

The impact of superficial zone damage on the tribological performance of cartilage: Implications for early intervention in osteoarthritis

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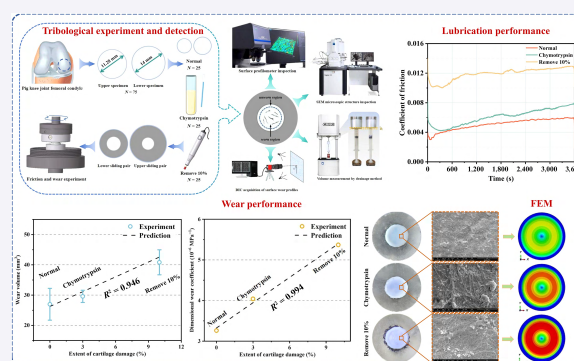
ABSTRACT: The superficial zone of the articular cartilage plays a vital role in maintaining low friction and wear within joints because of its excellent lubricating properties. While the tribological properties of cartilage with an intact superficial zone have been widely studied, it remains unclear how different degrees of superficial zone damage affect cartilage lubrication, friction, and wear. In this study, we conducted friction and wear experiments to assess changes in cartilage performance before and after damage and examined surface roughness and microstructure to understand lubrication and wear mechanisms. We also used finite element method (FEM) simulations to study the evolution of wear behavior in damaged cartilage. Research has shown that as damage to the superficial zone of cartilage worsens, its lubrication performance continuously decreases, leading to more severe wear. The cartilage friction coefficient exhibited varying degrees of dependence on pressure and speed at different periods, suggesting that superficial zone damage might cause a shift in the lubrication state of the cartilage. Furthermore, on the basis of the observed cartilage wear parameters and morphological features, we further confirmed that fatigue wear is the primary wear mode and proposed a numerical model for predicting cartilage wear volume on the basis of the degree of damage. These findings emphasize the importance of protecting and restoring the superficial zone in cartilage repair and regeneration strategies.

KEYWORDS: articular cartilage; superficial zone; lubrication; friction; wear

1 Introduction

Articular cartilage, as an anisotropic connective tissue covering joint surfaces, exhibits a distinctive layered structure along its thickness, comprising the superficial zone, middle zone, deep zone, and calcified zone [1, 2]. This unique structural organization enables cartilage to play a critical role in cushioning and lubrication during joint motion, allowing joints to withstand repetitive loading under low-friction conditions while maintaining long-term stability [2, 3]. Notably, the superficial zone, as the first line of defense at the contact interface, features densely arranged collagen fibers and an enrichment of surface lubrication

molecules, which are essential for maintaining the wear resistance and lubrication properties of cartilage [4]. However, this zone is particularly susceptible to microstructural damage under prolonged mechanical loading conditions, and given the extremely limited self-regenerative capacity of cartilage tissue, such damage can disrupt the lubricating barrier of the cartilage surface. This, in turn, triggers a vicious cycle of increased friction and wear, ultimately leading to the progressive degeneration of cartilage and the development of osteoarthritis [5]. Therefore, elucidating the relationship between superficial zone damage and cartilage friction–wear performance not only has scientific value for understanding cartilage degeneration but also provides a



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theoretical foundation for developing clinical prevention and treatment strategies.

Over the past century, research on cartilage tribology has led to the development of multiple lubrication theories. Early studies revealed that healthy cartilage exhibits an ultralow friction coefficient (0.001–0.02) and is significantly superior to engineered materials, thereby initiating the exploration of lubrication mechanisms [6, 7]. Since the 1930s, researchers have proposed several competing theories, including hydrodynamic lubrication [8], boundary lubrication [9], and weeping lubrication [10]. Among them, Walker's boosted lubrication theory provides key insights by demonstrating that a boundary lubrication layer composed of proteoglycans (macromolecules composed of a core protein and glycosaminoglycan chains, essential for cartilage hydration and load-bearing capacity) and lubricin (a glycoprotein secreted by synovial cells and chondrocytes that plays a key role in boundary lubrication by reducing friction on cartilage surfaces) exists on the cartilage surface, whereas small solutes penetrate the matrix [11], laying the foundation for understanding multiscale lubrication mechanisms.

With advancements in research, the scientific community has increasingly recognized that cartilage lubrication exhibits dynamic and composite characteristics. Through load–response experiments, Unsworth [12] demonstrated the transition between fluid film lubrication and boundary lubrication. Furthermore, Ateshian et al. [13–15] established a biphasic lubrication model that systematically elucidated the synergistic interactions between the solid and fluid phases. However, existing theories focus primarily on macroscopic mechanical responses or bulk matrix properties and lack in-depth analysis of the crucial role of the superficial zone. This zone not only exhibits a unique surface morphology but also possesses a compositional gradient and dynamic remodeling behavior that directly influences the interfacial lubrication performance. A significant portion of the ongoing debate arises from an incomplete understanding of the “structure–composition–function” coupling mechanisms in the superficial zone.

The superficial zone, as the outermost functional layer of articular cartilage, can be further divided into the mucus layer, the fine collagen fiber network layer, and the spindle-cell collagen fiber layer [16]. Serving as the core component of the cartilage lubrication system, the compositional gradient and structural characteristics of this zone directly determine the tribological properties of cartilage. The outermost mucus layer ($\approx 5\ \mu\text{m}$) exhibits a compositional gradient distinct from that of other regions of cartilage, with concentrations of lubricin and surface-active phospholipids (PLs); amphiphilic molecules consisting of hydrophilic heads and hydrophobic tails, key components of cell membranes and synovial fluid, and capable of forming an organized bilayer that contributes to boundary lubrication and friction reduction in cartilage lubrication) 2–3 orders of magnitude greater than those in deeper layers [17]. This mucus layer is composed of a highly ordered phospholipid bilayer, lubricin, hyaluronic acid (HA; a high-molecular-weight polysaccharide found in synovial fluid, which contributes to joint lubrication and cushioning by forming a viscous, protective layer on cartilage surfaces), and other boundary lubrication molecules [18]. Under physiological loading conditions (contact pressure: 1–4 MPa, sliding velocity: 1–20 mm·s^{−1}), these components work synergistically to achieve an ultralow friction coefficient ($\mu \approx 10^{-3}$) [1]. However, whether these lubrication structures accurately reflect the friction mechanisms of natural articular cartilage remains controversial. The specific contribution of the superficial zone to boundary lubrication and the potential shift in the

lubrication mode following damage remain unclear. Therefore, systematic investigations into the impact of superficial zone damage on lubrication performance are highly important.

This study aimed to investigate the impact of superficial zone damage on cartilage lubrication, friction, and wear behavior. Unlike traditional studies, we utilized actual cartilage tissue as the research subject and established damage models through enzymatic degradation and physical abrasion. Friction experiments were conducted to analyze changes in the lubrication mechanism before and after damage. Drainage methods, digital image correlation (DIC) imaging, surface profilometry, and scanning electron microscopy (SEM) were used to characterize the wear morphology of cartilage with varying degrees of damage. Wear volume, wear coefficient, and surface roughness were measured. Finally, finite element method (FEM) simulations were employed to model the evolution of wear behavior in damaged cartilage, revealing the effects of superficial zone damage on cartilage wear performance.

2 Materials and methods

2.1 Modeling and characterization of cartilage superficial zone damage

2.1.1 Cartilage sample preparation

As shown in Fig. 1, porcine knee cartilage was selected because of its high similarity to human knee cartilage in terms of its anatomical structure and biomechanical properties [19–21]. These joints were obtained from approximately eleven-month-old animals within 10 h post-slaughter from a local abattoir (Qianxihe, China). Full-thickness articular cartilage samples (with the calcified layer removed) were harvested from the central weight-bearing region of the medial femoral condyle using a micro bone saw and a cordless rotary tool, ensuring anatomical consistency across all samples for subsequent experimental procedures. During the modeling and characterization of the superficial zone of the cartilage, a custom-made punch was used to obtain circular cartilage samples with a diameter of $\phi = 14\ \text{mm}$. All samples were handled carefully during preparation to avoid damaging the cartilage surface. Following macroscopic examination of the prepared circular samples, only the crack-free cartilage samples, which exhibited uniform thickness and smooth surface topography, were selected for subsequent procedures. Unless otherwise specified, all the aforementioned and subsequent experiments were conducted in a controlled environment maintained at $25 \pm 1\ ^\circ\text{C}$ and 40%–60% relative humidity (RH).

2.1.2 Damage modeling of superficial zone of cartilage

This study established two distinct cartilage degeneration models (chymotrypsin digestion and physical abrasion) to investigate the effects of different degrees of superficial zone damage on the tribological properties of cartilage. The chymotrypsin digestion model primarily simulates the initial damage to the superficial zone, specifically the loss of the mucus zone. In contrast, the physical abrasion model simulates the extensive matrix damage that occurs as superficial zone damage progresses.

The first group of cartilage samples is referred to as the chymotrypsin group. Chymotrypsin (serine protease) was selected on the basis of its documented specificity for cleaving glycosylated domains of lubricin (PRG4) [22] while preserving collagen network integrity in deeper zones [23]. We modified the cartilage chymotrypsin digestion method of Kumar et al. [16] to specifically avoid the digestion of areas other than the superficial zone of

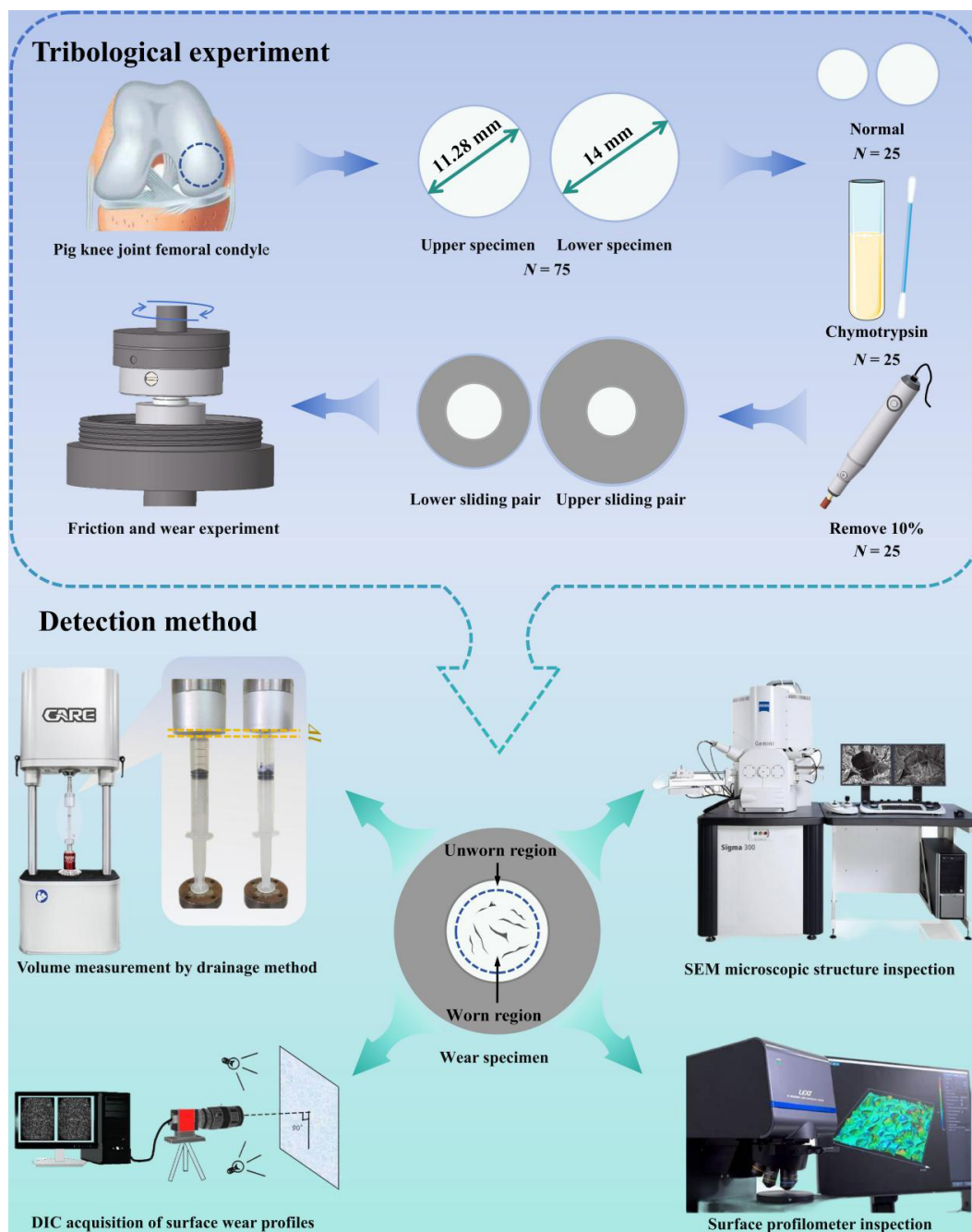


Fig. 1 Cartilage–cartilage friction and wear experiment and testing procedure.

cartilage. Chymotrypsin (GlpBio Technology Inc., USA) was prepared at a concentration of $50 \text{ units} \cdot \text{mL}^{-1}$. The specific operational procedures for creating the damage model in the chymotrypsin group are detailed in Table 1.

