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Trabecular microarchitecture and tensile strength of the humerus in diabetic Sprague Dawley rats consuming alcohol: a micro focus X-ray computed tomography case-control study

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SUMMARY

Diabetes and excessive alcohol consumption both negatively impact bone health independently. However, their combined effect is unclear. Therefore, this study aimed to investigate humerus trabecular morphometry and tensile strength in diabetic rats that consumed alcohol. Thirty-one (31) 10-week-old male Sprague-Dawley rats weighing 330g-370g were used. Rats were grouped as follows: untreated (control), which got no treatment (n=8); ALC, which consumed alcohol (n=8); and DB, diabetic group (n=7). The study also included an additional diabetic group that received alcohol, (DB+ALC) (n=8). Serum insulin and fasting blood glucose levels were used to confirm diabetes, which was induced by a high-fructose diet and streptozotocin. ALC rats received drinking water with 10% v/v alcohol daily until termination at 24 weeks of age. Afterwards, bilateral humeri were dissected and stored in 10% buffered formalin before osteometric measurements were recorded. Micro-focus X-ray computed tomography (Micro CT) scanning was then conducted to evaluate trabecular number (TbN), thickness (TbTh), spacing (TbSp), bone tissue volume ratio (BV/TV) before undergoing, and tensile strength tests.

The DB+ALC group had shorter humeri, although the bicondylar breadth was similar among all groups. The DB+ALC and the DB group showed diminished trabecular thickness (TbTh), but more trabeculae (TbN). The maximum and break force were the lowest in the DB+ALC group. Diabetic skeletopathy worsens with alcohol use, leading to shorter bones with lower trabecular thickness, maximum, and break force in the DB+ALC treated group. We suggest increased bone health monitoring for diabetic patients who consume alcohol.

Key words: Humerus – Cancellous bone – Diabetes mellitus – Alcoholics – Bone fractures – Tensile strength

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INTRODUCTION

The internal, spongy trabecular bone plays a vital role in skeletal health. Its intricate network acts as a support system, effectively distributing forces and preventing fractures, particularly in high-stress areas such as epiphysis and metaphysis of long bones (Oftadeh et al., 2015). Its porous structure also allows it to absorb impacts, protecting bones during locomotion (Morgan et al., 2018). Furthermore, its large surface area is critical for mineral exchange, and its unique architecture directly influences bone strength (Mulder et al., 2005). Diabetes (Murray and Coleman, 2019; Vilaca et al., 2020) and excessive alcohol consumption (Bhika et al., 2025) both negatively impact bone health, independently.

Alcohol abuse can significantly impact the health and structural integrity of the bones, leading to various vulnerabilities such as propensity to fracture and disturbances in trabecular organisation (Godos et al., 2022; Maurel et al., 2012). Research shows that excessive alcohol consumption adversely affects bone health through reduced absorption of important elements such as calcium and vitamin D, which are crucial for bone health and growth (Broulik et al., 2010; Godos et al., 2022). Reduced nutrient absorption increases the risk of osteoporosis over time (Balhara et al., 2022). Osteoporosis is characterized by a decline in bone tissue and microarchitecture, leading to a decrease in bone strength (Sözen et al., 2017). Also, alcohol interferes with the production of hormones, such as cortisol, which can promote bone resorption and inhibit bone formation (Noth and Walter, 1984). This creates an osteoporotic phenotype, with weakened bones, increased likelihood of fractures, and delayed fracture healing (Godos et al., 2022).

Poorly managed diabetes can also have detrimental effects on bone health (Compston, 2018), with diabetic individuals facing an elevated risk of developing diabetic skeletopathy due to hyperglyceamia, which can weaken the bones and make fractures more likely (Schousboe et al., 2022). Similar to alcohol, diabetes is also linked to increased osteoporosis; further jeopardizing bone health as a higher incidence of proximal humerus fractures is reported in people with diabetes (Liu et al., 2018; Schousboe et al., 2022; Vilaca et al., 2020). The increased bone fracture risk among diabetics is attributed to factors such as poor blood circulation, nerve damage, and changes in bone metabolism caused by the resultant dysregulation of elements such as calcium and phosphorus. Moreover, diabetes can impede the healing of fractures, resulting in longer recovery periods and an elevated risk of complications (Figeac et al., 2022).

