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Failure mechanisms of hydrogel-wet tissue adhesive interfaces: an energy dissipation perspective

Haitao Liu[1], Zhiyuan Meng[2], Rongrong Bao[3], Jing Rao[4, †], Mengxiao Chen[3, †], and Caofeng Pan[3, †]

[1]School of Materials Science and Engineering, Beihang University, Beijing 100191, China

[2]College of Biomedical Engineering & Instrument Science, Zhejiang University, Hangzhou 310027, China

[3]Beijing Key Laboratory for Atomic Manufacturing Equipment and Intelligent Sensing, Institute of Atomic Manufacturing, Beihang University, Beijing 100191, China

[4]School of Instrumentation and Optoelectronic Engineering, Beihang University, Beijing 100191, China

Abstract: Hydrogel-wet tissue adhesive systems hold great promise for biomedicine and bioelectronics, yet their practical application is severely restricted by interfacial failure in complex physiological environments. Such adhesives frequently suffer from fatigue, delamination and slippage well before reaching their theoretical adhesive strength, proving that conventional strength-based evaluation cannot reflect their actual stability. Classical fracture mechanics and viscoelastic adhesion theories describe failure through energy release rate, work of adhesion, and rate-dependent fracture energy, but they commonly incorporate dissipation into an effective fracture parameter and do not explicitly resolve how energy is stored at the interface, transferred into the bulk, and dissipated across the hydrated interface–bulk continuum. This review organizes current hydrogel-tissue adhesion strategies into a three-tier energy-regulation framework comprising interfacial buffering, interface-to-bulk energy transfer, and bulk dissipation. The framework is connected to measurable quantities, including energy release rate, effective fracture energy, fatigue threshold, interfacial stress concentration, transfer efficiency, and hysteresis loss, to provide semi-quantitative guidance for material design and comparison. Key failure modes, representative structural and molecular strategies, and a practical characterization workflow are discussed. Remaining challenges in parameter identification, cross-scale constitutive modeling, and in vivo validation are outlined.

Key words: hydrogel; wet tissue adhesion; interfacial failure; energy dissipation

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1. Introduction

Hydrogels have attracted extensive attention in the field of wet tissue adhesion due to their high water content[1], mechanical compliance[2], and excellent biocompatibility[3]. Their ability to form conformal contact with soft and dynamically changing biological tissues renders them promising for applications such as wound closure, tissue repair, and bio-integrated devices[4, 5]. Although initial adhesive strength has been significantly improved in recent years through various strategies including covalent bonding, hydrogen bonding, ionic interactions, and physical entanglement, achieving long-term stable adhesion in wet environments remains challenging[6–9]. Under actual physiological conditions, the hydrogel-tissue interface is continuously exposed to hydrated environments[10], cyclic deformations, and multiaxial stresses[11]. Under such complex conditions, the adhesive interface often fails gradually in the form of fatigue, progressive delamination, or interfacial slippage, rather than undergoing instantaneous fracture at the theoretical strength limit. These phenomena suggest that interfacial stability cannot be described solely by adhesive strength, but involves the dynamic evolution of mechanical responses in the coupled interface–bulk system. Conventional interfacial adhesion-strength-based evaluation mainly characterizes the maximum resistance of an interface to fracture under specific loading conditions, whereas long-term adhesion stability depends on how mechanical energy is distributed, transferred, and dissipated during continuous deformation and environmental perturbation. Most existing studies focus on enhancing interfacial interactions to improve initial adhesion performance[12, 13]. However, systems with similar adhesive strength often show significant differences in long-term stability, indicating that a single interface-strengthening strategy cannot explain or predict adhesive behavior. These observations have been demonstrated in several representative studies. For example, Yang *et al.* showed that topological adhesion markedly enhanced adhesion durability through topological interlocking and stress redistribution without proportionally increasing the initial adhesive strength[14]. Similarly, Wang *et al.* demonstrated that hydrogen-bond-pinned chain entanglement effectively suppressed interfacial crack propagation and prolonged service life by promoting stress delocalization rather than simply strengthening the interface[8]. Fang *et al.* further reported that fibrillar connected double-network hydrogels exhibited greatly improved fatigue resistance despite comparable static mechanical properties[9]. These studies collectively suggest

Correspondence to: J Rao, jingrao@buaa.edu.cn; M X Chen, mengxiaochen@buaa.edu.cn; C F Pan, pancaofeng@buaa.edu.cn
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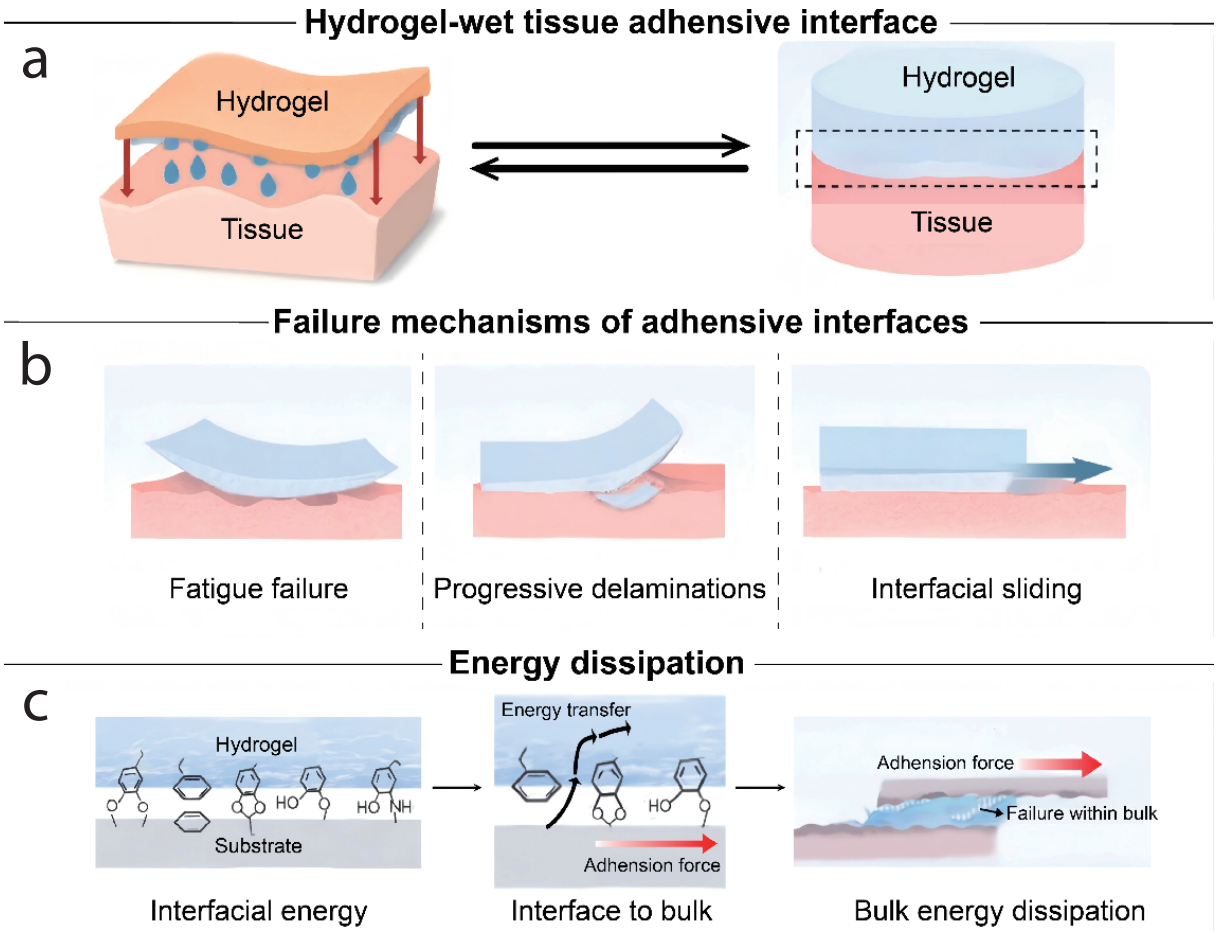