The second group is referred to as the remove 10% group. An electronic caliper (SYNTEK, China) was used to measure the thickness of the cartilage at least 5 times, and the average value was calculated. The cartilage surface was abraded using a cordless rotary tool until 10% of the cartilage thickness had been removed, retaining 90% of the original thickness. Additional technical details regarding the damage modeling procedures for the remove 10% group are outlined in Table 1.

2.1.3 Characterization using black Indian ink

Normal cartilage was used as the control group, whereas chymotrypsin-digested cartilage and 10% abraded cartilage were

used as the experimental groups. These three groups of cartilage were flattened and adhered to epoxy resin plates using ethyl cyanoacrylate. Black Indian ink (Phygene China) staining was used to visualize surface damage to the cartilage samples, indirectly providing evidence of superficial zone damage. The ink was uniformly applied to the cartilage surface and allowed to air dry naturally in a cool environment (20°C) for 6 min. Excess ink was subsequently removed by rinsing with phosphate-buffered saline (PBS).

2.1.4 Atomic force microscopy (AFM) imaging characterization

After the circular cartilage samples from the three experimental groups were blotted with tissue paper, they were immediately flattened and adhered to circular glass coverslips (1.5 cm in diameter) using ethyl cyanoacrylate. During the adhesion process, the articular cartilage surface was continuously moistened with

Table 1 Comparative protocol for cartilage degeneration models

Group	Chymotrypsin group	Remove 10% group
Core procedure	Cyclic enzyme application	Controlled thickness abrasion
Key steps	1. 50 units·mL ⁻¹ chymotrypsin solution preparation 2. Surface wiping (3 min)+static application (12 min) 3. 8 cycles (2 h total) 4. PBS rinse (≥ 5 min)	1. Cartilage thickness measurement (5 repeats) 2. Rotary abrasion (10% thickness removal) 3. PBS rinse (≥ 5 min)
Critical controls	1. Limited digestion to superficial zone 2. Modified from Kumar et al. [16]	1. Electronic caliper (SYNTEK, China) 2. 90% thickness retention
Duration	2 h active treatment	Until 10% thickness achieved
post-treatment	Residual enzyme removal	Abrasion debris elimination

PBS. The coverslip was then fixed onto a steel disk using double-sided adhesive tape, and the steel disk was magnetically attached to the piezoelectric scanner of an atomic force microscope (AFM; Nanoscope III, Digital Instruments, USA). The sample was subsequently immersed in PBS.

AFM imaging was performed using cantilevers with nominal spring constants of $k = 0.094$ and $0.022 \text{ N} \cdot \text{m}^{-1}$, which were equipped with spherical silicon nitride (Si_3N_4) probes. Imaging was conducted in contact mode, and after approximately 30 min of thermal stabilization, the drift of the cantilever deflection angle was minimized. To reduce sample deformation and probe contamination, the scan size was initially set to zero. Imaging was carried out in error signal mode, with both deflection and height signals collected simultaneously. The gain and scan rate were optimized to minimize the deflection signal. The scanning speed at all magnifications was approximately 1–3 lines per second, with frame sizes ranging from 80 nm to 1 μm .

The modulus was calculated using the Derjaguin–Muller–Toporov (DMT) model, with the calculation equation shown in Eq. (1):

$$F_L(i) = \frac{4}{3} E^* \sqrt{R} i^{3/2} + F_{\text{pull-off}} \quad (1)$$

where F_L is the load force; E is the reduced Young's modulus and $E^* = E/(1 - \nu^2)$; ν is the Poisson's ratio; R is the reduced radius; $1/R = 1/R_{\text{indenter}} + 1/R_{\text{surface}}$; i is the indentation depth; $F_{\text{pull-off}}$ is the force at the point of pull-off of the AFM probe, or the pull-off force for short. The pull-off force can easily be found from the force-indentation curves. The Poisson's ratio was based on our previous research and was 0.495 [24].

2.1.5 Characterization of hyaluronic acid immunohistochemical staining

Hyaluronic acid, a critical component of the cartilage surface lubrication interface, exhibits spatial distribution patterns that directly reflect damage-induced alterations in matrix integrity. This study utilized immunohistochemical staining to specifically detect HA expression profiles, aiming to uncover the loss dynamics of lubricating components in superficial injury models.

Sample processing adhered to standardized tissue fixation and dehydration protocols. Fresh cartilage samples were fixed in 4% paraformaldehyde (pH = 7.4, 48 h at 25 °C), followed by automated dehydration using a tissue processor (KH-TS, Hubei Xiaogan Kuohai Medical Technology Co., Ltd., China) with a graded ethanol series: 75% ethanol (60 min) → 85% ethanol (60 min) → 95% ethanol (60 min) → three cycles of absolute ethanol (60 min each). The tissues were cleared through xylene (three cycles, 30 min each) and infiltrated with paraffin at 60 °C (three cycles, 2 h each) using an embedding machine (KH-BL, Hubei Xiaogan Kuohai Medical Technology Co., Ltd., China). Sections of 3 μm thickness were cut with a microtome (HM325,

Thermo, USA), flattened on a tissue flotation bath (HI1210, Leica, Germany), and baked at 70 °C for 1 h in a thermostatic incubator (DH-250, Kewei Yongxing, China), followed by 80 °C oven curing.

The immunohistochemical procedure was conducted under light-protected conditions. Deparaffinized sections were subjected to antigen retrieval with 0.25% trypsin (T1320; Solarbio, China; 37 °C, 2 h). Endogenous peroxidase activity was blocked with 3% H_2O_2 (25 min, room temperature (RT), light-protected), and nonspecific binding was minimized using a goat serum blocking solution (ZLI-9022, Zhongshan Jinqiao, China). The samples were incubated with a rabbit anti-HA antibody (PAA182Ge01, Cloud-Clone, USA; 1:100 dilution) at 4 °C overnight, followed by incubation with an horseradish peroxidase (HRP)-conjugated secondary antibody (956D, Celpor, China; 60 min, RT). The 3,3'-diaminobenzidine (DAB) chromogenic reaction (DA1010, Solarbio, China) was microscopically monitored (BH-2, OLYMPUS, Japan), with hematoxylin staining (Jiangyuan Industrial Technology and Trade Corp., China) for 5–10 min and lithium carbonate (554-13-2, CSNpharm, China) blueing. Dehydrated sections were mounted with neutral resin (CS01-2424, Meivid, China) under cover glasses (CS01-2424, Meivid, China). Technical operations utilized a histochemical pen (YA0310, Solarbio, China) for area demarcation and adjustable pipettes (20–200 μL , Eppendorf, Germany) for precise reagent handling.

2.2 Cartilage–cartilage friction and wear experiments and detection

2.2.1 Preparation of cartilage friction and wear samples

The process of creating cartilage superficial zone damage models is described in Sections 2.1.1 and 2.1.2. For the cartilage–cartilage friction and wear experiments, a total of 150 circular cartilage samples harvested from the central weight-bearing region of the medial femoral condyle in porcine knee joints were paired into 75 friction pairs. Each pair comprised one large sample ($\phi = 14 \text{ mm}$) and one small sample ($\phi = 11.28 \text{ mm}$), as illustrated in Fig. 1. The 75 friction pairs were divided into three groups with 25 pairs in each group: the normal group (normal cartilage and normal cartilage), the chymotrypsin group (normal cartilage and chymotrypsin-treated cartilage), and the remove 10% group (normal cartilage and 10% removed cartilage). To avoid changes in the tribological properties of the cartilage samples due to being *ex vivo* [1], the time from extraction to the completion of the friction and wear experiments did not exceed 6 h. During this period, the samples were kept in PBS.

2.2.2 Friction and wear experiments

Cartilage friction and wear experiments were conducted using a vertical universal friction and wear testing machine (CTMM-1,

Jilin Guanteng Automation Technology Co., Ltd., China). As shown in Fig. 1, two custom sample fixtures were used, referred to as the upper cartilage fixture and the lower cartilage fixture, according to their positions. The primary function of the upper fixture was to rotate at a preset speed, whereas the lower fixture provided upward pressure and transmitted frictional force. The cartilage samples were flattened and securely bonded to the geometric center of the sample fixture using ethyl cyanoacrylate adhesive. A standardized bonding protocol was applied uniformly across all experimental groups to ensure consistency in the frictional contact area, with each sample precisely aligned within an identical adhesive region. This cartilage-on-cartilage friction and wear test design was based on the theoretical framework proposed by Forster and Fisher [25], which suggests that the biphasic nature and fluid load-bearing capacity of cartilage can be effectively evaluated even under static or quasi-static loading conditions.

To maintain cartilage hydration throughout the experiment, PBS was added to both surfaces of the cartilage before the experiment commenced. After the expected pressure was applied to the test machine, an additional PBS was injected with a syringe on the sides of the sample to form a continuous fluid film enveloping both sides of the sample. The entire experiment lasted 3,600 s, starting immediately after the target pressure load was reached.

To investigate the effects of superficial cartilage damage on cartilage lubrication under different pressures and velocities, five distinct operating conditions were established on the basis of the mechanical properties typical of knee joint physiology [26]. The specific operating conditions are detailed in Table 2. Each friction and wear experiment under different operating conditions was repeated five times.

2.2.3 Wear volume measurement

The macroscopic characterization of wear typically uses either wear volume or wear mass. Owing to the convenience of mass measurement, it is widely used in materials with high hardness and uniform density. However, as discussed by Fischenich et al. [27], cartilage is an anisotropic material with varying densities at different locations. More importantly, once cartilage is removed from a liquid environment, it continuously loses water. Therefore, wear mass cannot be used as a parameter to measure cartilage wear, making wear volume a more appropriate metric.

Owing to individual differences in cartilage, it is impossible to obtain completely uniform cartilage samples even with customized ring punchers. Therefore, we designed a cartilage wear volume measurement method based on the water displacement method. Specifically, a high-precision displacement detector in a microelectromagnetic dynamic mechanical testing system (M100, CARE, China) was used to measure the rise in liquid level within a syringe, which was then converted to the cartilage wear volume.

Importantly, to avoid measurement errors caused by cartilage compression, the samples to be tested were uniformly soaked in PBS for 1 h prior to wear volume measurement. As shown in

Fig. 1, an appropriate amount of PBS was drawn into the syringe, and the initial height l_1 was measured. After the piston was removed and the cartilage was placed into the syringe, the air in the syringe was slowly expelled, and the second height l_2 was measured. Thus, the displacement difference was given by $\Delta l = l_2 - l_1$. According to this method, the equation for calculating the wear volume of cartilage after wear is given by Eq. (2):

$$V_{\text{wear}} = (\Delta l_2 - \Delta l_1) \frac{\pi D^2}{4} \quad (2)$$

where V_{wear} represents the wear volume, measured in mm^3 ; Δl_1 represents the displacement difference before wear, and Δl_2 represents the displacement difference after wear, both of which are measured in mm; D represents the inner diameter of the syringe, measured in mm.