Diabetes and alcohol each disrupt bone health through distinct mechanisms. Diabetes impairs bone formation and microarchitecture, and neuropathy increases fall risk, collectively raising fracture susceptibility (Zamarioli et al., 2020). Alcohol reduces bone density through disrupted hormone balance and bone remodelling, and also elevates fall risk (Maurel et al., 2011). Many people who are diabetic are also known to abuse alcohol (Kim and Kim, 2012). The degree of impact is influenced by diabetes severity (Ndou et al., 2024) and alcohol consumption patterns (Pillay and Ndou, 2022). It remains unclear as to whether, when combined, these effects are amplified, substantially increasing osteoporosis and fracture risk.

The growing problem of diabetes and alcohol use in conjunction, driven by poor lifestyle choices, necessitates this research. The aim of this study was to assess trabecular microarchitecture and tensile strength in the humerus of diabetic rats consuming alcohol. The objective was to use Micro-CT to evaluate bone osteometry and trabecular morphometry, and tensile testing to measure bone strength. We hypothesized that alcohol consumption exacerbates the negative impact of diabetes on bone health. The understanding of this combined effect is essential for developing improved prevention and treatment strategies for diabetic patients.

MATERIALS AND METHODS

Study design

The study utilized 31 male Sprague-Dawley rats (10-weeks-old, 330-370g), housed individually in pathogen-free plastic cages at the University of the Witwatersrand's Central Animal Services.

They received ad libitum rodent diet and water, with controlled temperature (21-23oC) and a 12-hour light-dark cycle. The animals were randomly divided into the following groups: untreated (control), which received no treatment (n=8); alcohol (ALC), which consumed alcohol (n=8); diabetic (DB), the diabetic group (n=7); and a diabetic group that received alcohol (DB+ALC) (n=8). The Animal Ethics Committee of the University of the Witwatersrand, Johannesburg gave ethics approval for the study (AESC: 2018/011/58/C), and all the procedures met the standards of this committee.

The rats were placed on a rodent diet containing 20% fructose for two weeks. Subsequently, they received a single streptozotocin injection (40 mg/kg.ip) (Sigma, St. Louis, MO, USA) in freshly prepared 0.05 M citrate buffer (pH 4.5). At 12 weeks of age, rats in the ALC and DB+ALC groups consumed daily drinking water containing 10% v/v alcohol until termination at 24 weeks of age. Upon termination through pentobarbitone (250 mg/kg, ip), blood samples were collected via cardiac puncture into 4 ml serum-separating tubes to analyse terminal fasting blood glucose and insulin levels. Bilateral humeri were meticulously dissected and fixed in 10% buffered formalin for further processing.

Osteometric measurements

Once soft tissues were cleaned off the bilateral humeri, a digital caliper was used to measure the maximum length and the biomechanical length (Table 1 and Fig. 1).

Micro-focus X-ray Computed Tomography (Micro CT)

Humeri were scanned using a Nikon XTH 225/320 LC X-ray microtomography system secured in a floral oasis on a rotating manipulator. The X-ray settings were 100 kV and 100µA, with a capture rate of 0.5 frames per second for improved signal-to-noise ratio. Scans had a resolution of 35 µm. Parameters analysed included bone volume fraction (BV/TV), trabecular thickness (TbTh), spacing (TbSP) and trabecular number (TbN), using Volume Graphics®3.2 software.

Tensile strength testing (3-point bending)

A Shimadzu universal tester (Z-X S 200V E SSM346-57320-44, Shimadzu, South Africa) was used in order to assess the humeri tensile strength. Bones were positioned horizontally on two rounded supports, separated by 15 mm. A mediolateral force was applied at a constant rate of 3 mm/min until fracture. Load-displacement curves were generated, from which maximum force, break force, maximum displacement, and time to fracture were extracted.

Data analysis

The data were managed using Microsoft Excel, Office 365 (Microsoft Corporation ®), and analysed utilizing SPSS® Version 28 (IBM®). Multiple group comparisons of means fasting blood glucose, insulin levels, tensile strength and trabecular morphometric parameters were conducted using ANOVA with LSD post-hoc analysis. The significance level was set at a confidence interval of $p \leq 0.05$.

RESULTS

Fasting blood glucose

The terminal fasting blood glucose levels in the ALC group (76.68 mg/dL±14.98) was similar to untreated controls (mean=91.08 mg/dL±7.90) (p=0.960) (Fig. 2A). Conversely, the DB (244.8 mg/dL±30.79) and DB+ALC (249.6 mg/dL±109.73) groups had significantly higher terminal fasting blood glucose levels ($p<0.001$, for both groups compared to the untreated control) (Fig. 2A).