Fig. 1. (Color online) Failure mechanism and energy dissipation at hydrogel-wet tissue adhesive interface. (a) Schematic illustration of adhesion between hydrogel and tissue. (b) Failure mechanisms of the hydrogel-wet tissue adhesive interface: the progressive evolution from local damage accumulation to macroscopic interfacial failure under complex physiological loads. (c) Energy dissipation pathways of the hydrogel-wet tissue adhesive interface: the multistage mechanical energy regulation process across interfacial buffering, interface–bulk energy transfer, and bulk energy dissipation.

that long-term adhesion stability is governed by the regulation of stress transfer and energy dissipation rather than adhesive strength alone. This limitation highlights the need to revisit wet tissue adhesion from a more universal physical perspective[15, 16]. Energy-based analysis of adhesion is foundational rather than new. Griffith introduced the energy-release criterion for crack growth, Rivlin and Thomas adapted tearing energy to elastomers, Johnson–Kendall–Roberts and Kendall related surface energy and specimen geometry to adhesive contact and peeling, and Gent–Schultz and de Gennes emphasized the strong rate- and temperature-dependence introduced by viscoelastic losses[17]. In these classical descriptions, a crack propagates when the energy release rate G reaches an effective fracture energy Γ_{eff}; the measured Γ_{eff} can substantially exceed the thermodynamic work of adhesion because it includes crack-tip, interfacial, and bulk losses. These theories provide the essential mechanical basis for interpreting hydrogel-tissue adhesion[18–22].

Against this background, understanding adhesive behavior from an energy perspective provides a more unified analytical approach[23, 24]. As shown in Fig. 1, this perspective no longer treats the interface as an isolated boundary, but regards the adhesive system as an integrated whole coupled by the interface and the bulk, where the distribution and evolution of mechanical energy determine adhesive stability. We first categorize the major failure modes of hydrogel-wet tissue adhesive interfaces, including fatigue failure, progressive delamination, and interfacial slippage. Building upon these failure behaviors, we introduce an interface–bulk coupled energy-regulation framework and discusses how interfacial buffering, interface-to-bulk energy transfer, and bulk energy dissipation govern the initiation and evolution of failure. Finally, the implications of this framework for the rational design of stable hydrogel tissue adhesives are discussed.

2. Failure mechanisms of hydrogel-wet tissue adhesive interfaces

In complex physiological environments, hydrogel-wet tissue adhesive interfaces are usually under the coupling of continuous hydration and dynamic loads, exhibiting a series of common mechanical failure behaviors (Fig. 2(a))[25–29]. On the one hand, the hydrated layer on the tissue surface weakens effective molecular contact and reduces interfacial friction[30]; on the other hand, cyclic stretching, shear deformation, and multiaxial strain subject the interface to long-term non-uniform stress fields[31]. Under such conditions, stress gradually concentrates from the initial relatively uniform distribution to interfacial defects, edge regions, or incompletely wetted areas, accompanied by the progressive development of local relative displacement and interfacial separation. Therefore,

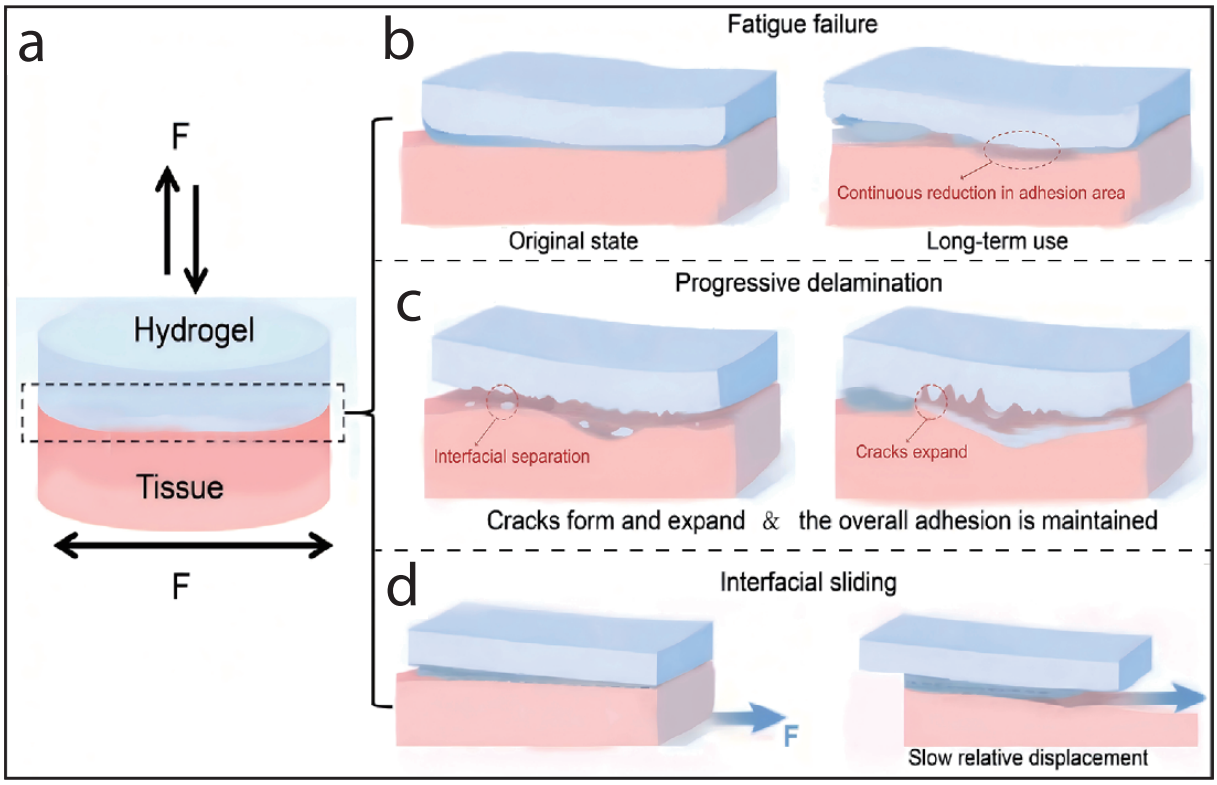


Fig. 2. (Color online) Schematic illustration of hydrogel-tissue adhesive interface failure mechanisms and classification. (a) Models demonstrating stress behavior caused by dynamic loads. (b)–(d) Three typical interfacial failure modes: fatigue failure from cyclic loading damage, progressive delamination from tensile/peeling crack propagation, and interface sliding from excessive shear stress.

interfacial failure is usually not instantaneous, but a progressive process evolving from local damage accumulation to macroscopic failure[32]. Based on their mechanical performance and evolution paths, the typical failure modes of wet tissue adhesive interfaces can be summarized into three categories: fatigue failure, progressive delamination, and interfacial slippage[33, 34].

