UNRAVELING THE INTERMITTENCY OF DAMAGE EVOLUTION FOR PREDICTING THE FAILURE OF QUASI-BRITTLE SOLIDS

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Abstract
We study the intermittent damage evolution preceding compressive failure using a non-local damage model accounting for material disorder and long-range elastic interactions. Our theoretical predictions are successfully compared with experiments carried on a model elasto-damageable 2D solid where damage evolution is tracked at both the global and local scale. Finally, we show how our understanding of these failure precursors can be harnessed for predicting the remaining lifetime of structures under compression.

1. Introduction
Compressive failure of quasi-brittle materials is preceded by a phase of damage accumulation characterized by bursts of activity. An intensification of the damage bursts is also observed as failure is approached. How this precursory activity impacts the emergence of the localization band at failure is still an open question [1]. A better understanding of this intermittency is therefore key to model how damage spreads and finally localizes during compressive failure and is of interest for applications of structural health monitoring.

Standard damage models of compressive failure consider damage growth in an effective homogeneous volume element, thereby missing the crucial role of material disorder. They also neglect the long-range interactions between material elements constituting the specimen, an effect that leads to cascades of damage events [2,3]. Here, we study damage accumulation in disordered solids using a micro-mechanical model [4-6] that encodes a feedback mechanism (shown in Fig. 1(a)) that captures damage cascades. Our theoretical approach incorporates a long-range interaction kernel derived from damage mechanics [6], see top inset of Fig. 1(b). We then compare our predictions with experiments performed on 2D cellular solids where damage localization is observed, see Fig. 1(c). We measure the energy dissipated by damage from both the global macroscopic response of the specimen and the local cell deformation using full field measurement of displacement and damage.

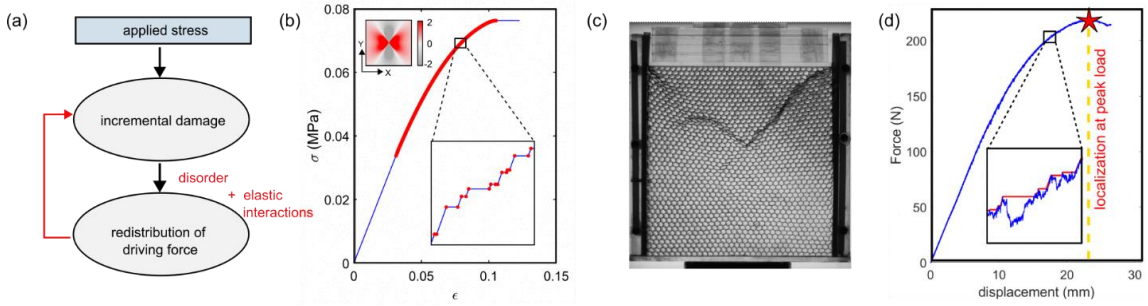

Fig.1: (a) Schematic of the feedback loop underlying damage evolution in heterogeneous solids. (b) The interaction kernel derived from damage mechanics [6] is shown in the top inset of (b). Mechanical response obtained from (b) theory and (d) experiments on 2D cellular solids, and their magnification in bottom insets. (c) Typical damage localization pattern recorded beyond peak load in our experiments.

2. Results
The cascades of damage events manifest as force plateaus in the force-displacement response as shown in insets of Fig. 1(b) and (d). They are described by remarkably robust scaling laws. For instance, the size of damage cascades S corresponding to force plateaus increases as damage grows. The approach to failure is described by a power-law, $S \sim \delta^{-\alpha}$ with $\alpha \approx 0.5$ as shown in Fig. 2(a) and 2(b) where δ is the normalized

distance to failure. At the individual cell level, damage cascades are shown to be composed of space and time coherent clusters, and their spatial extent ξ is observed to diverge as the specimen is driven towards failure in Fig.2(c). Interestingly, it reaches the specimen size at localization. The size and spatial extent are related to each other by the fractal dimension d_f as $S \sim \xi^{d_f}$, see insets of Fig. 2(a) and (b). Similarly, the duration T of the damage cascades is related to the spatial extent as $T \sim \xi^z$ where the dynamic exponent $z \approx 0.57$. In addition, we find the damage events within a single feedback loop as well as their activity rate can be described by power-laws. We also measure experimentally the long-range kernel describing the elastic interactions (see Fig. 2(d)) that is found to be in good agreement with the theoretical predictions shown in inset of Fig. 1(b).

Our findings thus reveal that intermittent damage evolution emerges from the complex interplay between long-range elastic interactions and material disorder providing a scaling description of various features. Their increase close to failure, however, can be attributed to the progressive loss of stability on approaching damage localization. As a result, we find that the distance to failure can be inferred from the statistics of precursors and therefore can be harnessed for predicting residual lifetime. As a proof of concept, we apply this new technique to our experimental data and show that it successfully predicts the time to failure, see Fig. 2(e). The application of this technique to the health monitoring of more complex structures will be discussed during the presentation.

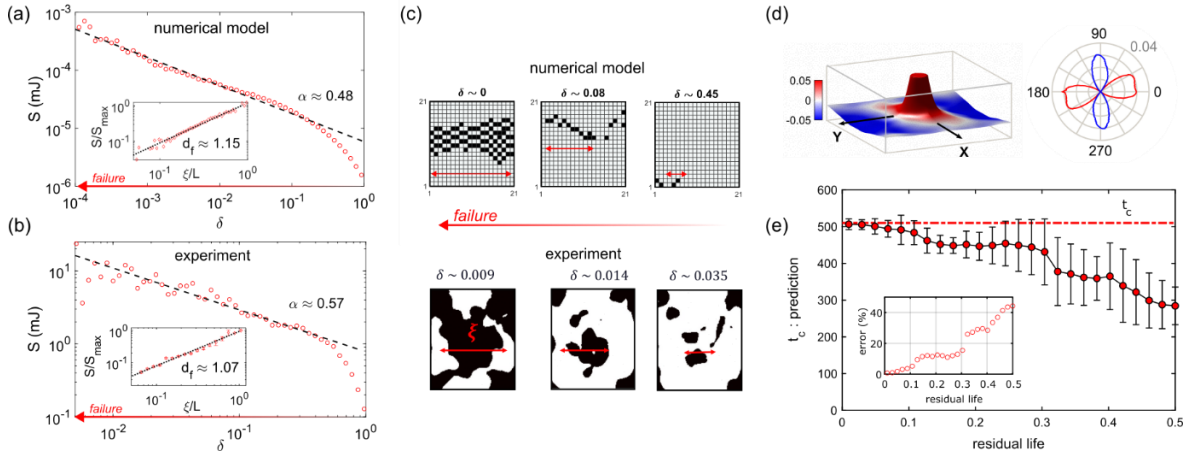

Fig.2: Variation of the average avalanche size with distance to failure in (a) theory and (b) experiments. The variation with the spatial extent providing the fractal dimension $d_f \approx 1.1$ is shown in the insets, respectively. (c) The maps of incremental damage growth at different distances to failure. (d) Map of the 2D auto-correlation of the incremental damage field and its angular variation at a fixed radius. (e) The predictions of time to failure and the error (inset) at different values of residual lifetime.

3. Conclusions

By accounting for the collective nature of damage evolution, we untangle the intermittent evolution of damage preceding compressive failure. We obtain scaling laws describing the non-stationary evolution of failure precursors as the specimen is driven towards failure and show that they can be harnessed for predicting the remaining lifetime.

References

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