Serum insulin concentration

The ALC group's terminal serum insulin levels (1152 pg/mL±150.3) were not significantly different from those of the untreated control group (1251 pg/mL±47.2) (p=0.491) (Fig. 2B). In contrast, insulin concentration for the DB (771.2 pg/mL±146.2) and DB+ALC (693 pg/mL±52.87) groups was significantly below that of the untreated control group (p=0.011 and $p<0.001$, respectively) (Fig. 2B).

Osteometric measurements

Table 1. Osteometric parameters.

Parameter		Description
1	**Maximum length**	Superior most point of the head to point most distant from it, measured parallel to the shaft.
2	**Biomechanical length**	Superior most point of the head to distal most point on lateral lip of trochlea.
3	**Bicondylar breadth**	Medial most point of medial epicondyle to lateral most point of lateral epicondyle.
4	**Vertical head diameter**	The greatest distance between the margins of the head in a paracoronal plane.

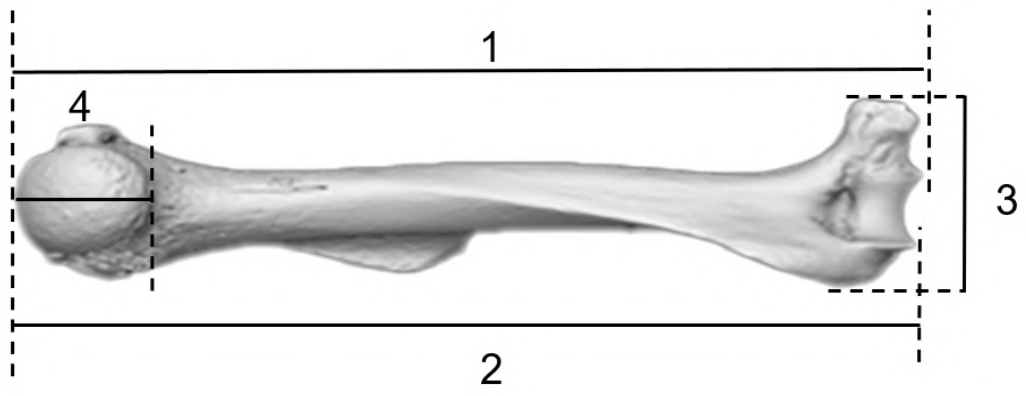


Fig. 1.- Osteometric measurements. Image showing measurements of the humerus taken with a digital caliper; maximum length (1), biomechanical length (2), bicondylar breadth (3) and vertical head diameter (4).

Maximum length

In comparison with the untreated controls, maximum bone length was shortest in the DB+ALC group and the DB groups (p=0.003 and p=0.017, respectively). Conversely, the maximum bone length in the ALC group was like that of the untreated controls (p=0.29) (Table 2). Moreover, the maximum bone length in this group (ALC) was similar to that observed in the DB and DB+ALC groups (p=0.169 and p=0.059, respectively). Additionally, the DB group had a similar length to that of the DB+ALC group (p=0.745) (Table 2).

Biomechanical length

When compared to the untreated control group, the biomechanical length was shortest for the DB+ALC and DB groups, (p=0.002 and p=0.014 respectively). In contrast, the ALC group displayed similar lengths to those of the untreated controls (p=0.282) (Table 2). This group (ALC) was sim-

Table 2. Osteometric measurements.

	UN		ALC		DB		DB+ALC	
Parameter	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
N	8		8		7		8	
[a]Maximum length (mm)	31.88	0.42	31.45	0.68	30.86	1.83	30.73	1.57
***p-value**			0.290		0.017		0.003	
[b]Biomechanical length (mm)	31.53	0.45	31.09	0.73	30.47	1.81	30.35	1.56
***p-value**			0.282		0.014		0.002	
[c]Bicondylar breadth (mm)	7.47	0.72	7.51	0.42	7.67	0.42	7.73	0.33
***p-value**			0.812		0.217		0.072	
[d]Vertical head diameter (mm)	5.50	0.31	5.76	0.39	5.56	0.47	5.71	0.32
***p-value**			0.051		0.680		0.077	

[a]Maximum length: ALC vs DB (p=0.169), DB+ALC (p=0.059); DB vs DB+ALC (p=0.745).
[b]Biomechanical length: ALC vs DB (p=0.146), DB+ALC (p=0.051); DB vs DB+ALC (p=0.764).
[c]Bicondylar breadth: ALC vs DB (p=0.315), DB+ALC (p=0.123); DB vs DB+ALC (p=0.695).
[d]Vertical head diameter: ALC vs DB (p=0.139), DB+ALC (p=0.711); DB vs DB+ALC (p=0.209). * Comparing each group to the untreated (UN). ALC, alcohol; DB, diabetic; DB+ALC, diabetes and alcohol.