2.2.4 Imaging and extraction of worn cartilage surface profile

To analyze the worn cartilage surface, a scalpel was used to cut the cartilage into four equal parts. The first cut was made carefully along a line passing through the center of the worn cartilage, followed by a second cut perpendicular to the first. One-quarter of the cartilage was then immersed in PBS for one hour to eliminate any deformation or shape artifacts introduced during cutting.

A non-contact three-dimensional (3D) full-field strain measurement system (XTDIC-CONST-HR, XTOP, China) was employed to capture high-resolution images of the cross-sections of the cartilage quarters. To minimize variability and ensure consistency in contour extraction, a polar coordinate system was established with the wear center as the origin, and contour profiles were extracted radially using GetData software. Specifically, one contour line was extracted from each of the four orthogonal directions (0° , 90° , 180° , and 270°), resulting in a total of four contour lines. Outliers were identified and excluded using K-means clustering, with contours exhibiting a root mean square error (RMSE) greater than 15% from the average being removed. To further enhance the consistency and smoothness of the extracted profile, the remaining curves were processed using a Savitzky–Golay smoothing filter to generate the final contour.

The extracted contour reflects the typical wear characteristics under the experimental conditions and was selected on the basis of its statistical representativeness and consistency across different trials.

2.2.5 Roughness measurement

The roughness of the worn and unworn regions of cartilage after wear was measured using an optical surface profiler (ContourX-100, BRUKER, Germany) with a $20\times$ objective lens. At least five different points were selected for measurement in each region. To ensure consistency in image acquisition positioning, standardized protocols were implemented as follows: Unworn regions were consistently imaged at the peripheral annular zone of the samples, whereas worn regions were imaged at an annular zone positioned 3.5 mm from the center of the circular samples. During each measurement, the imaging region was approximately $400\text{ }\mu\text{m} \times 400\text{ }\mu\text{m}$, and tilt correction was performed using the software's plane correction function. The roughness was calculated using Eq. (3):

$$S_a = \frac{1}{l} \int_0^l |y(x)| dx \quad (3)$$

where S_a is the arithmetic mean height of the cartilage surface, measured in μm . l is the measurement length, and $y(x)$ is the surface height function.

Table 2 Experimental conditions

Group	Operating condition	Parameter setting
A	1.0 MPa, 0.27 Hz	100 N, 17 r·min ⁻¹
B	1.5 MPa, 0.27 Hz	150 N, 17 r·min ⁻¹
C	2.0 MPa, 0.27 Hz	200 N, 17 r·min ⁻¹
D	1.0 MPa, 0.60 Hz	100 N, 36 r·min ⁻¹
E	1.0 MPa, 1.00 Hz	100 N, 60 r·min ⁻¹

2.2.6 Micromorphology analysis

The changes in the tribological properties of the cartilage were related to alterations in its microstructure. An environmental field emission scanning electron microscope (Quanta FEG 250, Thermo Fisher Scientific, USA) was used to observe the microstructure of worn and unworn regions of cartilage after wear tests under different superficial zone injuries. Immediately after completing the wear tests, the samples were immersed in anhydrous ethanol. After 24 h of immersion in anhydrous ethanol, the cartilage samples were fully dehydrated. The surfaces of the worn and unworn regions of the samples were subsequently scanned using SEM at 10 kV and 0.4 nA. To ensure consistency in image acquisition positioning, standardized protocols were implemented as follows: Unworn regions were consistently imaged at the peripheral annular zone of the samples, whereas worn regions were imaged at an annular zone positioned 3.5 mm from the center of the circular samples.

2.3 Theoretical model of cartilage wear

The common starting point for discussions of macroscopic wear is the Archard equation [28], which is generally expressed as Eq. (4):

$$V = K \frac{F_N}{H} s \quad (4)$$

where V is the wear volume (mm^3); s is the sliding distance (mm); K is the wear coefficient; F_N is the normal load (N); H is the material hardness (MPa). Typically, we treat K/H as a whole, referred to as the dimensional wear coefficient k , with units of MPa^{-1} . Thus, the Archard equation can be expressed as Eq. (5):

$$V = k F_N s \quad (5)$$

In typical pin-on-disk motion, the velocity at each point on the contact surface of the pin can be approximated as the same, and then the sliding distance s is constant. However, this simplification does not apply to disk-on-disk motion. Therefore, it is necessary to extend the Archard wear equation to accommodate the disk-on-disk wear motion. Notably, Archard stated that the equation assumed that the wear rate was independent of the apparent contact area [29]. In fact, this means that in disk-on-disk wear motion, points on the contact surface with the same velocity magnitude can be simplified as single points, even though the directions of their velocities differ (velocity as a vector has both magnitude and direction). Therefore, the entire disk can be considered an aggregate composed of countless microrings. A microring located x mm from the center has a sliding distance of $\omega x dt$ at time t , where ω is the rotational speed of the disk ($\text{rad}\cdot\text{s}^{-1}$).

In addition to the sliding distance s , another factor to consider in the Archard wear equation is the normal load F_N . To simplify the calculation, we make an assumption here. The pressure exerted on the disk is uniformly distributed over the contact surface, meaning that the pressure at every point on the contact surface is a constant value p . Therefore, the normal load on a microring located x mm from the center at time t is $2\pi x p dx$. Integrating over dx and dt , the macroscopic Archard wear equation for disk-on-disk wear is derived as Eq. (6):

$$V = k \iint 2\pi \omega x^2 p dx dt \quad (6)$$

where x is the distance from the center (mm), with a range of $0 \leq x \leq x_{\max}$. The value of $x_{\max}$ is obtained from the wear profile of the cartilage surface as described in Section 2.2.4. t represents time (s), ranging from $0 \leq t \leq 3,600$.

The dimensional wear coefficient k in Eq. (6) is crucial for assessing the impact of superficial cartilage damage on the tribological performance of cartilage. Here, we fit the relationship between k and surface damage to cartilage according to the results of the cartilage wear experiments, as shown in Eq. (7):

$$k = (0.207i + 3.325) \times 10^{-6} \quad (7)$$

where i represents the extent of cartilage damage (%). The extent of damage to the normal group cartilage was set to 0. Considering the degree of damage to the cartilage thickness, the extents of damage in the chymotrypsin group and the remove 10% group were set to 3% and 10%, respectively.

2.4 Finite element simulation of cartilage–cartilage wear

2.4.1 Finite element model establishment

Figure 2(a) shows that the main components of the finite element model are two cartilage discs with diameters of $\phi = 14$ and 11.28 mm, which are simplified to reduce computational effort. The material properties of the cartilage discs were derived from our previous study [24]. Specifically, the cartilage in the upper contact pair was simplified and modeled as a three-layer structure to reflect the layered mechanical properties of the cartilage. The Young's modulus of the superficial zone was set to 2.49 MPa, that of the middle zone was set to 4.21 MPa, that of the deep zone was set to 6.51 MPa, and the Poisson's ratio was uniformly set to 0.495. The Young's modulus of the cartilage in the lower contact pair was defined using the Abaqus subroutine USDFLD, where the field value was set to 0 at the beginning and 1 at the end. A linear relationship was established between the field value and the Young's modulus, which was controlled by the function in USDFLD. The boundary conditions are shown in Fig. 2(b). The arbitrary Lagrangian–Eulerian (ALE) adaptive control region was the wear surface. The mesh type was C3D8R, and the number of elements was determined after mesh independence verification, the details of which can be found in Section 3.

2.4.2 Embedding of theoretical model

In finite element analysis, wear is characterized by the wear depth dh at various locations. Assuming that the wear depth at point x at time t is $dh(x, t)$ and that the contact area at this point is an infinitesimally small dA , the following relationship is given by Eq. (8):

$$dh(x, t) = \frac{dV(x, t)}{dA} \quad (8)$$

where $dV(x, t)$ is obtained from Eq. (6). Owing to the limitations of simulation calculations, it is challenging to simulate every wear cycle process. Thus, a cycle jump technique [30] is needed, where one simulation cycle corresponds to ΔN cycles in the experimental analysis. Therefore, the final transformation is given by Eq. (9):

$$dh(x, t) = Nk\omega xp(x, t)dt \quad (9)$$

In the finite element simulation, the Umeshmotion subroutine was used to embed the wear height prediction model (Eq. (9)) into the finite element software for simulating cartilage–cartilage wear. This subroutine was written in Fortran. The core operation is as follows: Upon reaching the wear analysis step, the subroutine calls Eq. (9) to calculate the wear at each node within the incremental step. The computed data are then transmitted to the adaptive meshing module, which generates new nodes representing the worn surface and remaps the old mesh to form a new mesh,

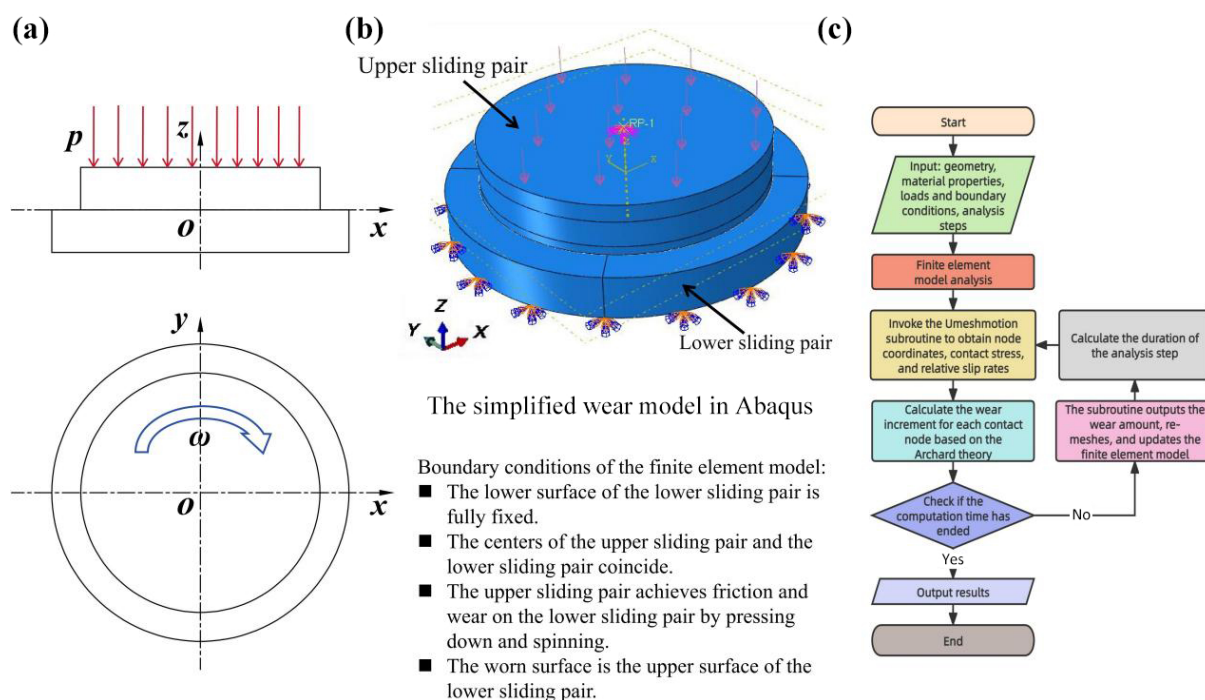


Fig. 2 Schematic diagram of finite element modeling of cartilage-cartilage wear. (a) Two-dimensional (2D) schematic. (b) 3D model and boundary conditions. (c) Uremshmotion subroutine workflow.

thereby completing one incremental analysis step. The detailed process is illustrated in Fig. 2(c).