Fatigue failure mainly occurs under low-amplitude cyclic loading (Fig. 2(b)). In a single loading cycle, the interface can generally maintain the overall adhesive state. However, with the increase of cycle numbers, irreversible damage gradually accumulates in the interfacial structure. This process is characterized by the continuous degradation of local adhesive regions and the gradual reduction of effective contact area, with damage accumulating dispersedly in space. When the accumulated damage reaches a critical level, the overall failure of the interface is eventually triggered[35]. Such fatigue-driven failure is commonly encountered in wearable bioelectronics, implantable sensors, and cardiac monitoring devices, a representative example is hydrogel-based wearable or implantable bioelectronic devices, such as ECG electrodes and cardiac patches, which are continuously subjected to heartbeat-, respiration-, or body-motion-induced cyclic deformation[36, 37]. Although each loading event is relatively small, repeated cycles can progressively deteriorate interfacial integrity and eventually result in adhesion failure. Progressive delamination usually occurs under continuous tensile or peeling loads (Fig. 2(c)). Debonding first occurs in local areas of the interface, and then damage propagates gradually along the interface direction, forming continuous cracks that spread to a larger range. This process has obvious spatial propagation characteristics, that is, evolving from local initiation to macroscopic interfacial separation, and finally leading to the overall peeling of the adhesive structure. Progressive delamination is frequently observed in wound closure, tissue sealing, and tissue repair applications, where tensile loading, tissue movement, or peeling forces can continuously drive crack propagation along the adhesive interface. For example, hydrogel wound dressings or tissue adhesive patches attached to skin or internal organs may experience localized debonding at the edge, which subsequently propagates under tissue movement or external tension, ultimately causing large-scale adhesive failure. Interfacial slippage mostly occurs under high shear stress conditions. When the shear load borne by the interface exceeds its static retention capacity, the system preferentially releases deformation through interfacial relative displacement instead of immediate fracture (Fig. 2(d)). In this process, the interface remains in contact but undergoes continuous slippage, accompanied by local structural adjustment and frictional dissipation. With the accumulation of slippage, interfacial performance gradually degrades, which may eventually evolve into delamination or even complete failure[38]. This failure mode is particularly relevant to soft bioelectronic interfaces and tissue-contact devices attached to highly mobile tissues, where substantial shear deformation generated by body movement can induce relative displacement at the hydrogel-tissue interface, and compromise signal stability and long-term adhesion reliability[39].

3. Energy dissipation pathways governing adhesion stability

A core conclusion from recent studies is that the durability of hydrogel-wet tissue adhesion is not determined by a sin-

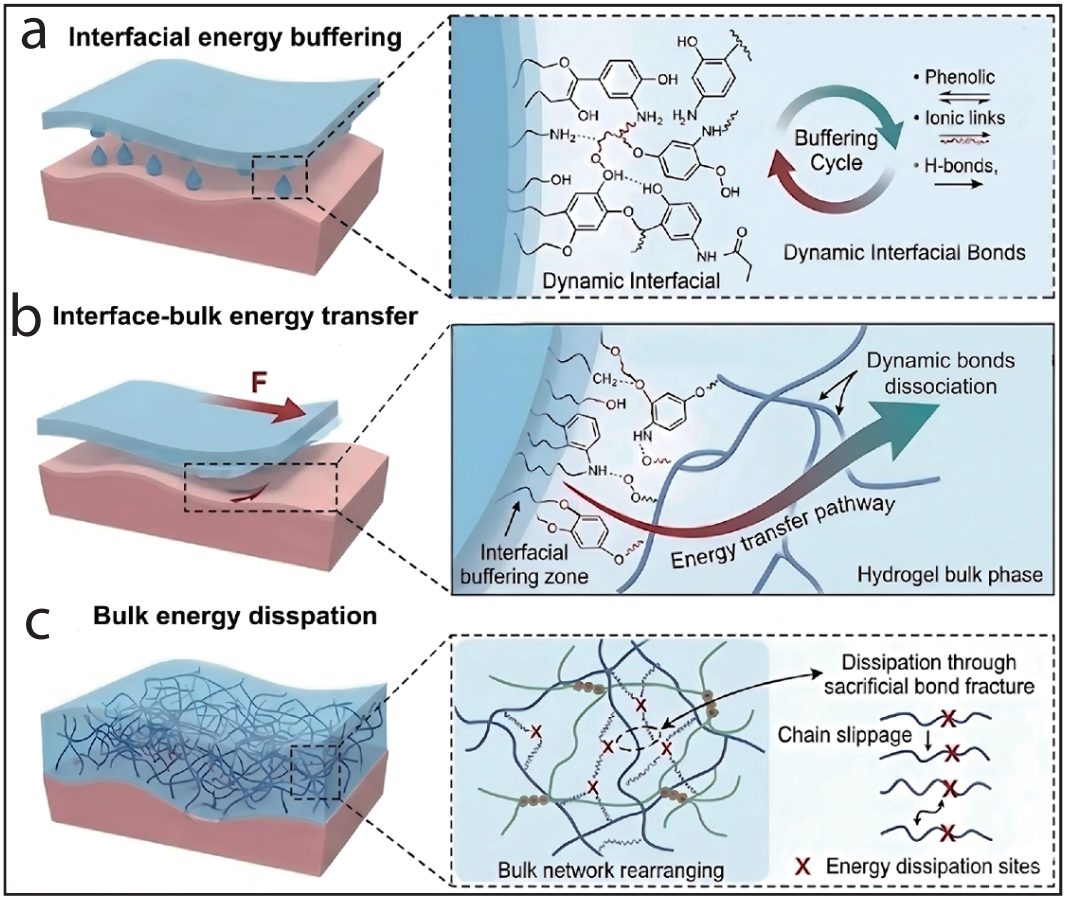


Fig. 3. (Color online) Energy dissipation pathways in the hydrogel-wet tissue adhesion interface. (a) Schematic of interfacial energy buffering through a buffering cycle involving the breaking and reformation of dynamic interfacial bonds (e.g., phenolic, ionic links, H-bonds). (b) Interface-to-bulk energy transfer, where energy is directed from the interfacial buffering zone into the hydrogel bulk phase, triggering dynamic bond dissociation. (c) Bulk energy dissipation via sacrificial bond fracture, chain slippage, and bulk network rearrangement at energy dissipation sites.

