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# Comparative Evaluation of COL1A2 Concentration in Completely Edentulous Patients with and Without Diabetes Mellitus

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**Abstract** **Background:** Type I collagen is a major structural component of bone and connective tissue and diabetes mellitus can alter collagen turnover, extracellular-matrix homeostasis and bone quality. COL1A2, which encodes the α2 chain of type I collagen, may therefore provide a biologically relevant marker for studying differences between diabetic and non-diabetic completely edentulous patients. **Aim:** To compare COL1A2 concentrations between completely edentulous patients with and without diabetes mellitus and to examine their relationship with age, HbA1c and Random Blood Sugar (RBS). **Methodology:** Sixty completely edentulous participants aged 40-80 years were included, with 30 diabetic and 30 non-diabetic participants. Diabetes status was defined in the source dataset using HbA1c and RBS criteria. COL1A2 concentration was reported in ng/mL. Group comparisons and correlations were recalculated from the 60 patient-level. **Results:** The diabetic group had a higher mean COL1A2 concentration than the non-diabetic group (504.12±57.55 vs. 328.99±45.18 ng/mL; Welch's t-test, $p<0.001$). Diabetic participants were also older on average (67.00±4.35 vs. 59.87±5.73 years; $p<0.001$). Gender distribution did not differ significantly between groups ($\chi^2 = 0.27$, $p = 0.604$). **Conclusion:** In the 60 observations, the reported COL1A2-labelled assay measurement was higher in the diabetes group than in the non-diabetes group. The marked age imbalance and the unresolved assay-analyte definition constrain biological interpretation; these cross-sectional data do not establish impaired collagen quality, altered bone remodeling or clinical prognosis.

**Key Words** COL1A2, Type I Collagen, Diabetes Mellitus, Complete Edentulism, Bone Quality, Collagen Metabolism, Prosthodontics, Infectious Disease, Public Health, Health Policy

## INTRODUCTION

Type I collagen is the predominant collagen in bone and forms an important part of the organic extracellular matrix. Its molecular organization and cross-linking contribute to the structural and mechanical properties of bone. Diabetes mellitus is associated with alterations in bone quality and collagen metabolism, including the accumulation of advanced glycation end products (AGEs), altered collagen cross-linking and changes in bone-cell activity. These mechanisms may affect bone remodeling even when changes in bone mineral density do not fully explain skeletal fragility [1,2].

COL1A2 encodes the α2 chain of type I collagen. Because collagen turnover is relevant to alveolar bone remodeling and the tissue environment of edentulous patients, differences in COL1A2 concentration may provide insight into the biological response associated with diabetes. However, a circulating or local COL1A2 concentration should be regarded as a biomarker rather than a direct measure of denture adaptation, residual-ridge resorption or prosthetic success [3,4].

Type I collagen is synthesized primarily by osteoblasts and is assembled into a fibrillar extracellular matrix that provides the organic framework for mineral deposition. The α1(I) and α2(I) chains are encoded by COL1A1 and COL1A2, respectively and their coordinated expression is essential for normal collagen fibril formation and tissue

integrity. Alterations in collagen synthesis, post-translational modification and degradation can therefore influence the mechanical and biological properties of bone [7,8].

In diabetes mellitus, persistent hyperglycaemia and oxidative stress promote non-enzymatic glycation of long-lived proteins such as type I collagen. Accumulation of advanced glycation end products (AGEs) can increase abnormal collagen cross-linking, alter fibril properties and interfere with normal bone-cell function. The AGE-RAGE pathway may additionally contribute to oxidative stress and inflammatory signaling, while diabetes-related changes in osteoblast and osteoclast activity can reduce bone turnover and impair bone quality. Importantly, these changes may occur even when conventional measures of bone mineral density do not show a proportionate reduction [9,10].

The oral and maxillofacial implications of diabetes are also clinically relevant. Diabetes has been associated with altered periodontal tissues, impaired healing and changes in the alveolar bone environment. In completely edentulous individuals, the biological quality of the residual ridge is particularly important because it contributes to denture support, stability and long-term adaptation. However, the relationship between diabetes, collagen biology and clinical prosthodontic outcomes remains incompletely understood.

Against this background, assessment of COL1A2 concentration may provide an exploratory biomarker-based approach to understanding collagen-related alterations in completely edentulous patients with diabetes. The present study therefore compares COL1A2 concentrations between diabetic and non-diabetic completely edentulous patients and evaluates their relationship with age and glycaemic indices.

