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Palatal Morphology and Health-Related Outcomes in Older Adults: A Retrospective Analysis Using Three-Dimensional Digital Dental Data

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Abstract

Background: Reduced transverse maxillary width has been linked to impaired nasal airway function, but whether palatal morphology relates to broader systemic health outcomes in adults — where craniofacial growth has stabilized — remains poorly characterized, in part because conventional linear measurements do not account for individual body size.

Methods and Findings: This retrospective observational study analyzed three-dimensional digital maxillary scans (Medit i700 intraoral scanner) from Caucasian patients aged 70–80 years attending two general dental practices in Melbourne, Australia. Intermolar width (IMW), intercanine width, and palatal depth were measured, and body-normalized composite indices — the Width-to-Palatal-Height Index (WPHI), Width-to-Palatal-Weight Index (WPWI), and Palatal-to-Body Ratio (PBR) — were derived. Associations with self-reported cancer, cardiovascular disease, obstructive sleep apnoea (OSA), and temporomandibular disorder (TMD) were assessed using Mann-Whitney U tests, point-biserial correlations, and logistic regression adjusted for age, sex, and smoking status. The primary analytic sample (S1, N = 37) excluded participants with prior orthodontic treatment; findings were replicated in the full sample (S2, N = 42). Lower WPHI and WPWI, reflecting a narrower and deeper palate relative to body size, were significantly associated with a history of cancer (WPHI: adjusted OR 4.15, p = .045; WPWI: adjusted OR 2.34, p = .047).

Cardiovascular disease showed a similar directional trend that did not reach significance (p = .086–.099). PBR was not associated with any outcome. OSA was instead associated with reduced absolute IMW (29.7 vs 33.3 mm, p = .072) rather than with body-normalized shape indices. No associations were identified for TMD.

Conclusions: Palatal shape relative to body size, rather than absolute palatal size, may be a functionally relevant marker of systemic health in older adults. A narrower, deeper vault in individuals with larger body size was associated with cancer history, whereas OSA related specifically to absolute transverse maxillary deficiency. These preliminary, hypothesis-generating findings derive from a modest retrospective sample and require validation in larger, prospectively designed cohorts.

Keywords

Palatal morphology; Maxillary width; Airway function; Craniofacial structure; Obstructive sleep apnoea; Cancer; digital dentistry; Morphometric indices.

Introduction

Nasal respiration warms, humidifies, and filters inhaled air, protecting the lower airway and supporting efficient gas exchange [1,2]. The hard palate forms the floor of the nasal cavity, so the transverse and vertical dimensions of the maxilla have a direct bearing on the space available for nasal airflow [2,3]. A narrow or high-arched palate reduces nasal cavity width and increases airway resistance, and has long been associated with a compensatory shift toward mouth breathing [3-5]. This shift can, in turn, further constrain maxillary growth, producing a self-reinforcing cycle between breathing pattern and craniofacial form [6-8].

Interest in the airway implications of maxillary morphology has grown substantially within dentistry and orthodontics, with treatment increasingly framed not only in terms of occlusion but also of respiratory function [9,10]. Advances in cone-beam computed tomography and intraoral scanning now allow palatal shape and dimension to be quantified with a precision that was not previously available in routine clinical settings [11,12]. A substantial evidence base links narrow maxillary form to reduced nasal cavity dimensions and elevated nasal resistance [13-15], and impaired nasal breathing has downstream associations with sleep-disordered breathing and, through that pathway, with cardiovascular and metabolic health [16-18].

