



Article

Biomechanics of Bruxism Potentially Determine the Sites of Severe TMJ Osteoarthritis

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Abstract: The objective of this study was to assess the osteoarthritis (OA) disease severity in 47 temporomandibular joints (TMJs) using a validated scale for gross signs of OA while noting the specific sites for profound disease on the donor condyle and fossa. A disease severity score of Grade 0–4, representing absent to severe disease, was awarded to each specimen's condyle and fossa by two blinded investigators who have demonstrated interrater reliability. The mandibular fossa was more pathological compared to the mandibular condyle (* $p = 0.001$). When the deepest focal lesions were qualitatively assessed, it was demonstrated that the mandibular fossa was more severely degenerated than the articular eminence in 58% of donors. In this subpopulation, 74% of the severe mandibular fossa pathology was seen on the deep articular surface. When the articular eminence was the most severely degenerated region of the fossa, it was equivalently likely to see severe focal lesions on the lateral eminence (35%) or equally distributed across the entire eminence (35%). The greatest disease severity was discovered in sites of overloading, which may be associated with paranormal mandibular movements and potentially bruxism. Patients with bruxism produce significant translational movements (grinding) in the upper joint compartment and heavy vertical loading (clenching). Theoretically, this amplifies pressure and inflammation on the lateral articular surfaces and in the deep fossa.

Keywords: temporomandibular joint; mandibular fossa; osteoarthritis; bruxism; cartilage degeneration



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1. Introduction

Temporomandibular joint disorders (TMDs) are the second most common musculoskeletal condition after lower back pain and may affect 5–12% of the population [1–3]. Osteoarthritis (OA) is the most common degenerative joint disease and is characterized by chronic degeneration of hard and soft tissues of the synovial joints. OA was believed to be exclusively related to cartilaginous “wear and tear”, but the complex pathogenesis is now better understood to be multifactorial and strongly correlated to altered inflammatory signaling and remodeling processes in the joint [4–7]. OA commonly presents in the temporomandibular joint (TMJ) and may cause symptoms such as joint pain, pain in the muscles of mastication, decreased range of mandibular motion, and other functional deviations; these symptoms classify the presentation of OA in the TMJ as a joint disorder TMD [3,8].

TMDs and chronic bruxism may produce irregular biomechanical alterations in the TMJ, which lead to OA by altering cellular communication in the cartilage and joint tissues [6–9]. Bruxism is a common parafunctional movement disorder characterized by grinding and clenching of the teeth, occurring regularly in adults during sleep. With respect to mandibular biomechanics, parafunctional movements are distinct from typical

occlusal stress exerted during mastication and swallowing. Movements of bruxism are non-functional oromandibular or lingual activities that include jaw clenching, tooth grinding or tapping, and biting of the cheeks, lips, or outside objects. Frequent sleep bruxism occurs in approximately 13% of the adult population. Awake bruxism is most prevalent in females compared to males, whereas sleep bruxism demonstrates no gender preference [10,11].

Bruxism develops chiefly due to pathophysiological and psychosocial factors. Disturbances in central neurotransmitter systems and the use of SSRIs (serotonin reuptake inhibitors) have been shown to be involved in the etiology of bruxism [11]. Disturbances in sleep represent a prominent pool of pathophysiological factors contributing to bruxism. Depression and increased levels of hostility, stress, and sensitivity have been shown to be psychosocial contributors to parafunctional bruxism movements [10,11]. Bruxism requires no treatment but often requires clinical management when problematic symptoms arise due to the non-functional bruxism habits [10]. Many patients experiencing bruxism or other TMDs producing abnormal biomechanics at the TMJ and can become vulnerable to the development of OA and its painful and function-limiting symptoms.

A previous study conducted by investigators drew conclusions on the comparable nature of OA disease severity in the TMJ and the knee joint in a cadaveric model. This was an unexpected finding given the TMJ is not a true weight bearing joint nor does it have the same histological cartilage type as the knee lining its articular surfaces. Based on a validated disease severity scale, cadaveric mandibular fossae of the TMJ developed statistically equivalent degeneration compared to donor-matched femoral condyles [8]. After this study, it was hypothesized that the human bite force, shown by Zhou and Ye (1994) to be 266 lbs at the first molar [12], was great enough to aid in the production of similar cartilage erosions and soft tissue changes in the TMJ as degenerations seen in knee joints of elderly cadaveric donors despite the resistant fibrocartilage phenotype of the TMJ articular surfaces. These findings drove the investigation to determine the specific sites of cartilage degeneration on the TMJ mandibular fossa and mandibular condyle.

There is a lack of educational emphasis on the TMJ in cadaveric dissection courses nationwide. Our team hypothesizes that the lack of educational emphasis on TMJ regional anatomy and biomechanics explains why the research on TMJ OA is so diminutive in the medical and dental literature pool. The objective of this study was to assess OA disease severity in a cohort of 47 cadaveric TMJs using a validated scale for OA gross symptoms while noting the specific sites for profound disease severity on the donor condyle and fossa. Once sites of disease severity were noted across the donor population, trends were evaluated to help determine which biomechanical activities or specific TMDs appeared to be most damaging to patient TMJ anatomy.

