Journal of Dental Research
2019, Vol. 98(10) 1096–1102

DOI: 10.1177/0022034519861037
journals.sagepub.com/home/jdr

Construction of a Biological Age Score to Predict Tooth Loss over 10 Years

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Abstract

The aim of the present study was to construct a biological age score reflecting one's physiologic capability and aging condition with respect to tooth loss over 10 y. From the follow-up to the population-based Study of Health in Pomerania (i.e., SHIP-2), 2,049 participants were studied for their baseline biomarker measures 10 y before (i.e., in SHIP-0). Metabolic and periodontal data were regressed onto chronological age to construct a score designated as "biological age." For either sex separately, the impact of this individualized score was used to predict tooth loss in the follow-up cohort in comparison with each participant's chronological age. Outcome data after 10 y with respect to tooth loss, periodontitis, obesity, and inflammation were shown to be better for biologically younger subjects than as expected by their chronological age, whereas for the older subjects, data were worse. Especially for tooth loss, a striking increase was observed in subjects whose biological age at baseline appeared to be higher than their chronological age. Biological age produced significantly better tooth loss predictions than chronological age ($P < 0.001$). Areas under receiver operating characteristic curves for tooth loss of ≥3 teeth in men during follow-up were 0.811 and 0.745 for biological and chronological age, respectively. For women, these figures were 0.788 and 0.724. For total tooth loss, areas under the curve were 0.890 and 0.749 in men and 0.872 and 0.752 in women. Biological age combines various measures into a single score and allows identifying individuals at increased risk of tooth loss.

Keywords: biomarkers, periodontitis, biological aging, edentulism, sex differences, prognostic factors

Introduction

Societies are currently facing a demographic change. As the population ages, the burden of periodontal disease and tooth loss is rising. Studies on the natural history of periodontitis performed in Sri Lankan tea plantation workers between 1970 and 1985 provided basic information on oral conditions unaffected by any prophylaxis or therapy over a period of 15 y (Löe et al. 1986). Approximately 10% of the study population showed rapid progression of periodontal disease and another 10%, no progression at all; for the remaining majority, moderate disease progression was identified. This observation was a first glimpse of a new view on the natural history of periodontitis as it became clear that susceptibility to the disease and resilience against it are very differently distributed among individuals.

Such individual characteristics of disease predisposition are a matter of life course epidemiology seeking to understand the development of chronic diseases. Individual characteristics developed in early life periods and effects of environmental exposures appear to affect biological functions and disease risks. A pathophysiologic model was suggested linking infectious exposure at earlier ages and environmental factors to inflammation, height, morbidity, and mortality at older ages (Crimmins and Finch 2006). Different studies support the assumption that oral health is continuously exposed to environmental and behavioral risks that lead to accumulated diseases in the dental tissues (Meisel et al. 2007; Correa et al. 2010; Holst and Schuller 2012). Exposure to both beneficial and adverse circumstances over the life course will vary for each individual and constitute a unique life exposure trajectory, which will manifest as different expressions of health, well-being, behavior, and learning skills. Tooth survival shows considerable variation among individuals of similar age due to the diversity in genetic makeup, life course fate, living habits, and environments.

From all these peculiarities, it became clear that people of identical age will express different disease characteristics. Substantial variability in clinical attachment loss rate and tooth loss exists among populations as well as among individuals

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A supplemental appendix to this article is available online.

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within a population (Brignardello-Petersen 2018; Needleman et al. 2018).

Thus, some may appear to be younger than their true (i.e., chronological) age with respect to their oral health state; others are more diseased than what would be expected at their age. From such considerations, a concept of "biological age" was developed. Though there is no simple index for aging, except chronological age by itself, researchers have developed statistical methods to construct different models for estimating biological age, which is thought to measure an individual's sum of positive and negative influences accumulating in a personal disease susceptibility (Levine 2013; Jia et al. 2017). The relative contributions of factors operating in fetal life, childhood, and adulthood to the risk of disease in middle age become more and more important during the life course or with increasing age. Though an important research issue, an individual's oral health in terms of biological age was rarely considered.