gle dissipation mechanism, but by the synergistic effect of multiple energy dissipation pathways across multiple spatial scales[40–42]. From an energy-balance perspective, adhesion stability depends on the competition between energy input, temporary storage at the interface, transfer into the bulk, and irreversible energy dissipation. Quantitative descriptors such as interfacial toughness, fatigue threshold and energy dissipation efficiency provide measurable indicators of different stages of this process. Classical fracture mechanics provides the system-level failure criterion, whereas the three-tier framework resolves the internal energy pathways that determine the effective resistance to failure. For an adhesive specimen with total potential energy Π and crack area A, the energy release rate is

$$G = -\frac{\partial \Pi}{\partial A}. \tag{1}$$

Crack advance is expected when $G \geqslant \Gamma_{\mathrm{eff}}(v, f, H)$, where Γ_{eff} depends on crack speed v, loading frequency f, hydration state H, geometry, and material history. For bookkeeping over a loading interval, the input work may be expressed as

$$W_{\mathrm{in}} = \Delta U_{\mathrm{i}} + \Delta U_{\mathrm{b}} + W_{\mathrm{d,i}} + W_{\mathrm{d,b}} + W_{\mathrm{crack}}. \tag{2}$$

Here, ΔU_{i} and ΔU_{b} are recoverable energies stored in the interfacial and bulk regions; $W_{\mathrm{d,i}}$ and $W_{\mathrm{d,b}}$ are irreversible interfacial and bulk losses; and W_{crack} is the energy consumed by damage and new crack area. Interface-to-bulk transfer is an internal energy flux, $J_{\mathrm{i}\to\mathrm{b}}$, rather than an additional sink in the global balance. Accordingly, interfacial buffering corresponds mainly to ΔU_{i}, interface-to-bulk transfer governs redistribution between ΔU_{i} and ΔU_{b}, and bulk dissipation contributes primarily to $W_{\mathrm{d,b}}$. Quantitative descriptors can be assigned to each stage. At peak load, interfacial buffering can be assessed from the recoverable strain energy within a defined interfacial zone and from the stress-concentration factor

$$K_{\sigma} = \frac{\sigma_{\max}}{\sigma_{\mathrm{nom}}}. \tag{3}$$

Transfer can be evaluated from full-field strain-energy maps using $\eta_{\mathrm{tr}} = U_{\mathrm{b}}/(U_{\mathrm{i}} + U_{\mathrm{b}})$, where U_{i} and U_{b} are the recoverable energies in predefined interfacial and bulk domains. Bulk dissipation can be represented by $\eta_{\mathrm{d}} = W_{\mathrm{hys}}/W_{\mathrm{load}}$, where W_{hys} is the cyclic hysteresis loss. These operational metrics do not constitute a universal constitutive model, because their values depend on geometry and the selected domain boundary; however, reporting the test geometry, interfacial-zone thickness, loading rate, hydration, and temperature makes cross-system comparison possible. As shown in Fig. 3, these energy dissipation pathways collectively form a multistage energy regulation system: Energy is first buffered at the interface through dynamic reversible bonding interactions (e.g., hydrogen bonds, ionic interactions, phenolic coordination), then trans-

