A,C,E,

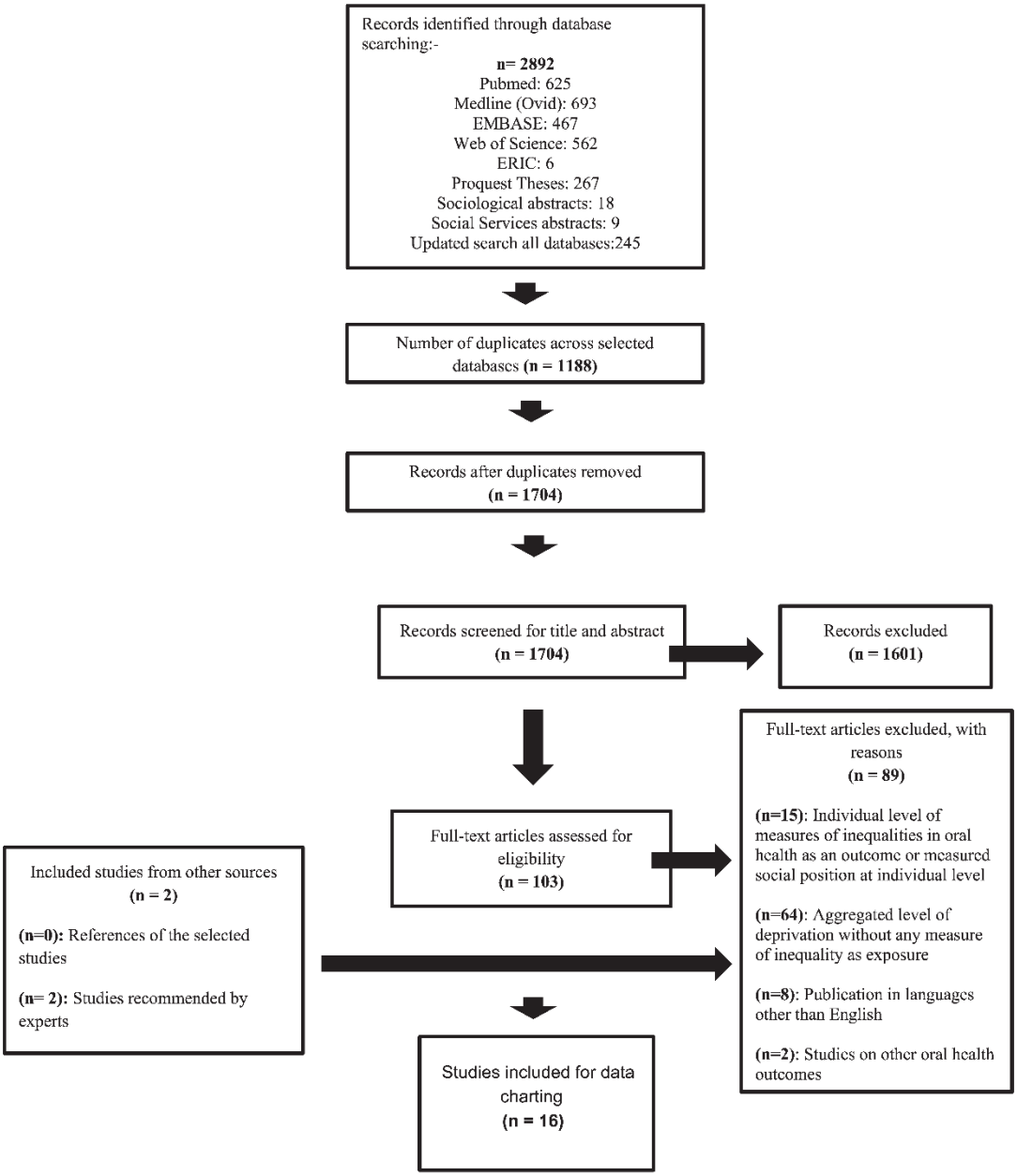

Fig. 2. Flow chart for the process of literature search to study selection.

Table 1
Descriptive summary of the selected studies.