2. Materials and Methods

2.1. Cadaveric Dissection and Accrual

Forty-seven intact TMJs from 25 cadaveric donors were harvested using classical dissection from skulls with calvaria and brains removed. The donor population was approximately 50% males and 50% females and between the ages of 69–107 years old. The TMJs were disarticulated from the skull by first removing the zygomatic arch, temporalis, and masseter muscles. The neck of the mandible was transected with an autopsy saw using a lateral approach, and the lateral pterygoid muscle was removed from its insertions on the pterygoid fovea, TMJ capsule, and articular disc. Two frontal plane cuts were made using external palpation landmarks at the anterior and posterior margins of the mandibular fossa of the temporal bone. Frontal plane cuts were extended medially to the depth of the foramen spinosum in the middle cranial fossa. Donors were obtained from the University of California Irvine Body Donation Program and the University of California San Diego Body Donation Program. This project (IRBNet ID 1681548-1) was reviewed by University of Nevada Las Vegas' Institutional Review Board in Las Vegas, NV, and was determined to be research not involving human subjects.

2.2. TMJ Photography

Each TMJ was photographed using a 12-megapixel iPhone 13 Pro camera. Each image included a drafting ruler for calibration. First, the TMJ condyle was photographed from the anterior, medial, lateral, and posterior views. Each condyle specimen was photographed on two different backgrounds to allow for better interpretation of the gross signs of OA. After disarticulation from the temporal bone, the mandibular fossa was placed on the benchtop and photographed from above on two different backgrounds, ensuring a clear view of the deep articular fossa and the articular eminence.

2.3. Utilization of the OA Disease Severity Scale

Articular cartilage lesioning was investigated on specimen mandibular condyles and mandibular fossae based on classifications used in Kovler et al. (2004) [13]. A validated OA disease severity scale was utilized to classify each joint surface as Grade 0–Grade 4 OA severity [13,14]. This OA disease severity scale has been used and validated in an array of synovial joints, including the TMJ [8]. “Grade 0 OA” represents “No OA,” this grade of disease severity has yet to be demonstrated in an elderly cadaveric donor TMJ to date. “Grade 1 OA” represents “Questionable OA”, where cartilage contour and exceptionally minor degenerative changes are indistinguishable. Fibrillations, superficial pitting, fraying, and splitting of the articular cartilage is representative of “Grade 2 OA” (Mild OA). Extensive ulceration or cartilage loss represents “Grade 3 OA” (Moderate OA). Specimens with large areas of complete cartilage degeneration accompanied by exposure or eburnation of bone is indicative of “Grade 4 OA” (Severe OA). Visible, gross alterations to the cartilage such as linear cracks or articular cartilage erosions could be associated with mild, moderate, or severe OA depending on the depth and coverage of damage [8,15–18].

2.4. Assessment of Gross Signs of OA and Donor Biomechanics

In previous investigations, chondral lesions were assessed using the validated scale for OA disease severity on the mandibular fossa and mandibular condyle without regard for the specific, focal sites of degeneration. Specific regions on specimen condyles and fossae were noted in this study as many presenting cartilage lesions do not cover the entirety of an individual specimen and may vary based on lesion etiology. The deepest focal lesions on specimen mandibular condyles can be assessed to present on the medial condyle, lateral condyle or across the entirety of the condyle. The deepest focal lesions on specimen mandibular fossae can be assessed to present on the articular eminence or on the true, more posteriorly located, fossa. The articular eminence may demonstrate its most severe signs of pathology on the medial aspect, the central aspect, the lateral aspect, or across the entirety of the articular eminence. The true mandibular fossa may demonstrate its most severe signs of pathology on the deepest aspect of the fossa, the lateral periphery, the medial periphery or on the entirety of the fossa (Figure 1). Previous investigations determined that the mandibular fossa demonstrates statistically more severe signs of OA pathology compared to the condyle [8]. Given the complex biomechanics of the TMJ, the articular eminence and articular fossa were further assessed in the consideration of their subregions.

To varying extents, all patients participate in the parafunctional activities that define bruxism, and, often, no clinical management is required. Bruxism is a diagnosis that considers more than the physical presentation of chondral lesions; a patient’s clinical history is taken into account. Given this consideration, the study assessed the biomechanics of parafunctional clenching and grinding activities that may generate overloading in the joint space and elicit signaling cascades associated with OA. Therefore, the assessment of the biomechanics was evaluated simultaneously with the assessment of focal, gross signs of OA on donor articular surfaces. The same focal lesions on the mandibular condyle (medial and lateral aspects) and mandibular fossa (articular eminence and mandibular fossa) of the temporal bone were evaluated. The articular eminence was further evaluated by reviewing the medial, central, and lateral regions. The true mandibular fossa was further evaluated

by reviewing the medial periphery, central/deep fossa, and lateral periphery. Given the mechanism of pathogenesis of OA, sites of fibrillation, pitting, erosion, and cortical bone exposure represent regions of biomechanical overloading. This overloading was then correlated with a potential bruxism diagnosis.