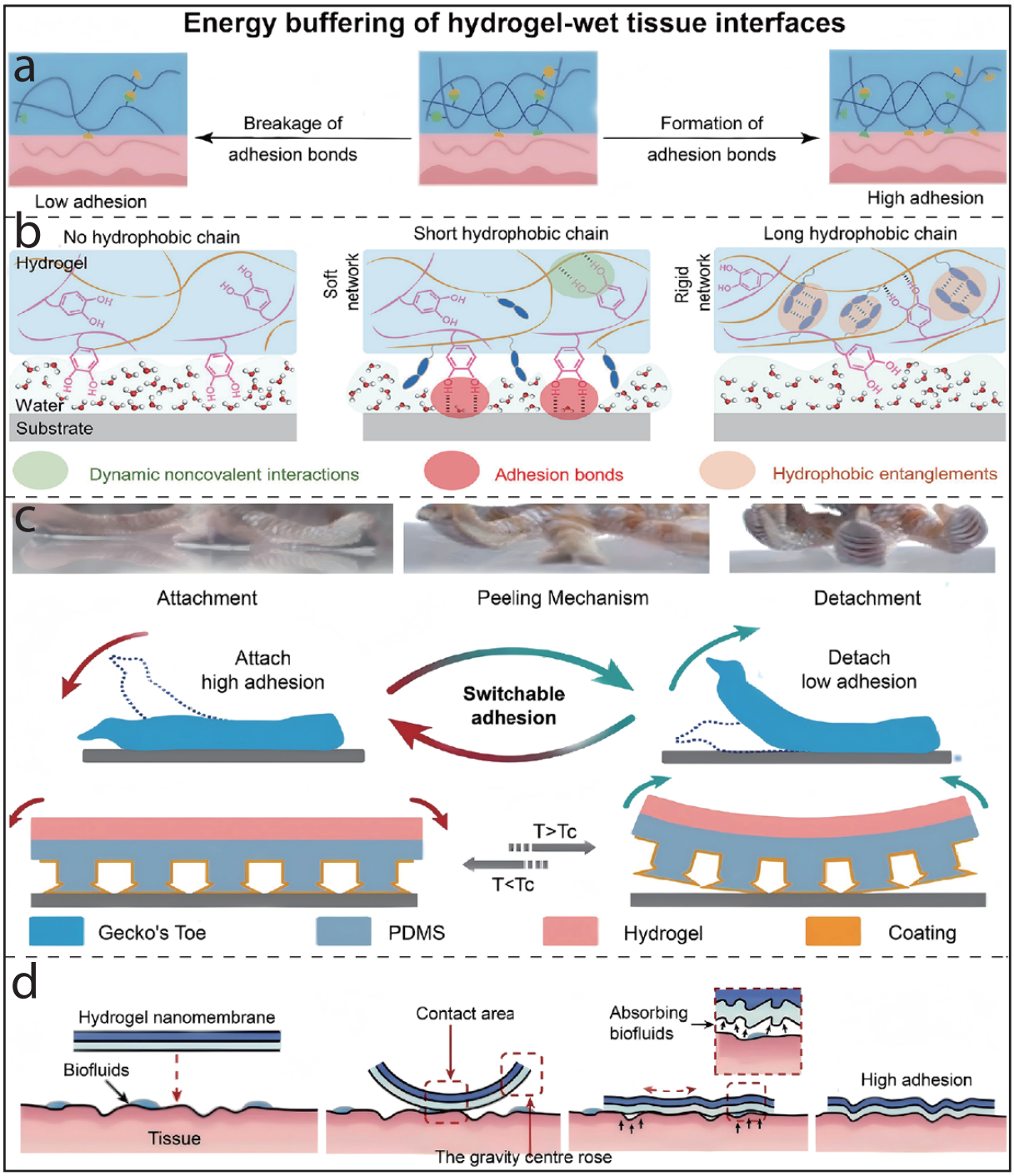


Fig. 4. (Color online) Energy buffering at the interface of adhesion. (a) Schematic illustration of dynamic adhesion bonds breaking and reformation at hydrogel-wet tissue interfaces. Reproduced with permission from Ref. [44] Copyright 2026, Royal Society of Chemistry. (b) The effect of hydrophobic chain length on interfacial interactions. Reproduced with permission from Ref. [46] Copyright 2023, American Chemical Society. (c) Repeated energy dissipation through interfacial structural rearrangements in biomimetic switchable adhesion. Reproduced with permission from Ref. [48] Copyright 2021, American Chemical Society. (d) The change of modulus delays interfacial energy release. Reproduced with permission from Ref. [50] Copyright 2025, Springer Nature.

ferred from the interfacial region to the hydrogel bulk, and finally dissipated through bulk network deformation, sacrificial bond rupture, and chain rearrangement. Therefore, this Section focuses on clarifying the energy dissipation pathways of hydrogel-wet tissue interfaces and how their coupling regulates the distribution, transfer, and dissipation of mechanical energy. From this perspective, different adhesion enhancement strategies can be systematically understood as ways to regulate interfacial buffering, interface–bulk energy transfer, and bulk dissipation, thereby determining the overall stability of the adhesive system.

3.1. Interfacial energy buffering

When adhering to wet tissue, the interface is the primary and most critical region for accommodating externally applied mechanical energy. Interfacial energy buffering refers to the reversible temporary storage and redistribution of externally applied mechanical energy within the interfacial region. Its primary function is to delay stress concentration and prevent premature interfacial failure before energy is trans-

ferred into the bulk. Unlike irreversible energy dissipation, buffering itself does not primarily consume mechanical energy, but temporarily accommodates it through elastic deformation and reversible molecular interactions, thereby providing sufficient time and spatial freedom for subsequent energy transfer and bulk dissipation. At this stage, energy is mainly confined to the interfacial region without significant diffusion into the bulk. The key to achieving interfacial buffering is to improve the dynamic response ability of the interface to external perturbations. It should be noted that different molecular interactions contribute to interfacial stability through distinct yet often overlapping mechanisms. Dynamic interactions such as hydrogen bonds, ionic interactions, and coordination bonds primarily function as reversible energy-dissipation units through repeated bond breaking and reformation, while simultaneously providing adhesive interactions. By contrast, hydrophobic interactions mainly enhance interfacial contact stability and resistance to interfacial water interference, although their reversible association and dissociation can also contribute to energy dissipation. Therefore, these interactions generally support both stable adhesion and energy dissipation, with their relative contributions depending on the interaction dynamics and interfacial structure[43].

At the molecular level, dynamic adhesive bonds (e.g., hydrogen bonds, ionic interactions, and coordination bonds) continuously break and reform under loading. The primary role of these reversible interactions during the buffering stage is to temporarily redistribute local stress and accommodate deformation rather than directly dissipate mechanical energy. Only when bond rupture is accompanied by viscoelastic relaxation, frictional sliding, or structural rearrangement does irreversible energy dissipation occur (Fig. 4(a))[44]. And this reversible bond exchange process enables the interface to dissipate mechanical energy in a time-dependent manner, converting local stress into distributed interfacial relaxation rather than sudden bond rupture[45]. On this basis, hydrophobic interactions provide additional buffering channels for the interface by maintaining interfacial contact stability and introducing molecular association and dissociation processes (Fig. 4(b)). Hydrophobic chains can penetrate or reorganize the interfacial hydrated layer, promoting more stable interfacial contact, while introducing physical entanglement and transient association. The length and density of hydrophobic chains determine the balance between interfacial anchoring strength and molecular mobility, thereby regulating the extent and rate of interfacial energy dissipation[46].

In addition, regulating the reconfigurability or local mechanical compliance of interfacial structures can further improve the adaptability of the interface to deformation, thereby delaying stress concentration to defect regions[47]. Zhang *et al.* obtained that bioinspired switchable adhesive systems can achieve repeated energy dissipation through reversible structural rearrangement during attachment-detachment cycles (Fig. 4(c))[48, 49]. In such systems, the interfacial contact configuration evolves dynamically under external stimuli, allowing energy to be dissipated not only through bond dynamics but also through mesoscopic structural transitions. Some hydrogel systems utilize interfacial self-modulus adaptation (Fig. 4(d)), which reduces the effective stiffness of the interfacial region under loading[50]. This modulus reduction can enhance interfacial compliance, thereby slowing down the energy release rate and alleviating stress concentration at critical sites[51]. The above strategies influence the initial distribution of mechanical energy by regulating the local response behavior of the interface, without involving its transfer or dissipation at larger scales. For design, a successful interfacial layer should lower K_σ and delay the point at which G reaches Γ_{eff} without sacrificing intrinsic wet adhesion or introducing excessive creep. Thus, interfacial buffering is best treated as a rate-dependent, reversible regulation stage, with irreversible losses reported separately. Therefore, interfacial buffering should be regarded as a reversible mechanical regulation process that delays stress concentration, whereas irreversible energy dissipation mainly occurs after energy is transferred into larger structural scales or coupled with viscoelastic deformation.

3.2. Interface–bulk energy transfer

Interface–bulk energy transfer refers to the process of redistributing externally input mechanical energy from the interfacial region to the hydrogel bulk, whose core function is to reduce the local energy density at the interface and guide energy into a larger volume of the material (Fig. 5(a)). In the global balance, transfer is not energy consumption; it is the internal flux $J_{i\to b}$ that connects interfacial storage to bulk storage and dissipation. This distinction separates the present intermediate tier from both reversible buffering and irreversible loss. Unlike interfacial energy buffering, this process is no longer limited to local interfacial responses, but to change the spatial distribution path of energy. Therefore, interface–bulk energy transfer constitutes a key intermediate link connecting interfacial responses and bulk dissipation.

