NUCLEATION AND PROPAGATION OF FRACTURE IN ELASTOMERS DURING POKER-CHIP EXPERIMENTS

Aditya Kumar^{1*}, Oscar Lopez-Pamies²

¹Georgia Institute of Technology, Atlanta, GA, USA, ²University of Illinois, Urbana-Champaign, IL, USA * Presenting Author email: aditya.kumar@ce.gatech.edu

Abstract

The poker-chip experiments of Gent and Lindley [1] – in which they bonded thin disks of elastomers to metal plates at two ends and applied tension – jump-started investigations into the phenomenon of cavitation. Despite their importance, these experiments and other similar experiments have yet to be fully explained. One likely reason for their elusiveness is that it had long been mistakenly presumed that cavitation in elastomers could be explained on the basis of an elastic instability. Another reason is that a unified nucleation and propagation fracture theory in large deformations to explain cavitation as a fracture phenomenon had not existed. Recently, Kumar, Francfort, and Lopez-Pamies [4] have introduced a comprehensive macroscopic phase-field theory for the nucleation and propagation of fracture in elastomers undergoing arbitrarily large quasistatic deformations. In this work, we quantitatively analyze the pokerchip experiments using this theory and showcase the theory's ability to model nucleation and propagation in a unified manner.

1. Introduction

Internal fracture in elastomers — because of the nature of their underlying macromolecular chains — initiates through a sudden irreversible growth of incipient defects into large cavities/cracks in response to external stimuli. This phenomenon has become known as cavitation. We credit the poker-chip experiments of Gent and Lindley [1] as the work that started fundamental investigations into this phenomenon. In these experiments, they bonded disks with various aspect ratios made out of natural rubber with a range of stiffnesses to metal plates at both ends and subjected them to tension as shown in Fig. 1(a).

Two schools of thought emerged from these experiments. First – the elasticity view – considered this phenomenon to be predominantly elastic growth of inherent defects. Second – the fracture view – considered this phenomemon to be governed by creation of new surfaces. While the first view has been popular, Lefèvre et al. [2] and Poulain et al. [3] have recently shown that cavitation is primarily a fracture phenomenon. Then, to analyze these experiments as a fracture phenomenon, there is a need for a unified theory of nucleation and propagation under large deformations. Kumar et al. [4] have recently put forth such a comprehensive theory of fracture – regularized, of phase-field type generalizing the work of Bourdin et al. [5] – that models the nucleation and propagation of fracture in elastomers undergoing arbitrarily large quasistatic deformations on equal footing.

2. Theory

The theory of Kumar et al. [4] posits that nucleation of fracture: (i) in the bulk is governed by the strength surface of the elastomer (and not just uniaxial tensile strength), (ii) from large pre-existing cracks is governed by the Griffith competition between its bulk elastic energy and surface fracture energy and is same as propagation, (iii) from boundary points, be them smooth or sharp, and small pre-existing cracks is governed by the interaction among the strength surface, the bulk elastic energy, and the surface fracture energy of the elastomer. These ingredients are integrated in a phase-field framework which results in a set of two partial differential equations that completely define the fracture process.

3. Results

Fig. 1 shows a few of the quantitative comparisons of the theory with the experimental results of Gent and Lindley [1]. In particular, Fig. 1(b) shows the comparison of post-mortem images of midplane of specimens from experiments with the theory. The theory is able to describe the change in number and shape of cavities

as the thickness of the poker-chip is increased. The results indicate a competition between nucleation (strength) and propagation (toughness) that determine the number of cavities. Fig. 1(c) show a comparison between normalized reaction force and deformation between experiment and theory. The theory is able to explain the initial elastic response, a plateau in the force-deformation response upon initiation of cavitation, followed by stiffening due to strain crystallization.



Fig.1 – Comparisons of theory [4] with poker-chip experiments. (a) Schematic, (b) post-mortem images of midplane of poker-chips, (c) normalized force vs stretch for two thicknesses.

3. Conclusions

A unified approach of studying fracture nucleation and propagation which can model the competition between nucleation and propagation processes is able to describe and explain the famed poker-chip experiments in elastomers.

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