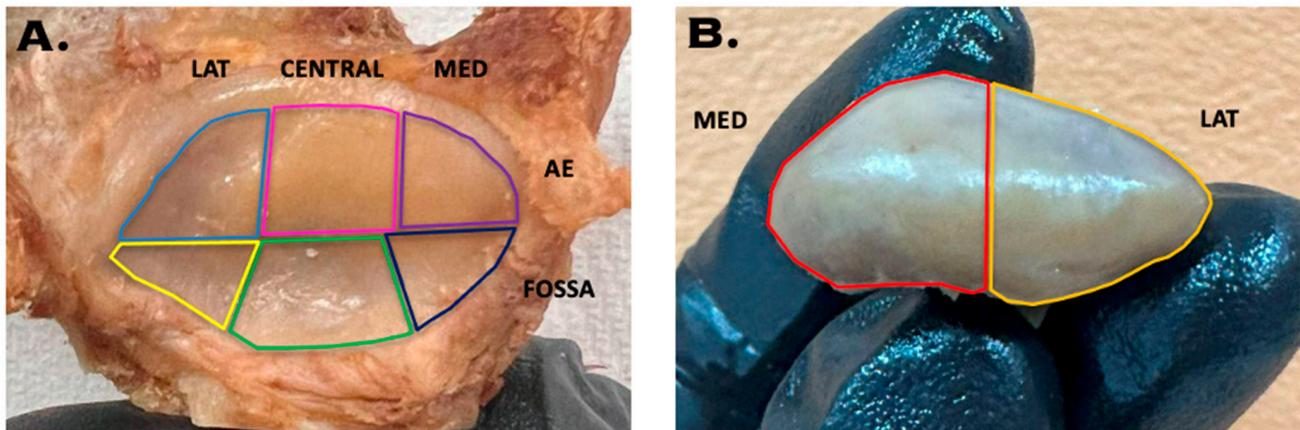


Figure 1. The focal regions of assessment on the articular surfaces of the TMJ. (A) Represents the focal regions of the articular eminence (top of image) and mandibular fossa (bottom of image). Blue, magenta, and purple traces represent the focal sites of assessment on the lateral, central, and medial articular eminence, respectively. Yellow, green, and navy traces represent the focal sites of assessment on the lateral, central/deep, and medial mandibular fossa, respectively. (B) Represents the focal regions of the mandibular condyle. The superior view of the condyle demonstrates the greatest proportion of the articular cartilage.

The validated disease severity scale was used by two blinded reviewers to assess the 47 TMJ specimens involved in this study. This is the first time the scale was utilized by a dental professional to score pathological specimens; previous studies conducted by our team left all specimen rating responsibilities to clinical anatomists with greater exposure to damaged cadaveric joint cartilage. Interrater reliability assessments were conducted to ensure raters were producing comparable scores on donor joints. Raters indicated the disease severity score for the mandibular condyle and mandibular fossa. Finally, a team of 4 healthcare professionals assessed and came to an agreement on the site of the most profound disease on the condyle or fossae.

2.5. Statistics

Statistical analysis was conducted using SPSS statistics software (version 27, IBM Corp, Chicago, IL, USA). Cohen's kappa test was used to estimate the interrater reliability of the two OA raters' condyle and fossa disease severity scale (DSS) scores. A score of 0.41–0.60 indicates moderate interrater reliability; 0.61–0.80 indicates substantial; and 0.81–1.00 indicates almost perfect agreement. Condyle and fossae cartilage disease severity ratings were compared using a paired two-sample *t*-test. A sample size calculation was performed to determine if the population size was adequate to compare interrater reliability between two raters at a 95% confidence level.

3. Results

3.1. Interrater Reliability Assessment and Sample Size Calculation

The disease severity scale utilized from Kovler et al. (2004) has been validated in an array of synovial joints that commonly experience OA [13,14]. Investigators involved in this project have previously validated the use of this scoring scale in the hands of doctoral-trained, clinical anatomists. This is the first investigation to incorporate clinicians—specifically, doctoral-trained dental professionals—into the blinded rating protocol for specimen review. Interrater reliability was demonstrated using Cohen’s kappa test when comparing the fossa and the condyle scores between the two blinded reviewers, one clinical anatomist and one licensed dentist. Kappa values were calculated at $K = 0.759$ and $K = 0.690$ for the interrater reliability for scoring on the fossae and the condyles, respectively. These values represent “substantial” interrater reliability based on the Cohen’s Kappa test classification system. After a sample size calculation was performed, it was determined that the population is adequate (>43 specimens) to compare interrater reliability between two raters at a 95% confidence level.

