

Oral Health Analytics in a ClinicalSpark Demonstration

1 **Title: Oral Function and Cardiometabolic Outcomes in Linked Health Records: A ClinicalSpark**
2 **Demonstration**

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5 **ABSTRACT**

6 Oral function has been associated with cardiometabolic health and mortality, but observational
7 associations do not establish the benefit of prosthetic treatment. This manuscript presents the existing
8 ClinicalSpark Studio demonstration of linked health-checkup, registry, medical-claims and dental-
9 claims analyses. The supplied summaries described 3,420,777 individuals and analyses of chewing
10 difficulty, prosthetic treatment, tooth-loss location and remaining teeth. Patient-level records,
11 executable analyses and validation evidence were unavailable. Reported outputs included a propensity-
12 matched mortality hazard ratio of 1.412 (95% confidence interval, 1.385–1.440), a prosthetic-treatment
13 hazard ratio of 0.428 (0.368–0.498), and five-year survival estimates of 97.4%, 94.1% and 84.2%
14 across decreasing tooth-count categories. A reported cardiovascular prediction model had an area
15 under the receiver operating characteristic curve of 0.842 (0.836–0.848). A mediation summary
16 attributed 31.4% of the reported effect to glycated hemoglobin and systolic blood pressure, although
17 its causal assumptions and effect scale were unspecified. Repeated subgroup counts, unclear
18 comparators, inconsistent balance summaries and missing follow-up and validation details limit
19 interpretation. These demonstration outputs illustrate an analytical framework but cannot establish
20 clinical risk estimates, causal pathways, predictive utility or a mortality benefit from prosthetic
21 rehabilitation.

22 **Keywords:** Health record linkage; Mastication; Tooth loss; Survival analysis; Clinical prediction

1.INTRODUCTION¹

Mastication is the process by which food is mechanically processed before swallowing. Its assessment can capture aspects of oral function that are not represented by tooth count alone. Self-reported chewing difficulty, measured masticatory performance, loss of occlusal support and prosthesis use describe related but distinct exposures. In the Shimane CoHRE study, lower masticatory performance and denture non-use in some tooth-loss groups were associated with hypertension in older adults, although the cross-sectional design limited conclusions about temporal direction. ¹

Longitudinal and cross-sectional studies also support examining oral function alongside metabolic health. The Suita study associated lower masticatory performance with subsequent metabolic syndrome in men. ² The Aichi Workers' Cohort Study reported a cross-sectional relationship between higher fasting blood glucose and chewing difficulty. ³ These observations motivate further investigation, but they do not establish whether impaired chewing precedes metabolic deterioration, reflects its consequences, or shares upstream causes with it. Differences in exposure measurement and study populations also constrain direct comparison between estimates.

Diet is one proposed link between oral function and systemic health. Marito and colleagues evaluated dietary intake, oral conditions and blood pressure in community-dwelling older adults and found associations involving posterior occlusal support and hypertension. Their observational results provide a rationale for examining dietary pathways, without establishing a causal mediation effect. ⁴ Separately, a Singapore cohort related perceived masticatory function to all-cause mortality and applied a doubly robust

¹**Abbreviations:** AUC, area under the curve; BMI, body mass index; CI, confidence interval; CVD, cardiovascular disease; GBDT, gradient boosted decision trees; HbA1c, glycated hemoglobin; HR, hazard ratio; OR, odds ratio; PSM, propensity score matching; RR, risk ratio; SBP, systolic blood pressure; SD, standard deviation; SMD, standardized mean difference.

causal-inference approach. That analysis concerned hypothetical preservation of perceived function and did not test the effect of dentures, bridges or implants.⁵

Linked health records could support complementary questions about oral exposures, disease occurrence, survival and prediction. Their interpretation depends on the validity of person-level linkage, clinical coding, exposure timing and follow-up. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement and its extension for routinely collected health data, RECORD, provide relevant reporting frameworks.^{6,7} Prediction requires an additional account of model development and evaluation, as described in TRIPOD+AI.⁸ A large number of source records cannot substitute for these design and reporting requirements.

In this manuscript, we present the existing ClinicalSpark Studio demonstration of oral-health analyses in a stated cohort of 3,420,777 individuals. We describe the supplied data model, summarize the reported descriptive, survival, matching, mediation and prediction outputs, and delimit the inferences those summaries can support. The work is a demonstration manuscript rather than independently validated clinical research. Its purpose is to provide a coherent account of the available analytical material while distinguishing reported outputs from verified observations and hypotheses from established effects.