Despite this evidence, important gaps remain. First, the great majority of studies examining palatal morphology and airway function have been conducted in children, adolescents, or patients undergoing active orthodontic treatment, whose craniofacial structures are still developing or have been therapeutically modified [7,9,10]. How naturally developed palatal form relates to health outcomes in adults — where growth has stabilized and structural variation reflects a lifetime of developmental and functional influence — is comparatively unexplored. Second, studies of "narrow palate" have relied almost exclusively on absolute linear measurements such as intermolar width, definitions of which vary considerably across the literature [19]. Absolute dimensions do not, by themselves, distinguish between a genuinely constrained palate and a proportionally smaller palate that simply belongs to a smaller-framed individual. Body-normalized or shape-based approaches, which are well established in general morphometrics [20,21] and in physiological performance testing [22], have rarely been applied to

craniofacial structure, even though twin studies suggest palatal dimensions are under substantial additive genetic control and likely represent stable developmental traits [23].

This study addresses these gaps by examining the relationship between palatal morphology and systemic health outcomes in an untreated adult population, using three-dimensional digital dental records collected as part of routine clinical care. In addition to conventional linear measurements, we introduce and apply exploratory composite indices that express palatal shape relative to body size, in order to test whether shape-based or size-based descriptors of the palate are more informative for systemic health associations. We hypothesized, on the basis of the anatomical continuity between the palate and the nasal floor [2,3] and established definitions of maxillary transverse deficiency [24,25], that a disproportionately narrow and deep palatal vault would be associated with adverse airway-related and systemic health outcomes.

Materials and Methods

Design and setting: This retrospective observational study used de-identified clinical records from patients attending two general dental practices in Melbourne, Australia. As all data were collected during routine care and no additional diagnostic procedures or patient contact were required, formal ethical approval was not sought, consistent with institutional and jurisdictional guidance for retrospective, de-identified clinical audits. All records were fully anonymized prior to analysis.

Participants: Eligible participants were Caucasian adults aged 70–80 years with a complete, sufficient-quality maxillary digital scan acquired using the Medit i700 wireless intraoral scanner (Medit Corp., Seoul, South Korea) as part of routine dental care. Participants were excluded if they had congenitally missing permanent teeth (excluding third molars), supernumerary teeth, clinically significant dental anomalies, or extensive restorations distorting palatal landmarks. Screening of 60 records yielded 42 eligible participants (Sample 2, S2). Five participants with self-reported orthodontic treatment history were additionally excluded to form the primary analytic sample (Sample 1, S1, N = 37; 20 male, 17 female; mean age 74.1 years, SD 3.4), because orthodontic intervention can alter maxillary transverse dimensions independently of natural craniofacial development. S2 (N = 42) served as a sensitivity analysis.

Palatal measurement: Intermolar width (IMW; distance between the lingual gingival margins of the maxillary first molars), intercanine width, and palatal depth (perpendicular distance from the deepest point of the palatal vault to the intermolar reference plane) were measured on each digital model using the calibrated digital caliper tools within the Medit Design App, to a resolution of 0.01 mm. All measurements were performed by a single examiner, in triplicate, with the mean of the three measurements used for analysis; this design reduced random measurement error but did not permit assessment of inter-examiner reliability.

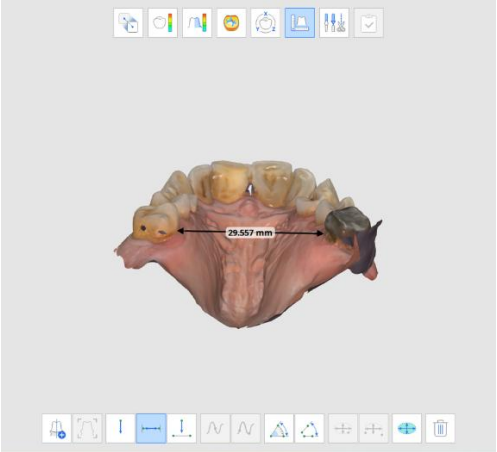

Figure 1: Intermolar Width Measurement in Medit Design App – Transverse view.