3.2. Condyle and Mandibular Fossa Disease Severity

In this 47-specimen population, the mandibular fossa was significantly more degenerated compared to the mandibular condyle ($p = 0.0001$; Figure 2). Figure 2 demonstrates an example donor where mandibular fossa degeneration was more severe than that of the donor-matched condyle. Figure 1A represents a donor fossa that was rated Grade 4 (Severe) OA, and Figure 1B represents a donor-matched condyle that was rated Grade 3 (Moderate) OA. This assessment was conducted based on a single rater’s disease severity scores opposed to averaged scores between the raters. The mandibular fossa received an average disease severity score of 3.36 ± 0.58 , and the mandibular condyle received an average disease severity score of 3.06 ± 0.93 . Only one specimen demonstrated more severe signs of degeneration on the condyle compared to the mandibular fossa of the temporal bone (1/47, 2%; Figure 3A).

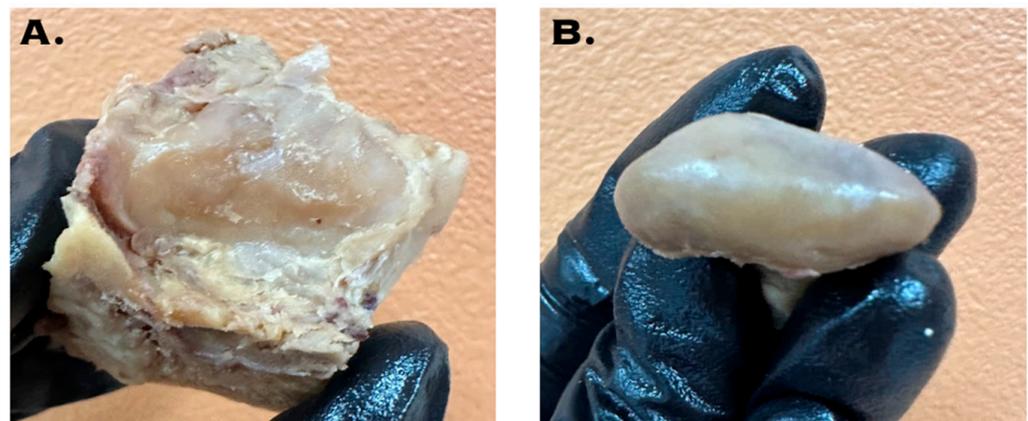


Figure 2. Example donor-matched mandibular fossa (A) and mandibular condyle (B) from a right TMJ. Specimen demonstrates preferential degeneration of the mandibular fossa compared to the condyle. Image (A) represents Grade 4 (Severe) OA and image (B) represents Grade 3 (Moderate) OA.