Code	Study	Study design/ analysis	Location	Population focus	Aggregate level	Oral health outcomes	Main Results (inequality – oral health)	Main Results (Theories)
A	Nadanovsky and Sheiham (1995)	Ecological/ correlations	18 industrialized countries	12-year-old children	Country	12-year-old DMFT Compound Annual Rate (%)	The higher the concentration of income in the top 20% income households in 1970–75, the lower the rate of DMFT reduction.	NA
B	Pattussi, Marcenes, Croucher, and Sheiham (2001)	Cross sectional/ correlations	Brazil	6–12-year-old school children	Intra-urban areas of Brasilia (Federal District)	Dental caries levels: the percent of children free of caries, mean DMF-T scores	GINI coefficient was negatively statistically significantly associated with both measures of dental caries experience, percent of caries free (P=0.003) and mean DMF-T scores (P=0.01).	NA
C	Hobdell et al. (2003)	Ecological/ correlations	99 countries (Dental caries) 44 countries (CPITN)	12 year olds (caries) and 35–44 year old adults (CPITN)	Country	Dental caries (DMFT) and destructive periodontal disease (CPITN)	Gini Index was positively and significantly correlated with dental caries and CPITN scores	NA
D	Peres et al. (2003)	Ecological/ correlations	São Paulo, Brazil	5–6 year old children	Cities and Town	Dental caries: mean DMFT	DMFT not associated with income inequality	NA
E	Bernabe, Sheiham, and Sabbah (2009)	Ecological, cross-sectional study/ correlations	High income countries	35–44 year old adults from rich countries	Country	Dental caries experience: untreated caries, missing teeth, filled teeth and DMFT; dental care index: restorative index, treatment index	Income inequality was significantly and inversely related to the number of filled teeth, DMFT score and provision of restorative treatment, but not to the number of decayed or missing teeth.	NA
F	Celeste, Nadanovsky, Ponce de Leon and Fritzell (2009)	Cross sectional/ multi-level	Brazil	15–19 and 35–44 year olds	Municipal level	Tooth loss: all natural teeth (yes/ no); untreated dental caries: number of teeth with untreated dental caries	Income inequality showed an effect after controlling for known confounders and mediators based on a priori postulated pathways with missing teeth and number of teeth with untreated decay. (VPC for at least one missing tooth—9.36%; Number of teeth with untreated caries=5.28%; Edentulism=9.08%; Number of teeth with untreated caries—4.37%)	Models representings social capital and health services did not change the Gini effect considerably
G	Bernabe and Hobdell (2010)	Cross sectional/ correlations	48 countries	5- to 6-year old children	Country	Dental Caries (DMFT Index)	The dmft index was significantly correlated with the Gini index in rich countries but not all countries	NA
H	Celeste and Nadanovsky (2010)	Cross sectional/ multi-level	Brazil	15–19 year olds	Municipal level	Number of missing teeth and number of decayed teeth	Municipal level public policies were the main explanation for the income inequality effects on oral health	Most of the Gini effect was explained by the number of years of water fluoridation and Scale of Municipal Public Policies (SMPP)
I	Sabbah, Sheiham and Bernabe (2010)	Ecological/ correlations	17 rich countries	Adults aged 35–44 years	Country	Periodontal disease: percentage of adults with periodontal pockets >4 mm 'Community Periodontal Index (CPI) 3 or 4' and with periodontal pockets >6 mm (CPI 4)	Higher levels of income inequality in rich countries were associated with higher levels of periodontal disease in adults, even after adjusting for measures of absolute national income	NA
J	Aida et al. (2011)	Cross sectional/ multi-level	Aichi, Japan	Older adults (65 and above)	District	Number of remaining natural teeth (having 20 or more teeth vs having 19 or less teeth)	Income inequality in communities was significantly associated with poor dental status. Income inequality was a major contributor to the variation in dental status between communities (Dental	Individual- and community-level non-volunteering and mistrust did not substantially reduce the odds for poorer dental status

							status, Variance=0.011, SE=0.012)	
K	Bernabe and Marcenes (2011)	Cross sectional/ multi-level	USA	18 years and above	State	Self-reported tooth loss: (none, 1–5, 6 or more but not all, and all teeth)	State Gini coefficient was associated with higher odds of reporting greater tooth loss. (Between state Variance – 0.025; SE – 0.005)	The state Gini coefficient remained significantly associated with tooth loss after adjustment for state dentist-to population ratio and percent receiving fluoridated water (neo-material) and individuals' marital status (social capital).
L	Celeste, Fritzell and Nadanovsky (2011)	Cross sectional/ multi-level	Brazil	35–44 year-olds adults	Municipal level	Untreated dental caries, edentulism, at least one site with CAL > 8 mm, bleeding or dental calculus	Lagged Gini showed no association with any outcome; current Gini was associated with untreated dental caries but not with edentulism and periodontal disease. (VPC for untreated dental caries = 3.6%)	NA
M	Vettore, Marques and Peres (2013)	Cross sectional/ multi-level	Brazil	Adults aged 35–44	State Capitals and Federal Districts	Periodontal disease: "Moderate to severe" periodontal disease; "Severe" periodontal disease	Income inequality was independently associated with "severe" periodontal disease (OR = 3.0, 95%CI 1.5;5.9); Variance = 0.101, SE = 0.044	NA
N	Vettore and Aqeeli (2015)	Cross sectional/ multi-level	Brazil	Adults aged 35–44	City	Oral Health Related Quality of Life (ORHQoL) measured by Oral Impacts on Daily Performance (OIDP)	Income inequality associated with emotional status, work and social contact. (Gini 1991; Variance = 0.070, SE = 0.021; Gini 2000; Variance = 0.072, SE – 0.021)	NA
O	Goulart and Vettore (2016)	Cross sectional/ Multi-level	Brazil	Adults aged 35 to 44	City	Tooth loss (Measured by M component of DMFT): Severe tooth loss (< 9 teeth) and lack of functional dentition (< 21 teeth)	Moderate and high increase in income inequality associated with both outcomes (Severe Tooth Loss – Variance = 0.104, SE = 0.055; Functional dentition, Variance = 0.189; SE = 0.061)	
P	Chalub, Martins, Ferreira and Vargas (2016)	Cross sectional/ multi-level	Brazil	Adults aged 35–44	Municipal level	Functional dentition (4 Definitions:- WHO functional dentition, Well distributed teeth, Functional dentition classified by aesthetics and occlusion, Functional dentition classified by esthetics, occlusion and periodontal status)	Income inequality was not associated with any definition of functional dentition	