In terms of occurrence conditions, energy transfer is usually activated when the interfacial buffering capacity becomes saturated. When the interface locally can no longer accommodate the input energy, mechanical energy will diffuse inward along the coupled structure between the interface and the bulk. This process requires continuous mechanical coupling between the interface and the bulk; otherwise, energy will be trapped in the interfacial region and induce local failure. For transfer paths, the diffusion of energy from the interface to the bulk relies on the synergistic effect of multiscale structures. First, the dynamic adjustment of interactions at the interface can trigger the deformation of adjacent polymer networks, transferring local stress from the interface to the interior[52]. Hui *et al.*[53] (Fig. 5(b)) demonstrated that a gradient foamed hydrogel containing hierarchical pore architectures can effectively enhance interface-to-bulk energy transfer. The continuous pore-size gradient creates a gradual transition in local stiffness, allowing externally applied stress to be redistributed progressively from the interfacial region into the hydrogel bulk instead of remaining localized at the adhesive interface. This continuous stress-transfer pathway reduces interfacial stress concentration while activating deformation over a larger material volume, thereby improving crack resistance and long-term adhesion stability. More importantly, this work illustrates that gradient architectures primarily function by enhancing the efficiency of mechanical energy transport, rather than directly dissipating energy, highlighting the distinct role of interface-to-bulk coupling within the overall energy-regulation framework. The gradient structure

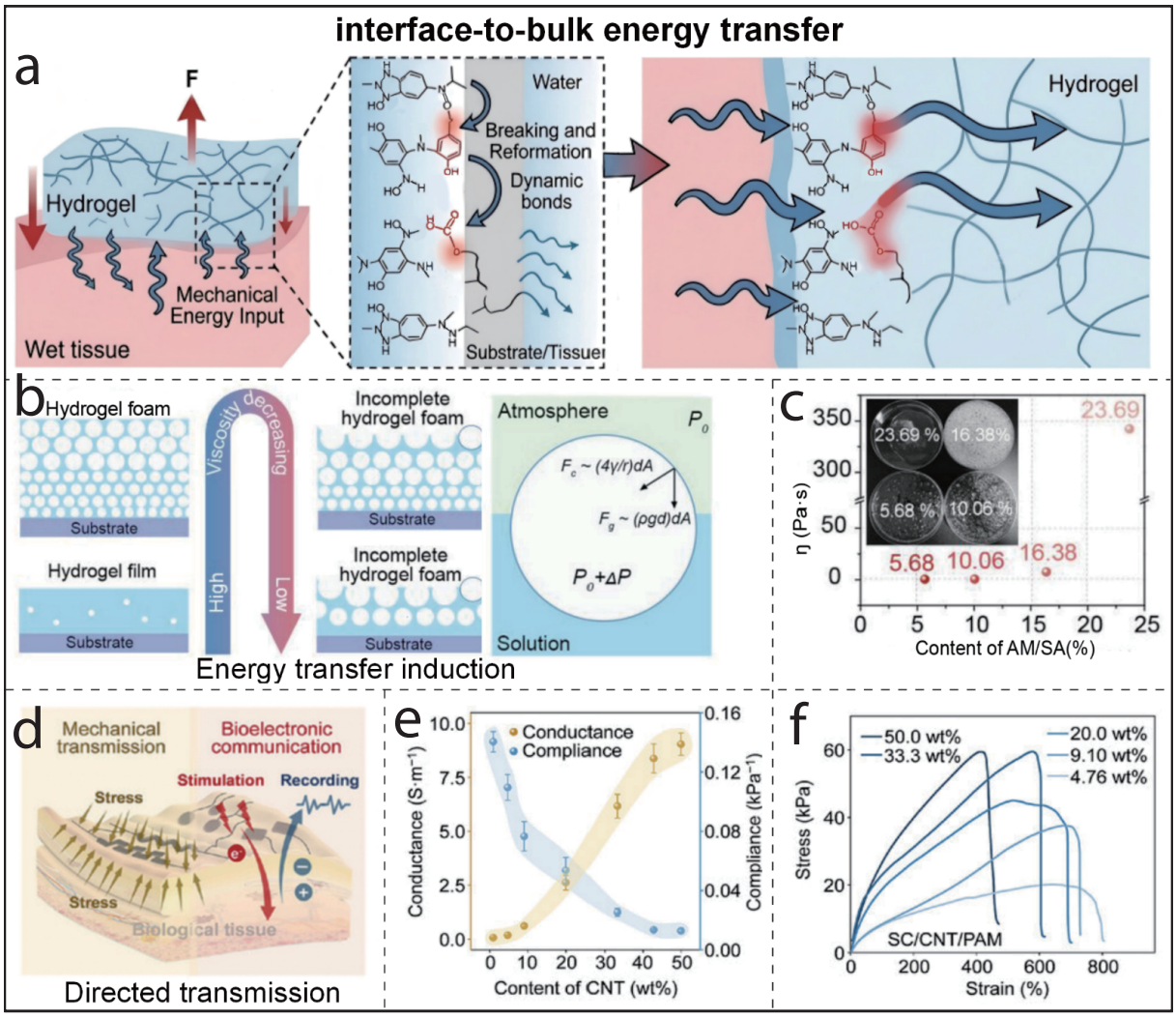


Fig. 5. (Color online) Energy transfer from interface to bulk. (a) Schematic illustration of energy transfer pathways induced by the breaking and reformation of dynamic interfacial bonds (e.g., H-bonds, coordination bonds) under mechanical energy input. (b) Infiltration and energy transfer induction process of hydrogel foam at the contact interface. Reproduced with permission from Ref. [53] Copyright 2024, John Wiley and Sons. (c) Regulation of system viscoelasticity by gel component (AM/SA) content to optimize energy transfer efficiency. Reproduced with permission from Ref. [53] Copyright 2024, John Wiley and Sons. (d)–(f) Directed transmission and mechanical adaptation: Including stress transmission in bioelectronic communication, regulation of compliance and conductance by CNT content, and stress-strain responses under various component ratios. Reproduced with permission from Ref. [54] Copyright 2026, John Wiley and Sons.

with progressively smaller pore sizes, combined with controlled hydrogel network configurations, facilitated energy transfer from the interface into the bulk phase (Fig. 5(c)).

Second, the viscoelastic response of the material determines the temporal matching ability of the bulk to the energy input from the interface, thereby affecting the efficiency of energy diffusion inward. In addition, when an interpenetrating structure or mechanical interlock forms between the hydrogel and the tissue, the stress transfer path at the interface is extended, allowing energy to cross the interface into a larger volume of the bulk region. In more complex systems, the introduction of anisotropic structures or functional components can also guide energy to propagate along specific directions, thereby improving the spatial efficiency of transfer. Tang *et al.*[54] (Fig. 5(d)) employed an *in-situ* generation strategy to construct a rapidly self-crosslinking, highly adhesive interface and a dynamic interpenetrating network phase, effectively integrating the dynamic bond interface with the interpenetrating network phase. This approach optimized both the mechanical adhesion energy and the stable transmission of bioelectrical signals from the interface to the bulk phase, while establishing an efficient dissipation pathway (Figs. 5(e) and 5(f)).

