News & views

Engineering

Strategic joints halt building collapse

Sarah L. Orton

A design principle for buildings incorporates components that can control the propagation of failure by isolating parts of the structure as they fail — offering a way to prevent a partial collapse snowballing into complete destruction. **See p.592**

In June 2021, the pool deck of Champlain Towers South, a residential building in Florida, suddenly gave way, triggering the progressive collapse of a substantial portion of the whole structure in a matter of seconds (see go.nature.com/3tux2ks). Most buildings aren't vulnerable to such extreme failure, but collapses still occur. Sometimes — albeit rarely — part of a building will be affected by severe weather, accidents, deterioration or even construction or design errors, and its failure instigates a domino effect that culminates in the collapse of the entire building, or a large section of it. But what if there were some way to prevent the dominoes from falling over? On page 592, Makoond *et al.*[1] report an addition to an engineer's armoury that can make buildings safer and more resilient by controlling the progression of collapse.

Structural collapse can be countered through two main approaches. First, the initial failure can be avoided, in the same way that road signs help to ensure that people do not crash their cars. Second, the propagation of that failure can be prevented, just as the safety systems in a car are designed to reduce the severity of a crash. Stopping the first domino, or the first failure, is the goal of most civil-engineering design. Building codes and standards are adhered to with the best knowledge and practices available to make buildings safe, with a target annual probability of failure of less than seven in ten million (ref. 2). But failures still happen. Makoond *et al.* therefore studied the second means of preventing collapse: limiting the extent of the damage.

The authors started by looking specifically at how to isolate collapse; that is, how to allow the damaged part of a building to fail without pulling down the rest of the structure. This is a new take on the issue of disproportionate collapse, in which a single small failure in a building's structure can lead to a much larger part of the building giving way. Current methods[3] for limiting such failures rely mainly on connecting parts of the structure so that missing components can be compensated for. This works in cases in which a single component (say, a column) is compromised, but what happens if more than one component fails? Connecting a building together might actually cause the collapsing part to bring down the rest of the building.

Makoond *et al.* therefore developed a kind of structural 'fuse' for buildings, which functions like a fuse in an electrical circuit, cutting off the collapsing region to save the rest of the structure (Fig. 1). They verified this design principle by constructing a two-storey concrete building consisting of precast columns with corbels (a type of bracket), and beams supporting concrete floors that were 'cast in place' (cast on site during construction). Each beam comprised a bottom layer that was precast, and a top layer that was cast in place. The 'fuse' consisted of partial-strength connections between the beams and the columns, and were made from steel dowel bars. The strength of the columns was also enhanced to ensure that the beams failed before the columns. This arrangement was designed to fail under a specific amount of load during a collapse,

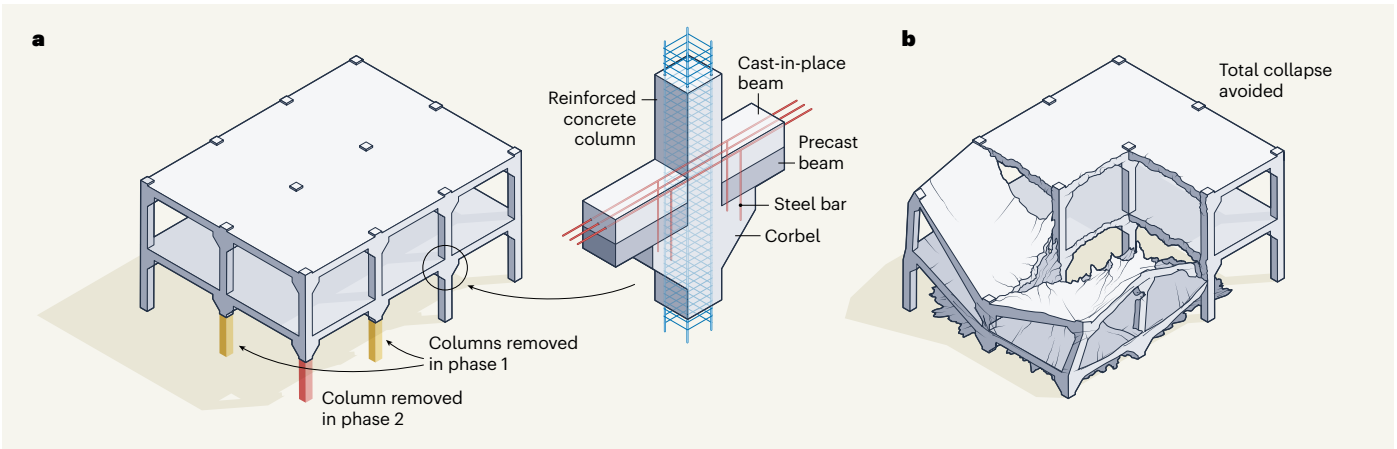

Figure 1 | Averting building collapse. Makoond *et al.*[1] developed a design principle that enables buildings to sustain partial damage without collapsing completely. They tested their approach by constructing a two-storey concrete building using precast columns with corbels (a type of bracket) and beams comprising a precast bottom layer and a top layer that was cast in place (on site during construction). A pair of steel bars connected the bottom of a beam to the corbel, and a second set of steel bars extended continuously along the top of the beam, passing through the column. The design was tested in two phases. **a**, When two non-adjacent columns were removed, the steel bars between the beams and the columns were strong enough to prevent collapse. **b**, When a corner column was also removed, the steel bars ruptured, but the design meant that the failure was isolated and prevented the whole building from collapsing.

allowing the collapsing part of the structure to break away so as to isolate the failure and prevent it from propagating.