## METHODS

### Study Design and Participants

This comparative observational study included 60 completely edentulous patients aged 40-80 years who attended Saveetha Dental College and Hospital, Chennai. The study period was reported as February 2024 to December 2024.

### Grouping

Thirty participants were classified as diabetic and 30 as non-diabetic. The diabetic group was described as having RBS >150 mg/dL and HbA1c >7%, whereas the non-diabetic group was described as having RBS 70-100 mg/dL and HbA1c <6%. The patient-level table contains the values used for the present statistical re-analysis.

### Exclusion Criteria

Smokers, individuals with systemic diseases other than diabetes that could affect oral health, participants taking vitamins A, C or E or antioxidant therapy within the preceding three months, individuals taking antibiotics and pregnant or lactating women were excluded.

### COL1A2 Measurement

The measurement was using a commercially available human quantitative sandwich ELISA kit. The reported procedure involved sample incubation, washing, HRP-conjugated detection antibody, substrate development, stop solution and optical-density measurement at 450 nm.

### Statistical Analysis

The 60 patient-level observations in the supplied dataset were independently recalculated. Continuous variables are presented as Mean±Standard deviation (SD). Welch's independent-samples t-test was used for the primary comparison of age and COL1A2 concentration because the groups differed in variability and age. Gender distribution was assessed using the chi-square test. Spearman correlation was used to examine the association between HbA1c and COL1A2. Statistical significance was set at $p<0.05$.

## RESULTS

### Demographic Characteristics

Demographic characteristics are shown in Table 1.

### Comparison Between Diabetic and Non-Diabetic Groups

The mean COL1A2 concentration was 175.13 ng/mL higher in the diabetic group, representing approximately 53.2% higher concentration relative to the non-diabetic group. The between-group difference was statistically significant ($p<0.001$) (Table 2).

The diabetic group was also older by approximately 7.13 years ($p<0.001$), indicating that age is a potential confounder and should be considered in future adjusted analyses.

Gender distribution was not significantly different between the two groups ($\chi^2 = 0.269$, $p = 0.604$). Thus, the observed COL1A2 difference is unlikely to be explained by a major imbalance in sex distribution within this sample (Table 3).

### Relationship Between Glycemic Indices and COL1A2

Across all 60 participants, HbA1c showed a strong positive Spearman correlation with COL1A2 ($\rho = 0.800$, $p<0.001$). However, this overall association should be interpreted cautiously because HbA1c and COL1A2 both differed strongly between the diabetic and non-diabetic groups. Within the diabetic group alone, the correlation was weak and non-significant ($\rho = -0.081$, $p = 0.671$). Within the non-diabetic group, the correlation was moderate ($\rho = 0.474$, $p = 0.008$).

The diabetic group showed a markedly higher mean COL1A2 concentration (504.12±57.55 ng/mL) than the non-diabetic group (328.99±45.18 ng/mL). This difference was statistically significant ($p<0.001$), indicating a strong between-group difference in COL1A2 concentration (Figure 1).

Table 1: Demographic Characteristics

| Parameter | Value |
|---|---|
| Total sample | 60 |
| Diabetic | 30 (50.0%) |
| Non-diabetic | 30 (50.0%) |
| Male | 27 (45.0%) |
| Female | 33 (55.0%) |
| Overall age | 63.43±6.20 years |
| Overall COL1A2 | 416.55±102.12 ng/mL |

Table 2: Comparison Between Diabetic and Non-Diabetic Groups

| Variable | Non-diabetic (n = 30) | Diabetic (n = 30) | p-value | Interpretation |
|---|---|---|---|---|
| Age (years) | 59.87±5.73 | 67.00±4.35 | 0.000001 | Significant |
| COL1A2 (ng/mL) | 328.99±45.18 | 504.12±57.55 | 1.50e-18 | Significant |

Table 3: Gender Distribution

| Gender | Non-diabetic (n = 30) | Diabetic (n = 30) |
|---|---|---|
| Female | 15 | 18 |
| Male | 15 | 12 |