2.EXPERIMENTAL PROCEDURES

2.1.Demonstration design and evidence provenance

The material consisted of a supplied manuscript containing aggregate tables and analysis descriptions attributed to ClinicalSpark Studio using Apache Spark. The present revision involved documentary synthesis and consistency checks; no patient-level records were

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accessed, no linkage was performed, and no clinical model was rerun. The origin of the demonstration values, including whether they were derived from real, simulated or otherwise illustrative records, was not established by the supplied material. Accordingly, all cohort sizes and effect estimates below are reported demonstration values. Institutional ethics review, consent or waiver, data-use authorization and protocol registration were not documented. The absence of those records does not establish exemption or approval.

2.2.Source tables and stated cohort

The described source tables contained 5,305,400 health-checkup records (exam_interview_processed), 13,114,600 registry records (tekiyo), 2,257,364,242 diagnosis records (receipt_diseases), 498,777,327 dental-claim items (receipt_dental_practice) and 1,421,861,940 tooth-level disease records (receipt_tooth_type_diseases). The stated final cohort comprised 3,420,777 individuals. Source rows and unique individuals are different units; these totals do not establish a participant flow. Geographic coverage, recruitment dates, eligibility criteria, linkage keys, linkage success, duplicate resolution and selection of a baseline examination were unspecified. No exclusion counts can therefore be inferred by subtracting source-row totals.

2.3.Oral exposures and subgroup definitions

The supplied classification defined a normal-chewing group by `sosyaku_code = 1` ($n = 2,816,907$) and impaired chewing by `sosyaku_code  $\geq$  2`. Impaired chewing was subdivided into a group with claims for dentures, bridges or implants ($n = 585,518$) and a group without recorded prosthetic treatment ($n = 18,352$). The questionnaire wording, assessment validity, treatment code lists, ascertainment window, prosthesis function and

time of treatment relative to baseline were unavailable. Absence of a treatment claim is not confirmation that a person had no prosthesis.

The draft separately classified remaining teeth as at least 20, 10–19 or fewer than 10, assigning the same three counts to those categories. It also described molar loss or disease at tooth positions 16–18, 26–28, 36–38 and 46–48, and anterior loss or disease at positions 11–13, 21–23, 31–33 and 41–43. Tooth disease and tooth absence were not operationally distinguished; mixed-site involvement, premolars and overlapping exposure categories were not resolved. No cross-tabulation established that the tooth-count, anatomical and prosthetic classifications identified the same individuals. The fewer-than-10-teeth category is termed severe tooth loss, because it does not imply complete edentulism.

2.4. Cardiometabolic and survival outcomes

The demonstration defined diabetes by HbA1c of at least 6.5% or fasting glucose of at least 126 mg/dL, and hypertension by SBP of at least 140 mmHg or diastolic blood pressure of at least 90 mmHg. These are the draft's classification rules; diagnostic confirmation, medication use and prior disease history were not specified. The diabetes summaries were prevalence comparisons, with no documented incident-diabetes analysis. A mortality flag was attributed to `tenki_kbn_code = 3` in diagnosis claims. Its agreement with a death registry, coverage of deaths outside observed care and handling of multiple claims were not established.

The proposed five-year cardiovascular outcome combined International Classification of Diseases, Tenth Revision codes I20–I25, I60–I69 and I50. These broad groups do not, by themselves, document incident myocardial infarction, acute stroke and heart failure events. The baseline washout period, first-event algorithm, index date, event adjudication, censoring date and competing-event handling were unspecified. The

115 available material therefore does not verify the incident outcome or a common five-year
116 observation period.

117 **2.5.Survival models and prosthetic comparisons**

118 The supplied methods named multivariable Cox models, a log-rank comparison and
119 Kaplan–Meier estimates. Table 2 reproduces coefficients labeled as unrestored impaired
120 chewing, prosthetic rehabilitation, SBP and BMI. The full adjustment set, exposure
121 coding, reference category, interactions, time origin, event counts, person-time and
122 proportional-hazards diagnostics were unavailable. In particular, the treatment coefficient
123 cannot be assumed to compare otherwise exchangeable treated and untreated participants.
124 The reported tooth-site and tooth-count analyses are retained separately because their
125 relationship to that model was not documented.