Although interface-to-bulk energy transfer cannot yet be directly quantified, its efficiency can be indirectly evaluated using several complementary mechanical descriptors. Digital image correlation (DIC) enables visualization of strain redistribution from the interface into the bulk, while fracture toughness and fatigue threshold reflect the effectiveness of stress delocalization during crack propagation[55]. In addition, hysteresis energy obtained from cyclic loading provides an indicator of the amount of mechanical energy transferred and subsequently dissipated within the bulk[56]. Combining these characterization methods offers a quantitative framework for evaluating interface-to-bulk energy transfer. Regarding functional outcomes, efficient energy transfer pathways can significantly reduce the degree of stress concentration in the interfacial region, redistributing the originally localized energy on a larger scale. This spatial redistribution process provides a prerequisite for subsequent internal energy responses of the

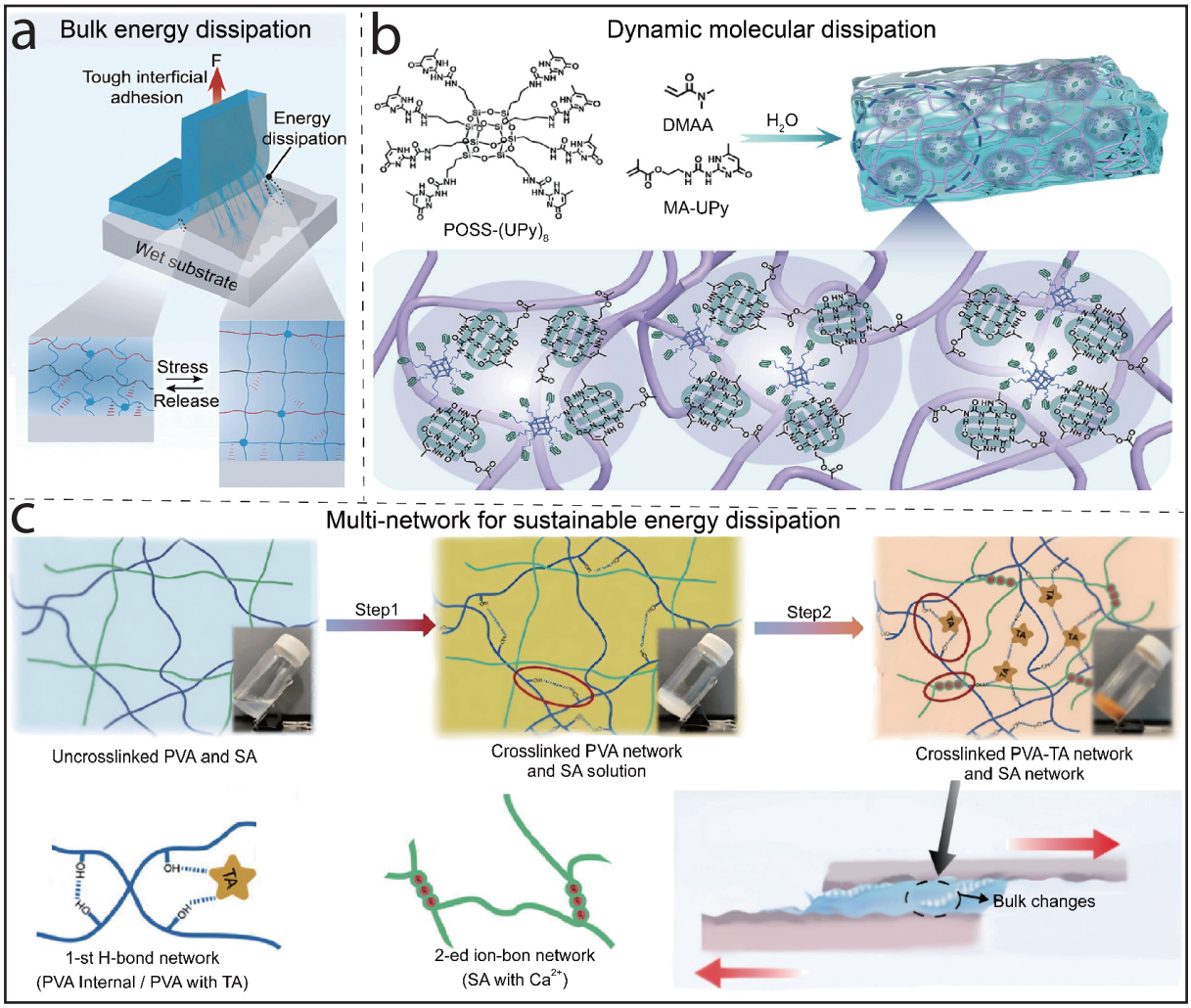


Fig. 6. (Color online) Dissipation of energy in the bulk. (a) Energy dissipation inspired by body network deformation and rearrangements. Reproduced with permission from Ref. [57] Copyright 2025, John Wiley and Sons. (b) Supramolecular network-enabled bulk energy dissipation via dynamic hydrogen-bonding clusters. Reproduced with permission from Ref. [58] Copyright 2025, John Wiley and Sons. (c) Hierarchical hydrogen- and ion-bond networks for sustainable bulk energy dissipation.

bulk and delays the propagation and evolution of interfacial defects. Conversely, when interface–bulk coupling is insufficient or transfer paths are restricted, mechanical energy will be trapped in the interfacial region for a long time, and premature failure may still occur even if the material bulk has good mechanical properties.

3.3. Bulk energy dissipation

When mechanical energy is transferred from the interface into the hydrogel's interior, it undergoes gradual conversion and attenuation within the material's bulk phase-a process termed bulk energy dissipation. Its fundamental role lies in providing sufficient volumetric and structural degrees of freedom, ensuring that the energy dissipated at the interface continues to dissipate rather than reaccumulating within the hierarchical framework of interface buffering-energy transfer-bulk dissipation. The bulk phase not only serves as the ultimate destination for energy but also determines whether the regulation achieved in the preceding two stages translates into a stable overall response. When the interface region can no longer accommodate incoming energy, mechanical energy inevitably diffuses into the bulk. Once energy enters the hydrogel bulk, viscoelastic deformation, sacrificial bond rupture, solvent-mediated relaxation, chain sliding, and network rearrangement can convert recoverable energy into heat or microstructural damage. Bulk dissipation is therefore represented by $W_{d,b}$ in Eq. (2), whereas the recoverable bulk energy ΔU_b may be returned during unloading or transferred back toward the interface. A stable design must dissipate sufficient energy to prevent re-localization while avoiding permanent deformation, excessive swelling, or damage accumulation. At this stage, the bulk's responsiveness dictates whether the energy is progressively attenuated or locally concentrated and fed back to the interface, ultimately triggering failure. Thus, bulk dissipation is not an isolated process but rather the final control mechanism within the entire energy regulation chain (Fig. 6).