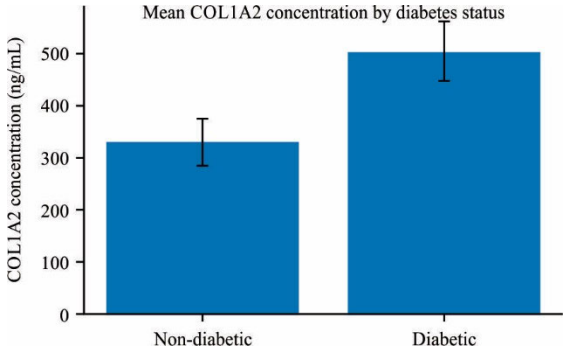



Figure 1: Mean COL1A2 Concentration in Diabetic and Non-Diabetic Participants

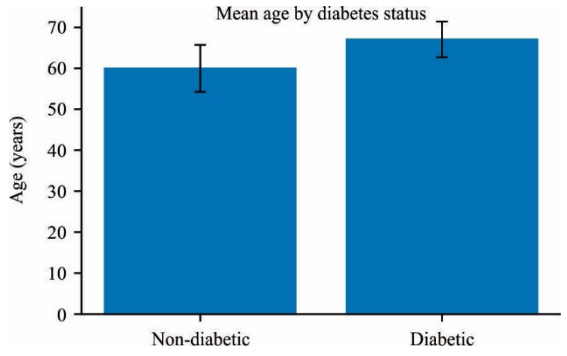



Figure 2: Mean Age of Diabetic and Non-Diabetic Participants

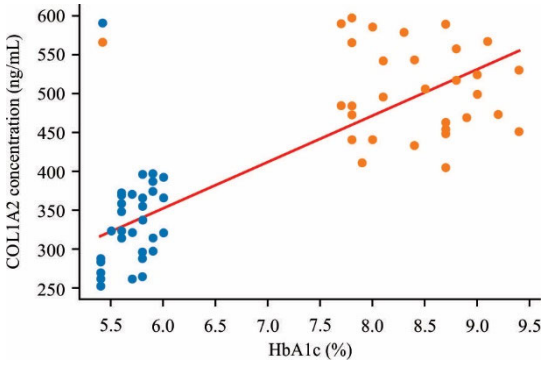



Figure 3: Relationship Between HbA1c and COL1A2 Concentration

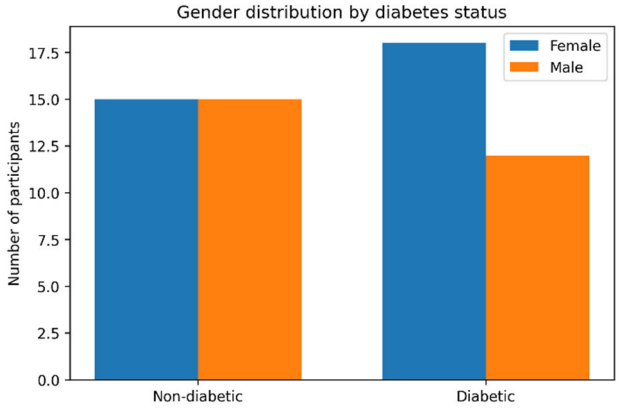



Figure 4: Gender distribution by diabetes status

The diabetic participants were older on average (67.00±4.35 years) than the non-diabetic participants (59.87±5.73 years). The difference was statistically significant ($p<0.001$), identifying age as a potential confounding factor in the comparison of COL1A2 concentrations (Figure 2).

Across all 60 participants, HbA1c demonstrated a strong positive association with COL1A2 (Spearman $\rho = 0.800$, $p<0.001$). However, the overall relationship should be interpreted cautiously because it is influenced by the marked separation between diabetic and non-diabetic groups (Figure 3).

Female and male participants were reasonably balanced across the two groups, with no statistically significant difference in gender distribution ($\chi^2 = 0.269$, $p = 0.604$). Therefore, a major sex imbalance is unlikely to explain the observed difference in COL1A2 concentration (Figure 4).

### Overall Graphical Findings

COL1A2 concentration was substantially higher in diabetic participants, while the diabetic group was also significantly older. HbA1c showed a strong overall positive association with COL1A2, whereas gender distribution was not significantly different between groups.

## DISCUSSION

The principal finding of this study was a markedly higher COL1A2 concentration among completely edentulous participants with diabetes compared with those without diabetes. The difference was large and statistically significant. This observation is biologically plausible in the context of diabetes-related alterations in extracellular-matrix turnover, collagen modification and bone quality although the present study cannot establish the mechanism responsible for the elevated concentration [11,12].