G&l). Nine studies pertained to Brazil (IDs B,D,H,L,M,N,O&P), with three of these (IDs F,G&L) reporting overlapping outcomes for two identical population groups from the same survey. The selected studies included ages five years and upwards. All 16 studies were cross-sectional with seven assessing the association by correlations while nine conducted a multi-level analysis. Random parameters were reported in six out of nine multi-level studies. The geographic unit of analysis ranged from municipal level to country level. Among the selected studies, nine were designed specifically to test associations between inequality and oral health while seven were exploratory studies which tested inequality as one of the contextual factors. Oral health outcomes tested included dental caries ($n=9$), tooth loss ($n=8$), periodontal disease/outcomes ($n=4$) and oral health related quality of life ($n=1$) (Table 1).

3.2. Theories – type and extent in the selected studies

Overall, there were 48 uses of seven types of social theories in the selected studies including psychosocial (13-IDs C,E,F,G,H,I,J,K,L, M&N), behavioural (10-IDs A,C,D,E,G,K,L,M,N&P), neo-material (10-IDs D,F,G,H,I,K,L,N,O&P), social capital (6-IDs B,E,F,I,J&N), social cohesion (4-IDs B,E,K&O), material (3-ID D,F&N) and social support (2-IDs K&M) (Table 2). This includes all theories that were either directly mentioned by authors in the text or in which the text appeared to describe one of these theories. Six of theories were directly mentioned; psychosocial (11-IDs C,E,F,G,H,I,K,L,M&N,P), behavioural (7-IDs C,E,G,L,M,N&P), social capital (5-IDs E,F,I,J&N), social cohesion (4-IDs B,E,K,&O), material (3-IDs F,N,P) and neo-material (2-IDs F&H) (Table 2).

Four studies (IDs F,H,J&K) explicitly tested the theories as mediators or pathways between social inequalities and population oral health outcomes (Table 2, Appendix 2). Three studies (IDs B, M&P) discussed at least one construct that was consistent with a theory in the introduction and discussion, but did not test it. Seven studies (IDs C,E,G,I,L,N&O) used theories for post-hoc explanations to either discuss their findings or to stimulate further discussion. Three studies had no theoretical basis at all (ID A&D) (Table 2).

3.3. Within category differences between studies

Despite testing theories as mediators, differences according to the explicitness and comprehensibility regarding the theories were observed among the four explicitly theory based studies (IDs F,H,J&K). While (IDs F,H&J) explicitly stated that they intended to test the potential of one or alternate theoretical models, in explaining the associations between income inequality and health outcomes, (ID K) only incorporated theories within the modelling strategy and stated that it accounted for diverse set of individual and state level factors. In terms of comprehensibility, while (IDs F, H,K) included multiple theoretical models, (ID J) only tested the potential of social capital to explain the relationship. The studies categorized under 'post-hoc' group differed in the way that while studies (IDs E&O) only introduced theories in the introduction to justify testing for inequality – oral health association, studies (IDs I&L) used theories in both introduction and discussion to justify the objective and potential explanations for their findings. Finally, studies (IDs C,G&N) only discussed theories in the discussion as potential explanations for their findings. Such between study differences were not observed under those identified with 'no theory' and 'theoretical construct used'.

3.4. Measurement of social inequality in the selected studies

All the selected studies used income inequality as the measure of area-level social inequality. 15 out of the 16 selected studies used the Gini Index as a measure of income inequality, while one study (ID E) used both the Gini index and the 20:20% (ratio of total annual household income received by the richest 20% of the population to that received by the poorest 20%). Only one study (ID A) used the percentage of national income earned by the top 20% as the measure of area-level social income inequality (Table 3).

4. Discussion

All but one of the selected studies mentioned at least one theoretical pathway between social inequality and population oral health; however, theories were seldom explicitly stated and tested. Psychosocial theory was most frequently used. Income inequality was the only measure of inequality reported and always measured on a stratificational scale.