With increasing age, humans experience many constraints, such as different diseases and also tooth loss. In the oldest, tooth loss may finally result in edentulism. Besides caries, periodontitis is a major cause of tooth loss. Periodontitis is an inflammatory disease of the gums associated with systemic responses. Elevated blood levels of markers of inflammation, including C-reactive protein and fibrinogen, are observed in periodontitis, indicating systemic reactions to the local inflammation within the oral cavity (Linden et al. 2008). Elevated concentrations of these inflammatory markers are also associated with different systemic conditions, such as metabolic syndrome (López et al. 2012). Metabolic syndrome refers to the concomitant occurrence of cardiovascular risk factors, comprising dyslipidemia, elevated blood pressure, obesity, disturbed glucose homeostasis, and a proinflammatory state; associations with periodontitis are obvious (Lamster and Pagan 2017).

This study aimed at evaluating the impact of an individual age score—the biological age—on tooth loss over a 10-y period. Biological age was constructed from a panel of biomarkers of the population-based study SHIP-0 (Study of Health in Pomerania) at baseline, and it was compared with chronological age in predicting tooth loss during the 10 y up to the follow-up study SHIP-2. To construct the score, we used indicators of metabolic syndrome and periodontitis.

Materials and Methods

Study Population

SHIP is a population-based prospective cohort study in the northeast region of Germany. Extensive protocols on the SHIP study design and recruitment have been published elsewhere (John et al. 2001; Hensel et al. 2003). Of 6,265 eligible subjects, 4,308 participated in the baseline examinations from 1997 to 2001 (SHIP-0), and of these, 2,333 subjects participated in the 11-y follow-up examinations executed from 2008 to 2012 (SHIP-2). All participants gave written informed consent, and the study protocol was approved a priori by the local Ethics Committee. The STROBE guidelines were followed in the reporting of this observational study. We excluded all participants who were edentulous at baseline, had missing data at baseline, and were no longer participating in follow-up after 11 y. Thus, finally 2,049 subjects were included: 974 male and 1,075 female.

Paraclinical and Periodontal Assessments

For obesity-related factors, measurements were taken under standardized conditions. Body weight was measured to the nearest 0.1 kg on a decimal scale (S20; Soehnle), height to the nearest 1 cm, and waist and hip circumferences to the nearest 0.5 cm. Waist girth was measured at the midpoint between the lower ribs and the iliac crest. Hip circumference was measured horizontally at the level of the largest lateral extension of the hips or over the buttocks. For laboratory measurements, nonfasting blood samples were drawn from the cubital vein in the supine position. Fibrinogen concentrations were assayed according to Clauss (Electra 1600; Instrumentation Laboratory). HbA1c was measured via high-performance liquid chromatography (ClinRep HbA1c; Recipe Chemicals + Instruments GmbH). Total cholesterol, low-density lipoprotein (LDL), and high-density lipoprotein were measured with a Hitachi 704 analyzer (Roche). Clinical attachment level (CAL) was assessed at 4 sites per tooth (distobuccal, mesiobuccal, midbuccal, midlingual/midpalatal) with a periodontal probe (PCP11; Hu-Friedy). CAL represents the distance from the cementoenamel junction to the bottom of the periodontal pocket. CAL was not measured if determining the cementoenamel junction was vague (e.g., wedge-shaped defects, fillings, or crown margins). Resting systolic and diastolic blood pressure was measured 3 times at the right arm of seated participants with a digital BP monitor (HEM-705CP; Omron Corporation), with each reading being followed by a rest period of 3 min. The mean of the second and third measurements was calculated and used for the calculation of pulse pressure. Hand grip strength was measured by a handheld Smedley-type dynamometer used for diagnostic purposes (Scandidact) and indicated in kilograms. Body fat mass was assessed by bioelectrical impedance analysis.