126 **2.6.P propensity score matching**

127 The described propensity score model used logistic regression with age, sex, baseline
128 BMI and SBP. Matching was reported as nearest-neighbor, one-to-one, without
129 replacement, with a caliper equal to 0.2 SD of the logit propensity score. The matched
130 population contained 585,518 participants in each group. The target estimand, eligible
131 impaired population, overlap assessment, missing-data handling, matching order and
132 treatment of paired observations in outcome models were unspecified. Supplied
133 standardized mean differences are retained in Table 6 as reported diagnostics, not
134 independently verified evidence that confounding was removed.

135 **2.7.Mediation analysis**

136 The draft attributed an additive decomposition of a total effect to a component labeled
137 direct and indirect components through HbA1c and SBP, using a module named

causal_dag. The effect scale, regression models, exposure and mediator timing, mediator dependence, exposure–mediator interactions and uncertainty estimation were not supplied. No evidence established the identification assumptions needed for a causal interpretation. The reported components are therefore described as a demonstration decomposition; the residual component is not assigned to inflammation, autonomic function or any other unmeasured mechanism.

2.8.Cardiovascular prediction

A gradient boosted decision tree model was described as predicting five-year cardiovascular events using six oral and metabolic variables. The draft supplied discrimination, sensitivity, specificity, F1 score and relative feature importance. Development and evaluation sample sizes, event prevalence, patient-level partitioning, temporal separation, tuning, hyperparameters, missing-data preprocessing and the classification threshold were unavailable. Calibration, external evaluation and clinical utility were not reported. Neither the stated confidence interval nor the relative feature-importance values can be independently reconstructed from the supplied summaries.

2.9.Statistical reporting

Continuous summaries are reported as mean $\pm$ SD and supplied effect estimates with their stated 95% confidence intervals. P values and test statistics in the accompanying tables are transcribed from the draft; no new significance tests were performed. The original inference procedures, multiplicity adjustment and confidence-interval methods were not documented. Arithmetic checks addressed internal consistency only and did not validate the underlying analyses. Reporting was organized with reference to STROBE, RECORD and TRIPOD+AI, without claiming complete compliance.⁶⁻⁸ ClinicalSpark Studio and

161 Apache Spark versions, dependencies, execution settings and random seeds were
162 unavailable.

163 3.RESULTS

164 3.1.Reported metabolic summaries differed across chewing and prosthetic groups

165 The three reported chewing/prosthetic groups summed to the stated cohort of 3,420,777.
166 Mean HbA1c was $5.63 \pm 0.62\%$, $5.73 \pm 0.69\%$ and $5.80 \pm 1.15\%$ in the normal-chewing,
167 impaired-with-prosthetics and impaired-without-recorded-prosthetics groups,
168 respectively (Table 1). Corresponding SBP values were 124.79 ± 17.60 , 127.43 ± 17.80
169 and 127.61 ± 18.06 mmHg. Diastolic blood pressure and BMI summaries were also
170 higher in the group without recorded prosthetics than in the normal-chewing group (Table
171 1). No verified statistical comparison or complete confounder profile accompanied these
172 descriptive differences.

173 3.2.Mortality models reported contrasting coefficients for impairment and 174 prosthetic treatment

175 The draft reported crude mortality proportions of 3.673% for unrestored impaired
176 chewing and 0.683% for normal chewing, without defining a common observation period.
177 Its Cox-model summary gave an HR of 4.846 (95% CI, 4.176–5.623) for unrestored
178 impairment and 0.428 (0.368–0.498) for prosthetic rehabilitation (Table 2). SBP and BMI
179 coefficients were 1.013 (1.012–1.013) per mmHg and 0.947 (0.941–0.953) per kg/m²,
180 respectively. The treatment coefficient corresponds arithmetically to a 57.2% lower
181 relative hazard under its unspecified model contrast, but does not demonstrate treatment
182 benefit. The HR must also be distinguished from the ratio of crude mortality proportions.

3.3. Tooth-site summaries lacked a verified common comparator

The anatomical analysis reported diabetes in 2,560 of 18,352 individuals classified as having molar loss (13.95%) and 59,485 of 585,518 classified as having anterior loss (10.16%; Table 3). The corresponding reported RRs were 1.752 (95% CI, 1.690–1.817) and 1.355 (1.344–1.367), with ORs of 1.874 (1.797–1.955) and 1.395 (1.382–1.409). The control prevalence and adjustment were unspecified, preventing verification of a common reference population.