2.5 Statistical analysis

All the results of the experiments are presented as the means $\pm$ standard deviations. Statistical analysis of differences between different groups was conducted using GraphPad 9.5 statistical software. One-way analysis of variance (ANOVA) was performed for each pair of data, and Fisher's least significant difference (LSD) test was subsequently used for post hoc analysis. The p -values for the differences between groups were labeled as follows: p -value > 0.05 , $*p$ -value < 0.05 , $**p$ -value < 0.01 , and $***p$ -value < 0.001 .

3 Results

3.1 Modeling and characterization of superficial zone damage to cartilage

3.1.1 Characterization using black Indian ink

Figure 3(a) shows the characterization of superficial cartilage damage using black India ink staining. The results are discussed in terms of visual and tactile aspects. Before the superficial zone damage treatment, the normal group exhibited a certain degree of surface gloss and a slippery feel. After superficial layer damage, a duller surface color with reduced gloss and a sticky feel appeared in the chymotrypsin group. The remove 10% group appeared visually dull and non-reflective, with a rough tactile sensation.

To better illustrate the reflectivity of the cartilage surface, we captured side-view images of the cartilage during both the black India ink staining and washing stages. After being stained with India ink and air-dried for 6 min, the cartilage surface of the normal group still showed noticeable gloss. The chymotrypsin group exhibited a wavy sheen on the cartilage surface, whereas the surface of the remove 10% group was completely dry and non-reflective. After washing with PBS, the surface of the cartilage in

the normal group remained clear and glossy. The chymotrypsin group showed pitted damage to the cartilage surface, whereas the remove 10% group exhibited highly pronounced directional scratch damage.

3.1.2 AFM imaging characterization

Figure 3(b) shows the surface morphology of the cartilage superficial zone after damage, which was captured using AFM. Figure 3(c) presents the related parameters of each cartilage group measured by AFM, including R_a , R_q , and modulus. Importantly, the modulus measured by probe-based methods such as AFM typically represents only the local modulus, and here, it specifically represents the modulus at the measured superficial zone of the cartilage. AFM imaging revealed that the surface of untreated cartilage was the smoothest, with the lowest surface roughness and the highest modulus. The surface of the chymotrypsin group exhibited slight fibrotic structures. Compared with those of the normal group, the R_a value increased by 13.78% (p -value = 0.83), the R_q value increased by 15.26% (p -value = 0.82), and the modulus decreased by 54.23% (p -value = 0.0068). The surfaces of the remove 10% group showed extensive fibrotic structures. Compared with those of the normal group, the R_a value increased by 40.19% (p -value = 0.27), the R_q value increased by 61.84% (p -value = 0.1), and the modulus decreased by 95.48% (p -value = 0.0003).

3.1.3 Characterization of hyaluronic acid immunohistochemical staining

Figure 3(d) shows the immunohistochemical staining of hyaluronic acid in the superficial damage model, where brown-yellow indicates positive expression of hyaluronic acid. According to the $1\times$ magnification images of the cartilage cross-sections in the normal group, hyaluronic acid was absent in most regions of the cartilage, except for a thin layer in the superficial zone. At $25\times$ magnification, HA-positive expression was observed in the outermost layer of the normal cartilage, with a thickness of

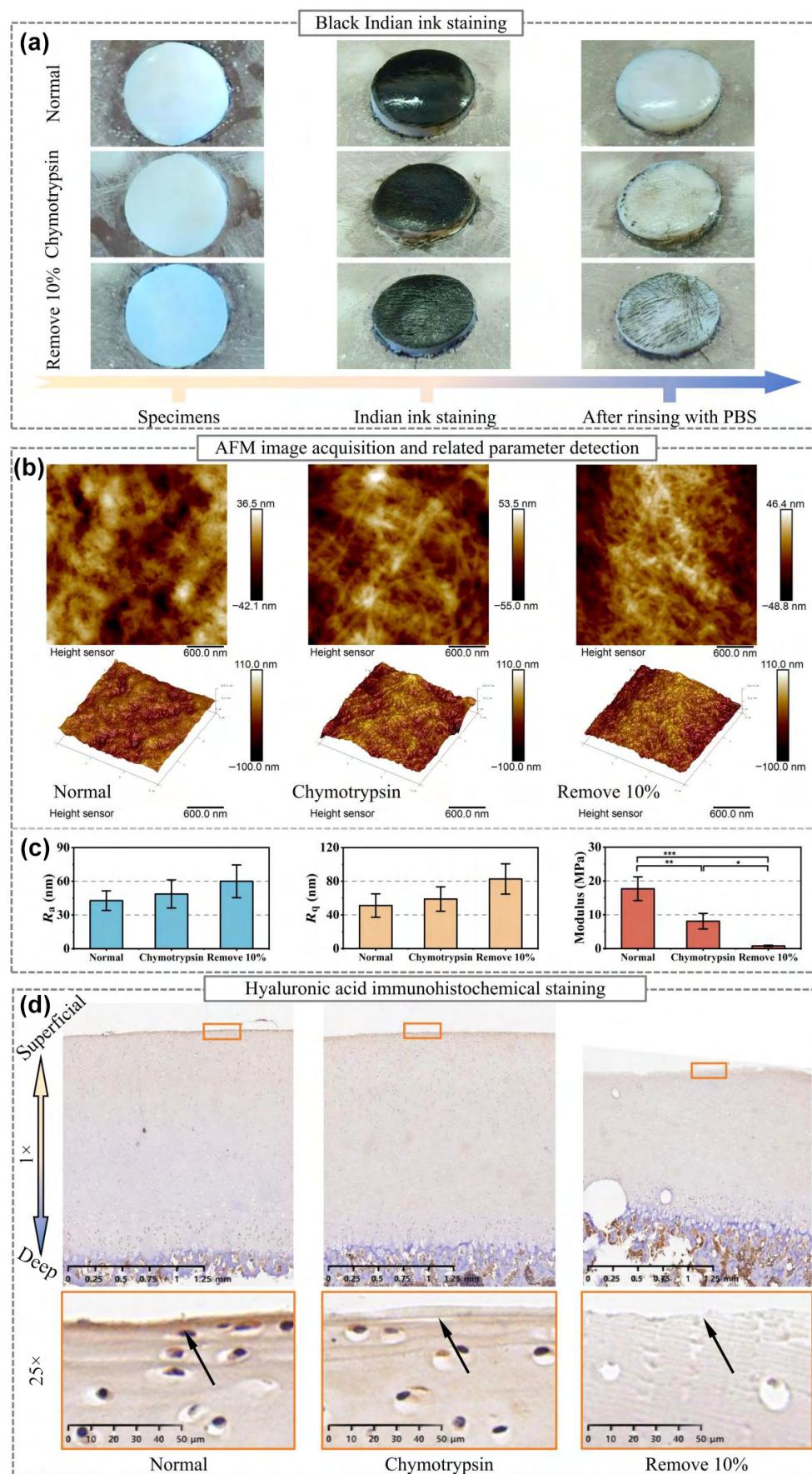


Fig. 3 Characterization of superficial zone cartilage damage model. (a) Superficial zone damage model characterized by black India ink. (b) Superficial zone damage model characterized by AFM imaging. (c) Cartilage-related parameters measured by AFM. (d) Superficial zone damage model characterized by hyaluronic acid immunohistochemical staining.

approximately 5 μm . In contrast, no HA-positive expression was detected in the superficial zone of either the chymotrypsin group or the remove 10% group.

3.2 Effects of superficial damage on cartilage lubrication performance

3.2.1 Effects of superficial damage on cartilage friction coefficient

Details of the friction and wear apparatus are shown in Fig. 4(a). The upper friction component is primarily responsible for rotation, whereas the lower component mainly provides upward normal pressure, with both components in full contact and friction in a PBS-rich environment.

As shown in Figs. 4(b)–4(f), the general trend of the friction coefficient was that it decreased rapidly and briefly in the early stages, then slowly increased over time and eventually stabilized. Among the different damage groups, the remove 10% group consistently exhibited the highest friction coefficient, followed by the chymotrypsin group, with the normal group having the lowest friction coefficient. Clearly, superficial zone damage was the main reason for the differences in friction coefficients between the groups.

Given that most studies on cartilage lubrication performance tend to shorten the friction time to several seconds to a few minutes, we deemed it necessary to quantify and discuss the friction coefficients obtained in this study in different phases [31–34]. As shown in Fig. 4(d), we extracted two stages from the entire 3,600-s friction test for averaging. Specifically, stage I represents the initial 600 s (initial average friction coefficient), and stage II represents the final 600 s (final average friction coefficient). Figures 4(g)–4(j) show the initial and final average friction coefficients under different pressures and rotational speeds.

Specifically, the more severe the damage is, the greater the increase in the friction coefficient, both in the initial and final stages. For example, under conditions of 1 MPa and 0.3 Hz in the initial stage, the chymotrypsin group increased by 18.64% compared with the normal group (p -value = 0.28), whereas the remove 10% group increased by 172.97% (p -value < 0.0001). In the final stage under the same conditions, the chymotrypsin group showed an increase of 27.6% compared with the normal group (p -value = 0.019), whereas the remove 10% group increased by 117.21% (p -value < 0.0001).

3.2.2 Effects of pressure on cartilage friction coefficient

As shown in Fig. 4(g), with increasing pressure, the initial friction coefficient decreased more significantly, and this reduction became more pronounced as the degree of damage increased. Specifically, for the initial friction coefficient, pressures of 1.5 and 2 MPa reduced the friction coefficient by 19.42% (p -value = 0.49) and 35.96% (p -value = 0.14), respectively, compared with 1 MPa in the normal group. Similarly, in the chymotrypsin group, the friction coefficient decreased by 19.03% (p -value = 0.37) and 25% (p -value = 0.21), respectively, whereas in the remove 10% group, the friction coefficient decreased by 10.19% (p -value = 0.15) and 43.37% (p -value = 0.0002), respectively.

For the final friction coefficients, the differences among the friction coefficients at 1, 1.5, and 2 MPa were minimal, generally less than 5%. In the normal group, the friction coefficient increased by 2.39% (p -value = 0.94) and 4.43% (p -value = 0.81) at 1.5 and 2 MPa, respectively, compared with that at 1 MPa. In the chymotrypsin group, the friction coefficient at 1.5 MPa decreased by 1.87% (p -value = 0.95) compared with that at 1 MPa, whereas

the friction coefficient at 2 MPa increased by 1.2% (p -value = 0.98). In the remove 10% group, the friction coefficient at 1.5 and 2 MPa increased by 2.51% (p -value = 0.81) and 0.71% (p -value = 0.98), respectively, compared with that at 1 MPa.

3.2.3 Effect of rotational speed on cartilage friction coefficient

As shown in Figs. 4(d)–4(f), with increasing rotational speed, the increase in the friction coefficient became more gradual, and the duration of low friction coefficients was extended. Specifically, in terms of the initial friction coefficients, rotational speeds of 0.6 and 1 Hz reduced the friction coefficient by 9.45% (p -value = 0.8) and 17.59% (p -value = 0.5), respectively, compared with that at 0.3 Hz in the normal group. Similarly, in the chymotrypsin group, the friction coefficient decreased by 3.54% (p -value = 0.97) and 10.84% (p -value = 0.75), respectively, whereas in the remove 10% group, the friction coefficient decreased by 3.85% (p -value = 0.81) and 8.08% (p -value = 0.43), respectively.

In terms of the final friction coefficients, rotational speeds of 0.6 and 1 Hz increased the friction coefficient by 11.24% (p -value = 0.37) and 23.68% (p -value = 0.048), respectively, compared with that at 0.3 Hz in the normal group. In the chymotrypsin group, the friction coefficient at 0.6 Hz increased by 1.33% (p -value = 0.98) compared with that at 0.3 Hz, whereas at 1 Hz, it decreased by 1.74% (p -value = 0.97). In the remove 10% group, the friction coefficient at 0.6 and 1 Hz increased by 1.1% (p -value = 0.97) and 1.25% (p -value = 0.96), respectively, compared with that at 0.3 Hz.