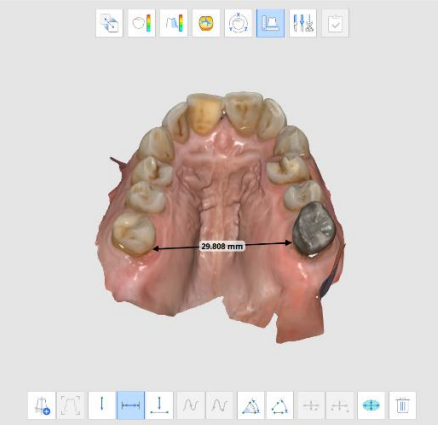

Figure 2: Intermolar width measurement in Medit Design App – occlusal view.

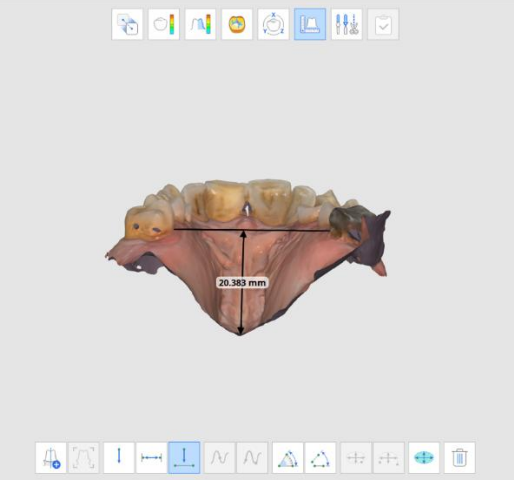

Figure 3: Palatal depth measurement in Medit Design App.

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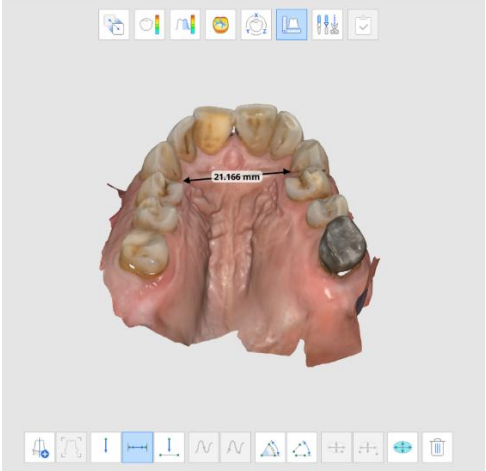

Figure 4: Intercanine width measurement in Medit Design App.

Derived indices: A Palatal Size Index (PSI = IMW × intercanine width × depth) approximated palatal volume. Because craniofacial dimensions scale with body frame, PSI was normalized by body surface area (estimated using the Du Bois formula [26]) to yield the Palatal-to-Body Ratio (PBR = PSI / BSA), a body-normalized proxy for palatal capacity. Palatal shape was expressed as the Width-to-Palatal-Height Ratio (WPH = IMW / depth), and two body-normalized shape indices were derived: the Width-to-Palatal-Height Index (WPHI = WPH / [height (m) × weight (kg)]) and the simpler Width-to-Palatal-Weight Index (WPWI = WPH / weight). Lower WPHI/WPWI values indicate a disproportionately narrow and deep palate relative to body size. These indices are exploratory and have not previously been described in the craniofacial literature; they draw on established morphometric and normalization principles [20–22].

Outcomes and covariates: Health outcome and anthropometric data (height, weight, smoking status, medical history) were extracted from patient questionnaires and clinical records completed at the time of examination. Four primary outcomes were defined a priori: self-reported history of cancer, cardiovascular disease, obstructive sleep apnoea (OSA), and temporomandibular disorder (TMD). TMD status was a binary composite based on the Diagnostic Criteria for Temporomandibular Disorders [27], scored positive if any of jaw pain, masticatory muscle pain, joint sounds, jaw locking, or associated headache/earache was reported. Six supplementary outcomes (anxiety/depression, snoring, stroke, sinus problems, gum disease, allergies) were examined exploratorily.