Reported mortality HRs were 4.846 (95% CI, 4.176–5.623) for molar loss and 2.074 (1.979–2.173) for anterior loss (Table 4). Their point-estimate ratio is approximately 2.34, but no covariance, direct contrast or contrast-specific confidence interval was supplied. Significance of the individual coefficients does not establish significance of their difference. The repeated group counts and unrestored-impairment HR across these analyses leave their independence unresolved.

3.4. Prediction performance was reported without an identifiable evaluation sample

The cardiovascular model summary reported a receiver operating characteristic AUC of 0.842 (95% CI, 0.836–0.848), sensitivity of 78.4%, specificity of 76.1% and F1 score of 0.749 (Table 5). Relative feature importance was 28.4% for molar loss, 24.1% for SBP, 19.7% for chewing difficulty, 15.2% for HbA1c, 7.8% for BMI and 4.8% for prosthetic rehabilitation. Molar loss and chewing difficulty together accounted for 48.1% of the reported importance, rather than 48.1% of disease causation or preventable events. Without an evaluation sample, threshold and validation protocol, these values do not establish out-of-sample performance.

3.5.The matched analysis reported residual mortality associations

The stated matched population comprised 1,171,036 individuals, with 585,518 in each group. The draft reported post-match SMDs of 0.002 for age, 0.000 for sex, 0.001 for SBP and 0.000 for BMI, and mortality estimates of RR 1.284 (95% CI, 1.268–1.300) and HR 1.412 (1.385–1.440; Table 6). However, the impaired matched count equals the prosthetically treated subgroup, rather than all participants with impaired chewing. Moreover, some prematch SMDs were not reproducible under conventional pooled-variance calculations from the displayed summaries. These issues preclude treating the matching report as verified covariate balance or an independent confirmation of the mortality association.

3.6.The decomposition did not identify biological pathways

The supplied total coefficient of 0.0412 was decomposed into 0.0283 labeled direct, 0.0071 through HbA1c and 0.0058 through SBP (Table 7). Reported contributions were 68.6%, 17.2% and 14.2%, respectively, with 31.4% attributed to the two mediators together. The displayed coefficients sum to the total, although the percentages cannot all be reproduced exactly from their rounded values. Unrounded output was unavailable. These numerical allocations do not establish the fraction of cardiovascular risk caused by metabolic changes, nor do they identify a direct inflammatory or neural pathway.

3.7.Reported survival declined across tooth-count categories

Five-year survival was reported as 97.4% (95% CI, 97.3–97.5%) for at least 20 teeth, 94.1% (93.9–94.3%) for 10–19 teeth and 84.2% (83.4–85.0%) for fewer than 10 teeth (Table 8). The corresponding HRs were 1.000 as reference, 1.684 (1.642–1.727) and 4.846 (4.176–5.623). The difference between the first and last survival estimates was 13.2

percentage points. These summaries describe an ordered pattern within the demonstration; they cannot establish a dose–response relationship without verified group derivation, event times, censoring and numbers at risk.

4.DISCUSSION

Published observational studies provide a reason to investigate oral function alongside metabolic health and survival. The Shimane and Aichi studies found cross-sectional associations, while the Suita study and Singapore mortality analysis addressed longitudinal questions.^{1-3,5} The present ClinicalSpark demonstration presents a broader set of linked-record outputs, but no independent access to the records or executable analyses was available. Its numbers therefore cannot be treated as replication of those studies or as additional clinical evidence. The most defensible contribution is a coherent description of the proposed analytical questions and the evidence needed to interpret them.

Exposure definition is a central limitation. Subjective chewing difficulty, diseased teeth, absent teeth, posterior occlusal support and prosthetic treatment are not interchangeable clinical states. Their identical or overlapping demonstration counts require a documented derivation rather than an assumption of equivalence. A claims record establishes that a coded service or diagnosis was recorded, but the draft did not show how it verified functional restoration, tooth absence or current prosthesis use. RECORD emphasizes transparent coding, selection and linkage descriptions for precisely this reason.⁷ These uncertainties affect both the reported associations and the meaning of the predictors entered into the model.

