detailed mechanisms by which bonds break could be important for the formation and evolution of fracture lances.

The study of how materials fail is crucial to our ability to engineer better ones — for example, to create lighter, stronger and tougher materials. The emergence of instability fracture mechanisms such as those studied by Pons and Karma tend to make it more difficult for cracks to spread because they increase the energy required for a crack to move forwards, thereby enhancing a material's overall resistance to catastrophic failure. The design of novel materials by deliberately invoking instability fracture mechanisms — perhaps through the creation of material structures that induce local combined tension–tear loading — could provide a powerful strategy to increase a structure's resistance to failure without the need to introduce additional material components, instead relying solely on structural changes.

An intriguing direction in which Pons and Karma's idea could be taken is the study of failure in biological materials — for example, spider silk, nacre and bone. In these materials, better resistance to fracture is achieved not through the addition of stronger materials but rather by reliance on a hierarchy of structures, in which each hierarchical level has its own fracture mechanism. The synergistic effect of fracture mechanisms at all levels, attained through a seamless merger of structure and material, achieves an overall performance that vastly exceeds that of each individual level. Although we rely on strong element bonding in the design of most engineered materials — such as covalent bonds in glass or polymers, or metallic bonds in steel — bonding in biological materials is often weaker. For example, spider silk is one of the strongest materials known (stronger than steel), yet its strength lies in the cooperation of extremely weak hydrogen bonding between protein molecules[9]. The effective use of multiple structural levels enables biological materials such as silks to achieve high performance without relying on strong bonding.

From a slightly different viewpoint, the breakdown of biological materials' capacity to effectively withstand fracture can lead to injury and disease, as observed, for example, in osteogenesis imperfecta (brittle-bone disease). As such, the study of fracture could also help us to understand the mechanisms that underlie severe diseases and perhaps provide new pathways for treatment.

Pons and Karma's work[2] shows that a computational approach, validated by experiment, is a powerful tool in explaining fundamental issues in materials failure. The emergence of computational materials models that involve multiple scales[10], from the atomic to the macroscopic, holds great promise in elucidating the complex facets of how materials fail, and could lead to exciting breakthroughs in our understanding of material breakdown in engineering, geology and biology. ■

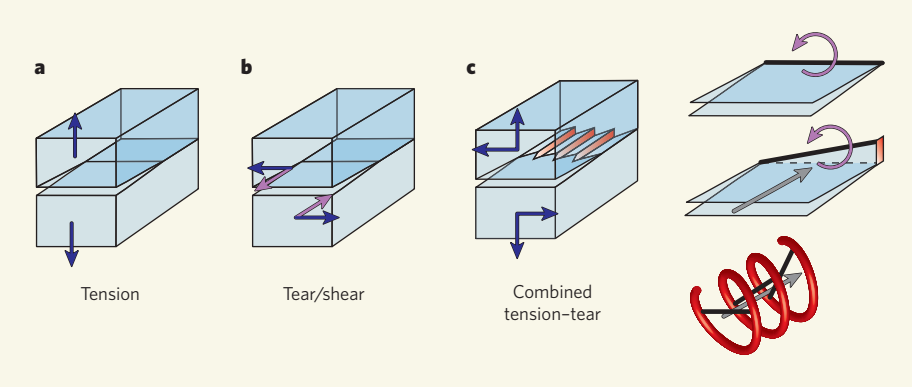

Figure 2 | Cracked specimen under different types of loading. **a**, Tension. **b**, Tear/shear (dark blue arrows for tear, pink arrows for shear). **c**, Combined tension–tear; under mixed tension–tear loading, an initially planar crack splits into several daughter cracks, or fracture 'lances' (red). This splitting is caused by a helical rotation of the crack front (thick black lines in right panel) around the principal direction of crack propagation (grey arrow). Pons and Karma's computational study[2] describes the three-dimensional details of crack-front evolution under such mixed tension–tear loading.

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DEVELOPMENTAL GENETICS

Time for teeth

The age at which babies cut their first tooth very much depends on the age at which their parents did. But what genes — or general genome-wide differences, for that matter — mediate variations in this trait? Demetris Pillas and his colleagues set out to address this question, and others, by searching for genetic markers that affect primary-tooth development during infancy (D. Pillas *et al. PLoS Genet.* **6,** e1000856; 2010).

The authors investigated records from two groups of subjects: one consisting of more than 4,500 individuals from northern Finland born in the mid-1960s, and the other made up of more than 1,500 individuals living in Britain and born in the early 1990s. They then performed genome-wide association studies on DNA extracted from blood samples taken from these individuals, looking for the genetic markers called single nucleotide polymorphisms (SNPs) that affect normal tooth development.

SNPs in five genomic regions (loci) stood out as showing a strong association not just with a delay in the time of first-tooth eruption but also with fewer teeth at the age of one year. SNPs within another five loci also seemed to associate with these traits, albeit to a lesser extent. The SNPs in the five top-ranked loci are within or near genes that have been implicated in growth and organ development, if not directly in tooth development, suggesting that tooth development is not an isolated event but is related to other developmental processes.

The extra benefit offered by Pillas and colleagues' subject groups was that, as well as providing records of infancy, the individuals were followed into adulthood. Taking advantage of this resource, the authors investigated how their top ten SNPs correlated with dental complications later in life. One SNP, rs6504340, which is located in the *HOXB* cluster of development-regulating genes,

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showed a strong association with a requirement for orthodontic treatment by the age of 30.

The authors point out the need for similar studies in other population groups to further establish the associations of these SNPs with tooth development. Given the link with other developmental processes, such studies might unveil connections with certain diseases in later life.

Sadaf Shadan