Crack initiation in viscoelastic materials

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In viscoelastic materials, individually short-lived bonds collectively result in a mechanical resistance which is long-lived but finite, as ultimately cracks appear. Here we provide a microscopic mechanism by which cracks emerge from the nonlinear local bond dynamics. This mechanism is different from crack initiation in solids, which is governed by a competition between elastic and adhesion energy. We provide and numerically verify analytical equations for the dependence of the critical crack length on the bond kinetics and applied stress.

Liquids cannot fracture, but solids can. We consider the intermediate case: viscoelastic materials. These materials are made of filaments or particles interconnected by short-lived bonds. This design theme of transient networks is commonly used in both natural and man-made materials such as cytoskeletal polymer networks in cells \textsuperscript{[1]}, physical gels \textsuperscript{[2]}, associative and telechelic polymers \textsuperscript{[3]}, and colloidal gels \textsuperscript{[4]}. The molecular dynamics of transient networks lead to interesting macroscopic mechanics: at times shorter than the bond lifetime the material behaves like a solid \textsuperscript{[1]}, while on longer time scales the bonds reorganize and the material deforms viscoelastically \textsuperscript{[1] \textsuperscript{[5] \textsuperscript{[6]}}. As a result, transient networks are much more deformable than permanent networks \textsuperscript{[7]}. However, viscoelastic materials can resist mechanical stress only for a limited time, after which the system suddenly loses its mechanical percolation, a process which is known as fracturing \textsuperscript{[3] \textsuperscript{[8] \textsuperscript{[9] \textsuperscript{[10]}}. This raises the question how we can design transient networks such that the robustness against stress is optimized, which requires an understanding of the mechanism by which transient networks fracture.

Fracturing of viscoelastic materials is often explained by the Griffith theory of crack initiation in brittle solids \textsuperscript{[3] \textsuperscript{[8] \textsuperscript{[11] \textsuperscript{[12]}}. The Griffith theory predicts that beyond a critical stress, initial defects will grow into macroscopic cracks as the elastic energy released by the crack dominates the surface energy required for separation \textsuperscript{[15]}. However, this framework was originally developed for solids, and assumes defects are either static or growing. This assumption is clearly invalid for viscoelastic materials, as defects are not static entities but instead continuously appear and heal \textsuperscript{[16]}. Therefore, viscoelastic materials require a framework which takes into account the reversible bond dynamics.

The seminal work of Bell on cellular adhesion provides such a framework of reversible bond dynamics under force \textsuperscript{[17]}, and has received considerable attention in studying small-scale systems such as protein clusters which provide cellular adhesion \textsuperscript{[18] \textsuperscript{[19]}, fracturing of a single colloidal strand \textsuperscript{[9]} and protein unfolding \textsuperscript{[20]}. In all of these works, force is assumed to be homogeneously distributed across all bonds, which appears to be a realistic assumption for microscopically small systems. Contrarily, in the context of viscoelastic materials, theoretical \textsuperscript{[21] \textsuperscript{[22] and experimental work on both synthetic gels \textsuperscript{[23]} and biopolymer networks \textsuperscript{[24]} has revealed non-affine deformations upon application of global stress \textsuperscript{[25]}. Indeed, imaging of various networks under stress showed inhomogeneities of the local force which are strongly correlated in space \textsuperscript{[26] \textsuperscript{[27]}. These inhomogeneities might be negligible when considering bulk properties such as the average bond lifetime under stress \textsuperscript{[29] \textsuperscript{[30]}, but likely play a key role in crack initiation. In situ imaging of stressed viscoelastic materials revealed that fracturing occurs via well-defined cracks \textsuperscript{[3] \textsuperscript{[8] \textsuperscript{[9] \textsuperscript{[10]}} rather than via the homogeneous degradation expected from the Bell model \textsuperscript{[17] \textsuperscript{[20] \textsuperscript{[29] \textsuperscript{[30] \textsuperscript{[31] \textsuperscript{[32]}}}, suggesting local rather than global load sharing. At which length scale does the global load sharing assumption become inaccurate? And what determines the fracturing properties of a system of reversible bonds under load beyond this length scale?

To answer these questions we developed a minimal model that includes reversible bond dynamics (figure \textsuperscript{[1a]} in the simplest possible ‘material’ that is capable of exhibiting spatial inhomogeneity required for studying crack initiation: bonds distributed over a 1D-space, subject to mechanical stress. To account for inhomogeneous load sharing, we assume a force distribution that depends on the local bond spacing (figure \textsuperscript{[1b]}). We show that this minimal model system exhibits spontaneous crack initiation and subsequent fracture, in a manner that is consistent with experimental observations in wide range of viscoelastic materials \textsuperscript{[3] \textsuperscript{[11] \textsuperscript{[31] \textsuperscript{[32]}}. We verify our results by comparison with a mechanical model. We study the process of crack initiation in more detail by locally ‘ablating’ bonds (figure \textsuperscript{[1c]}), which reveals a critical crack length beyond which fracturing occurs. We provide analytical equations describing the process of crack initiation on basis of the nonlinear bond dynamics, and predict the dependence of the critical crack length on both bond properties and applied stress. Our work reveals that the process of crack initiation in viscoelastic materials is fundamentally different from that in traditional solids, as a consequence of the reversible bond dynamics.

