MATERIALS SCIENCE

# Bioinspired improvement of laminated glass

## Laminated glass with a microstructure inspired by nacre has a higher impact resistance

*By* **Kyriaki Corinna Datsiou**

Glass has a distinctive set of optical, thermal, mechanical, chemical, and electrical properties that are useful in many applications. Yet, the inherent brittleness of glass can limit its use in load-bearing applications for which structural failure has serious consequences (for example, large public structures). Preexisting surface flaws can propagate when combined mechanical and environmental loads put glass under tension and trigger sudden catastrophic failure. In structural applications, laminated glass—that is, two or more glass plates adhesively bonded with thin polymer interlayers—is used to retain fragments in the event of unexpected fracture. The fractured laminated glass not only maintains user safety but also partly retains the failed unit's stiffness and structural integrity because the fragments interlock. On page 1260 of this issue, Yin *et al.* (*1*) present a bio-inspired glass-polymer composite with superior damage tolerance to laminated glass, reconfirming that nature can be a source of inspiration for technological improvements of materials.

Nature has evolved many impact-tolerant materials (*2*), including nacre (mother-of-pearl) (*3*), which protects the soft bodies of mollusks from predators and environmental aggravation. Nacre has a complex hierarchical structure in which flat polygonal "bricks" (tablets) of calcium carbonate (aragonite) are embedded in an organic protein "mortar" and arranged in columns or sheets (see the figure). Early studies showed that nacre has improved fracture toughness and ductility compared with its constituent materials (*4*, *5*).

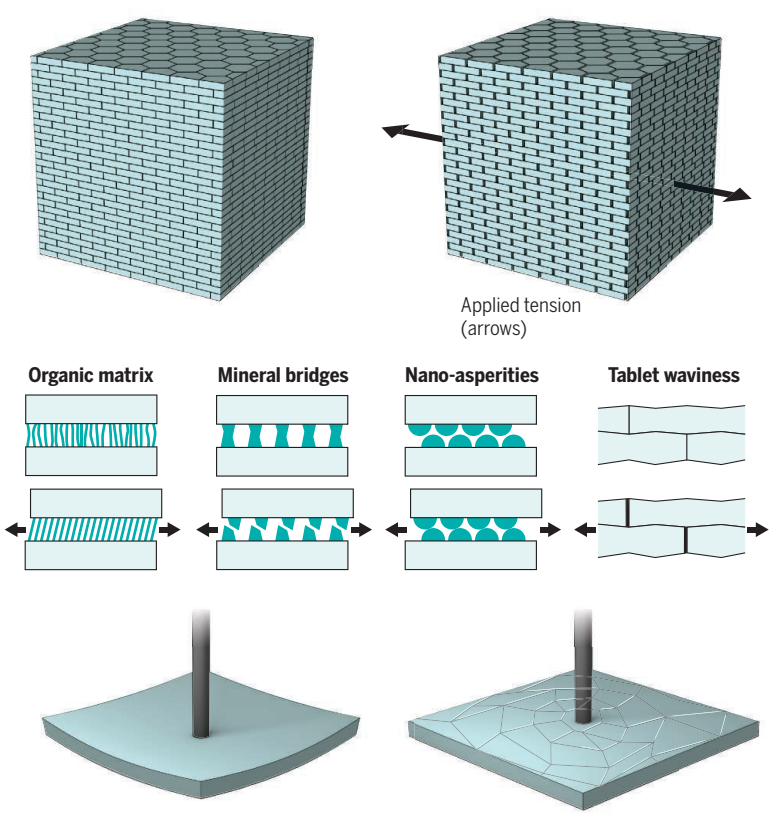
The main defense of nacre against impact is a collective tablet sliding mechanism (*2*, *6*). Under tensile loads, progressive shear deformation develops through large volumes of the material. Excessive sliding of individual tablets and consequent localized failure at small strains is prevented through strain-hardening mechanisms related to tablet waviness and nanoasperities that trigger interlocking, the presence of mineral bridges, and the viscoelastic response of the organic matrix (see the figure). These mechanisms gradually spread the deformation through the nacre architecture. The coupled action of the local extensions at all sliding sites generates large strains within the material and improves its ability to absorb energy during impact.

Yin *et al.* translated this effective yet complex architecture to the manufacture of impact-resistant laminated glass. They engraved the outlines of tablets with a pulsed ultraviolet laser beam in hexagonal or square patterns on borosilicate glass plates of 220-μm thickness. These engraved glass plates were then laminated with 125-μm-thick ethylene vinyl acetate interlayers, and during this process, the tablets separated. The resulting transparent, nacre-like glass sheet had only a marginally lower light transmission than that of conventional laminated glass.

Despite attempts to predict and improve the performance of conventional laminated glass (*7*–*11*), the glass elements in these units remain brittle, with limited deformation capability. Perhaps one of its most important limitations is that replacement, which is often costly, is the only solution in case of fracture. By contrast, Yin *et al.* present robust evidence that nacre-like glass has improved ductility and may reduce impact-related failures. Nacre-like glass can dissipate 2.5 to 4 times more energy than conventional laminated glass and 15 to 24 times more energy than plain borosilicate glass. The enhanced dissipation in nacre-like glass is associated

### Imparting impact resistance

Yin *et al*. improved the impact resistance of conventional laminated glass by patterning thin sheets into tablets like the aragonite tablets in nacre.