Statistical analysis: All analyses were conducted in R version 4.4 [28] using a reproducible, parameterized analytic pipeline. Associations between six palatal predictors (IMW, depth, WPH, WPHI, WPWI, PBR) and each outcome were assessed using Mann-Whitney U tests, selected because small sample sizes precluded reliable verification of normality [29], and point-biserial correlations. Logistic regression estimated crude and adjusted odds ratios (age, sex, and smoking status; body mass index was omitted from models using body-normalized predictors to avoid structural collinearity [30]). Events-per-variable (EPV) ratios were computed for every model; adjusted models were fitted only where EPV exceeded 2, and Firth penalised-likelihood regression [31,32] was applied where separation was detected. Spearman correlations [33] characterized inter-predictor relationships. No correction for multiple comparisons was applied,

consistent with the exploratory, hypothesis-generating nature of this pilot analysis [34]; results with $p < .10$ are reported as marginal trends.

Results

Sample characteristics: Descriptive characteristics of the primary sample (S1, N = 37) are shown in (Table 1). Cancer was the most prevalent primary outcome (11/37, 29.7%), followed by heart disease and TMD (7/37 each, 18.9%) and OSA (5/37, 13.5%). Males were taller and had higher body surface area than females, as expected, but raw palatal dimensions did not differ significantly by sex (all $p > .18$), and body normalization successfully removed sex-related variation in the composite indices (WPHI $p = .157$; WPWI $p = .279$; PBR $p = .556$), supporting pooled analysis. WPHI and WPWI were strongly correlated with one another ($\rho = .983$) and with body weight ($|\rho| > .83$), but were uncorrelated with PBR ($\rho = -.08$ to $-.11$), confirming that the shape-based and volume-based indices capture distinct dimensions of palatal morphology.

Variable	Value
Age, years, mean (SD)	74.1 (3.4)
Sex, male/female	20 / 17
Height, m, mean (SD)	1.70 (0.10)
Weight, kg, mean (SD)	75.8 (17.6)
BMI, kg/m², mean (SD)	26.4 (5.5)
Intermolar width, mm, mean (SD)	32.8 (4.0)
Intercanine width, mm, mean (SD)	23.1 (3.3)
Palatal depth, mm, mean (SD)	15.1 (3.0)
WPHI, mean (SD)	0.020 (0.008)
WPWI, mean (SD)	0.032 (0.013)
PBR, mean (SD)	6189 (1512)
Cancer, n (%)	11 (29.7)
Cardiovascular disease, n (%)	7 (18.9)
Obstructive sleep apnoea, n (%)	5 (13.5)
TMD, n (%)	7 (18.9)

Table 1: Descriptive characteristics of the primary analytic sample (S1, N = 37).

Cancer: Lower WPHI and WPWI values were significantly associated with a history of cancer (Table 2). Each 0.01-unit decrease in WPHI was associated with a nearly four-fold increase in cancer odds (crude OR 3.95, 95% CI 1.04–15.04, $p = .044$), an association that persisted after adjustment for age, sex, and smoking status (adjusted OR 4.15, $p = .045$). WPWI produced comparable estimates (crude OR 2.22, 95% CI 1.00–

4.92, p = .050; adjusted OR 2.34, p = .047). Raw IMW and depth showed no association with cancer (both p > .20), and PBR showed no trend in either sample (S1 r = .05, p = .77; S2 r = -.01, p = .98), indicating that the association was specific to vault shape rather than overall palatal volume. Inclusion of the five orthodontic-treated participants in the sensitivity sample (S2, N = 42) attenuated the cancer associations (WPHI crude OR 2.22, p = .104), consistent with four of the five treated individuals having markedly narrow IMW suggestive of pre-treatment constriction rather than natural morphology.

Cardiovascular disease: Participants with cardiovascular disease showed lower WPHI (0.015 vs 0.020, p = .086) and WPWI (0.026 vs 0.034, p = .099) than unaffected participants, directionally consistent with the cancer findings but not statistically significant, with wide confidence intervals reflecting the small number of cases (n = 7). PBR again showed no association (p = .968).