We initialize a one-dimensional (1D) network with $N$
equally spaced binding sites using periodic boundary conditions, each bond having a probability $K$ to start in a closed state. Next we model the dynamics of the bonds with a kinetic Monte Carlo scheme using the following bond dynamics:

$$K = \frac{k_{on}}{k_{on} + k_{off,0}} \quad (1)$$

where $k_{on}$ is the rate of bond closing and $k_{off,0}$ the rate of bond opening in the absence of force (figure 1a). We normalize time by the on-rate, $k_{on}$. The off-rate increases exponentially with the applied force $f$ on the bond in keeping with the Bell model $[17]$

$$k_{off}(f_i) = k_{off,0} \cdot \exp\left(\frac{f_i}{f_{1/e}}\right) \quad (2)$$

where $f_{1/e}$ is the force where the off-rate has fallen to 1/e of $k_{off,0}$. We calculate the force per bond $f_i$ via

$$f_i = \alpha_i \cdot \sigma \quad (3)$$

where $\sigma$ is the stress on the system and $\alpha$ is a yet to be defined stress intensity factor per bond. In global load sharing, the applied stress is equally divided over all bonds. To investigate the effect of inhomogeneous force distribution as present in any network under stress $[26-28]$, we investigate a local load sharing model. In this model, we assume that the force distribution is dependent on the distance $l_i$ of a bond to its nearest neighbor on both sides (figure 1b). Explicitly, we define a stress intensity factor $\alpha$ on a closed bond at site $i$ by:

$$\alpha_i = \begin{cases} \frac{N \cdot \sum_{i} l_i}{\sum_{i} n_i} & \text{Local} \\ \frac{\sigma}{\sum_{i} n_i} & \text{Global} \end{cases} \quad (4)$$

where $n_i$ equals 1 when the bond is closed and 0 when the bond is open. Note that in both modes of load sharing the total amount of force is independent of the bond fraction and normalized by the system size, $\sum_i f_i = \sigma$. We normalize the applied stress by the bond force sensitivity $f_{1/e}$.

As shown in figure 2a, the fraction of closed bonds fluctuates over time, until it drops precipitously to zero at a certain moment that we denote as the rupture time. The rupture time is exponentially distributed - indicative of the stochastic nature of fracturing $[35]$. To test the sensitivity of the average rupture time to the applied stress, we perform simulations at a fixed network size and fixed bond affinity for different levels of stress ($N = 20$, $K = 0.9$, $\sigma = 0.5 \ldots 2$). For both local and global load sharing, we find that the average rupture time shows two distinct regimes with a transition around $<t_{rupt}> \approx 1$ (figure 2a). As we will explain later on, these regimes correspond to a metastable network at low stress and an unstable network at high stress. Importantly, networks with local load sharing are markedly less robust than globally load sharing networks, with smaller average rupture times at all stresses.

To test how the system size influences the average rupture time, we perform simulations for networks with $N$ varying between 5 and 100 (figure 2b). In case of global load sharing, we see that the average rupture time monotonically increases with system size, as the relative fluctuations of the fraction of closed bonds ($\sum_i n_i(t)$) decreases $[9, 17, 19]$. In case of local load sharing, we find similar rupture times as compared to globally load sharing networks for small $N$. But strikingly, beyond a critical length (around $N = 12$ for these conditions), we find that only in case of local load sharing the rupture time decreases with increasing system size, according to $<t_{rupt}> \sim N^{-1}$ (inset of figure 2b). This dependence suggests a constant crack initiation rate for every 12 bonds at this particular stress. Indeed, kymographs of simulations using local load sharing reveal that fracturing proceeds via cracks rather than homogeneous degradation (figure 2d).

To understand what sets this critical length for crack initiation, we performed ‘ablation experiments’ (figure 1c): first we equilibrate the network under stress, next we remove all bonds in $l_{ablate}$ adjacent positions, then we study whether bond ablation triggered network fracturing. We chose the system size $N=l_{ablate}$-10, such that
the system is large compared to the number of ablated bonds, yet small enough to allow for equilibration without spontaneous crack initiation. Figure 3a shows how fracturing becomes more likely upon increasing the ablation size \( l_{\text{ablate}} \), and that the required ablation size \( l_{\text{ablate}} \) to initiate fracturing decreases with the applied stress \( \sigma \). Figure 3b shows that an increase of bond affinity \( K \) increases the critical ablation size \( l_{\text{ablate}} \) required for triggering fracture.