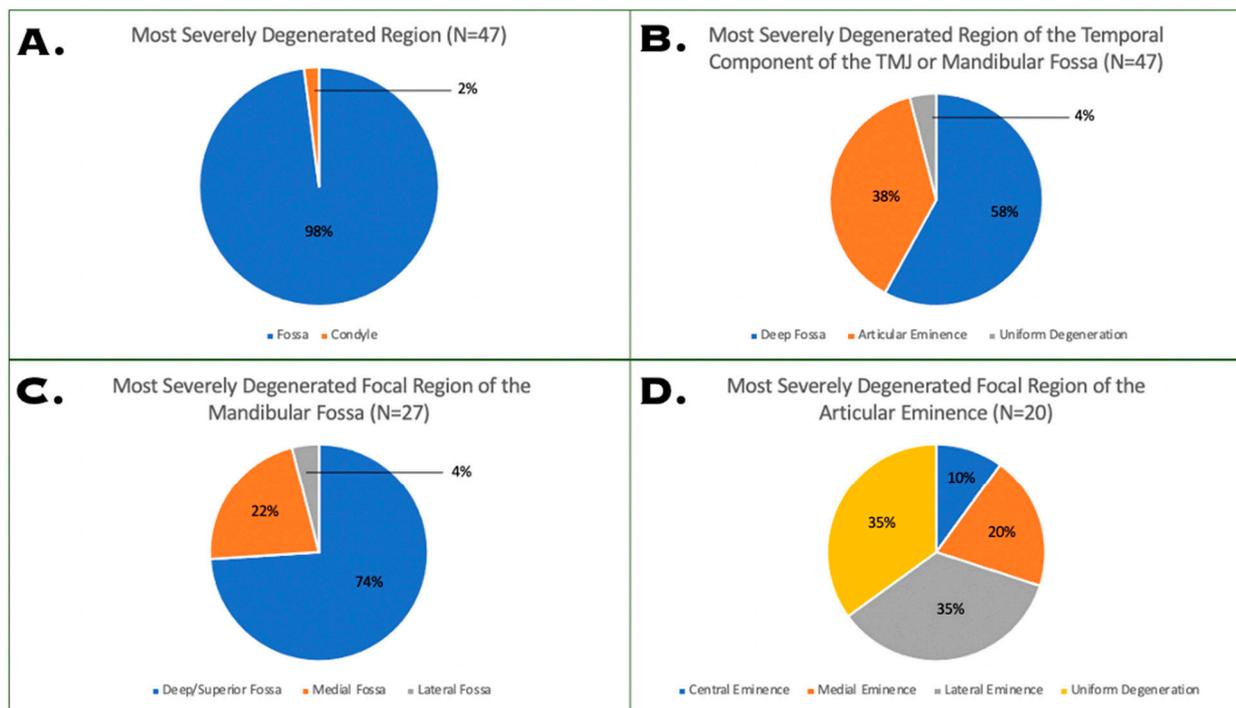


Figure 3. Histogram representations of the regions and focal regions of degeneration on the mandibular fossa. (A) Histogram representing the site of greatest OA severity, written as percentages (N = 47), for the mandibular fossa (blue) and mandibular condyle (orange). (B) Histogram representing the site of greatest OA severity on the components of the mandibular fossa (deep fossa, medial fossa, lateral fossa). (C) Histogram representing the focal regions of the superior mandibular fossa of greatest OA severity. This evaluation only considered the specimens where the mandibular fossa was determined to be the most degenerated component of the TMJ (58%; n = 27). (D) Histogram representing the focal regions of the articular eminence with the greatest OA severity. This evaluation only considered the specimens where the articular eminence was determined to be the most degenerated component of the TMJ (38%; n = 20).

3.3. Focal Lesion Disease Severity

When the specific regions of the mandibular fossa were qualitatively assessed for the most severely degenerated focal sites it was demonstrated that the mandibular fossa was more severely degenerated than the articular eminence in 58% of donors (Figure 3B). In addition to the assessment of fibrillations, cartilage erosions, bony exposure, or eburnation, the surface area coverage of degenerations across the fossa or the articular eminence were considered when determining which focal site demonstrated the highest degree of severity. In the subpopulation with more pathological mandibular fossa compared to the articular eminence, 74% of the severe mandibular fossa pathology was seen on the deep articular surface opposed to the medial or lateral periphery of the fossa (Figure 3C). When the articular eminence was the most pathological region of the fossa, it was equivalently likely to see severe focal lesions on the lateral eminence (35%) or equally distributed across the entire eminence (35%; Figure 3D).

Figure 4 demonstrates a sample specimen from the subpopulation of donors with the most severe focal site of degeneration located on the deep mandibular fossa compared to the articular eminence. Images A–C are representative donor fossa and condyle from a 94-year-old female. These images demonstrate Grade 4 (Severe) OA on the fossa and condyle. The condyle of this donor demonstrated a deep defect on the posterosuperior aspect of the condyle, which is not pictured. Image B highlights the approximate surface area assessed for the articular eminence (red) and the deep mandibular fossa (yellow). Dashed lines represent the approximate regions of greatest disease severity on the articular

cartilage of each region. Deep bone exposure and eburnation are demonstrated within the fossa whereas only shallow erosions are present on the articular eminence. Images D–F are representative donor fossa and condyle from a 69-year-old female. These images demonstrate Grade 4 (Severe) OA on the fossa and Grade 2 (Mild) OA on the condyle. Image E highlights the approximate surface area assessed for the articular eminence (red) and the deep mandibular fossa (yellow). This image clearly demonstrates substantial cartilage erosions in the deep fossa (yellow dashed region) and only mild fibrillations on the eminence (fibrillations present across eminence). The condyles present with lesser degeneration compared to the fossa in each case; the 69-year-old’s condyle is scored as mild, and the extreme severity of OA seen on the 94-year-old’s fossa is evidently worse than the severity on the condyle despite receiving the same Grade 4 rating.