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with the uniaxial and biaxial tablet-sliding mechanism, as confirmed by the authors through micro-computed tomography, and is also related to the viscoelastic response of the polymer matrix.

Although the high ductility of nacre-like glass will be useful in many applications, it comes at the cost of reduced stiffness and strength. As a result, deformation limits in building design standards could be easily exceeded, which could compromise user comfort or trigger buckling phenomena that could lead to structural instability and collapse. To address these issues, Yin *et al.* propose the addition of a plain glass plate as a front layer to the nacre-like architecture. As an example, they demonstrate that this configuration can increase the unit's strength to 85 to 90% that of laminated glass. However, additional glass plates will be prone to fracture in a fashion similar to that of the glass elements in laminated units.

Barthelat and Zhu (*12*) showed that stiffness, strength, and toughness of natural nacre is a function of tablet geometry (aspect ratio, waviness, and nanoasperities), tablet overlap, and the presence of mineral bridges (*12*). Hence, methodological advances to more closely mimic natural nacre could be used to optimize the performance of synthetic nacre-like glass reported by Yin *et al.* However, common problems associated with conventional laminated glass could still hold, such as delamination, edge stability, and polymer degradation driven by environmental conditions during its service life. Deciphering these effects will improve the understanding on how impact-resistant, transparent, and durable bioinspired glass can be efficiently produced. Then, systematic design procedures can be developed to support the use of nacre-like glass in real-world structural applications. These insights will provide the basis for extending the use of glass to new and more challenging applications. ■

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NEUROSCIENCE

# Releasing the brake on eating

## Obesity alters neuronal gene expression and activity that may influence overeating

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Obesity is a global health problem that contributes to the increased incidence of other diseases, including type 2 diabetes, cardiovascular disease, autoimmune disorders, and cancer. Overeating is the largest determinant of obesity (*1*), yet we understand very little of the neural mechanisms underlying why individuals continue to consume food regardless of satiety. The lateral hypothalamic area (LHA) is a key region of the brain that coordinates diverse physiological functions related to survival—including responses to stress, drinking, and energy homeostasis, in order to maintain a physiological equilibrium in a changing environment. The LHA receives a variety of peripheral inputs about current energy needs and integrates these with centrally provided information to coordinate behavior. On page 1271 of this issue, Rossi *et al.* (*2*) demonstrate how glutamatergic neurons of the LHA respond to an obesogenic diet of high-fat chow, potentially explaining overeating.

***"Rossi et al. provide a promising hypothesis that diet-induced obesity impairs the...brake on food intake"***

Rossi *et al.* examined how obesity affects gene expression (transcriptome) of single LHA neurons in mice. Although they observed altered gene expression in many LHA cell types, the strongest obesity-induced transcriptomic changes occurred in vesicular glutamate transporter type-2 (VGLUT2)–expressing neurons; this broad neuronal population in the LHA uses glutamate as its fast transmitter. Consistently, gene expression changes in LHA$^{VGLUT2}$ neurons were strongly associated with body mass index in humans. When the authors profiled which genes were changed in LHA$^{VGLUT2}$ neurons, they found that obesity altered multiple genes involved in the regulation of neuronal activity. To follow up on the functional impact of these gene-level changes, Rossi *et al.* used two-photon imaging of calcium activity, a proxy of neuronal activity, in individual LHA$^{VGLUT2}$ neurons prior to obesity, early in exposure to an obesogenic diet (2 weeks), and after the development of obesity (12 weeks). They found decreased calcium activity in response to sucrose consumption early in the diet exposure. This suggests that these effects may reflect the increased caloric load rather than the development of obesity.

Reduced basal calcium activity was observed once obesity developed, which is consistent with reduced LHA$^{VGLUT2}$ neuronal activity observed in obese mice. These effects may be due to a change in intrinsic activity of LHA$^{VGLUT2}$ neurons rather than a change in synaptic input, because diet-induced obesity reduced the amplitude and duration, but not the frequency, of baseline calcium events; decreased neuronal excitability; and did not alter synaptic efficacy. Therefore, although decreased sucrose intake and LHA$^{VGLUT2}$ calcium responses to sucrose occurred early in the high-fat diet exposure, changes in intrinsic activity of LHA$^{VGLUT2}$ neurons only occurred after the development of obesity. Future work could elucidate the mechanism of obesity-induced suppression of LHA$^{VGLUT2}$ neuronal activity.

LHA$^{VGLUT2}$ neurons represent 15 distinct glutamatergic neurons defined by expression of neuropeptides, transcription factors, and other genes (*3*). For example, hypocretin, neurotensin, melanin-concentrating hormone, and somatostatin-expressing LHA neurons all express glutamate as their fast neurotransmitter (*3*) yet, when activated, may have functionally different sequelae. The putative function of LHA$^{VGLUT2}$ neurons has been explored previously. Activation of LHA$^{VGLUT2}$ neurons in mice produces avoidance or escape behaviors and suppresses feeding even when mice have been deprived of food (*4*–*6*). These different functions may result from the activation of either neurochemically distinct subpopulations or different projection targets of LHA$^{VGLUT2}$ neurons (*5*–*9*).

Rossi *et al.* show that calcium activity in LHA$^{VGLUT2}$ neurons of fasted mice is

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