To quantitatively understand the ablation data, we define crack length \( L \) as the largest bond distance \( l_i \) in the system. In case of global load sharing, the force on bonds at the edge of the crack stays independent of \( L \) as long as \( L \ll N \), so ablation does not induce fracture. By contrast, in case of local load sharing, the force on the bond at the edge of the crack is \( f = \sigma \cdot L \cdot \frac{1}{2} \) (the factor \( \frac{1}{2} \) is because the load on the hole is shared by the bonds at both ends). Thus, \( k_{\text{off}} \) exponentially grows with the crack size due to a linearly increasing force, whereas the chance of rebinding increases only linearly due to a larger area in which re-binding can occur. As a result, for large enough \( l_{\text{ablate}} \), bond unzipping will occur for any system under stress.

We are interested in the length \( L_{\text{unstable}} \) at which the crack becomes unstable. As a first order approximation, we can find the fixed points of crack length \( \*L \) by calculating the length at which the rates of bond opening and closing are equal (figure 4b):

\[
2 \cdot k_{\text{off}}(\frac{1}{2} \cdot \sigma \cdot \*L) \approx k_{\text{on}} \cdot \*L
\]

This condition is met at the average bond distance at equilibrium, \( L_{\text{stable}} \), and the bond distance at the unstable point, \( L_{\text{unstable}} \):

\[
L_{\text{stable}}(\sigma, K) \approx 2 \cdot \frac{W_0(-\sigma \cdot (\frac{1}{K} - 1))}{-\sigma}
\]

\[
L_{\text{unstable}}(\sigma, K) \approx 2 \cdot \frac{W_1(-\sigma \cdot (\frac{1}{K} - 1))}{-\sigma}
\]

where \( W \) is the Lambert W function with \( W_0 \) the main branch and \( W_1 \) the second branch \([30]\). Note that the network transitions from metastable to unstable at \( L_{\text{stable}} = L_{\text{unstable}} \) (seen as a change in slope in figure 2b at around \( t_{\text{rupt}} \approx 1 \)). For local load sharing, the transition from a metastable to an unstable network occurs beyond a critical bond-to-bond distance \( L_{\text{unstable}} \), whereas for global load sharing this transition occurs beyond a critical fraction of open bonds and therefore explains the continuous increase of rupture time as function of time \([9, 17–19]\).

To test equation 7, we show in figure 4b that all ablation data can be successfully collapsed onto a single master curve using a normalized ablation size \( \frac{l_{\text{ablate}}}{L_{\text{stable}}} \). To compare equation 7 with both the ablation data and the typical length scale observed in figure 2b, we first define a critical ablation length, \( l_{\text{crit}} \), which we obtain by fitting the size dependence of the rupture probability \( \phi_{\text{rupt}} \) to a sigmoidal function \( \phi_{\text{rupt}} = \frac{1}{1 + e^{-\frac{l_{\text{ablate}}}{l_{\text{crit}}}}} \) at each applied stress and at bond affinity \( K = 0.9 \). We can now combine the critical ablation length \( l_{\text{crit}} \) from figure 3a with the optimal system size \( N \) found in figure 2a and conclude that all these data are well-described by equation 7 (figure 4c).

Up to now, theoretical work on transient network fracturing has been limited to the assumption of global load sharing \([9, 17–19]\). We find that \( t_{\text{rupt}} \) is insensitive to this assumption for microscopic systems (up to approximately 10-100 bonds, see equation 7 and figure 2d). However, fracturing of larger system follows fundamentally different rules, in which the notion of local load sharing becomes important. Our study investigates the idealized limit of fully localized load sharing, but we observe similar behavior of a typical fracture length when the load distribution is simulated via a mechanical model \([35]\).

Our model predicts features which are different from global load sharing, but consistent with experimental observations on a wide range of viscoelastic materials. First,
As a function of crack size, the on-rate increases linearly, whereas the off-rate increase exponentially. As a result, the crack becomes unstable after \( \frac{L_{\text{ablate}}}{L_{\text{unstable}}} \) or bond affinity \( k_0 \). Control ablation experiments using the same parameter with global load sharing never showed fracturing.

rupting of macroscopic viscoelastic materials proceeds via spontaneous crack initiation, different from the homogeneous failure predicted by global load sharing models \([7, 18, 19]\). This prediction is borne out by experimental observations of a wide range of viscoelastic materials \([3, 11, 31, 32]\). Second, the model of global load sharing predicts that the rupture time strongly increases with the system size. As a result, delayed fracturing (\( \frac{t_{\text{rupt}}}{t_{\text{rupt}}} \gg k_{\text{on}} \)) would only be experimentally observable very close to the critical stress for any macroscopic system. Instead, delayed fracture is experimentally observed for many different viscoelastic materials over a wide range of stresses \([3, 10]\). We find that in case of local load sharing, the dependence of \( \frac{t_{\text{rupt}}}{t_{\text{rupt}}} \) on \( \sigma \) does not diverge upon increasing system size \( N \). Thus, our model for the first time explains why delayed fracturing is readily observable on laboratory timescales over a wide range of stresses in experiments.