This is a clever idea, but the design of the fuse is key. Too weak and the building could turn into a house of cards; too strong and the collapsing part of the structure could pull down the entire building. Makoond and colleagues therefore developed the concept of hierarchy-based-collapse isolation, which essentially limits the extent of the collapse. When the initial area of damage is small (for example, affecting a single column), the building should be able to redistribute the load, and the fuse is just strong enough to prevent further collapse. The idea is that, in practice, the single damaged column would be noticed and repaired before more damage could occur. However, if the original area of damage spans several columns, the authors' fuses are weak enough to break, thereby stopping the whole building from collapsing.

Makoond *et al.* subjected their precast concrete building to two phases of testing. In the first phase, they removed two columns that were not adjacent to each other, one at a time. The fuses were strong enough to compensate for the missing columns and prevent collapse. In the second phase, the authors took out a corner column that was positioned between those removed in the first phase. This initiated a collapse in all of the areas directly supported by the missing columns, but not – thanks to the authors' fuses – in the rest of the building. These experiments also provided valuable data for Makoond and colleagues' computational models of collapsing buildings, which engineers can use to better understand how buildings fail.

This study shows that the hierarchy-based-collapse approach can work well in precast buildings. However, fuses will need to be custom designed for other building types, such as those for which the building frame is cast during construction, and those that contain concrete floor slabs with no beams. If such a system had been in place in Champlain Towers South (an example of the latter type of structure), it is possible that the initial collapse would not have propagated across almost half of the building. Although the details are a long way from being easily implemented, Makoond and colleagues' approach will ultimately make buildings more resilient. It therefore fulfils the main objective of structural engineering, which is to protect the safety of the public[4].

Sarah L. Orton is in the Department of Civil and Environmental Engineering, University of Missouri, Columbia, Missouri 65211, USA.
e-mail: ortons@missouri.edu

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4. American Society of Civil Engineers. *Code of Ethics* (ASCE, 2020).

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Molecular biology

Tumours form without genetic mutations

Anne-Kathrin Classen

Researchers find that brief and reversible inhibition of a gene-silencing mechanism leads to irreversible tumour formation in fruit flies, challenging the idea that cancer is caused only by permanent changes to DNA. **See p.688**

The formation of tumours and progression to cancer are usually thought to be driven by the accumulation of permanent genetic mutations. Specific genetic mutations that have been linked to cancer often alter gene-expression programs, promoting changes in a host of cellular functions, including proliferation, differentiation, metabolism and survival[1,2]. On page 688, Parreno *et al.*[3] challenge the idea that tumours arise only from permanent genetic mutations. Using fruit flies (*Drosophila melanogaster*), they demonstrate that transiently disrupting mechanisms that regulate gene expression without making changes to the DNA sequence – a process known as epigenetic regulation – is sufficient to establish gene-expression programs that support tumour initiation and progression.

Epigenetic mechanisms maintain gene-expression patterns throughout a cell's divisions, even if the original environment in which these patterns were established changes. Biochemical modifications (such as the addition of a methyl group) to DNA or histone proteins (around which DNA is packaged as chromatin) allow genes to be activated or repressed in a heritable manner. Alterations to DNA-methylation and histone-modification patterns throughout the genome have been associated with various aspects of cancer, and so epigenetic modifications represent non-genetic but potentially heritable adaptations that promote tumour growth and progression. Beyond this, these modifications can be valuable biomarkers for the diagnosis of cancer, and potential therapeutic targets for its treatment[4,5].

However, epigenetic changes in tumours cannot always be attributed to permanent mutations in genes encoding proteins that carry out epigenetic modifications, such as histone modifiers, DNA-methylation enzymes and chromatin-remodelling proteins. In some cases, tumours can develop without any identifiable mutations being present[6]. These puzzling observations suggest that epigenetic alterations can function as crucial non-genetic drivers of disease, yet experimental evidence for this has been lacking.

Parreno and colleagues investigated whether tumours could arise from transient dysfunction of components of a family of gene-silencing proteins called the Polycomb group. Polycomb group proteins are essential for the determination of cell fate: they epigenetically repress genes that control differentiation by (among other things) methylating histones in patterns that are established during embryonic development. Mutations in Polycomb group proteins have been linked to various human cancers[7]. Because Polycomb group proteins are evolutionarily conserved from fruit flies to humans, it is not surprising that mutations in these proteins also promote tumour formation in fruit-fly tissues by deregulating genes that control cell fate and proliferation[8,9]. The simplicity of fruit-fly Polycomb group proteins and tumour-suppression mechanisms allowed Parreno *et al.* to test *in vivo* whether cellular reprogramming sufficient for tumour initiation can be driven by purely epigenetic mechanisms.

A gene-silencing technique called RNA interference allowed the authors to reversibly reduce the levels of two members of the Polycomb group, referred to as PH proteins, in a tissue of the developing fruit-fly larva called the imaginal disc. Strikingly, transient loss of PH proteins at an early stage of development induced the formation of tumours that were characterized by abnormal tissue architecture, excessive growth and loss of cell differentiation – but were not associated with any specific permanent mutations. Importantly, Parreno *et al.* saw that these tumours remained stable even though PH protein