Obstructive sleep apnoea: OSA presented a distinct predictor profile. Participants with OSA had narrower absolute intermolar width than those without (29.7 vs 33.3 mm, p = .072; point-biserial r = -.311, p = .061) and lower WPH (1.89 vs 2.32, p = .095), whereas the body-normalized shape indices WPHI and WPWI showed no association (p = .234 and .162 respectively). This pattern — an association with absolute width but not with body-normalized shape — was the reverse of that seen for cancer and cardiovascular disease.

TMD and supplementary outcomes: No palatal index was associated with TMD (all p > .25). Among supplementary outcomes, sinus problems showed a marginal association with greater palatal depth (r = .289, p = .078), and snoring, the most prevalent outcome (54.1%), showed no association with any palatal index.

Outcome	Predictor	Crude OR (95% CI)	Crude p	Adjusted OR	Adjusted p
Cancer	WPHI (per 0.01 decrease)	3.95 (1.04–15.04)	.044	4.15	.045
Cancer	WPWI (per 0.01 decrease)	2.22 (1.00–4.92)	.050	2.34	.047
Cancer	PBR (per 1000 decrease)	0.93 (0.58–1.48)	.762	—	—
Cardiovascular disease	WPHI (per 0.01 decrease)	2.05 (0.62–6.81)	.240	—	—
Cardiovascular disease	WPWI (per 0.01 decrease)	1.65 (0.76–3.58)	.202	—	—
OSA	IMW (per 1 mm decrease)	1.23 (0.97–1.56)	.083	—	—
OSA	WPH (per 1 unit decrease)	4.14 (0.55–30.95)	.166	—	—
TMD	WPHI (per 0.01 decrease)	0.77 (0.28–2.11)	.614	—	—

Table 2: Associations between palatal morphometric indices and primary health outcomes, logistic regression (S1, N = 37).

Adjusted models included age, sex, and ever-smoker status and were fitted only where events-per-variable exceeded 2; dashes indicate models not fitted or not materially different from the crude estimate. OR, odds ratio; CI, confidence interval; OSA, obstructive sleep apnoea; TMD, temporomandibular disorder.

Discussion

The principal finding of this study is that body-normalized indices of palatal shape — WPHI and WPWI — were significantly associated with a history of cancer, while the body-normalized volume index PBR was not associated with any outcome examined. This dissociation suggests that the proportional relationship between palatal width and depth, rather than overall palatal capacity, may be the more functionally relevant descriptor of maxillary morphology in relation to systemic health, consistent with broader principles in morphometrics in which shape and size are treated as independent dimensions of anatomical variation [20,21]. Obstructive sleep apnoea showed the opposite pattern, being associated with absolute transverse width (IMW) rather than with proportional shape, indicating that different palatal characteristics may be relevant to different health outcomes depending on the underlying mechanism.

A plausible, though speculative, explanatory pathway links palatal shape to systemic outcomes through chronic airway function. Because the hard palate forms the nasal floor, a narrow, high-arched vault reduces the space available for nasal airflow and increases resistance at anatomically critical sites such as the internal nasal valve and inferior turbinate [3,35,36]. According to basic fluid-dynamic principles, even small reductions in airway calibre can produce disproportionate increases in resistance [36], favoring a compensatory shift toward oral breathing and bypassing the nasal contribution to humidification, filtration, and nitric-oxide-mediated pulmonary Vaso regulation [37]. Chronic or intermittent reductions in oxygenation associated with disordered breathing have been linked to oxidative stress, endothelial dysfunction, and systemic inflammation [38–41], and, at the cellular level, to stabilization of hypoxia-inducible factors that regulate angiogenesis, glycolytic metabolism, and cell survival pathways implicated in tumour biology [42–45]. Within this framework, a disproportionately narrow and deep palate could act as a structural marker of a lifetime of subtly altered airway function and oxygenation, rather than as a direct cause of malignancy. Because this study was cross-sectional and observational, causality cannot be established, and shared early-life developmental influences on both craniofacial growth and long-term disease susceptibility remain an equally plausible explanation for the observed association [46].