Although social theories are often mentioned in studies of social ecology in social (oral) epidemiology and have drawn interest over time, the lack of explicit theoretical basis among selected studies substantiates the findings from the study by Baker and Gibson (2014). Using a qualitative methodology the current study observed that theories were mostly used for a post-hoc explanation of results rather than being explicitly stated or incorporated in analytical models. Furthermore, differences were also observed in the context in which theories were used in a post-hoc manner. When theories were tested for mediation, the studies differed according to their comprehensibility and explicitness. The differences in descriptive and explanatory objectives of the selected studies may be a potential explanation for such differences. Most of the selected studies were designed to test the empirical association between inequality and oral health, rather than to explain them. On the other hand, some studies were exploratory and included inequality as one of the exposures. A very small proportion of studies aimed to test any theoretical pathways. As a scoping review, the current study did not draw conclusions on the associations between inequality and population oral health. However, summary of findings (Table 1) suggests an association between income inequality and multiple oral health outcomes. Considering that theories form a strong basis for choosing appropriate strategies to reduce ill effects of inequalities on population oral health, findings from the current review highlights the lack of theory and underscores the necessity for explicit theoretical basis in future studies.

The different theoretical pathways have key implications for the pathogenesis of different oral health outcomes. For example, fluoride intake affects the risk of experiencing caries, but is not considered to be causative for periodontal disease. Even within one oral disease, caries, intermediate and proximal factors that affect the risk of having disease in the first place (e.g. sugar, fluoride) are not the same as the risk of losing a tooth due to caries (add access to timely dental care to sugar and fluoride). This highlights the need for outcome-specific theoretical models to explain the associations and for robust data collection based on outcome specific theoretical models. This would make research recognize the heterogeneity of etiologies and these may be important for which pathways matter for different conditions. Furthermore, inequality is a true ecological variable (Diez-Roux, Link, & Northridge, 2000), and the intervening mechanisms or resources may differentially impact individual and population health status (Rose, 1992). Therefore, the theoretical models demand more clarifications when differentiating between ecological relationship between inequality and population health, and contextual effects of inequality on individual health. Use of direct acyclic graphs (DAGs) (Fleischer & Diez-Roux, 2008) to identify *a-priori* confounders and mediators can also help in this process.

The predominance of psychosocial theory including depletion of psychosocial assets such as social capital and social support theory in oral health literature is mirrored in its use in research in general

Table 2
Analysis of the theoretical basis of selected studies assessing the association between area level social inequality and population health outcomes.

Study	Type of theory/theories: Direct (*) and Inferred (#)	Explicitly theory-based	Some conceptual basis	Theoretical construct used	Post-hoc	No Theory
A	No extract					+
B	Social cohesion* and social capital#			+		
C	Behavioural* and psychosocial*				+	
D	Behavioural#, material# and neo-material#					+
E	Social capital*, social cohesion*, psychosocial* and behavioural*				+	
F	Social capital*, material*, psychosocial*, neo-material*	+				
G	Behavioural*, psychosocial* and neo-material#				+	
H	Psychosocial*, neo-material*	+				
I	Psychosocial*, social capital* and neo-material#				+	
J	Social capital* and psychosocial#	+				
K	Social cohesion*, psychosocial* and behavioural*, neo-material# and social support#	+				
L	Psychosocial*, behavioural* and neo-material#				+	
M	Psychosocial*, behavioural* and social support#			+		
N	Material*, social capital*, behavioural*, psychosocial* and neo-material#				+	
O	Social cohesion*, neo-material# and psychosocial#				+	
P	Behavioural* and psychosocial*			+		

health literature (Islam, Gerdtham, Gullberg, Lindstrom & Merlo, 2008; Murayama, Fujiwara & Kawachi, 2012; Oksanen et al., 2008; Robert, 2001). The use of social capital, social cohesion, social network and social support along with the levels at which they are conceptualized needs more clarification. First, social capital is a broader concept which includes both social cohesion and social network (Mackenbach et al., 2016). Second, these inter-personal constructs and resources may mean different things at the individual and contextual level. Finally, their potential explanatory power may differ for different outcomes. This is substantiated by the evidence showing community-level structural social capital to attenuate the odds of inequality for poorer self-rated health but having no substantial impact on the odds for worse dental status (Aida et al., 2011). The limited explicit attention to the neo-material pathway within the selected studies could be due to the lack of clarity on its conceptualisation and measurement. The definition of neo-material theory contains two important elements: 'structural factors differentiating equal and unequal societies' and 'systematic underinvestment in public policies and health care' (Lynch et al., 2000). In terms of public health policies and health care, some of the key determinants of oral diseases in the population may include infrastructures such as access to dental health care, water fluoridation, food supply and population-level tobacco control measures (Watt, 2012). Under the neo-material interpretations of the inequality – health relationship, it is argued that the historical, cultural, political and economic processes, which lead to inequality, may also shape the nature and availability of health supportive infrastructure (Lynch et al., 2004). In eight out of the ten selected studies where neo-material explanations were inferred, policy determinants such as water fluoridation; social spending and public investment, and, dentist to population ratio were mentioned; but the pathway was not identified as neo-material. It should be noted that all selected studies conducted a secondary analysis which limits conceptualization of the theoretical pathways as the investigators are restricted to use the available variables and examine only a few constructs.