The prosthetic-treatment coefficient requires particular restraint. The reported HR of 0.428 (Table 2) may appear compatible with lower mortality, but its reference category

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and treatment timing were not established. Differences in access to care, general health, frailty, socioeconomic conditions and treatment eligibility could influence both prosthesis receipt and survival. If treatment were classified using postbaseline claims while follow-up began earlier, survival to treatment could also bias the comparison. Matching on age, sex, BMI and SBP would not by itself address these concerns. An appropriate evaluation would need an explicit time zero, a treatment strategy, eligible comparison participants, measured confounders and a reproducible analysis of the intended contrast. No mortality-prevention or reimbursement recommendation follows from the supplied coefficient.

The tooth-site and tooth-count results face additional interpretive constraints. Dividing two HR point estimates can summarize their numerical relation, but it cannot replace a model-based direct comparison with valid uncertainty. Likewise, five-year Kaplan–Meier survival and crude mortality proportions need not coincide when follow-up and censoring differ. Their discrepancy here cannot be resolved because the relevant observation periods and denominators were absent. Even a correctly estimated ordered survival pattern would not isolate the effect of losing a particular tooth category from age, disease severity, smoking, nutritional status or other correlated characteristics. The demonstration does not establish that molars have a quantitatively greater causal effect on mortality than anterior teeth.

Dietary pathways remain plausible research hypotheses. The oral-health and dietary measurements in Marito and colleagues' cross-sectional study motivate studying nutrition together with blood pressure, but do not validate the demonstration's mediation fractions.

⁴ No dietary intake, inflammatory biomarker, autonomic measure or longitudinal mediator trajectory was supplied here. A residual coefficient after including HbA1c and SBP cannot be labeled an inflammatory pathway simply because those mediators leave part of an

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276 association unexplained. The reported use of SBP both as a matching covariate and as a
277 mediator also requires a clear distinction between baseline and subsequent measurements.
278 Without that temporal distinction and an identified effect scale, the decomposition has no
279 established causal interpretation.

280 The prediction summary addresses a different question from the etiologic analyses. A
281 high reported AUC does not demonstrate calibration, clinical usefulness or
282 generalizability. The absence of a documented test population prevents assessing whether
283 feature construction, repeated records or tuning could have introduced information
284 leakage. Feature importance also depends on the fitted algorithm and the chosen
285 importance metric; the reported percentages do not quantify the benefit of intervening on
286 a feature. TRIPOD+AI calls for sufficient detail on development data, evaluation,
287 performance and model specification to permit appraisal. ⁸ Those requirements remain
288 unmet by the aggregate performance values alone.

289 Several limitations apply to the entire demonstration. The population, calendar period,
290 ethics and data-use documentation, linkage process, event definitions, missing-data
291 strategy and analytical code were unavailable. Its confidence intervals and very small
292 reported P values cannot be verified and do not compensate for those gaps. The present
293 revision checked documentation, reference metadata and arithmetic relationships rather
294 than clinical validity. A reproducible study would require the missing design evidence and
295 independent evaluation before conclusions could extend beyond the demonstration. As
296 presented, the material illustrates how oral-health exposures might be examined within
297 linked records, while leaving associations, mechanisms, prognostic value and treatment
298 effects unresolved.

299 **ACKNOWLEDGMENTS**

300 Funding sources and acknowledgments were not documented in the supplied
301 demonstration material.

302 **AUTHOR CONTRIBUTIONS**

303 The collective author label is retained from the supplied draft. Individual contributor
304 identities, roles and approval of this manuscript were not documented.

305 **COMPETING INTERESTS**

306 A competing-interest declaration was not supplied; absence of a declaration should not
307 be interpreted as absence of competing interests.

308 **DATA AND MATERIALS AVAILABILITY**

309 The material available for this manuscript comprised the supplied demonstration draft
310 and its aggregate summaries. Patient-level data, linkage and analysis code, fitted models
311 and execution logs for the reported clinical analyses were unavailable. The revised
312 manuscript and accompanying Tables 1–8 preserve the reported summaries but do not
313 constitute an independently reproducible clinical dataset.

314 **SUPPLEMENTARY MATERIALS**

315 Tables 1–8 are supplied in a separate editable table document, with corresponding
316 Markdown source. All values in these tables are reported demonstration outputs with the
317 qualifications specified in their captions and notes.

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