Notably, the present sample did not show a significant association between palatal shape indices and OSA itself, even though OSA has repeatedly been linked to intermittent hypoxia and cardiovascular strain in the wider literature [43,44]. This may partly reflect underdiagnosis of OSA in older populations [47,48], which would bias any true association toward the null, and it is also consistent with the interpretation that OSA is governed primarily by absolute airway geometry — the physical space available for airflow — rather than by proportional relationships that only become relevant across longer, cumulative timescales [49,50]. Reduced intermolar width has previously been associated with airway collapsibility and reduced tongue space in OSA [51,52], and the present findings, though not statistically significant after adjustment, are directionally consistent with this literature.

These findings should be interpreted within the broader interventional literature on maxillary expansion, which provides indirect but consistent support for a structural link between maxillary width and nasal airway function: widening the maxilla has repeatedly been shown to increase nasal cavity volume and reduce airway resistance in treated populations [53,54,55]. The present study extends this evidence by examining naturally occurring variation in untreated adults, a population that has received comparatively little attention relative to children, adolescents, and treated cohorts [9,10].

Strengths and limitations: Strengths of this study include the use of precise three-dimensional digital measurements, real-world clinical data reflective of general dental practice, Standardised measurement and analytic protocols, and the exclusion of orthodontically treated individuals from the primary analysis to isolate naturally developed morphology. The principal limitation is sample size: with as few as five OSA cases and seven cardiovascular cases, the study was powered to detect only medium-to-large effects, and several adjusted models had low events-per-variable ratios, meaning adjusted estimates should be regarded as indicative rather than definitive. The retrospective, cross-sectional design precludes causal inference; health outcomes were self-reported without medical record verification or diagnostic subtyping (for example, cancer type was not recorded, which likely diluted any organ-system-specific association); and measurements were performed by a single examiner without formal inter-rater reliability assessment. The sample was restricted to Caucasian individuals aged 70–80 years from two practices in one city, and palatal morphology is known to vary across ethnic groups [56], limiting generalizability. WPHI and WPWI are novel, exploratory indices that have not previously been validated externally, and their strong inverse correlation with body weight means that some of the observed association could, in principle, reflect a body-weight pathway rather than palatal geometry per se; future work should attempt to disentangle these components.

Clinical and research implications: These findings suggest that dental practitioners' access to precise digital maxillary records may have value beyond occlusal assessment, offering a potential — if presently exploratory — window into airway-related structural risk relevant to interdisciplinary, preventive care [10,57]. More immediately, the results support continued development and external validation of body-normalized craniofacial indices, and highlight the value of distinguishing palatal shape from palatal size in future morphometric research [20,21]. Larger, prospective studies with medical record-verified outcomes, cancer subtyping, objective airway assessment (for example CBCT-derived airway volume), and formal reliability testing are needed to confirm these associations and clarify the biological mechanisms involved.

Conclusion

In this retrospective analysis of digital maxillary scans from older Australian adults, body-normalized indices of palatal shape — but not a body-normalized index of palatal volume — were associated with a history of cancer, while obstructive sleep apnoea was associated with absolute transverse maxillary width rather than shape. These preliminary findings support a conceptual distinction between shape-based and size-based descriptors of craniofacial structure and suggest that palatal morphology, assessed through routine digital dental records, may warrant further investigation as a marker of long-term airway function and systemic health. Confirmation in larger, prospectively designed, and more diverse cohorts is required before any clinical application can be considered.

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Ethics approval and consent

This was a retrospective study.

Conflict of interest

The authors declare no conflict of interest.

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