3.3 Effects of superficial damage on cartilage wear performance

3.3.1 Macroscopic parameter characterization of wear

The tribological properties of cartilage include lubrication, friction, and wear. This section explores the wear properties of cartilage. Figures 5(a)–5(c) show upper surface images of the lower friction components of the cartilage after wear under 1 MPa and 0.27 Hz conditions. The normal group's cartilage surface showed no distinct boundary between the worn and unworn regions, whereas the chymotrypsin group and the remove 10% group displayed varying degrees of wear marks, with the remove 10% group showing deeper and more pronounced wear regions.

Figure 5(d) shows the dimensionless wear coefficient (k) for cartilage with different degrees of superficial damage, which was calculated using Eq. (6) from Section 2.3. The normal group had a wear coefficient of 3.26 MPa⁻¹. Compared with the normal group, the chymotrypsin group and the remove 10% group exhibited increasing trends, with wear coefficients of 4.04 and 5.37 MPa⁻¹, reflecting increases of 23.93% and 64.72%, respectively. Additionally, k increased linearly with the degree of superficial damage ($R^2 = 0.994$).

Figure 5(e) shows the wear volume of cartilage with different degrees of superficial damage. The normal group had a wear volume of 26.97 ± 5.26 mm³. Compared with the normal group, the chymotrypsin group and the remove 10% group showed increasing trends, with wear volumes of 29.58 ± 2.02 and 40.84 ± 4.13 mm³, increasing by 9.67% (p -value = 0.72) and 51.43% (p -value = 0.013), respectively. Additionally, we applied the cartilage wear prediction model (Eq. (6)) to estimate the wear volumes for the different damage groups and compared them with the experimental results. Good agreement was observed between the predicted and experimental data ($R^2 = 0.946$), further validating the accuracy of our theoretical model for predicting wear in cartilage with varying degrees of superficial damage.

Examining the wear contours of cartilage under specific

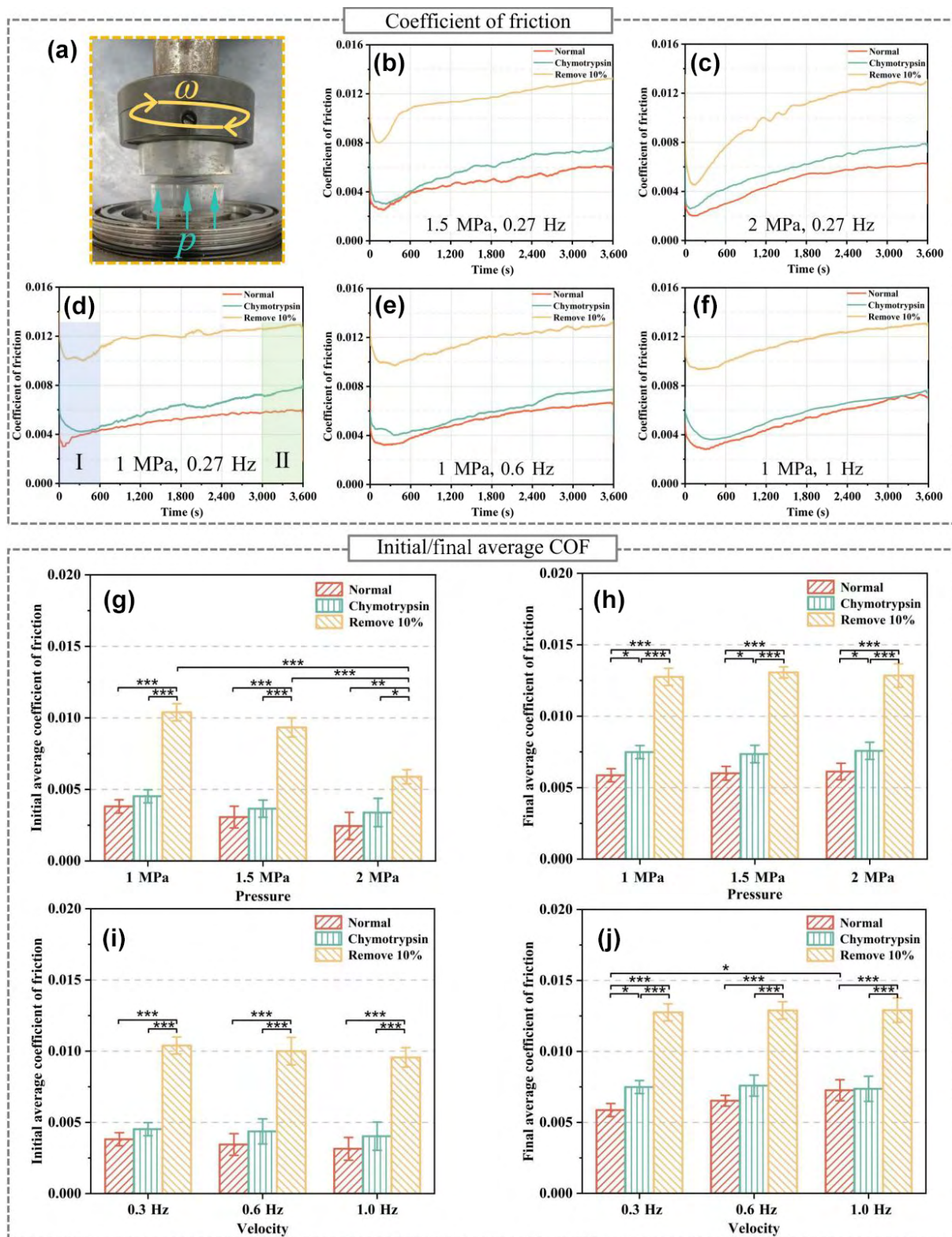


Fig. 4 Macroscopic characterization of cartilage lubrication under superficial damage. (a) Schematic of the cartilage–cartilage friction and wear testing apparatus. (b–f) Friction coefficients of cartilage under superficial damage at different pressures and rotational speeds. (g–j) Initial and final average friction coefficients of cartilage under superficial damage at various pressures and speeds.

pressures and speeds allowed for more intuitive observations of the wear conditions across different groups. Figure 5(f) shows a schematic diagram of upper surface wear contour imaging of the cartilage. Figures 5(g)–5(i) display the upper surface wear contours of the normal group, the chymotrypsin group, and the remove 10% group, respectively. The normal group showed

minimal changes in surface contour with a smaller wear depth, whereas the chymotrypsin group and the remove 10% group exhibited more pronounced depressions. The remove 10% group, in particular, showed the deepest wear and largest wear depth, indicating the most severe wear. Notably, the edge of the wear region was generally farther from the center than the initial radius

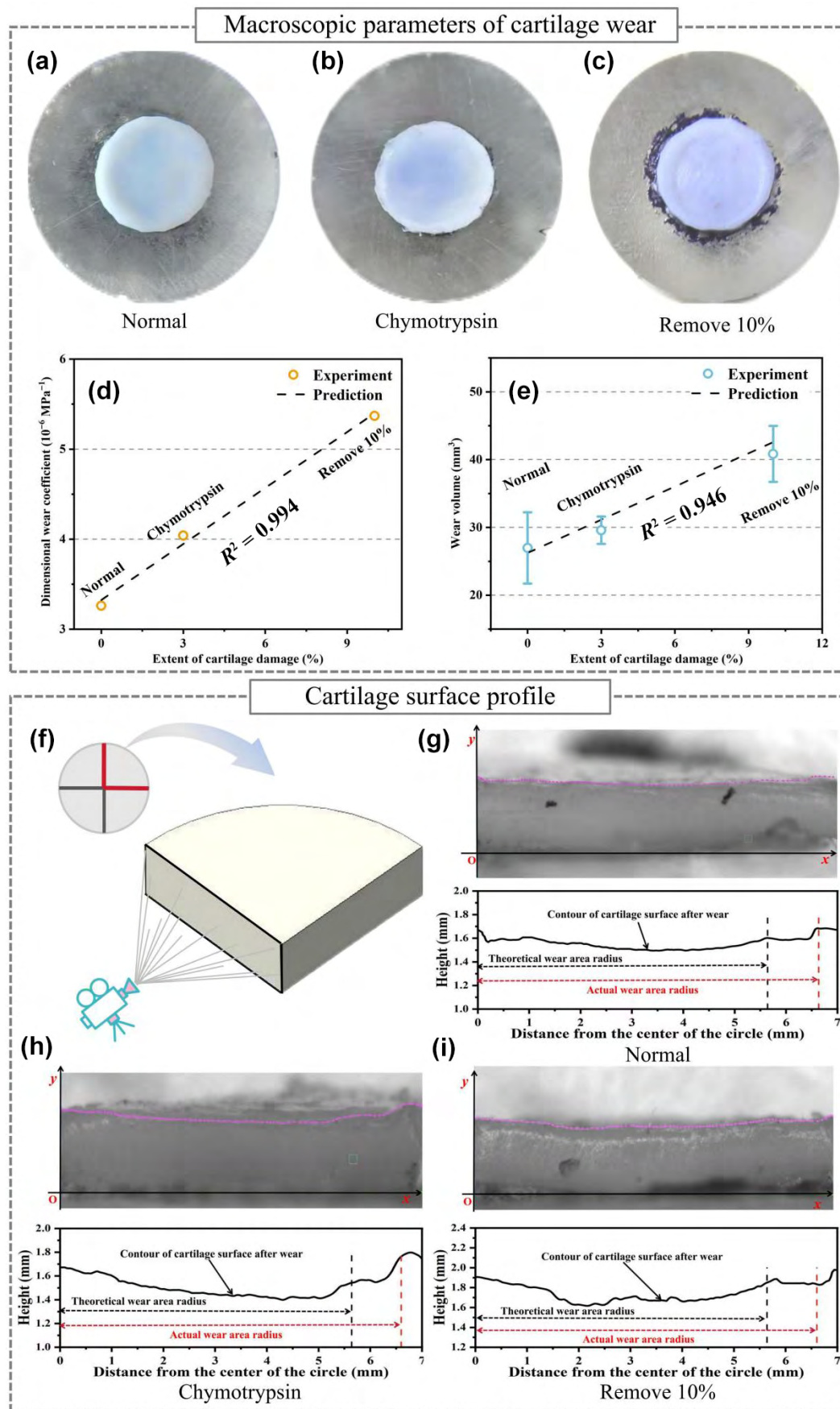


Fig. 5 Macroscopic characterization of cartilage wear under superficial damage. (a–c) Surface images of cartilage wear under superficial damage at 1 MPa and 0.27 Hz. (d) Average dimensionless wear coefficient and fitted curve under 1 MPa and 0.27 Hz. (e) Actual wear volume and predicted curve under 1 MPa and 0.27 Hz. (f) Schematic diagram of DIC imaging of cartilage surface contour. (g–i) Upper surface contours of cartilage wear under superficial damage at 1 MPa and 0.27 Hz.

of the upper friction component, which was primarily due to the significant lateral deformation of the cartilage in the upper friction component.

3.3.2 Microscopic morphological characterization of wear

Regardless of damage modeling or frictional wear, cartilage generally exhibits changes in roughness at the mesoscale. To enhance the accuracy of roughness comparison before and after cartilage wear, both worn and unworn regions were designated on the same sample. Additionally, to minimize artifacts from the irregular shape of the cartilage, five measurement points were selected within both the worn and unworn regions (Fig. 6(a)).