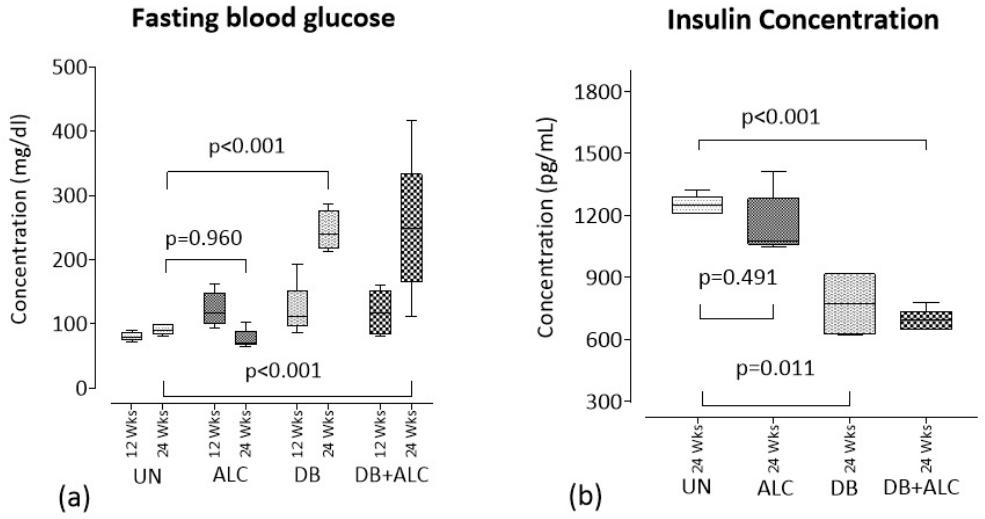


Fig. 2.- Fasting blood glucose and serum insulin concentration. Box-and-whisker plots illustrate a) Fasting blood glucose at 12 and 24 weeks, b) Serum insulin at termination. The box shows the range of values between 25th and 75th percentiles and the line in the box is the mean. Whiskers show the lowest and highest values, indicating the overall data range. Fasting blood glucose at 12 weeks: ALC vs DB (p>0.999), DB+ALC (p=0.999), DB vs DB+ALC (p=0.999). Fasting blood glucose at 24 weeks of age: ALC vs DB (p=0.001), DB+ALC (p=0.001), DB vs DB+ALC (p=0.993). Serum insulin concentration: ALC vs DB (p=0.057), DB+ALC (p=0.016), DB vs DB+ALC (p=0.651). UN, untreated; ALC, alcohol; DB, diabetic; DB+ALC, diabetes and alcohol.

ilar to the DB and DB+ALC groups (p=0.146 and p<0.051, respectively). However, the diabetic group (DB) did not exhibit a significant difference in biomechanical length in comparison to the DB+ALC group (p=0.764) (Table 2).

Vertical Head Diameter

The vertical head diameter was unaffected in all experimental groups, as the ALC (mean=5.76±0.39), DB (mean=5.56±0.47), and DB+ALC (mean=5.71±0.32) groups showed similarities to the untreated controls (mean=5.50±0.31) (p=0.051, p=0.680 and p=0.077, respectively). A similar vertical head diameter was observed between the ALC and the DB groups (p=0.139) (Table 2). The DB+ALC group was similar to the ALC group in length (p=0.711). The vertical head diameter in the DB group was similar to that of the DB+ALC group (p=0.209) (Table 2).

Bicondylar breadth

When compared to the untreated group (mean=7.47±0.72), the bi-condylar breadth was not affected in any of the groups; ALC (mean=7.51±0.42), DB (mean=7.67±0.42) and DB+ALC (mean=7.73±0.33) (p=0.812, p=0.217 and p=0.072 for all groups respectively) (Table 2). The ALC group had a similar bi-condylar breadth to that of the DB and DB+ALC groups (p=0.315 and p=0.123, respectively). This is also the case of the DB group than the DB+ALC groups (p=0.695) (Table 2).