Tooth Loss

The number of natural teeth present was determined, excluding third molars. Tooth loss was assessed as the difference between the number of teeth at baseline minus that at follow-up. Moreover, dichotomous tooth loss was defined as any tooth loss versus no tooth loss (yes/no). Additionally, tooth loss was stratified into 3 dichotomous categories: no tooth loss at all, 1 to 2 teeth lost, and >2 teeth lost during the follow-up period.

Statistical Analyses

Means and standard deviations were computed for continuous variables, whereas frequency distributions were assessed for

Table 1. Baseline Characteristics of the Participants with a Complete Data Set (N = 2,049).

	Male (n = 974)	Female (n = 1,075)	P Value
Age, y	46.2 ± 13.4	44.7 ± 13.2	0.016
Cholesterol, mmol/L			
HDL	1.3 ± 0.3	1.6 ± 0.4	<0.001
LDL	3.7 ± 1.2	3.4 ± 1.2	<0.001
Triglycerides, mmol/L	2.1 ± 1.4	1.4 ± 0.8	<0.001
Waist circumference, cm	94.4 ± 11.1	80.5 ± 12.0	<0.001
Height, cm	176.8 ± 6.6	164.1 ± 6.7	<0.001
BMI, kg/m²	27.5 ± 3.8	26.1 ± 4.9	<0.001
Subjects with obesity, n (%)	341 (35.0)	317 (29.5)	0.009
Pressure, mm Hg			
Pulse	53.6 ± 11.8	45.5 ± 12.3	<0.001
Arterial	104.3 ± 12.6	95.7 ± 12.4	<0.001
Subjects with HbA1c ≥6.5%, n (%)	56 (5.7)	26 (2.4)	<0.001
HbA1c, %	5.4 ± 0.9	5.2 ± 0.6	<0.001
CAL, mm	2.6 ± 1.8	2.1 ± 1.5	<0.001
PPD, mm	2.5 ± 0.7	2.3 ± 0.6	<0.001
No. of teeth, edentulous excluded	22.1 ± 6.0	21.9 ± 6.0	0.17
CRP, mg/L	2.1 ± 4.5	2.7 ± 4.1	<0.001
Fibrinogen, g/L	2.8 ± 0.6	2.9 ± 0.6	<0.001
Education, <10th grade, n (%)	261 (26.8)	238 (22.1)	0.013
Pure alcohol, mL/wk	153 ± 165	40 ± 58	<0.001
Pack-years smoked	10.2 ± 15.2	3.7 ± 7.6	<0.001

Values presented as mean ± SD unless otherwise noted. BMI, body mass index; CAL, clinical attachment level; CRP, C-reactive protein; HDL, high-density lipoprotein; LDL, low-density lipoprotein; PPD, probing pocket depth.

categorical variables. Multiple linear regressions were used to construct biological age, separated for either sex. After a correlation analysis, correlation coefficients were kept with ≤0.7 and >0.2 between predictors and age for at least 1 sex. Chronological age was regressed against the selected biomarkers HbA1c, LDL, triglycerides, fibrinogen, waist circumference, height, pulse pressure, and CAL via multiple linear regression. Finally, the predicted age figure was corrected by adding a z score defined as $(CA - mCA) \times (1 - b)$, where CA is the subject's chronological age, mCA the mean of age, and b is the regression coefficient of predicted age on chronological age. This procedure results in a slope of 1 and intercept of 0 between biological and chronological age. Methodological details of model building are given in these references (Nakamura et al. 1989; Jia et al. 2017; Kang et al. 2017). Outcome calculations were made comparing individuals whose estimated biological age was less than, equal to, or greater than their self-reported chronological age. Receiver operating characteristic (ROC) analyses were performed to test equality of areas under the curve (AUCs) for tooth loss. Two-sided P values <0.05 were considered statistically significant. All analyses were performed with STATA/SE 14.0 (StataCorp).