Under the same conditions, the roughness of the worn region was generally greater than that of the unworn region (Fig. 6(b)). The normal group had a roughness of $2.941 \pm 0.396 \mu\text{m}$ in the unworn region and $3.946 \pm 0.269 \mu\text{m}$ in the worn region, reflecting a 34.2% increase ($p\text{-value} = 0.022$). The chymotrypsin group exhibited a roughness of $3.408 \pm 0.266 \mu\text{m}$ in the unworn region and $4.245 \pm 0.294 \mu\text{m}$ in the worn region, an increase of 24.6% ($p\text{-value} = 0.021$). The remove 10% group had a roughness of $4.012 \pm 0.154 \mu\text{m}$ in the unworn region and $4.573 \pm 0.339 \mu\text{m}$ in the worn region, increasing by 13.9% ($p\text{-value} = 0.059$). For the same region, whether worn or unworn, roughness tends to increase with damage severity. In the unworn region, compared with the normal group, the chymotrypsin group and the remove 10% group exhibited increases of 15.88% ($p\text{-value} = 0.2$) and 36.42% ($p\text{-value} = 0.0094$), respectively. In the worn region, they increased by 7.58% ($p\text{-value} = 0.49$) and 15.89% ($p\text{-value} = 0.097$), respectively.

The 3D morphology of the worn and unworn cartilage regions in Fig. 6(c) shows that as the damage severity increased or when comparing the pre-wear and post-wear conditions, the surface morphology of the damaged and worn samples exhibited greater fluctuations. This effect was particularly evident in the worn region of the remove 10% group.

Figures 6(d)–6(i) show SEM images of the worn and unworn regions of cartilage, with magnifications of 1,000 $\times$, 10,000 $\times$, 20,000 $\times$, and 30,000 $\times$. The observations focused on the upper surface of the lower friction component.

Figures 6(d) and 6(e) illustrate the morphology of the unworn and worn regions in the normal group. At 1,000 $\times$ magnification, the surface of the cartilage in the normal group appeared relatively flat, but differences became evident at higher magnifications. Specifically, at increasing magnifications, the unworn region of the normal group showed dense, cloud-like structures layered on the cartilage surface, which will be further discussed in Section 4 (Fig. 6(d)). In the worn region, these cloud-like structures were largely diminished and replaced by numerous small granular structures. At 30,000 $\times$ magnification, not only were small granular structures visible, but faint fiber structures were also observed beneath them in the worn region of the normal group (Fig. 6(e)).

Figures 6(f) and 6(g) depict the morphology of the unworn and worn regions in the chymotrypsin group. At 1,000 $\times$ magnification, the cartilage in the chymotrypsin group, whether worn or unworn, showed disordered folds and debris-like substances. The unworn region exhibited thin cloud-like structures and increasingly distinct fiber structures (Fig. 6(f)), resembling the state after the dissolution and detachment of the dense cloud-like structures observed in the normal unworn region (Fig. 6(d)). This phenomenon is likely attributable to the specific degradation of lubricin by chymotrypsin. However, under sustained shear forces, the microstructure of the worn region in the chymotrypsin group significantly changed, with numerous

disordered fibers and debris particles adhering to the fibers, whereas the previously attached cloud-like structures were no longer visible (Fig. 6(g)).

Figures 6(h) and 6(i) show the morphology of the unworn and worn regions in the remove 10% group. At 1000 $\times$ magnification, scratches were visible on the surface of the unworn region (Fig. 6(h)), whereas folds were more prominent in the worn region (Fig. 6(i)). At higher magnifications, the unworn region showed some granular structures and densely packed directional fibers (Fig. 6(h)). In contrast, the worn region was predominantly composed of fibers, which was particularly evident at 30,000 $\times$ magnification. Although dense fibers were also observed at high magnification in Fig. 6(g), the most notable difference was that no granular structures adhered to the fiber bundles in Fig. 6(i), making the overall appearance denser and more uniform.

3.4 Finite element simulation results

3.4.1 Validation of finite element model for assessing cartilage wear with superficial damage

In this finite element simulation of cartilage wear, the entire wear process was divided into three stages, each occupying 1 s in the simulation. The first stage (1 s) was the compression stage, which aimed primarily to ensure full contact of the cartilage. The second stage (1 s) was the wear stage, corresponding to 3,600 s of actual wear, which mainly simulated the cartilage wear process. The third stage (1 s) was the rebound stage, aimed at minimizing the judgment error of wear volume caused by elastic deformation of the cartilage (in finite element simulation, wear was considered irreversible deformation).

Figure 7(a) shows the relationships among the average contact stress on the upper surface of the lower friction component at the end of the first stage, the central processing unit (CPU) computation time, and the number of contact surface nodes. It is generally understood that sparser meshing is more likely to lead to distortion in finite element simulation results. Therefore, it is crucial to balance computational accuracy and time costs. In this finite element simulation study, a contact surface node count of 1,946 was chosen to ensure both computational accuracy and minimize time costs as much as possible.

Figure 7(b) shows the wear volume obtained from finite element simulations, along with a comparison of the wear volumes predicted by the experimental and theoretical models. The simulated wear volume was extracted at the end of the unloading stage. All comparisons were made against the average values of the corresponding real experimental groups. The error of the simulation model for the normal group was 7.1%, whereas the error of the theoretical model was 2.28%. For the chymotrypsin group, the simulation model had an error of 4.78%, and the theoretical model had an error of 5.23%. In the remove 10% group, the simulation model error was 2.84%, and the theoretical model error was 4.22%. The RMSE of the simulation model was 1.53 mm^3 , whereas that of the theoretical model was 1.38 mm^3 . Both the finite element model and the theoretical model closely match the real wear experiments in terms of wear volume.

Compared with the wear contours in Fig. 7(c), the simulated results were similar to the basic trends observed in real contours, with greater wear corresponding to more severe superficial damage to the cartilage. In particular, the wear depth in the remove 10% group reached a maximum of 0.287 mm. These results confirm the validity of the finite element model established in this study.

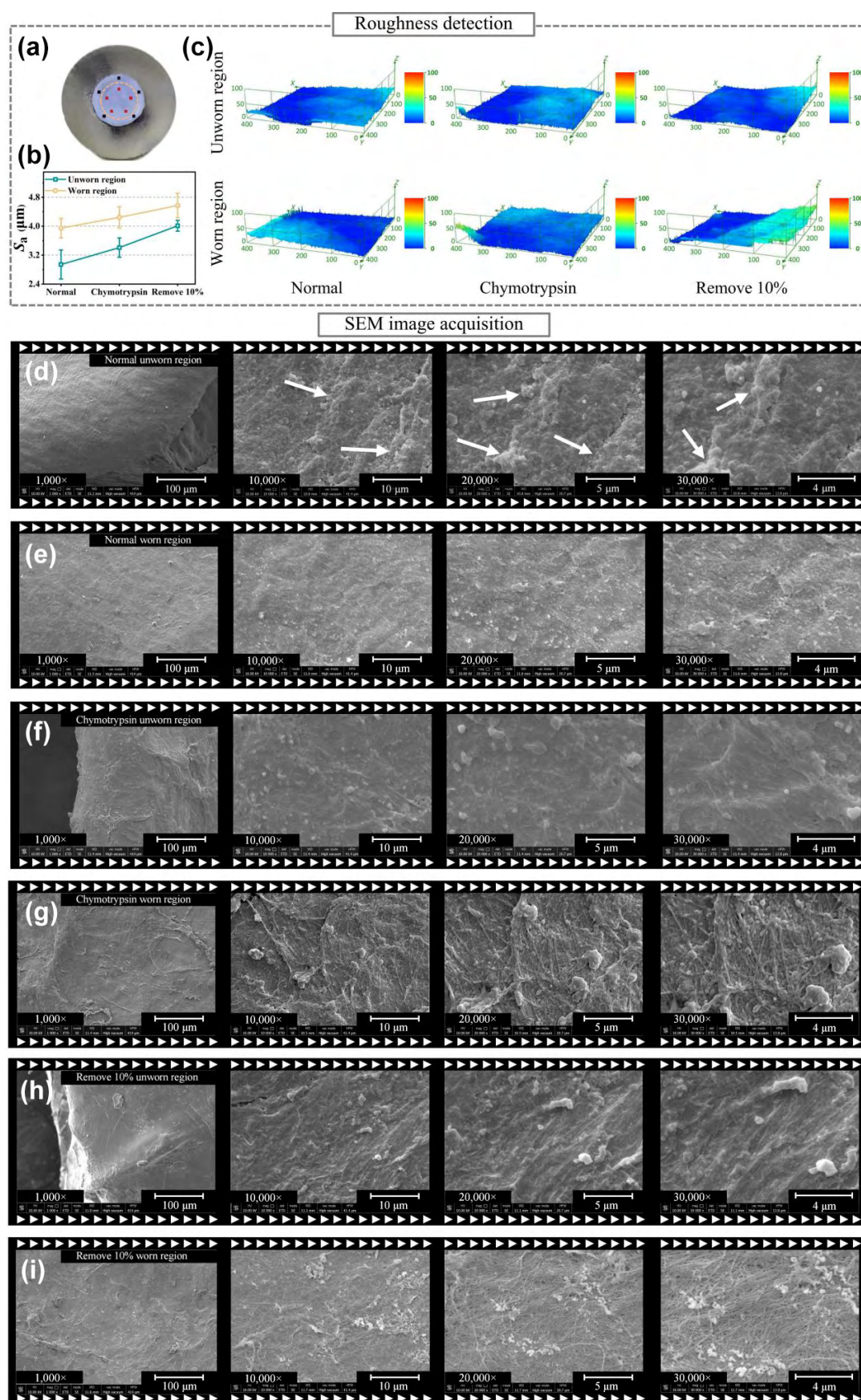


Fig. 6 Microscopic morphology of cartilage wear under superficial damage. (a) Schematic of cartilage roughness measurement points. (b) Roughness of worn and unworn cartilage regions. (c) 3D morphology of worn and unworn cartilage regions. (d) SEM image of unworn region in normal group. (e) SEM image of worn region in normal group. (f) SEM image of unworn region in chymotrypsin group. (g) SEM image of worn region in chymotrypsin group. (h) SEM image of unworn region in remove 10% group. (i) SEM image of worn region in remove 10% group.