Trabecular Morphometry

Bone volume fraction (BV/TV)

The DB, ALC, and DB+ALC groups exhibited a similar bone volume ratio (BV/TV) to that of the untreated control group (p=0.085, p=0.078, and p=0.119, respectively) (Table 3 and Figs. 3A-D). The DB and DB+ALC groups showed a similar bone volume ratio (BV/TV) to that of the ALC group, (p=0.984 and p=0.708). Additionally, the DB group exhibited BV/TV similar to that of the DB+ALC group (p=0.703) (Table 3).

Trabeculae thickness (TbTh)

Reduced trabecular thickness was observed in the DB+ALC, and DB groups compared to the untreated controls (p=0.022 and p=0.007, respectively). In contrast, the ALC group exhibited no significant difference compared to the untreated control group (p=0.359) (Table 3 and Figs. 3C and D). Moreover, similar trabeculae thickness was observed between the ALC and the DB or DB+ALC group (p=0.065 and p=0.189). Likewise, similar

Table 3. Trabecular morphometric measurements.

	UN		ALC		DB		DB+ALC	
Parameter	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
N	8		8		7		8	
[a]BV/TV	0.25	0.08	0.21	0.05	0.21	0.06	0.22	0.04
***p-value**			0.078		0.085		0.119	
[b]TbTh (mm)	0.32	0.09	0.28	0.10	0.20	0.05	0.23	0.10
***p-value**			0.359		0.007		0.022	
[c]TbN (numerals)	0.84	0.33	0.85	0.44	1.13	0.35	1.07	0.28
***p-value**			0.942		0.013		0.024	
[d]TbSp (mm)	1.06	0.55	1.06	0.39	0.77	0.32	0.79	0.30
***p-value**			0.982		0.092		0.073	

[a]BV/TV: ALC vs DB (p=0.984), DB+ALC (p=0.708); DB vs DB+ALC (p=0.703).
[b]TbTh: ALC vs DB (p=0.065), DB+ALC (p=0.189); DB vs DB+ALC (p=0.448).
[c]TbN: ALC vs DB (p=0.016), DB+ALC (p=0.029); DB vs DB+ALC (p=0.578).
[d]TbSp: ALC vs DB (p=0.088), DB+ALC (p=0.070); DB vs DB+ALC (p=0.910).
* Comparing each group to the untreated. ALC, alcohol; DB, diabetic; DB+ALC, diabetes and alcohol. BV/TV, bone volume fraction; TbTh, trabecular thickness; TbN, trabecular number; TbSp, trabecular spacing.

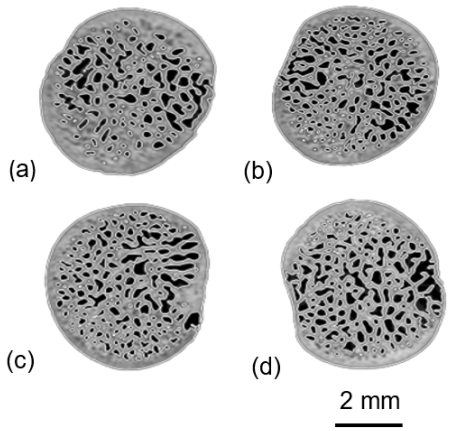


Fig. 3.- Micro-focus x-ray CT cross-sections of the proximal humerus: (a) Untreated control, (b) alcohol only (ALC), (c) DB and (d) DB+ALC. ALC, alcohol; DB, diabetic; DB+ALC, diabetes and alcohol.

trabeculae thickness was also observed between the DB and DB+ALC group (p=0.448) (Table 3 and Figs. 3C and D).

Trabeculae number (TbN)

The DB+ALC and the DB groups had significantly more trabeculae than the untreated control group (p=0.024 and p=0.013, respectively). Conversely, the ALC group did not show statistically significant differences in trabecular number compared to the untreated control group (p=0.94) (Table 3). However, this group (ALC) exhibited significantly fewer trabeculae than the DB and DB+ALC groups (p=0.016 and p=0.029, respectively). Regarding diabetes, the DB group exhibited similar trabeculae thickness to the DB+ALC group (p=0.578) (Table 3 and Figs. 3C and D).