All the selected studies conceptualized social inequality as income inequality. Social inequality contains structured and recurrent patterns of unequal distributions of goods, wealth, opportunities, rewards, and punishments. It is argued in the literature that income inequality may not capture all dimensions in which social inequality can occur such as those canvassed widely in the health inequalities literature including gender, ethnicity, indigenous status, education and economic position/wealth (Costa-Font & Hernández-Quevedo, 2012; Krieger, 1999; Bartley, 2004). Economic inequality is one dimension in which social inequality may occur. In addition, using income inequality does not capture all aspects of economic inequality (Sen, 1992, 1997, 1999). The value of income is entirely as a means to realizing individual achievements and freedoms. Income is not the only means by which this is achieved. The other means

Table 3
Conceptual and measurement alternatives used to measure social inequality in the selected studies.

Study[a]	Type of social inequality	Area based quantitative measure of inequality	Categorization of inequality variable
A	National distribution of income	Percentage of national income	Percentage of national income earned by the top 20%
B	Income inequality	Gini Index	Continuous measure of Gini
C	Income inequality	Gini Index	Continuous measure of Gini
D	Income inequality	Gini Index	Continuous measure of Gini
E	Income inequality	(1) Gini Index (2) 20:20: Ratio of the total annual household income received by the richest 20% of the population to that received by the poorest 20%	Continuous measure of Gini
F	Income inequality	Gini Index	A change of 10 points in the Gini scale
G	Income inequality	Gini Index	Continuous measure of Gini
H	Income inequality	Gini Index	A change of 0.46 points in Gini: difference between the Gini value of the lowest and the highest Brazilian municipalities
I	Income inequality	Gini coefficient and the ratio between annual income of richest and poorest 20% of the population (20:20 ratio)	Continuous measure of Gini
J	Income inequality	Gini Index	0.1 point difference in Gini coefficient
K	Income inequality	Gini Index	Per 0.05 unit increase (or 5%) in the Gini coefficient
L	Income inequality	Gini Index	A change of 10 points in the Gini scale
M	Income inequality	Gini Index	Tertiles of distribution into low, moderate and high
N	Income inequality	Gini Index	Tertiles of distribution into low, moderate and high
O	Income inequality	Gini Index	Tertiles of distribution into low, moderate and high and then change in Gini over time by categorizing into (Stable, reduction, moderate increase and high increase)
P	Income inequality	Gini Index	Tertiles of distribution

[a] All studies assessed social status rather than social class, and examined inequalities on a stratificational or gradational scale.

include rights, liberties and opportunities and wealth, and the social bases of self-respect (Sen, 1997). Whether or not a particular level of income provides economic equality depends on a range of factors, including personal characteristics, environmental conditions, variations in social climate, differences in local commodity requirement and the distribution of income within a family. Further, while many studies of individual social position explore the impact of belonging to one group or another within these dimensions, ecological studies have examined social inequalities and population health primarily using the dimension of income inequality (Navarro, 2009). The use of income inequality to measure social inequality only captures one dimension of social inequality. As such it risks ignoring the underlying class relations, power dynamics and exploitation (Muntaner & Lynch, 1999), which are responsible for generating these income inequalities. Muntaner and Lynch (1999) further stated that a measure of class exploitation can be measured at any aggregate level and is more informative due to its explicit social mechanism.

The current review had several strengths and some limitations. This study scoped the area-level inequality oral health literature using a novel and robust methodology. The use of deductive content analysis using qualitative software for critical evaluation of the theoretical basis of empirical studies has not been published elsewhere. The search strategy of the current scoping review included a wide range of electronic databases as well as grey literature. A limitation includes that potentially relevant studies ($n=8$) (Appendix 1) could not be included in the review as they were not published in English. Furthermore, the scoping review assessed the use of theory in a specific research question which is the association between area level social inequalities and population oral health, and some of the evaluated social theories (behavioural, material and psychosocial) are also used to explain health inequalities within societies (Bartley, 2004). The individual level oral health inequalities literature was not assessed in our review for the use of theory and future studies may use the current methodology to address this question. Finally, the evaluated social epidemiological theories included those which were generalizable across societies which may lead to the lack of inclusion of context specific explanations.