Type I collagen is a major organic component of bone and its cross-linking contributes to the material properties of the bone matrix. Diabetes is associated with accumulation of AGEs and non-enzymatic collagen cross-links, which can alter collagen structure and impair bone quality. Importantly, diabetic skeletal fragility may occur even when bone mineral density is not substantially reduced, highlighting the importance of matrix quality rather than mineral density alone. [13,14]

The elevated COL1A2 concentration observed in the diabetic group should therefore not be interpreted simply as evidence of better collagen formation. A higher measured concentration can reflect altered synthesis, turnover, extracellular-matrix remodeling, impaired degradation or differences in the biological compartment sampled. Reviews of diabetic bone disease describe complex changes involving osteoblast and osteoclast function, collagen glycation, AGE-RAGE signaling and reduced bone turnover. The present findings are consistent with the concept that diabetes is accompanied by measurable changes in collagen-related biology but additional biomarkers are required to determine whether those changes represent increased formation, altered degradation or pathological remodeling. [15,16]

The diabetic group was significantly older than the non-diabetic group. Age itself influences collagen cross-linking and accumulation of AGEs and therefore may confound the association between diabetes and COL1A2. The observed difference in age is particularly important because the study population consisted of older adults. Future studies should use age-matched groups or multivariable regression models incorporating age, sex, duration of diabetes, glycemic control, medications and other relevant metabolic variables. [17,18]

Gender distribution was similar between groups and did not reach statistical significance. This supports reasonable balance with respect to sex in the present sample, although the sample size is too small to exclude modest sex-specific effects. [19,20]

The positive overall association between HbA1c and COL1A2 was strong; however, the absence of a significant correlation within the diabetic group suggests that the overall relationship may largely reflect the difference between diabetic and non-diabetic participants rather than a dose-response relationship between glycemic control and COL1A2 among diabetic patients. [21,22,23,24,25] This distinction is important and supports cautious interpretation of the correlation analysis.

From a prosthodontic perspective, the findings may be relevant because diabetes can influence residual alveolar bone quality and tissue healing. Nevertheless, no direct clinical outcomes such as residual-ridge resorption, denture retention, mucosal healing, patient satisfaction or implant/denture survival were measured. Consequently, a direct relationship between COL1A2 and prosthetic success cannot be claimed from this dataset.

## CONCLUSIONS

Within the 60-patient dataset provided, completely edentulous participants with diabetes had significantly higher COL1A2 concentrations than non-diabetic participants. The diabetic group was also significantly older, while gender distribution was similar between groups. The findings suggest an association between diabetes and altered collagen-related biology in completely edentulous patients. However, the study does not establish causality or demonstrate that COL1A2 concentration directly predicts bone loss or prosthodontic treatment outcome. Larger, age-matched and clinically longitudinal studies are required.

## Limitations

### Additional Measurement Limitations

The identity, analytical specificity and calibration of the ELISA-labelled COL1A2 analyte must be documented before interpreting the concentration as circulating collagen-chain protein.

The study used a relatively small sample of 60 participants from a single institution, limiting statistical power and generalizability. The diabetic and non-diabetic groups were not age-matched; the diabetic group was significantly older, creating potential confounding. The study was cross-sectional, so temporal and causal relationships between diabetes, COL1A2 and oral/prosthetic outcomes cannot be established. No direct prosthodontic or radiographic outcomes, such as residual ridge resorption, denture retention, mucosal healing or patient-reported treatment success, were measured.

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Supplementary Table1: Statistical Dataset Used for the Revised Figures