The most basic dissipation capacity originates from the continuous deformation response of the network under external loads. The stretching, slippage, and network rearrangement of polymer chains gradually convert stored elastic energy into dissipated energy, achieving progressive stress relaxation rather than instantaneous release (Fig. 6(a))[57]. This process is characterized by typical viscoelastic behavior, enabling the material to continuously "absorb" input energy during sustained loading without sudden instability. At a

finer scale, the introduction of dynamic supramolecular interactions can significantly enhance the local energy dissipation capacity. Reversible hydrogen bond clusters and other noncovalent interactions break and reform repeatedly under stress, forming distributed "sacrificial units" (Fig. 6(b))[58]. These units can be repeatedly activated at different locations, allowing energy to be continuously consumed discretely inside the bulk, thereby avoiding structural damage caused by local energy concentration[59]. Multi-network or hierarchical structures further improve the dissipation efficiency and stability of the bulk[60, 61]. Networks with different stiffness and bonding characteristics respond sequentially during deformation: rigid or highly cross-linked networks mainly bear loads, while flexible or dynamic networks dissipate energy through chain migration, bond rupture, and structural rearrangement (Fig. 6(c))[62]. This hierarchical activation mechanism allows energy to be gradually absorbed at different scales and effectively avoids concentrated release. More importantly, the hierarchical structure not only enhances the dissipation capacity but also improves the energy "import efficiency" from the interface to the bulk by expanding the volume involved in deformation.

In summary, the bulk energy dissipation of hydrogels is determined by the synergy of continuous deformation, distributed sacrificial units, and hierarchical structures[63, 64]. These mechanisms work together to enable the mechanical energy transferred into the bulk to be continuously attenuated at a larger scale, thereby preventing energy from re-concentrating at the interface. In the hierarchical energy regulation framework, bulk dissipation is not only the final destination of energy but also determines the stability of the overall adhesive system under complex loads through coupling with interfacial buffering and energy transfer processes.

4. Conclusion and outlook

The long-term stability of hydrogel-wet tissue adhesive interfaces can be fundamentally understood as a multiscale mechanical problem governed by energy regulation. Rather than being dictated by a single parameter such as adhesive strength, interfacial stability emerges from the coordinated interplay of three tightly coupled processes: interfacial energy buffering, interface-to-bulk energy transfer, and bulk energy dissipation. Interfacial buffering mitigates local stress concentration and delays the onset of failure, energy transfer enables the redistribution of accumulated energy away from the interface, and bulk dissipation ensures that the transferred energy is continuously attenuated rather than re-localized. Stable adhesion is therefore achieved only when these three processes are properly matched across temporal and spatial scales, allowing the system to dynamically accommodate complex mechanical loading and wet physiological environments. The framework yields a practical quantitative workflow. Interfacial performance should be assessed by K_σ and rate-dependent storage and relaxation; transfer by η_{tr} from full-field energy maps; bulk response by η_d, recovery, and Γ_0. Stable adhesion requires these descriptors to be matched rather than maximized independently. Strong interfacial bonding with poor transfer can preserve a high stress concentration, whereas efficient transfer into a damage-prone bulk can accelerate fatigue. Importantly, these three processes should not be optimized independently, but balanced as an integrated energy-regulation chain. Interfacial buffering determines whether local energy accumulation can be delayed, interface-to-bulk energy transfer governs whether energy can effectively leave the interfacial region, and bulk dissipation determines whether the transferred energy can be continuously attenuated. Overemphasizing a single mechanism may not necessarily improve adhesion stability. For instance, strong interfacial buffering without efficient energy transfer may still result in local energy accumulation, whereas efficient transfer without sufficient bulk dissipation may cause energy reconcentration within the material. Therefore, the rational design of stable hydrogel tissue adhesives should focus on coordinating these three processes across multiple length and time scales, enabling continuous energy dissipation throughout the interface–bulk continuum. In this sense, hydrogel-tissue adhesive interfaces should be viewed not as an isolated interfacial property but as a continuous, coupled energy dissipation process spanning the interface and the bulk, providing a unified physical basis for integrating interfacial chemistry with bulk mechanics. Despite this conceptual clarity, several critical challenges remain. Quantitative methods capable of directly resolving energy distribution and transfer across the interface–bulk continuum are still lacking, limiting mechanistic understanding to indirect interpretations. Future studies should establish quantitative evaluation frameworks that combine energy-regulation analysis with representative performance metrics, such as interfacial toughness, fatigue threshold, and energy dissipation efficiency. These parameters can provide complementary information on crack resistance, long-term durability, and energy attenuation capability, thereby enabling a more rigorous assessment of adhesion stability. Moreover, a unified cross-scale framework linking molecular dynamics, network evolution, and macroscopic mechanical behavior has yet to be established. Future studies should further establish quantitative constitutive relationships linking molecular interactions, energy transfer efficiency, and macroscopic adhesion performance across multiple length scales. Correlating molecular interaction dynamics and structural evolution with measurable parameters such as interfacial toughness, fatigue resistance, and energy dissipation efficiency will be essential for developing predictive models that bridge material design and long-term adhesive performance. Current studies also largely rely on simplified loading scenarios, leaving the effects of multiaxial stresses, cyclic deformation, and dynamic tissue remodeling insufficiently understood under realistic physiological conditions. Addressing these gaps will require the convergence of advanced quantitative characterization, robust cross-scale modeling, and validation in complex in vivo environments, ultimately enabling the transition from empirical optimization to rational design of hydrogel-based adhesives with long-term reliability.

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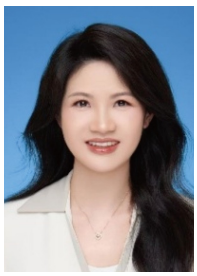
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Haitao Liu received his B.E. degree from Wuhan University of Technology in 2025. He is currently pursuing his M.S. degree at Beihang University. His research focuses on flexible sensors, flexible electronics, and intelligent sensing systems.



Mengxiao Chen is an associate professor at Beihang University. She received her B.S. degree in physics from Northeastern University 2012; and Ph.D. degree in physics from Beijing Institute of Nanoenergy and Nanosystems, CAS, in 2017. Then she joined Nanyang Technological University as a research fellow, and worked at the College of Biomedical Engineering & Instrument Science at Zhejiang University in Hangzhou as a Tenure-track Professor. Her main research interests include soft electronics, bioinspired electronics, and novel functional fiber devices.



Jing Rao is a professor at Beihang University. She received her Ph.D. from Nanyang Technological University and was a Humboldt Research Fellowship holder at the Technical University of Munich in Germany. She was also an assistant professor at the University of New South Wales in Australia. Her main research areas are non-destructive testing, flexible sensors, and structural health monitoring.



Caofeng Pan is a distinguished Professor at Beihang University, and awarded of the National Science Fund for Distinguished Young Scholars. Prof. Pan earned his bachelor's (2005) and doctoral (2010) degrees from the School of Materials Science and Engineering, Tsinghua University. He subsequently conducted postdoctoral research at the Georgia Institute of Technology, USA. From 2013 to 2023, he served as a professor and group leader at the University of Chinese Academy of Sciences and the Beijing Institute of Nanoenergy and Nanosystems, CAS. Since 2023, he has been serving as a distinguished professor and leads a research group at the Institute of Atomic Manufacturing, Beihang University. Prof. Pan's research focuses on atomic-level manufacturing and low-dimensional semiconductor materials/device for sensing applications.