The model makes several concrete predictions that can be tested experimentally by applying shear stress on viscoelastic materials. Firstly, we predict that the average rupture time measured at constant stress will be inversely proportional to the system size (figure 2c) as the crack initiation rate is constant per volume. Secondly, the presence of a critical crack length can be measured directly by performing laser ablation on viscoelastic materials under stress, a technique that is common in physical studies of cell and tissue tension \([37]\). Thirdly, the dependence of the critical crack length on the applied stress and bond kinetics (Equation 7) can be tested experimentally. The bond kinetics can for instance be experimentally controlled by changing the temperature in cross linked actin networks \([38]\) or salt conditions in polyelectrolyte gels \([39]\).

Our framework for understanding the crack initiation process in viscoelastic materials can be used to rationally design more robust materials. We have considered evenly distributed bonds. For future work, it would be interesting to investigate the effect of inhomogeneity under local load sharing. It is interesting to note that cellular adhesion proteins are not randomly distributed but clustered with a well-defined size \([40]\). Simulations have shown that an intermediate degree of clustering is optimal for preventing fracturing \([41, 42]\), although the nature of this optimum remained poorly understood. We speculate that this optimal clustering density is related to the critical length scale for crack initiation and that this strategy of clustering bonds is an interesting design principle for synthetic materials.

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Supplementary Information

To validate the behavior of cracks in viscoelastic materials obtained with the local load sharing assumption, we implemented a 2D mechanical model (figure S5) based on Finite Element Analysis (FEA) [43]. The transient bonds, modeled by a linear elastic material with a Young’s modulus $E$ and Poisson’s ratio $\nu$, are modeled as elastic bodies fixed at their bottom and attached to an upper body with the same material properties. Both the bonds and the elastic body are discretized using bilinear square elements consisting of four nodes. The elastic body consists of $h \cdot N$ elements, where $h$ is the height of the elastic body expressed in terms of the in number of elements and $N$ is the number of bonds. Periodic boundary conditions are applied to the left and right boundaries. To apply tension to the bonds, a vertical displacement is applied to the upper boundary of the solid part, until a force $\sigma_{\text{FEA}}$ is reached. A Monte Carlo scheme similar to the one used for the local load sharing model is applied to determine the transient behavior of the bonds, using equation 2 and 3 from the main text, and we define the stress intensity factor value $\alpha_i$ for FEA as:

$$\alpha_i = \frac{U_i}{\sum_i U_i} N$$

where $U_i$ is the bonds’ elastic strain energy density which can be found by integrating the stress vector on the bond $\{s\}_i$ for the strain vector $\{e\}_i$ according to:

$$U_i = \int \{s\}_i^T \{de\}_i = \frac{1}{2} \begin{bmatrix} s_{xx} & s_{yy} & s_{xy} \end{bmatrix}_i \begin{bmatrix} e_{xx} \\ e_{yy} \\ e_{xy} \end{bmatrix}_i$$

Figure S6 shows how the elastic body redistributes the applied force on the bonds. Importantly, we observe a stress intensity distribution for FEA that is comparable to that of the local load sharing assumption using the settings of $h = N$ and $E_{\text{body}} = E_{\text{bond}}$. For future work it will be interesting to vary $h$ and/or $E_{\text{bond}}/E_{\text{body}}$ and test the effect on the stress distribution and subsequent fracturing behavior.
FIG. 5. **Finite element model** Finite element model representation of an elastic body (blue) of total thickness $h$ with random distribution of bonds (green) at rest (left) and stretched (right). The deformation, due to the applied force $\sigma_{\text{FEA}}$ on the elastic body, is exaggerated for clarity.

FIG. 6. **Comparison of load distribution** Stress intensity factor comparison of different modes of load sharing (global, local, FEA) for the bond distribution shown in Figure S5.

FIG. 7. **FEA verifies main qualitative difference between local and global load sharing** Unlike global load sharing but similar to local load sharing (main text figure 2c), the average rupture time in FEA is peaked for intermediate system size $N$. 
FIG. 8. **Rupture times are exponentially distributed**

We performed fracturing simulations under local load sharing for 1D networks (see main text figure 1) using identical parameters ($\sigma = 0.7, K = 0.9, N = 20, 1000$ repeats) and recorded $\tau_{\text{rupt}}$. The distribution of rupture times is exponential, suggesting a stochastic process.