| ID | Group | Gender | Age | HbA1c (%) | RBS (mg/dL) | COL1A2 (ng/mL) |
|---|---|---|---|---|---|---|
| 1 | Diabetic | Female | 73 | 9.4 | 179 | 451.14 |
| 2 | Non-Diabetic | Female | 65 | 5.9 | 90 | 297.67 |
| 3 | Diabetic | Female | 69 | 8.8 | 171 | 558.07 |
| 4 | Non-Diabetic | Male | 60 | 5.9 | 96 | 374.49 |
| 5 | Diabetic | Female | 69 | 9.0 | 203 | 524.42 |
| 6 | Non-Diabetic | Female | 61 | 5.7 | 83 | 261.32 |
| 7 | Diabetic | Female | 65 | 8.7 | 191 | 589.42 |
| 8 | Non-Diabetic | Male | 69 | 5.4 | 95 | 252.50 |
| 9 | Diabetic | Male | 65 | 8.0 | 161 | 585.68 |
| 10 | Non-Diabetic | Female | 67 | 5.6 | 97 | 348.30 |
| 11 | Diabetic | Male | 72 | 8.1 | 202 | 495.95 |
| 12 | Non-Diabetic | Female | 61 | 6.0 | 89 | 366.02 |
| 13 | Diabetic | Female | 69 | 7.8 | 198 | 483.96 |
| 14 | Non-Diabetic | Male | 59 | 5.8 | 88 | 355.23 |
| 15 | Diabetic | Male | 62 | 8.4 | 203 | 543.43 |
| 16 | Non-Diabetic | Male | 66 | 5.9 | 98 | 396.71 |
| 17 | Diabetic | Male | 67 | 8.7 | 164 | 448.55 |
| 18 | Non-Diabetic | Male | 53 | 5.8 | 88 | 396.28 |
| 19 | Diabetic | Male | 62 | 8.0 | 193 | 440.87 |
| 20 | Non-Diabetic | Male | 51 | 5.4 | 95 | 269.05 |
| 21 | Diabetic | Female | 61 | 7.7 | 197 | 590.06 |
| 22 | Non-Diabetic | Female | 59 | 5.9 | 84 | 314.13 |
| 23 | Diabetic | Male | 60 | 8.1 | 164 | 542.30 |
| 24 | Non-Diabetic | Female | 60 | 5.8 | 80 | 296.13 |
| 25 | Diabetic | Male | 69 | 8.7 | 200 | 405.32 |
| 26 | Non-Diabetic | Female | 61 | 5.6 | 95 | 369.32 |
| 27 | Diabetic | Female | 72 | 9.1 | 172 | 567.18 |
| 28 | Non-Diabetic | Female | 56 | 5.7 | 83 | 370.62 |
| 29 | Diabetic | Male | 61 | 9.2 | 168 | 473.40 |
| 30 | Non-Diabetic | Male | 68 | 6.0 | 91 | 392.38 |
| 31 | Diabetic | Male | 67 | 9.0 | 196 | 499.42 |
| 32 | Non-Diabetic | Female | 68 | 5.4 | 94 | 287.13 |
| 33 | Diabetic | Female | 75 | 8.3 | 182 | 578.87 |
| 34 | Non-Diabetic | Female | 69 | 5.6 | 90 | 358.72 |
| 35 | Diabetic | Female | 70 | 8.7 | 169 | 453.91 |
| 36 | Non-Diabetic | Male | 54 | 5.4 | 97 | 284.05 |
| 37 | Diabetic | Female | 60 | 7.7 | 204 | 484.77 |
| 38 | Non-Diabetic | Male | 65 | 5.8 | 95 | 365.52 |
| 39 | Diabetic | Female | 67 | 8.7 | 206 | 462.96 |
| 40 | Non-Diabetic | Female | 56 | 5.8 | 87 | 264.93 |
| 41 | Diabetic | Female | 73 | 7.8 | 205 | 440.98 |
| 42 | Non-Diabetic | Male | 52 | 5.6 | 93 | 314.14 |
| 43 | Diabetic | Female | 67 | 8.9 | 192 | 469.49 |
| 44 | Non-Diabetic | Male | 57 | 5.4 | 82 | 262.68 |
| 45 | Diabetic | Female | 67 | 8.5 | 181 | 505.74 |
| 46 | Non-Diabetic | Male | 61 | 5.8 | 85 | 287.75 |
| 47 | Diabetic | Male | 64 | 7.8 | 194 | 472.86 |
| 48 | Non-Diabetic | Male | 57 | 5.7 | 88 | 321.21 |
| 49 | Diabetic | Female | 71 | 7.9 | 161 | 411.05 |
| 50 | Non-Diabetic | Male | 60 | 5.9 | 96 | 386.93 |
| 51 | Diabetic | Male | 71 | 9.4 | 220 | 530.17 |
| 52 | Non-Diabetic | Male | 54 | 5.8 | 81 | 337.58 |
| 53 | Diabetic | Female | 70 | 7.8 | 176 | 597.19 |
| 54 | Non-Diabetic | Female | 53 | 6.0 | 95 | 321.08 |
| 55 | Diabetic | Female | 60 | 8.8 | 176 | 517.30 |
| 56 | Non-Diabetic | Female | 57 | 5.6 | 84 | 322.41 |
| 57 | Diabetic | Male | 64 | 8.4 | 196 | 433.31 |
| 58 | Non-Diabetic | Female | 50 | 5.6 | 89 | 372.03 |
| 59 | Diabetic | Female | 68 | 7.8 | 183 | 565.77 |
| 60 | Non-Diabetic | Female | 67 | 5.5 | 98 | 323.34 |