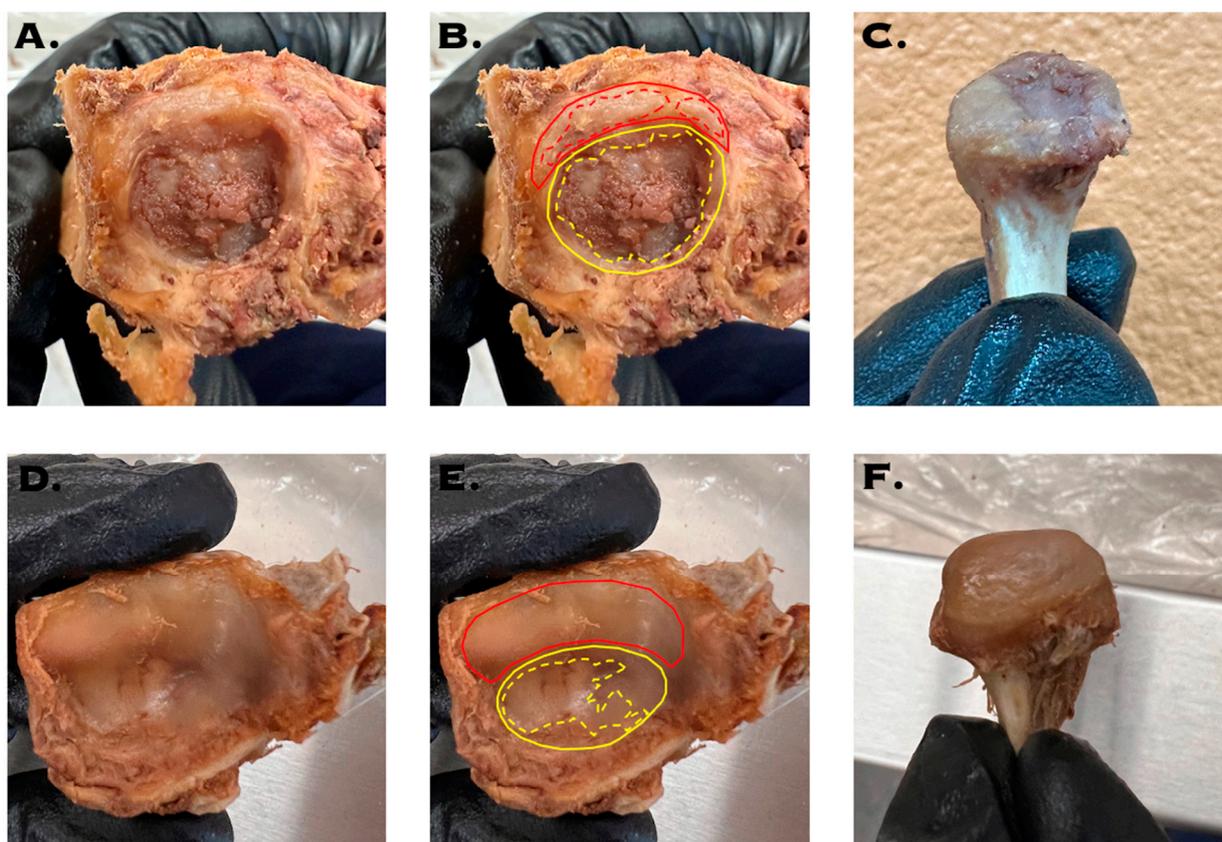


Figure 4. Sample donor images from the subpopulation of specimens with the most severe focal sites existing on the deep mandibular fossa opposed to the articular eminence. Images (A–C) are photographs of the TMJ anatomy of a 94-year-old female donor. Images (D–F) are photographs of the TMJ anatomy of a 69-year-old female donor. The approximate margins of the articular eminence (red) and mandibular fossa (yellow) are traced in a solid line. The most severe cartilage defects are traced in each mandibular fossa with a dashed line (yellow).

There were no focal sites on the condyle that appeared to be most prone to severe degeneration. There were two condyle specimens that received “Grade 1” scores for disease severity, implying it is questionable as to whether there is even OA pathology present. These specimens were excluded from frequency data on condylar cartilage degeneration. Out of 45 specimens, 19% demonstrated the most severe focal pathology on the medial condyle (8/45), 33% on the lateral condyle (15/45), and 48% uniformly across the frontal plane of the mandibular condyle (21/45). There was a slight partiality to see the lateral side of the condyle demonstrate the most severe signs of OA on the articular surface.

4. Discussion

Given the frequency of TMDs it is interesting there is such sparse research occurring on TMJ OA, especially in cadaveric joints. The TMJ is often left out of foundational cadaveric dissection courses in doctoral healthcare programs in the United States so investigators of TMDs or OA typically do not have the experiences needed to move their work into the cadaveric arena. This study is novel, being based on the deeper visualization and assessment of cartilage defects compared to the more plentiful radiological studies currently in the literature pool.

It is hypothesized that the mandibular fossa demonstrates greater disease severity compared to the mandibular condyle based on the anatomy of the upper joint compartment. This joint compartment is responsible for the translational movements of the TMJ whereas the lower joint compartment is responsible for the hinge joint activity. The upper joint compartment lacks collateral ligament reinforcement and possesses capsular connections that are laxer compared to the lower joint compartment [19]. This anatomy and its biomechanical effects are evident in dissection based on the fact the articular disc always stays more tightly adhered to the condyle with joint disarticulation. Laxity of the upper compartment creates an environment where biomechanical alterations are achievable.

In normal chewing, and to a greater degree in patients with bruxism, there are significant lateral, translational movements occurring in the upper joint compartment [8]. Clenching, also seen with bruxism, amplifies pressure and potentially inflammation in the deepest regions of the mandibular fossa [10,11]. In a population with such a high average disease severity score (mandibular fossa = $3.36 \pm 0.58/4$; mandibular condyle = $3.06 \pm 0.93/4$) for TMJ OA, it is understandable why deep and asymmetrical fossa degeneration is a trend. Demonstration of asymmetrical degeneration is parsimoniously indicative of paranormal translational movements and biomechanical loading in the TMJ.