Trabeculae spacing (TbSp)

No significance was detected in trabecular spacing (TbSp) when comparing the untreated control group to the DB+ALC and DB groups (p=0.073 and p=0.092 respectively). Additionally, similar trabecular spacing was observed between the ALC group and the untreated group (p=0.982) (Table 3 and Fig. 3). The group similarity trend continued when comparing TbSp in the ALC group to the DB and DB+ALC groups (p=0.088 and p=0.070, respectively). Moreover, the DB group exhibited similar trabecular spacing to the DB+ALC group

(p=0.910) (Table 3 and Figs. 3C and D).

Tensile strength

Maximum force

The untreated control group had a similar maximum force to the ALC, and DB groups (p=0.116 and p=0.111, respectively). However, the DB+ALC group had a lower maximum force than the untreated control group (p=0.012) (Table 4). The DB group and the DB+ALC group demonstrated similar maximum forces compared to the ALC group, with p-values of 0.939 and 0.421, respectively. Additionally, the DB group exhibited a similar maximum force to the DB+ALC group, with a p-value of 0.491 (Table 4).

Break force

The break force in the ALC and DB group was like that of the untreated control group (p=0.078 and p=0.135, respectively). In contrast, the DB+ALC group had a lower break force than that of the untreated control group (p=0.016) (Table 4). No break-force differences were detected when comparing the ALC group to the DB and DB+ALC groups (p=0.830 and p=0.628, respectively). Similarly, the DB group showed no break force differences when compared to the DB+ALC group (p=0.484) (Table 4).

Displacement

When compared to the untreated control group (mean=3 mm±0.90), the ALC, DB, and DB+ALC groups had a lower displacement value (p=0.001, p=0.019 and p<0.001) (Table 4). In contrast, the ALC group had displacement values similar to those of the DB and DB+ALC groups (p=0.348 and p=0.339, respectively). However, the DB+ALC group had significantly lower displacement values compared to the in the DB group (p=0.019) (Table 4).

Time to fracture

The time to fracture in ALC, DB and DB+ALC groups was significantly lower than that of the untreated control group (p=0.001, p=0.018 and p<0.001) (Table 4). Group similarities were observed when comparing the ALC group with the DB and DB+ALC groups (p=0.368 and p=0.312, respectively). However, the DB+ALC group exhibited significantly less time to fracture compared to the in the DB group (p=0.019) (Table 4).

DISCUSSION

The study investigated bone size, trabecular bone structural organisation, and bone strength of the humerus in diabetic rats consuming alcohol. The humerus was selected for this study because it is cited as one of the skeletal sites affected by alcohol (Saville, 1975) and diabetic skeletopathy (Inzerillo and Epstein, 2004). The hypoinsulinemia and hyperglycaemia recorded in the present study are consistent with a diabetic state.

Table 4. Tensile strength of the humerus.

	UN		ALC		DB		DB+ALC	
Parameter	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
N	8		8		7		8	
[a]Maximum force (N)	110.19	12.75	98.56	14.34	97.97	32.47	93.14	28.55
***p-value**			0.116		0.111		0.012	
[b]Break force (N)	109.73	13.59	96.00	17.84	97.72	32.39	92.58	28.87
***p-value**			0.078		0.135		0.016	
[c]Displacement (mm)	3.00	0.90	1.91	0.57	2.22	1.31	1.53	0.57
***p-value**			0.001		0.019		<0.001	
[d]Time to fracture (Sec)	59.99	17.92	38.42	11.25	44.33	26.13	30.66	11.50
***p-value**			0.001		0.018		<0.001	

[a]Maximum force: ALC vs DB (p=0.421), DB+ALC (p=0.939); DB vs DB+ALC (p=0.491).
[b]Break force: ALC vs DB (p=0.830), DB+ALC (p=0.628); DB vs DB+ALC (p=0.484).
[c]Displacement: ALC vs DB (p=0.348), DB+ALC (p=0.339); DB vs DB+ALC (p=0.019).
[d]Time to fracture: ALC vs DB (p=0.368), DB+ALC (p=0.312); DB vs DB+ALC (p=0.019).
* Comparing each group to the untreated. UN, untreated group, alcohol; DB, diabetic; DB+ALC, diabetes and alcohol.