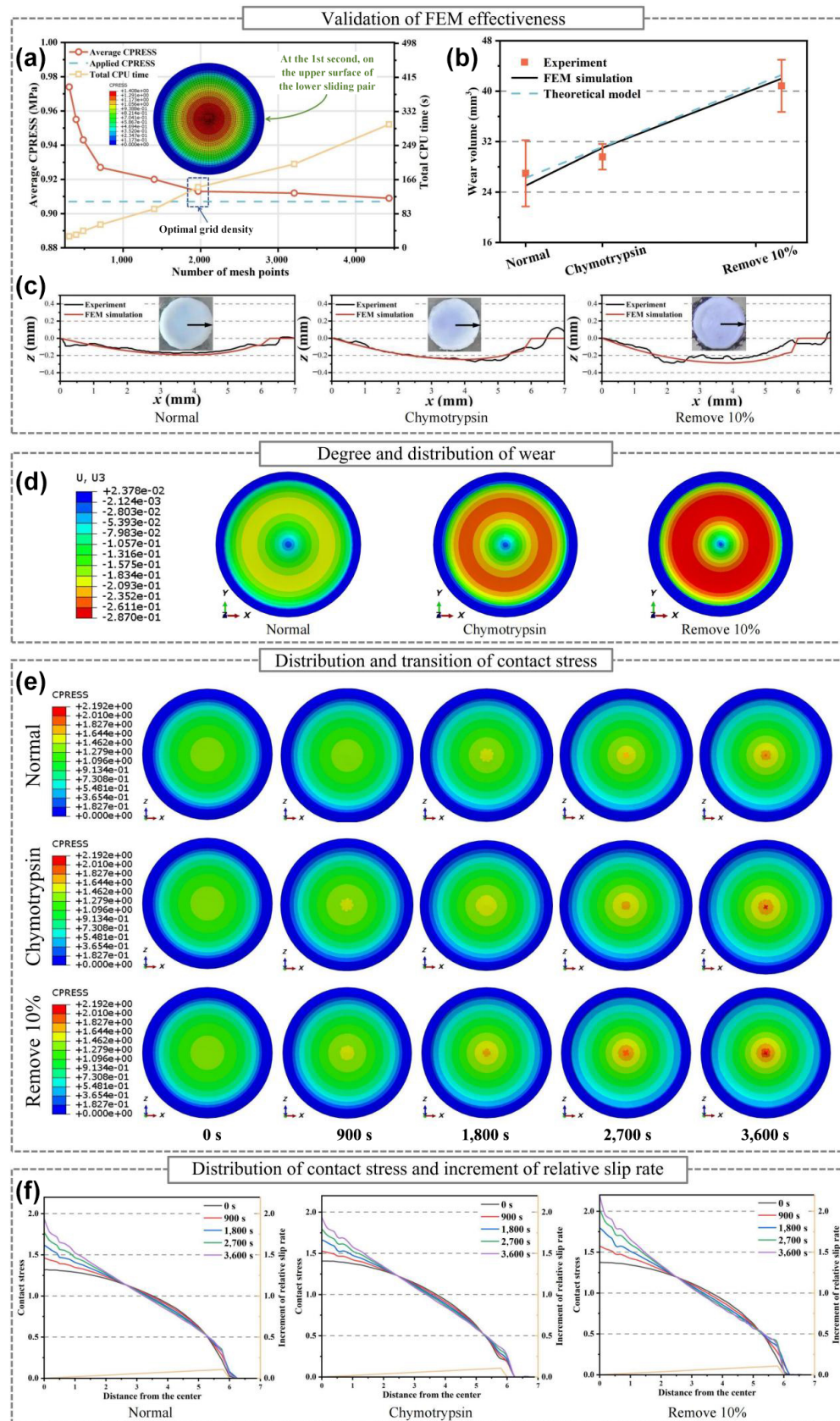


Fig. 7 Finite element simulation of cartilage wear under superficial damage. (a) Mesh independence verification. (b) Comparison of finite element-simulated wear volume, theoretical model-predicted wear volume, and actual experimental results. (c) Comparison of simulated and actual wear contours of upper surface for normal group, chymotrypsin group, and remove 10% group. (d) Simulation results of extent of wear on upper surface of cartilage. (e) Distribution and evolution of contact stress within 3,600 s of wear simulation. (f) Distribution of contact stress and relative sliding rate increase during 3,600 s of wear simulation. In the finite element contour plots, CPRESS denotes contact pressure, U displacement, and U3 displacement in the third direction, corresponding to the vertical displacement used to evaluate surface deformation/wear depth.

3.4.2 Changes in cartilage surface and contact stress during wear process

On the basis of the finite element wear model established above, we further analyzed the effects of superficial zone damage on the cartilage surface and contact stress. Figure 7(d) shows the simulated results of the wear depth on the cartilage surface. As indicated in the legend, areas with colors closer to blue represent shallower wear, whereas areas closer to red indicate deeper wear. On the basis of the wear depth range shown in the legend, we consider a wear depth h in the range of $0.155 \text{ mm} < h < 0.287 \text{ mm}$ to indicate deep wear. The proportion of the surface area in the normal group within this range was 41.46%, while it was 59.76% in the chymotrypsin group and 66.83% in the remove 10% group.

Figure 7(e) shows the distribution of the contact stress and its variation over time. The distribution of contact stress was not uniform, with a trend of stress concentrating toward the center even from the initial moment. As wear progressed, cartilage with more severe superficial damage exhibited more pronounced stress concentrations. Figure 7(f) provides a quantitative analysis of the distribution of the contact stress and the relative increase in the sliding rate. As shown, more severe damage led to a greater increase in the contact stress over time, whereas the relative increase in the sliding rate was not affected by the degree of damage or time. At the beginning of the wear process, the contact stresses at the center for the normal, chymotrypsin, and remove 10% group samples were 1.31, 1.35, and 1.37 MPa, respectively. By the end of the wear process, the contact stress at the center increased by 45.51% in the normal group, 48.2% in the chymotrypsin group, and 57.27% in the remove 10% group.

4 Discussion

Osteoarthritis and cartilage motion injuries are globally persistent and severe diseases that significantly impact quality of life. These diseases often begin with superficial zone damage to the cartilage. Therefore, exploring how superficial zone damage affects the tribological properties of cartilage is crucial. In this study, we characterized different degrees of superficial zone damage in cartilage and conducted friction and wear experiments under varying pressures and speeds on damaged cartilage samples. We also used FEM simulations to overcome some limitations of real-world experiments.

In this study, the main methods for creating superficial zone damage models were enzymatic degradation with chymotrypsin and physical abrasion using a cordless rotary tool. Unlike Graindorge et al. [35], who used sodium dodecyl sulfate (SDS) treatment to remove surface-adsorbed components while preserving lubrication and enabling self-repair via synovial fluid adsorption, our approach directly disrupted PRG4 anchoring and the collagen framework, leading to irreversible lubrication loss. The primary principle of chymotrypsin degradation is to specifically destroy lubricin (PRG4), significantly reducing the binding force between the upper mucus zone and the lower fibrous layer, leading to phenomena similar to mucus zone dissolution and extensive peeling from the cartilage surface. This can be observed in the comparison between Figs. 6(d) and 6(f). Similarly, Kumar et al. [16] reported that the use of chymotrypsin significantly increases the friction coefficient of cartilage but did not analyze the specific mechanism behind this increase. Zappone et al. [22], in their study on lubricin, subsequently provided a detailed explanation of the mechanism by which chymotrypsin affects lubricin. These findings revealed that chymotrypsin specifically cleaves the C-terminal domain of PRG4 (including the

hemopexin-like (HPX) homology region), thereby disrupting its anchoring function. This cleavage leads to conformational changes in the mucin domain of PRG4, causing it to detach and lose its ability to stabilize the complex through electrostatic interactions with HA/PL via negatively charged glycan chains [22]. Although this study was based on the method of applying lubricin to mica substrates, we believe that the findings regarding lubricin function and targeted destruction by chymotrypsin are accurate. Furthermore, we observed that lubricin in cartilage also exhibited end-anchoring failure under the action of chymotrypsin. Notably, the hydrolytic activity of chymotrypsin may also contribute to the secondary degradation of hyaluronan-binding proteins in the superficial zone. As reported by Blumberg and Ogston [36], the use of chymotrypsin or trypsin alone failed to demonstrate hydrolytic activity toward hyaluronan-protein complexes. However, Ogston and Sherman [37] later reported that their combined use could produce hydrolytic effects.

With respect to damage characterization, Indian ink, which has a high affinity for the fibers in articular cartilage, was used in this study to indicate the degree of fiberization and damage to the cartilage surface [38]. Macroscopically, normal cartilage is characterized by its glossy surface, whether it is stained with Indian ink or not. It also feels very smooth, attributed to the mucus zone in the superficial zone. The chymotrypsin-treated cartilage surface showed ink-stained speckles and a sticky feel, consistent with the theory of Zappone et al. [22]. Chymotrypsin rapidly desorbs the terminal domain of lubricin, releasing the non-adhesive mucinous domain [1]. The scratches and rough feel observed in physically abraded cartilage are attributed to the exposure of collagen fibers due to superficial zone damage (Fig. 3(a)). The observations in Figs. 3(b), 6(d), 6(f), and 6(h) are consistent with macroscopic damage principles; intact cartilage in the normal group ensures a smooth surface, whereas fibrous structures are exposed in both the chymotrypsin group and the remove 10% group.

In a study by Seror et al. [39], which explored how PLs form an appropriate boundary layer on the cartilage surface, they successfully simulated a friction coefficient close to that of the cartilage surface on mica using a mixture of HA and PL. On the basis of this evidence, they proposed that the cartilage boundary layer primarily consists of HA and PL, which are commonly found in cartilage and synovial fluid. Crucially, lubricin (PRG4) was identified as the key factor in maintaining the stability of this boundary layer on the cartilage surface. Furthermore, their study revealed the formation and stabilization mechanisms of this composite boundary layer: HA forms a surface-anchoring network by hydrogen bonding with the sialylated O-glycans of the mucin domain of lubricin. Moreover, the N/C-terminal domains of lubricin are anchored to the cartilage collagen matrix through hydrophobic interactions. PL is embedded in the PRG4–collagen complex via its hydrophobic tail, while its phosphocholine headgroup interacts with the carboxyl groups of HA through charge–dipole interactions, forming a stable HA–PL interfacial layer [39]. The lubricating function of this interfacial layer relies on the high hydration capacity of the phosphocholine headgroup (binding 12–15 water molecules) and its fast dynamic exchange rate (10^8 s^{-1}). Under shear-induced fluidization of the hydration layer, this interfacial layer achieves ultralow friction ($\mu \approx 0.001$) [1]. Our findings on superficial zone damage modeling are highly consistent with this mechanism. As shown in Fig. 3(d), HA expression disappears from the cartilage surface following chymotrypsin digestion or physical abrasion, indicating that HA is indeed a key component of the boundary layer in normal

cartilage. Furthermore, the cloud-like substance enriched on the cartilage surface (marked by white arrows in Fig. 6(d)) is most likely PL, as supported by the characteristic morphology of dehydrated PL observed in SEM images [40]. Notably, PL did not reappear after subsequent wear or direct damage modeling, suggesting that the presence of PL depends on the integrity of the PRG4–HA interfacial layer. This series of structural evidence confirms that the cartilage boundary layer is primarily composed of HA and PL, with lubricin playing a central role in maintaining the structural stability and lubricating function of this boundary layer. Notably, in cartilage structural studies, the boundary layer is often referred to as the mucus zone. The concept of the boundary layer originates from cartilage lubrication studies, whereas the mucus zone comes from cartilage structural studies. Therefore, the above discussion on the structure of the boundary layer also applies to the mucus zone.

The coefficient of friction (COF) is a parameter commonly used to evaluate the lubrication state of cartilage. An important basis for determining the lubrication state of cartilage is the correlation between the COF and factors such as pressure and sliding speed [31, 33, 41]. In this study, we observed a gradual increase in the COF with increasing friction time (Figs. 4(b)–4(f)). This phenomenon essentially reflects the dynamic transition in the biphasic solid–fluid load distribution under an external load. According to the biphasic theory proposed by Mow et al. [42], the initial external load is primarily borne by the interstitial fluid pressure. In this state, solid–solid contact is minimal, and the COF remains at a low level (approximately 0.003–0.005 for normal cartilage). During loading, as time progresses, interstitial fluid continuously seeps out through the pores of the cartilage surface, reducing the fluid phase load contribution from > 80% initially to < 20% [43]. This process leads to a phase transition in the lubrication mechanism. Specifically, during the fluid film lubrication-dominant phase, the poroelastic pressure maintains fluid support, keeping the COF at a low level due to the sufficient thickness of the fluid film (Figs. 4(g) and 4(i)). In the mixed lubrication transition phase, the loss of the fluid phase leads to a rapid increase in solid-phase contact, during which the viscoelastic contribution of the boundary layer material gradually strengthens. In the boundary lubrication steady-state phase, the interstitial fluid pressure almost completely dissipated, and the COF stabilized (approximately 0.006–0.008 for normal cartilage) (Figs. 4(h) and 4(j)).

With respect to the effect of the sliding speed on the lubrication state, the early reduction in the COF with increasing speed (Fig. 4(i)) reveals the compensatory effect of the migrating contact area (MCA) on fluid film lubrication [43]. In normal cartilage, an increase in sliding speed increases the Péclet number ($P_e = V_a/H_{+A}k$, where V is the sliding speed; a is the contact radius; H_{+A} is the aggregate modulus; k is the permeability) from 0.5×10^4 to 2×10^4 ($V \approx 5\text{--}17.72 \text{ mm}\cdot\text{s}^{-1}$, $a \approx 2.82 \text{ mm}$, $H_{+A} = 13 \text{ MPa}$ [44], $k = 2.29 \times 10^{-16} \text{ m}^2\cdot(\text{Pa}\cdot\text{s})^{-1}$), far exceeding the critical value $P_e = 1$. Of course, in the spin–squeeze motion, this range represents only the Péclet number at $r/2$; in fact, there are differences between low-speed and high-speed regions within the entire cartilage contact area. In the low-speed region near the center, $P_e < 1$, and the fluid film cannot be effectively maintained. In the high-speed region away from the center, $P_e > 1$, where the migrating contact speed is significantly faster than the rate of interstitial fluid outflow, enhancing the fluid film lubrication effect. The increase in sliding speed expands the $P_e > 1$ region, which explains why the growth trend of the COF becomes more gradual in Figs. 4(d)–4(f) and why the average COF decreases with increasing speed, as shown in Fig. 4(i).