Results

Table 1 presents the typical sex differences in body shape, clinical chemistry, blood pressure, periodontal measures, and markers of inflammation as observed in this population at baseline. As expected, women in the general population showed fewer risks of systemic diseases as well as better periodontal health than men in most of the measured variables. Also, behavioral factors, such as smoking or education, and HbA1c indicating glucose homoeostasis were in favor of women. No difference was noticed in the number of teeth present between men and women. From this list, biomarkers were selected and regressed on age to construct a score representative for biological age.

Table 2 displays the resulting regressions separately for men and women. This was necessary due to the profound differences between the sexes with which the variables are related to age. Such differences are especially remarkable in HbA1c, LDL, triglycerides, and fibrinogen. As shown by the mean variance inflation factors, multicollinearity was largely avoided. Pulse pressure was included to have both systolic and diastolic blood pressure included but avoiding their collinearity. Thereafter, the correction was applied as described in the Materials and Methods section. Finally, we verified the correspondence of the so-constructed biological age with chronological age. This is shown in the Figure with the distribution of the differences calculated as biological age minus chronological age. It follows that a minus sign of this difference represents individuals who appeared to be younger than their true age (i.e., chronological age). Concurrently, a positive difference represents those appearing older than their chronological age.

The age differences between biological and chronological age were categorized into tertiles, as indicated in the Figure (lower panel). These tertiles comprise the younger, matching, and older subjects in comparison with their chronological age.

Table 2. Multiple Regressions on Chronological Age by Sex: All Independent Variables Assessed at Baseline.

	Male (n = 974)	P Value	Female (n = 1,075)	P Value
HbA1c, %	0.22 (–0.53 to 0.96)	0.57	2.10 (1.12 to 3.07)	<0.001
LDL cholesterol, mmol/L	0.63 (0.09 to 1.18)	0.022	1.71 (1.19 to 2.23)	<0.001
Triglycerides, mmol/L	−0.72 (–1.18 to –0.26)	0.002	−0.05 (–0.82 to 0.72)	0.90
Fibrinogen, g/L	1.27 (0.27 to 2.26)	0.013	–0.65 (–1.58 to 0.27)	0.17
Waist circumference, cm	0.30 (0.24 to 0.37)	<0.001	0.14 (0.08 to 0.20)	<0.001
Height, cm	–0.52 (–0.62 to –0.43)	<0.001	–0.31 (–0.39 to –0.22)	<0.001
Mean CAL, mm	3.11 (2.73 to 3.49)	<0.001	3.51 (3.10 to 3.93)	<0.001
Pulse pressure, mm Hg	0.12 (0.07 to 0.17)	<0.001	0.25 (0.21 to 0.30)	<0.001
Variability explained, R^2	0.488		0.545	
Mean variance inflation factor	1.18		1.31	

Values are presented as β coefficients with 95% CI.
CAL, clinical attachment level; LDL, low-density lipoprotein.

Outcome data after 10 y with respect to tooth loss, periodontitis, obesity, and inflammation are shown in Table 3 for men and Table 4 for women. Hand grip strength was included as a proxy for the general health status. In all the measures estimated, the outcome for the younger cohort was better than that for the matching subjects, and for the older, it was worst. Especially for tooth loss, a striking increase was observed in subjects whose biological age at baseline appeared to be higher than their chronological age. This was true for men and women likewise, though it appeared different between the sexes. Frequency of risk factors, such as smoking, poor education, or obesity, clusters in subjects older than their chronological age. For a more refined subdivision into quintiles of the age difference between biological and chronological age, see Appendix Tables 1 and 2.

ROC curves were used to determine whether biological age might be a better predictor of tooth loss than the mere chronological age. Appendix Table 3 displays the resulting AUCs in comparison of both age categories: biological and chronological (all differences, $P < 0.001$). Biological age performs better in each case in men and in women as well. The most striking differences in AUCs were observed with respect to higher tooth loss of >2 teeth: in men, the AUC was 0.75 versus 0.81 for chronological age and biological age, respectively; in women, 0.74 and 0.79. The figures with respect to total tooth loss were 0.75 versus 0.89 in men and 0.75 versus 0.87 in women. For illustration, the comparative ROC curves for edentulism are shown in the Appendix Figure.