Several early, hallmark studies on TMJ morphometrics found that OA predominantly affected the lateral aspect of the TMJ [20–26]. After reassessing the same 115 TMJs from Oberg et al. (1971), Hansson and Oberg hypothesized that the lateral localization of degeneration is based on the anatomy and biomechanics of the TMJ. The TMJ condyle is located lateral to the temporal component of the joint and the lateral pole of the condyle lies superior to the medial pole of the condyle; this stresses the lateral aspect of the joint preferentially [26]. Additional sources are also in agreement regarding biomechanical causes of lateral-side degeneration. The 42nd Edition of Gray's Anatomy discusses how the human mandible uses lateral movement to create shearing force that "enhances the effectiveness of the power stroke of mastication". Bruxism is also associated with lateral movements of the jaw, but with significantly increased bite forces [27]. In finite element analyses of the TMJ, both Perez del Palomar and Doblare (2006) and Sagl et al. (2022) found that lateral movements of the jaw generated the highest loads on the lateral portions of the joint, especially the lateral aspect of the fibrocartilage disc [28,29].

Lateral focal OA severity in the TMJ appears to be a well-supported trend, although studies—including this one—have discussed specimens that demonstrate pathology uniformly across the articular surfaces of the TMJ or pathology preferentially expressed on the medial aspect of the joint. In a study assessing bilateral joints from single donors (N = 22), data showed that 41% of edentulous donors showed different pathology bilaterally. The most common location for focal OA severity may often be the lateral aspect of the joint but the contralateral joint could reveal OA findings on the medial and central parts. The concept of transversal shift of the mandible is considered in donors where asymmetrical signs of OA are seen on dissection and are due to chronic overloading. This concept indicates that chronic overloading occurring in the lateral joint space would be accompanied by medial and/or central overloading in the contralateral joint [30].

Data showed that lateral degeneration on the articular eminence was seen in 35% of specimens. This is the largest individual site of severe OA lesioning and is equivalent in prevalence to specimens showing uniform degeneration across the entire eminence. Figure 5 demonstrates an example mandibular fossa with preferential degeneration on the

lateral side of the articular eminence (A) and equivalent wear across the entire articular eminence (B). Additionally, the remaining donors demonstrated medial degeneration (20%) or central degeneration (10%) as the site of most severe OA lesioning on the articular eminence (Figure 3D). The population may show this breakdown due to the biomechanics of transversal shift; one TMJ heavily degenerates laterally, while the contralateral joint degenerates medially and centrally. Considering the fact that the majority of donors contributed bilateral TMJs to the study, this breakdown also helps assure investigators that bruxism is the most likely initiator of OA in the joint space. There are only three ways to chronically overload the TMJ: internal derangement, transversal shift, and hypertrophy of the muscles of mastication [30]. Two of the mentioned mechanisms of chronic overloading (transversal shift and muscular hypertrophy) point to bruxism as a cause of overloading and generation of the altered inflammatory signaling seen in OA patients. Transversal shift is glaringly present in patients that grind their teeth, whereas muscles of mastication hypertrophy with chronic clenching of the teeth. Beyond these convincing biomechanical justifications pointing to bruxism as the underlying cause of OA, bruxism is also wildly more common compared to TMJ trauma, disc pathologies, or systemic metabolic, inflammatory, and autoimmune diseases. Bruxism is the most parsimonious explanation for the disease findings.

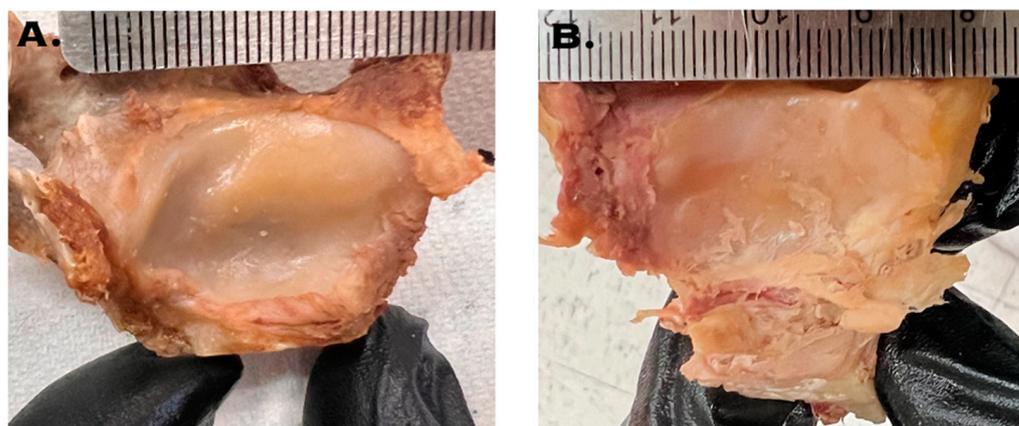


Figure 5. Example mandibular fossa with preferential degeneration on the lateral side of the articular eminence (A) and equivalent wear across the entire articular eminence (B). Image (A) represents a right mandibular fossa and image (B) represents a left mandibular fossa.

Further investigation should be carried out to determine if preferential wear on the lateral aspect of the eminence is more common than medial or central wear based on morphometrics of the fossa. Figure 6 exhibits stark contrast in the inclination of the articular eminence in two arthritic TMJ specimens. Anatomical variation of the fossa depth, eminence inclination, overall fossa width, and the ratio of fossa to condyle width may be contributing to lateral eminence wear in a greater percentage of the population.