4.1. Research implications and conclusions

The need for more robust empirical testing of pathways in the association between area-level social inequality and population oral health has emerged as one of the main research implications from this review. With the importance placed on the conceptualization and measurement of social inequality, an understanding of how other societal measures of inequality such as labour market inequality (Muntaner, Chung, Benach & Ng, 2012) and rate of exploitation (Muntaner et al., 2002) affect population oral health would complement the research on the income inequality hypothesis. Systematic reviews and meta-analysis should be conducted to summarize evidence on the inequality – oral health relationship. However, it is conceivable that meta-analysis may not be appropriate as different pathways operate to different extents in different contexts. Finally, outcome specific theoretical models would provide insight to potential interventions to reduce the public health burden of oral diseases associated with inequality. With growing income and social inequalities globally, this research is an important line of investigation to reduce the overall public health burden of oral diseases.

Acknowledgements

This work is supported by an International Postgraduate Research Scholarship and Australian Postgraduate Award, awarded to Ankur Singh for a doctoral degree at The University of Adelaide. HSS is supported by a scholarship award from the Brazilian government agency CAPES (HSS - process BEX 13774-13-1) for a doctoral degree. The authors of the manuscript would like to acknowledge Mr Michael Draper, Research Librarian at The University of Adelaide, for helping develop the search strategy for the selected databases and in identifying grey literature. The authors also acknowledge critical comments on the manuscript by Dr Catherine Chittleborough, School of Public Health, The University of Adelaide.

Appendix A. Supplementary material

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.ssmph.2016.06.001.

References

Aida, J., Kondo, K., Kondo, N., Watt, R. G., Sheiham, A., & Tsakos, G. (2011). Income inequality, social capital and self-rated health and dental status in older Japanese. *Social Science and Medicine, 73*, 1561–1568.

Arksey, H., & O'Malley, L. (2005). Scoping studies: Towards a methodological framework. *International Journal of Social Research Methodology, 8*, 19–32.

Baker, S. R., & Gibson, B. G. (2014). Social oral epidemi(olog)^{2}y where next: one small step or one giant leap? *Community Dentistry and Oral Epidemiology, 42*(6), 481–494.

Bartley, M. (2004). *Health inequality: An introduction to theories, concepts, and methods*. Cambridge, UK: Polity Press.

Bernabe, E., & Hobdell, M. H. (2010). Is income inequality related to childhood dental caries in rich countries? *Journal of the American Dental Association, 141*, 143–149.

Bernabe, E., & Marcenes, W. (2011). Income inequality and tooth loss in the United States. *Journal of Dental Research, 90*, 724–729.

Bernabe, E., Sheiham, A., & Sabbah, W. (2009). Income, income inequality, dental caries and dental care levels: an ecological study in rich countries. *Caries Research, 43*, 294–301.

Campbell, M., Egan, M., Lorenc, T., Bond, L., Popham, F., Fenton, C., et al. (2014). Considering methodological options for reviews of theory: illustrated by a review of theories linking income and health. *Systematic Reviews, 3*, 114.

Celeste, R. K., Fritzell, J., & Nadanovsky, P. (2011). The relationship between levels of income inequality and dental caries and periodontal diseases. *Cad Saude Publica, 27*, 1111–1120.

Celeste, R. K., & Nadanovsky, P. (2010). How much of the income inequality effect can be explained by public policy? Evidence from oral health in Brazil. *Health Policy, 97*, 250–258.

Celeste, R. K., Nadanovsky, P., Ponce de Leon, A., & Fritzell, J. (2009). The individual and contextual pathways between oral health and income inequality in Brazilian adolescents and adults. *Social Science and Medicine, 69*, 1468–1475.

Chalub, L. L., Martins, C. C., Ferreira, R. C., & Vargas, A. M. (2016). Functional dentition in Brazilian adults: An investigation of Social Determinants of Health (SDH) using a multilevel approach. *Plos One, 11*, e0148859.

Coburn, D. (2000). Income inequality, social cohesion and the health status of populations: the role of neo-liberalism. *Social Science and Medicine, 51*, 135–146.

Costa-Font, J., & Hernández-Quevedo, C. (2012). Measuring inequalities in health: What do we know? What do we need to know? *Health Policy, 106*, 195–206.

Davies, P., Walker, A. E., & Grimshaw, J. M. (2010). A systematic review of the use of theory in the design of guideline dissemination and implementation strategies and interpretation of the results of rigorous evaluations. *Implementation Science* (p. 5), 5.

Diez-Roux, A. V., Link, B. G., & Northridge, M. E. (2000). A multilevel analysis of income inequality and cardiovascular disease risk factors. *Social Science Medicine, 50*, 673–687.

Fleischer, N., & Diez-Roux, A. (2008). Using directed acyclic graphs to guide analyses of neighbourhood health effects: An introduction. *Journal of Epidemiology Community Health, 62*, 842–846.