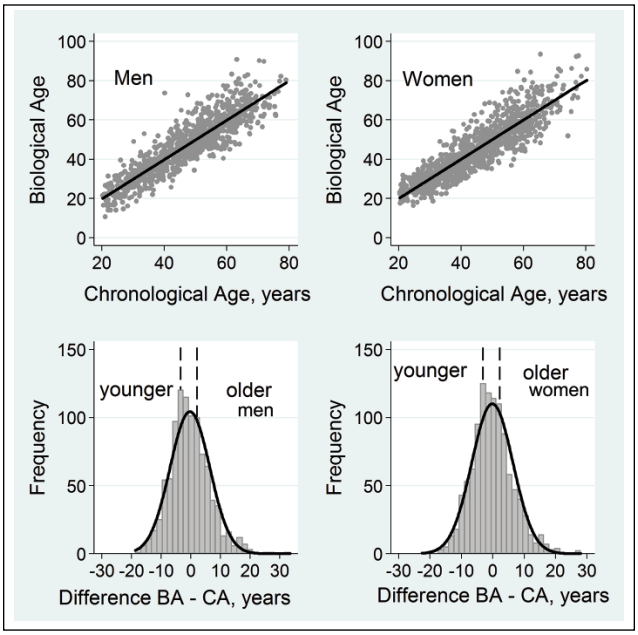

Figure. Regression of biological age on chronological age in men and women, with each individual's spot and regression lines (upper panel). Frequency histograms of differences, biological age (BA) minus chronological age (CA); dashed lines separate the tertiles of these differences (lower panel).

Discussion

The prevalence of periodontitis and tooth loss rises with age. However, there is much debate about the question whether age by itself is a risk factor of tooth loss. Many of the risk factors associated with periodontitis and tooth loss are shared with systemic diseases that are highly prevalent with increasing age (Persson 2018). In this study, we related a set of such factors as biomarkers to age in an attempt to construct an individual age score designated as biological age. Such a labeling may be unsatisfactory in view of the restricted number of predictor variables included. Nevertheless, biological age is considered a mean to evaluate an individual's physiologic capability and aging condition as compared with others of the same age group (Borkan and Norris 1980). As shown in the Results section, there are significant differences in outcome after 10 y of follow-up depending on an individual's biological age in relation to the chronological age. Tooth loss and edentulism were more accurately predicted by biological age than by chronological age. Many retained teeth may be an indicator of positive oral health behavior throughout the life course (Holm-Pedersen et al. 2007).

The individual parameters employed here to construct the biological age model have all features as biomarkers of periodontitis (Table 2). Age thresholds for periodontitis risks have recently been published with risk caused by diabetes, hypertension, obesity, and associated symptoms (Han and Park 2017). Data from the National Health and Nutrition Examination Survey revealed a significant association between the number

Table 3. Parameters Assessed after 10 y in Men by Biological Age Tertiles Obtained from Baseline.