Cohen's kappa test produces stronger scores on the mandibular condyle when assessing the most recently dissected subpopulation (N = 16 TMJs, eight donors). A kappa value of $K = 1.0$ was achieved in the assessment, representing perfect agreement between raters' scorings. Our team identified several limitations that prevent perfect interrater reliability on every assessment. A major limitation was the variable extent of tissue drying that occurred on a specimen based on how long it remained in the donor prior to dissection. Tissue drying was also accelerated if the joint capsule was opened and the rating of the cartilage degeneration on the fossa or condyle was delayed. There were a variable number of photographs taken of each specimen, which made the review and rating of the donor TMJs variable between the two raters; different images may have been more deeply scrutinized by each reviewer. The background on which the specimens were photographed was not always consistent. For the subpopulation of specimens from the most recent cohort

of donors, dissections were performed earlier to prevent drying, and photography was conducted on two different backgrounds. Photography was also made more regimented by photographing the condyle from medial, lateral, anterior, posterior, and superior views as opposed to a random mix of the mentioned views with additional oblique views that highlight pathologies that were apparent when handling the specimen.



Figure 6. Sample paired mandibular fossae from two different donors. (A) represents anatomical variation of the articular eminence where the inclination is more gradual or less steep. This donor demonstrates Mild OA based on visible fibrillations. (B) represents anatomical variation of the articular eminence with a greater inclination. This donor demonstrates Severe OA based on larger cartilage erosions and bony exposures.

This study has additional limitations that warrant mentioning. It is challenging to acquire cadavers for research purposes so accumulating a sizeable donor pool takes time; investigators aim to expand the donor pool in future undertakings. It is also challenging to acquire donors younger than 60 years old. Body donor programs for education and research typically accept donors that are ineligible for tissue and transplant donation based on their age and the state of their organs. The age range included in the specimen population is a limitation in the study and leaves investigators inquiring into the pathogenesis of TMJ OA in more youthful patients. Longitudinal studies on living patients should be carried out and findings should be used to complement the results from the study at hand. This could allow for a clearer assessment of patient biomechanics and may elucidate whether bruxism is the concrete diagnosis for the prevalent signs of disease present at sites of overloading within the joint space. All donors used in the educational dissection laboratory where the study was carried out were included in the study; selection for donor distribution is handled by the body donor programs and is random. By including all available specimens, potential selection bias is decreased.

There is a minor limitation in attributing the articular cartilage pathology to TMJ OA. As previously discussed, there are only three ways to develop chronic overloading in the TMJ and two of the three (transversal shift and muscular hypertrophy) point to paraneural bruxism activities. The body donor program provides a clinical history for each donor with primary and secondary causes of death and any additional history a patient may

be interested in disclosing. No clinical histories associated with our donor pool indicate additional pathologies or injuries that would have encouraged rapid degeneration of the TMJ and its supportive anatomy such as metabolic bone diseases, trauma, autoimmune conditions (ex. rheumatoid arthritis), internal derangement syndromes, or connective tissue diseases. Even if left off of a donor's clinical history, these health issues are rare. Some investigators choose to hedge their bets on this topic and use terms such as "osteoarthritis" or "degenerative joint disease" in place of TMJ OA when assessing morphometric changes to the articular cartilage. This team of investigators prefers to apply the principle of parsimony and the likely pathogenesis to our interpretation of findings for maximum impact. Previous publications cite that the fields of OA and orthopedics research support classifying focal lesions on the knee joint articular cartilage as OA opposed to other various degenerative joint diseases [9,14]. This cadaveric research program on the TMJ stems from our investigations of the knee joint, so a similar justification is applied to the TMJ cartilage.

As a future direction, investigators plan to assess the wear patterns and other dental findings of the donors incorporated in this study. Abe et al. (2009) found that total tooth wear scores showed modest diagnostic value in discriminating sleep bruxers from the control group in young adults [31]. The presentation of generalized tooth wear would further affirm that paranormal biomechanics associated with bruxism are driving the cartilage degeneration seen in the TMJ. Identifying specific sites of disease severity in the TMJ will help improve the clinical management of patients experiencing joint pain, orofacial pain, and decreases in TMJ range of motion, which can ultimately lead to malnutrition, metabolic pathologies, and reduced quality of life. This study's findings suggest that macroscopic evidence of TMJ OA is common in the elderly population. Awareness of this issue can lead dental professionals to place more importance on screening for TMDs. By increasing attention to signs and symptoms of parafunctional habits such as bruxism, early detection and appropriate management can be accomplished. Patients may benefit from reversible, conservative treatment options earlier in life. Taken with future clinical investigations and larger-scale cadaveric studies, these findings may help elucidate common focal sites of TMJ OA. Clinicians may work and prescribe to preliminary curve behaviors leading to the damages, or later pursue more invasive adjustments to prevent exacerbation to severe mandibular and temporal bone destruction.

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