Goulart, M. A., & Vettore, M. V. (2016). Is the relative increase in income inequality related to tooth loss in middle-aged adults? *Journal of Public Health Dentistry, 76*, 65–75.

Hobdell, M. H., Oliveira, E. R., Bautista, R., Myburgh, N. G., Lalloo, R., Narendran, S., et al. (2003). Oral diseases and socio-economic status (SES). *British Dental Journal, 194*, 91–96 (discussion 88).

Honjo, K. (2004). Social epidemiology: Definition, history, and research examples. *Environmental Health and Preventive Medicine, 9*, 193–199.

Islam, M. K., Gerdtham, U. G., Gullberg, B., Lindstrom, M., & Merlo, J. (2008). Social capital externalities and mortality in Sweden. *Economics and Human Biology, 6*, 19–42.

Kawachi, I., & Kennedy, B. P. (1999). Income inequality and health: Pathways and mechanisms. *Health Services Research, 34*, 215–227.

Kawachi, I., & Berkman, L. (2000). *Social epidemiology*. New York Oxford: Oxford University Press.

Kawachi, I., Kennedy, B. P., Lochner, K., & Prothrow-Stith, D. (1997). Social capital, income inequality, and mortality. *American Journal of Public Health, 87*, 1491–1498.

Kondo, N., Sembajwe, G., Kawachi, I., van Dam, R. M., Subramanian, S. V., & Yamagata, Z. (2009). Income inequality, mortality, and self rated health: meta-analysis of multilevel studies. *British Medical Journal, 339*, 1178–1181.

Krieger, N. (1999). *Embodying inequality: A review of concepts, measures, and methods for studying health consequences of discrimination* (pp. 295-352).

Krieger, N. (2011). *Epidemiology and the people's health: Theory and context*. Oxford University Press.

Krieger, N. (2014). Got Theory? On the 21st c. CE rise of explicit use of epidemiologic theories of disease distribution: A review and ecosocial analysis. *Current Epidemiology Reports, 1*, 45–56.

Levac, D., Colquhoun, H., & O'Brien, K. K. (2010). Scoping studies: Advancing the methodology. *Implementation Science* (p. 5), 5.

Listl, S., Galloway, J., Mossey, P. A., & Marcenes, W. (2015). Global economic impact of dental diseases. *Journal of Dental Research, 94*, 1355–1361.

Lynch, J., & Kaplan, G. A. (1997). Understanding how inequality in the distribution of income affects health. *Journal of Health Psychology, 2*, 297–314.

Lynch, J., Smith, G. D., Harper, S., Hillemeier, M., Ross, N., Kaplan, G. A., et al. (2004). Is income inequality a determinant of population health? Part 1. A systematic review. *Milbank Quarterly, 82*, 5–99.

Lynch, J. W., Smith, G. D., Kaplan, G. A., & House, J. S. (2000). Income inequality and mortality: Importance to health of individual income, psychosocial environment, or material conditions. *British Medical Journal, 320*, 1200–1204.

Macintyre, S. (1997). The black report and beyond what are the issues? *Social Science Medicine, 44*, 723–745.

Mackenbach, J. D., Lakerveld, J., van Lenthe, F. J., Kawachi, I., McKee, M., Rutter, H., et al. (2016). Neighbourhood social capital: measurement issues and associations with health outcomes. *Obesity Reviews, 17*(Suppl. 1), S96–S107.

Marcenes, W., Kassebaum, N. J., Flaxman, E., Naghavi, A., Lopez, M., Murray, A., et al. (2013). Global burden of oral conditions in 1990–2010: A systematic analysis. *Journal of Dental Research, 92*, 592–597.

Marmot, M., & Wilkinson, R. (2000). Psychosocial and material pathways in the relation between income and health: A response to Lynch et al. *British Medical Journal, 322*, 1233–1236.

Muntaner, C., Borrell, C., Vanroelen, C., Chung, H., Benach, J., Kim, I. H., et al. (2010). Employment relations, social class and health: A review and analysis of conceptual and measurement alternatives. *Social Science and Medicine, 71*, 2130–2140.

Muntaner, C., Chung, H., Benach, J., & Ng, E. (2012). Hierarchical cluster analysis of labour market regulations and population health: a taxonomy of low- and middle-income countries. *BMC Public Health, 12*, 286.

Muntaner, C., Lynch, J., M., H. Lee, H. J., David, R., Benach, J., et al. (2002). Economic inequality, working-class power, social capital, and cause-specific mortality in wealthy countries. *International Journal of Health Services, 32*, 629–656.

Muntaner, C., Lynch, J., & Oates, G. L. (1999). The social class determinants of income inequality and social cohesion. *International Journal of Health Services: Planning, Administration, Evaluation, 29*, 699.

Muntaner, C., & Lynch, J. (1999). Income inequality, social cohesion, and class relations: A critique of Wilkinson's Neo-Durkheimian research programme. *International Journal of Health Services, 29*, 59–81.