	Men, Mean ± SD or *n* (%)		
	Younger (*n* = 325)	Matching (*n* = 325)	Older (*n* = 324)
Chronological age, baseline	47.8 ± 13.6	43.7 ± 13.6	47.1 ± 12.6
Biological age, baseline	40.7 ± 13.3	42.9 ± 13.6	54.1 ± 14.4
Actual age, 10-y follow-up	58.4 ± 13.3	54.3 ± 13.5	57.6 ± 12.5
No. of teeth	22.3 ± 5.5	22.0 ± 6.2	16.4 ± 9.3
No. of teeth lost	1.1 ± 2.0	1.2 ± 2.1	3.5 ± 4.3
Some teeth lost, yes	151 (46.5)	136 (41.8)	234 (72.2)
>2 teeth lost	45 (13.8)	56 (17.2)	139 (42.9)
No. of edentulous	2 (0.6)	2 (0.6)	27 (8.3)
PPD, mm	2.5 ± 0.4	2.6 ± 0.5	3.0 ± 0.7
CAL, mm	2.5 ± 1.1	2.8 ± 1.5	4.1 ± 1.9
BMI, kg/m^2	26.9 ± 3.1	28.5 ± 3.6	30.8 ± 4.4
Obesity, BMI ≥30 kg/m²	57 (17.5)	104 (32.0)	180 (55.7)
Body fat mass, %	22.5 ± 5.0	24.3 ± 4.7	25.9 ± 5.3
Hand grip strength, kg	47.1 ± 9.3	47.4 ± 10.1	44.4 ± 8.3
No. of current smokers	38 (11.7)	66 (20.3)	73 (22.5)
Poor education, <10th grade	73 (22.5)	74 (22.8)	114 (35.2)
Fibrinogen, g/L	2.8 ± 0.7	3.0 ± 0.7	3.2 ± 0.8
CRP, mg/L	1.4 ± 1.5	1.6 ± 1.6	2.1 ± 2.0

Comparisons between age groups for all listed figures, $P < 0.001$.
BMI, body mass index; CAL, clinical attachment level; CRP, C-reactive protein; PPD, probing pocket depth.

Table 4. Parameters Assessed after 10 y in Women by Biological Age Tertiles Obtained from Baseline.

	Women, Mean ± SD or *n* (%)		
	Younger (*n* = 359)	Matching (*n* = 358)	Older (*n* = 358)
Chronological age, baseline	46.4 ± 12.1	42.0 ± 13.1	45.8 ± 14.0
Biological age, baseline	39.6 ± 11.9	41.5 ± 13.0	52.8 ± 15.7
Actual age, 10-y follow-up	57.0 ± 11.9	52.6 ± 12.9	56.4 ± 13.9
No. of teeth	22.3 ± 5.4	21.5 ± 6.1	17.3 ± 8.5
No. of teeth lost	0.8 ± 1.4	1.2 ± 2.4	2.3 ± 3.1
Some teeth lost, yes	145 (40.4)	164 (45.8)	228 (63.7)
>2 teeth lost	45 (12.5)	55 (15.4)	122 (34.1)
No. of edentulous	0 (0)	3 (0.8)	16 (4.5)
PPD, mm	2.4 ± 0.4	2.5 ± 0.4	2.8 ± 0.8
CAL, mm	2.3 ± 1.1	2.4 ± 1.3	3.4 ± 1.8
BMI, kg/m^2	25.8 ± 4.2	27.4 ± 5.0	29.9 ± 5.9
Obesity, BMI ≥30 kg/m²	52 (14.5)	99 (27.7)	166 (46.6)
Body fat mass, %	32.1 ± 5.8	33.8 ± 6.6	36.3 ± 6.7
Hand grip strength, kg	27.4 ± 6.5	28.1 ± 5.9	26.6 ± 6.0
No. of current smokers	52 (14.5)	67 (18.7)	75 (21.0)
Poor education, <10th grade	69 (19.2)	61 (17.0)	108 (30.2)
Fibrinogen, g/L	3.0 ± 0.7	3.2 ± 0.7	3.4 ± 0.8
CRP, mg/L	1.6 ± 1.6	1.9 ± 1.7	2.5 ± 2.2

Comparisons between age groups for all listed figures, $P < 0.001$.
BMI, body mass index; CAL, clinical attachment level; CRP, C-reactive protein; PPD, probing pocket depth.