Therefore, the present study successfully induced diabetes, as in previous studies (Wilson and Islam, 2012). In the diabetic states, the humerus showed a decrease in size, which was further worsened by alcohol. Diabetes alone (DB), or when confounded by alcohol intake (DB+ALC), disrupted trabecular bone organization, as indicated by a higher trabecular number (TbN) with reduced trabecular thickness (TbTh) in these animals (DB, DB+ALC). Additionally, the study also found that diabetes reduced bone strength.

Measures of bone size

In the current study, shorter bone lengths were found in the diabetic group (DB). This finding corroborates previous studies on Zucker Diabetic Sprague Dawley (ZDSD) rats (Reinwald et al., 2009) and KK-Ay Mouse (Xu et al., 2014), which reported shorter bones among diabetic animals. When compared to the untreated control, the diabetic animals that consumed alcohol (DB+ALC) exhibited shorter bones. Although there are no comparable studies that investigated the combined effects of chronic alcohol consumption and diabetes on bone, these findings are aligned with scientific reports on the individual effects of alcohol and diabetes on bone length (Xu et al., 2014). The bone length reduction reported in the present study may be a consequence of reduced mineral density emanating from the detrimental effects of diabetes on bone (Zhang et al., 2022), compounded by the loss of bone mineral density caused by alcohol (Maurel et al., 2011).

The lack of significant differences in the vertical head diameter and the bicondylar breadth while the length was reduced in diabetes animals (DB) and diabetic animals consuming alcohol (DB+ALC) suggests that appositional growth in the epiphyseal ends of the bone may have been spared. However, bone longitudinal growth (length) in the diaphysis is most likely affected due to its dependence on the epiphyseal growth plate. In alignment with this proposition, a recent study in gestational alcohol exposure reported that alcohol inhibited the expression of transforming growth factor beta-1 in the growth plate (TGFB-1) (Pillay et al., 2024); as this cytokine (TGFB-1) is known to promote bone growth (Rahman et al., 2015), it is likely, that alcohol in the present study may have disturbed the TGFB-1 pathway, thereby delaying bone shaft lengthening. Similarly, another study, reported increased expression of TRAP in diabetic rats accompanied by shorter bones (Tolosa et al., 2013).

Trabecular Morphometry

The current study showed no significant changes in bone tissue ratio (BV/TV) and trabecular spacing (TbSp) across the groups. These findings are aligned with previous reports on the detrimental effects of diabetes (Kawashima et al., 2009) and alcohol (Maurel et al., 2012) on trabecular morphometric parameters. Both the diabetic (DB) and diabetic with alcohol (DB+ALC) groups showed increased trabecular number (TbN), indicating disrupted trabecular organization and porous bone. This disruption in humeral trabecular architecture possibly contributes to the increased fracture risk observed with diabetes and alcohol. Trabecular thickness (TbTh) in the DB+ALC group reflected influences from both alcohol and diabetes, showing similarities to both the ALC and DB groups.

Bone tensile strength

Both the maximum and break forces were low in diabetic rats taking alcohol (DB+ALC). This means that these bones require less force to be weakened and subsequently fracture. Therefore, these findings show the adverse effects of the multi-morbidity of alcohol consumption among diabetics. Our results support the known phenomenon of increased susceptibility to bone fracture among diabetics (Vilaca et al., 2020) and alcoholics (Sampson, 1998).The alcohol only (ALC) rats, diabetic rats (DB), and diabetic rats with alcohol consumption (DB+ALC) displayed a reduction in break time and displacement. When this is considered together with the observed decrease in maximum and break force in diabetic rats taking alcohol (DB+ALC), it shows that alcohol reduced bone strength in diabetic rats. Furthermore, the lower displacement and time to fracture values in the diabetic rats simultaneously taking alcohol (DB+ALC) in comparison to the diabetic group (DB) illustrates the exacerbated effects of alcohol

consumption in diabetic animals on bone integrity. Viewed together, the tensile strength findings demonstrate that excessive alcohol consumption exacerbate bone weakness among diabetics.

Limitations of study

Only male rats were used in the current study; therefore, the results should be interpreted with caution when applying them to both sexes.

CONCLUSION

This study demonstrates significant alterations in trabecular morphometry, bone strength, and biomechanical length across groups, with diabetic rats, especially those exposed to alcohol, exhibiting the most pronounced changes. Diabetes alone compromised bone health, and alcohol consumption further intensified these negative effects. These findings underscore the need for targeted interventions and further research to understand and mitigate skeletal fragility in diabetic individuals who consume alcohol.

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