Murayama, H., Fujiwara, Y., & Kawachi, I. (2012). Social capital and health: A review of prospective multilevel studies. *Journal of Epidemiology, 22*, 179–187.

Nadanovsky, P., & Sheiham, A. (1995). Relative contribution of dental services to the changes in caries levels of 12-year-old children in 18 industrialized countries in the 1970s and early 1980s. *Community Dentistry and Oral Epidemiology, 23*, 331–339.

Navarro, V. (2002). A critique of social capital. *International Journal of Health Services, 32*, 423–432.

Navarro, V. (2009). What we mean by social determinants of health. *International Journal of Health Services, 39*, 423–441.

Oksanen, T., Kouvonen, A., Kivimäki, M., Pentti, J., Virtanen, M., Linna, A., et al. (2008). Social capital at work as a predictor of employee health: Multilevel evidence from work units in Finland. *Social Science and Medicine, 66*, 637–649.

Pattussi, M. P., Marcenes, W., Croucher, R., & Sheiham, A. (2001). Social deprivation, income inequality, social cohesion and dental caries in Brazilian school children. *Social Science and Medicine, 53*, 915–925.

Peres, M. A., Peres, K. G., Antunes, J. L., Junqueira, S. R., Frazao, P., & Narvai, P. C. (2003). The association between socioeconomic development at the town level and the distribution of dental caries in Brazilian children. *Revista Panamericana de Salud Publica, 14*, 149–157.

Pickett, K. E., & Wilkinson, R. G. (2015a). Income inequality and health: A causal review. *Social Science and Medicine, 128*, 316–326.

Pickett, K. E., & Wilkinson, R. G. (2015b). Recalibrating Rambotti: Disentangling concepts of poverty and inequality. *Social Science and Medicine, 139*, 132–134.

Riva, M., Gauvin, L., & Barnett, T. A. (2007). Toward the next generation of research into small area effects on health: A synthesis of multilevel investigations published since July 1998. *Journal of Epidemiology and Community Health, 61*, 853–861.

Robert, D. P. (2001). Social capital: Measurement and consequences. *Isuma: Canadian Journal of Policy Research* (p. 2), 2.

Rodgers, G. B. (1979). Income and inequality as determinants of mortality: An international cross-section analysis. *Population Studies, 33*, 343–351.

Rose, G. (1992). *The strategy of preventive medicine*. Oxford: Oxford University Press.

Sabbah, W., Sheiham, A., & Bernabe, E. (2010). Income inequality and periodontal diseases in rich countries: An ecological cross-sectional study. *International Dental Journal, 60*, 370–374.

Sen, A. (1992). *Inequality Reexamined*. Oxford: Oxford University Press.

Sen, A. (1997). From income inequality to economic inequality. *Southern Economic Journal, 64*, 384–401.

Sen, A. (1999). *Development as freedom*. New York: Knopf.

Singh, A., Harford, J., Watt, R. G., & Peres, M. A. (2015). The role of theories in explaining the association between social inequalities and population oral health: A scoping review protocol. *JBI Database of Systematic Reviews Implementation Reports, 13*, 11.

Subramanian, S. V., & Kawachi, I. (2004). Income inequality and health: What have we learned so far? *Epidemiologic Reviews, 26*, 78–91.

Subramanian, S. V., & Kawachi, I. (2006). Whose health is affected by income inequality? A multilevel interaction analysis of contemporaneous and lagged effects of state income inequality on individual self-rated health in the United States. *Health Place, 12*, 141–156.

Townsend, P., Davidson, N., & Black, D. S. (1982). *Inequalities in health: The Black report*. Harmondsworth: Penguin.

Vettore, M. V., Marques, R. A. D., & Peres, M. A. (2013). Social inequalities and periodontal disease: Multilevel approach in SBBrasil 2010 survey. *Revista de Saude Publica, 47*, 29–39.

Vettore, M. V., & Aqeeli, A. (2015). The roles of contextual and individual social determinants of oral health-related quality of life in Brazilian adults. *Quality of Life Research*. , http://dx.doi.org/10.1007/s11136-015-1118-0.
Wagstaff, A., & Doorslaer, E. V. (2000). Income inequality and health: What does the literature tell us? *Annual Review of Public Health*, *21*, 543–567.
Watt, R. G. (2012). Social determinants of oral health inequalities: Implications for action. *Community Dentistry and Oral Epidemiology*, *40*(Suppl. 2), S44–S48.
Wilkinson, R. G. (1997). Socioeconomic determinants of health. Health inequalities: Relative or absolute material standards? *British Medical Journal*, *314*, 591–595.
Wilkinson, R., & Pickett, K. E. (2006). Income inequality and population health: A review and explanation of the evidence. *Social Science and Medicine*, *62*, 1768–1784.