of teeth and the parameters of metabolic syndrome (Zhu and Hollis 2015). HbA1c is associated with periodontitis and indicates glycemic control (Kocher et al. 2018). Markers of dyslipidemia, with LDL and triglycerides included here, are highly correlated with obesity and periodontitis (Lee et al. 2013; Cury et al. 2018). Markers of inflammation, such as C-reactive protein, fibrinogen, or white blood cell counts, were considered links between systemic inflammatory diseases and inflammation in periodontitis and also for tooth loss (Meisel et al. 2007; Pink et al. 2015). Predisposition to inflammatory diseases might be associated with impaired length growth and impose a lifelong inflammatory burden also reflected by severity of periodontitis (Meisel et al. 2007; Shim and Han 2018). Thus, height was also included in the regression analysis on chronological age. Indicators of obesity, such as body mass index, waist circumference, or visceral fat masses, are also related to age, and numerous studies are engaged in evaluating the association with periodontitis (Kangas et al. 2017; Cury

et al. 2018). High blood pressure, a risk factor for cardiovascular diseases, is included in the symptoms cluster of metabolic syndrome. It is also related to age and shows association with periodontitis (Ollikainen et al. 2014). CAL by itself accumulates with the aging process, thereby summing up all the positive and negative influences on inflammatory episodes during a lifetime (Needleman et al. 2018). Models of biological age to facilitate individualization of metabolic syndrome were published elsewhere (Kang et al. 2017).

The construction of an aging score has to account for the majority of changes that occur with age. As different organs may age at different rates, the selection of biomarkers is crucial and may vary with the outcome of interest. Here, we selected those that are correlated to age and periodontitis or tooth loss as well. Numerous such aging biomarkers were employed in the past, mostly selected for the outcome under study or for the easy application in clinical practice (Jia et al. 2017). Essential preconditions for any biological age are that it is better predictive than chronological age, is reproducible, and shows significant differences among individuals of identical chronological age. Genetic and/or molecular indicators will be more important in the future (Hanson et al. 2016). To our knowledge, no attempts have been made up to now to find a reflection of one's age in relation to calendric age with respect to tooth loss. Nevertheless, dental indicators were used to determine the biological age of the living or for determining the age of the dead (Sengupta et al. 1999; Jankauskas et al. 2001).

This study included only those subjects who showed up at follow-up after 10 y, but it used their measures at baseline. Thus, this was a selection, and the criterion of a general population was biased. Moreover, to assess tooth loss, all subjects being edentulous at baseline were excluded. This population employed in the SHIP-0 may have genetic, environmental, and socioeconomic characteristics not comparable to other populations and therefore may not be applicable to other populations. A major limitation of this study is the rather arbitrary selection of parameters related to age. Further studies should focus on the most reliable and predictive factors. In view of the studies reporting tooth loss as a predictor of mortality, applying biological age for tooth loss may offer a tool to disentangle this relationship (Vedin et al. 2017).

In conclusion, the positive and negative summing up of risk effects in a person at a particular time point offers a chance to predict his or her future trajectory (Levine 2013; Kang et al. 2017). A score for biological age offers the possibility to assess the dental state, especially future tooth loss, on an individual basis long before loss of a tooth becomes unpreventable. This or similar scores may serve for improved communication with patients, as it offers a comparison of the state of the individual dental health with others of identical chronological age and sex.

Author Contributions

P. Meisel, contributed to conception and data analysis, drafted and critically revised the manuscript; C. Pink, contributed to design and data analysis, critically revised the manuscript; M. Nauck, contributed to data analysis, critically revised the manuscript; H. Völzke, contributed to design and data acquisition, critically revised the manuscript; T. Kocher, contributed to conception and data interpretation, critically revised the manuscript. All authors gave final approval and agree to be accountable for all aspects of the work.

Acknowledgments

The authors acknowledge the participation of the study subjects of the Study of Health in Pomerania. The Study of Health in Pomerania is part of the Community Medicine Research network of the University Medicine Greifswald, Germany, which is supported by the Federal State of Mecklenburg–West Pomerania. The authors declare no potential conflicts of interest with respect to the authorship and/or publication of this article.

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