The early decrease in the COF with increasing pressure is also attributed to the pressurization mechanism of the interstitial fluid (Fig. 4(g)). In normal cartilage, an increase in pressure (from 1 to 2 MPa) rapidly increases the poroelastic pressure p generated by solid-phase compression ($p = H_{+A}\nabla u$, where ∇u is the volumetric strain), thereby driving interstitial fluid to rapidly seep out and form a fluid film [38]. However, in the superficial layer damage group, high pressure exacerbates the irreversible decline in the lubrication mechanism. The increased porosity of the superficial layer causes the outflow rate of interstitial fluid to exceed the interfacial anchoring rate, leading to rapid dissipation of the fluid film. Simultaneously, superficial layer damage results in a rapid reduction in the coverage of lubricin, leading to severe friction caused by direct contact with collagen fibers. Although high pressure can still temporarily increase the thickness of the fluid film, the COF in the damaged group increases significantly over a short period (Figs. 4(b)–4(d)). This demonstrates that the superficial layer not only serves as a permeability barrier but also functions as a strategic reserve layer for interfacial lubrication molecules. Its integrity determines the robustness of cartilage lubrication under high-pressure conditions.

In our study, the primary factor driving differences in COF was varying degrees of superficial cartilage damage. After eliminating the effects of pressure and speed on interstitial fluid, the underlying nature of superficial damage becomes clear. More severe superficial damage leads to a higher COF at any stage of the friction process, indicating that boundary friction dominates the entire process. The nature of boundary friction depends on the structural composition of the boundary layer [1]. As previously discussed, the boundary layer of intact cartilage is formed by a complex of PL and HA, with the phospholipid bilayer playing a key role in maintaining low friction. This specialized boundary lubrication, generated by the phospholipid bilayer, is commonly referred to as hydration lubrication [18, 45]. Notably, digestion by chymotrypsin or direct physical abrasion partially or completely disrupts the boundary layer responsible for hydration lubrication. This disruption causes a transition at the frictional interface from a phospholipid bilayer to a fibrous layer, which is the primary cause of the increased COF. At the molecular level, this transition can be explained by interfacial energy theory. Following chymotrypsin treatment, the anchoring function of lubricin (PRG4) is lost, leading to dissociation of the HA–PL complex due to the loss of bridging interactions [39]. Once hydration lubrication fails, the frictional interface becomes dominated by adhesion between collagen fibers. In normal cartilage, the hydrated layer formed by phosphatidylcholine headgroups lowers the interfacial energy ($\Delta\gamma \approx 10 \text{ mJ}\cdot\text{m}^{-2}$), thereby suppressing adhesion. However, after damage, polar groups such as hydroxyproline become exposed, increasing the interfacial energy to approximately $\Delta\gamma \approx 50 \text{ mJ}\cdot\text{m}^{-2}$ [1]. This trend in adhesion strength aligns with the interfacial energy theory for rough hydrophilic surfaces [46]. On the basis of the Johnson–Kendall–Roberts (JKR) theory, adhesion forces significantly influence surface friction in cartilage with flexible surfaces. The damaged phospholipid bilayer can no longer effectively mitigate the increase in adhesion forces between contact surfaces, leading to a further increase in the COF. Therefore, the destruction of the boundary layer and the increase in interfacial adhesion are the key mechanisms driving the increase in the COF.

The final COF of the normal group in Fig. 4(j) appears to be an anomaly in the overall trend, as the final COF should generally be independent of speed due to boundary lubrication dominance. In fact, the increase in the COF here highlights another issue in the cartilage tribology system: whether the COF is independent of

wear. For example, Oungoulouian et al. [47] considered friction and wear to be directly related. However, many scholars argue that a low COF does not necessarily indicate low wear [48]. On the basis of evidence from this study and our previous layered cartilage theory [24], we believe that these views are not contradictory but depend on the timing of observation. Cartilage, an anisotropic tissue with distinct layered structures, has boundary friction characteristics highly dependent on the material properties of the boundary layer at the contact site [1]. As shown in Fig. 6(e), even at a speed of 1 Hz, the mucus zone of the cartilage thinned. This implies that wear becomes more severe at higher speeds, and once the mucus zone fails, the COF will eventually approximate that of the chymotrypsin-treated group. This does not mean that the chymotrypsin or the remove 10% group does not experience wear; rather, the fibrous layer in the superficial zone is thicker and more resistant to complete erosion than the mucus zone is.

Similarly, the investigation of wear under superficial cartilage damage is also within the scope of this study. Our data show that, on a macroscopic level, more severe superficial damage leads to greater wear volume and higher dimensionless wear coefficients (Figs. 5(d) and 5(e)). The measurement of wear volume and the dimensionless wear coefficient is commonly used in studies on the wear of prostheses made of alloys or polytetrafluoroethylene (PTFE) in cartilage repair [49]. Measuring the wear volume of cartilage itself is challenging because of individual differences in cartilage and its irregular shape [41]. In our study, we attempted a drainage-based method to convert the irregular wear volume of cartilage into an easily detectable rise in water level. The data show that this method is effective.

As shown in Fig. 5(d), the dimensionless wear coefficient calculated on the basis of the built theoretical wear model essentially represents the wear resistance of cartilage under superficial zone damage. The increase in the dimensionless wear coefficient indicates that once the superficial zone is damaged, the wear rate of the cartilage accelerates. In other words, the more severe the superficial zone damage is, the greater the acceleration of cartilage wear. Even normal cartilage clearly has a wear rate. Therefore, cartilage in an active state in the body should have a self-repair equilibrium. The mainstream view is that the wear of normal adult cartilage can be compensated for by the matrix components synthesized by chondrocytes [50]. However, the occurrence of cartilage degeneration or osteoarthritis disrupts the balance between degradation and synthesis in chondrocytes, leading to cartilage tissue damage [51]. Chondrocytes may also be lost during wear and thus be unable to maintain tissue homeostasis in microdamaged areas [52]. First, we believe that the view of wear equilibrium maintenance in normal cartilage is reasonable, but our data reflect that further wear of damaged cartilage is also an accelerated process without the involvement of chondrocytes. Even if osteoarthritis does not affect the balance of matrix metabolism or cause cell death in microdamaged areas, accelerated wear of cartilage after superficial zone damage still occurs. This is more akin to the wear properties of the solid-phase matrix of cartilage itself.

An increase in cartilage surface roughness has been widely demonstrated as a typical characteristic of cartilage under wear [38, 52]. This study further validates this classical conclusion through topographic analysis (Figs. 6(a)–6(c)). Notably, in cartilage samples with pre-existing damage in the superficial zone, traditional wear processes can still induce roughness changes comparable to those in intact tissue. This result suggests that even when the superficial zone of the cartilage is damaged, its wear behavior still follows the general trend of increasing roughness. In fact, superficial zone damage represents a stage of macroscopic

wear, which explains why it results in similar coarsening of surface topography.

On a microscopic level, wear occurs in cartilage regardless of superficial zone damage. Notably, the wear zone images of the chymotrypsin group and the remove 10% group show exposed and dense fibers, but the fiber morphology is different (Figs. 6(g) and 6(i)). This is more related to the current wear depth. The wear depth of the remove 10% group was closer to that of the middle zone fibers, making it difficult to observe residual superficial zone material in the fibers. Additionally, since no grooves or transfer films were observed on the cartilage surface after wear, the most likely wear type under the cartilage–cartilage compression spin motion mode is fatigue wear [53]. The most significant phenomenon is that the material on the worn surface peels off in sheets, and the good lubrication performance of the cartilage itself effectively prevents adhesive and abrasive wear.

In this study, finite element simulations were used to supplement data that cannot be observed in real time during cartilage wear experiments. First, the use of the finite element subroutine based on the modified Archard wear model for cartilage wear modeling is feasible. The simulated wear volumes and surface wear contours closely match the experimental results (Figs. 7(b) and 7(c)). Second, regarding the formation mechanism of the wear contours, the distribution of contact stress and relative sliding rate increments in Figs. 7(e) and 7(f) explain why the lowest wear point does not occur at the wear boundary. As shown in Fig. 7(f), the contact stress decreases with increasing distance from the center, whereas the relative sliding rate increases in the opposite manner, making it unlikely for their product to peak at the edge. Currently, modeling cartilage on the basis of the Archard wear model is not uncommon. For example, Li et al. [54] successfully established a knee cartilage wear model to explain the mechanisms of osteoarthritis onset. However, the main issue with these studies is the lack of real experimental data to validate the accuracy of the models. In our research, we achieved mutual validation among experiments, theoretical models, and finite element models, with the results showing a high degree of consistency. Therefore, we believe that extending the finite element model in this study to a predictive model for real knee joint cartilage wear is feasible.

This study bridges biomedical engineering and clinical practice by revealing the multiscale tribological mechanisms of cartilage. Structurally inspired mucus zone insights enable optimized biomimetic lubricants and self-healing joint coatings through gradient material designs, enhancing artificial joint durability. Clinically, identified boundary-layer wear patterns transform cartilage degeneration diagnostics, enabling early detection through composition monitoring instead of conventional imaging. Targeted mucus region protection strategies facilitate precise, minimally invasive interventions. Quantitative fatigue wear analysis supports personalized osteoarthritis warning models, shifting disease management from reactive treatment to proactive prevention. These advances establish an integrated “prevention–diagnosis–treatment” clinical framework.

Notably, this study has certain limitations. For example, cartilage lubrication is often influenced by the composition of the lubricating fluid. Some studies have noted that different sliding fluid compositions significantly affect the COF and can directly influence cartilage lubrication performance [32, 41, 55]. In terms of wear characterization parameters, some studies have used the contents of glycosaminoglycans (GAGs) and collagen to characterize cartilage wear [56, 57]. Future studies should also investigate these histological wear characterization parameters.

5 Conclusions

The superficial zone of cartilage is critical to its exceptional tribological performance. In this study, a cartilage superficial zone damage model was established using chymotrypsin enzymolysis and 10% physical polishing, which systematically revealed lubrication and wear mechanisms following superficial zone damage. These findings indicate that the mucus zone, which is primarily composed of a hyaluronic acid-phosphatidylcholine complex with lubricin that enhances its adhesion to the cartilage surface, is essential for maintaining low-friction conditions. Once compromised, a sharp increase in the friction coefficient occurs. Cartilage predominantly operates in a mixed lubrication regime with boundary lubrication dominance, whereas interstitial fluid-mediated fluid film lubrication plays a crucial role in the early stage of friction. Fatigue wear is identified as the primary failure mode, with a significant dependence on the integrity of the boundary zone. These insights not only provide a molecular-level reference for the design of biomimetic joint lubricants but also suggest that the dynamic lubrication mechanisms of cartilage can be replicated using gradient-permeability biomaterials. Notably, preserving the integrity of the superficial zone may offer a new strategy for delaying osteoarthritis progression, while the detection of related components holds promise for early osteoarthritis diagnosis.

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Declaration of competing interest

The authors have no competing interests to declare that are relevant to the content of this article.

Electronic Supplementary Material

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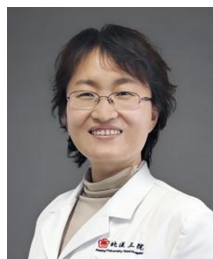
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