

in a confined space known as an acoustic cavity – and then repeatedly drop small spheres of insulating oxides onto a plate made from the same material. Each time the sphere bounced off the plate, it was caught in the acoustic cavity, in which an oscillating electric field caused it to accelerate. The charge the sphere had acquired through its contact with the plate affected its acceleration. It could thus be estimated by tracking the sphere's trajectory using a high-speed camera. The charging of the objects could not be explained fully by adsorbed-water contamination, but the authors showed that their results were consistent with the asymmetric formation of an adventitious carbon layer on the two objects.

These were technically demanding experiments: the carbonaceous adsorbates that were found to be responsible for the asymmetric charge transfer are present in air at parts-per-trillion levels, and the charge exchanged per collision was on the order of 10^5 electrons. This corresponds to around 16 femtocoulombs (1 femtocoulomb is 10^{-15} coulombs), a charge level that lies at the practical detection limit of state-of-the-art charge-measuring electrometers. To put this into perspective, a femtocoulomb is so small that a smartphone in standby mode transfers this charge in about one-trillionth of a second. The authors' use of high-speed imaging to indirectly measure charge circumvented this limitation.

In the authors' experiments, the contact force between the ball and the plate could be controlled by fixing the height of the fall; the number of collisions was counted carefully; the time evolution of the adsorbed carbon layer was accounted for; and large data sets were collected without the test objects coming into contact with anything other than air. If both surfaces were covered by naturally occurring carbon, then the charging of the two objects was unpredictable, but if one surface was cleaned, then charges moved as predicted by the triboelectric series. If the sphere was cleaned, it acquired a more negative charge compared with experiments in which the plate was cleaned (Fig. 1).

This effect was observed for both spherical and flat objects and persisted even when the sample roughness was altered by a factor of more than 100. These observations indicate that although mechanical and deformation-related factors must be considered, naturally adsorbed carbon impurities are a source of symmetry-breaking that is responsible, more generally, for contact electrification.

The authors have unveiled this symmetry-breaking factor for insulating oxides, including several glasses, alumina (Al_2O_3) and zirconia (ZrO_2). Such materials are naturally occurring and widespread, but because adventitious carbon is ubiquitous, to the point that it is deposited on surfaces even under ultra-high-vacuum conditions, it is likely that this effect

can be observed in a broad range of materials, including plastics and semiconductors.

Fascination with triboelectricity dates back to antiquity⁹, and its systematic study was among the earliest activities of modern science. The authors' findings show that triboelectric charging is fundamentally an interfacial phenomenon, shaped by chemical as well as mechanical factors.

The work of Grosjean and colleagues presents several important multidisciplinary questions. For instance, it remains unclear how, at the molecular level, a film of adventitious carbon, varying widely in both composition and coverage, guides the direction of charge transfer. Further work is also needed to investigate whether different insulating materials accumulate different types of carbon contaminant and whether adsorption of atmospheric carbon alone can modify a material's contact electrification in a given context. Triboelectric charging has also been associated with heating¹⁰ and catalysis¹¹, and the potential role of adsorbed carbon in these processes is intriguing.

Contact electrification has broad practical significance. As well as widespread natural phenomena, it underpins technologies such

as laser printing, mineral processing and industrial exhaust treatment¹². Grosjean and colleagues' work is important because it identifies a key factor that has limited scientists' ability to control and model triboelectric charging in a predictable manner.

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Medical research

Gene editing for a developmental disorder

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An approach that specifically targets a single nucleotide corrected behavioural abnormalities in an animal model of Snijders Blok–Campeau syndrome. **See p.785**

Genetic variants in the *CHD3* gene underlie a neurodevelopmental disorder called Snijders Blok–Campeau syndrome. On page 785, Yang *et al.*¹ have developed a gene-editing approach – directly altering a mutated base in a DNA sequence – that restores *CHD3* function in mice harbouring a mutation that is commonly observed in children with the disorder. These promising results might pave the way for the development of an effective clinical treatment.

Neurodevelopment can be compared to a symphony, with each gene acting as an instrument. At the podium, functioning like a conductor, are proteins that ensure genes are expressed at precise times, with the right intensity. Some of these are called chromatin modifiers, which unpack chromatin – the complex of DNA and protein that stores genetic information – to enable gene expression. One such class of chromatin modifiers are encoded

by the *CHD* genes, which have essential roles in neurodevelopment. Each of the nine CHD proteins has complementary but distinct functions². It is therefore unsurprising that mutations in any of these genes can affect neurodevelopment, leading to disorders that affect roughly 17 in every 100,000 births worldwide³.

Snijders Blok–Campeau syndrome, which is associated with mutations in the *CHD3* gene⁴, was first described in 2018. Children with this condition have developmental delays, intellectual disability, poor motor coordination, difficulty with vision and speech, distinct facial features, and a subset have altered brain structure. Remarkably, similar clinical hallmarks arise regardless of whether the gene was duplicated, deleted or has a type of mutation called a missense mutation.

Yang and colleagues focused on the most

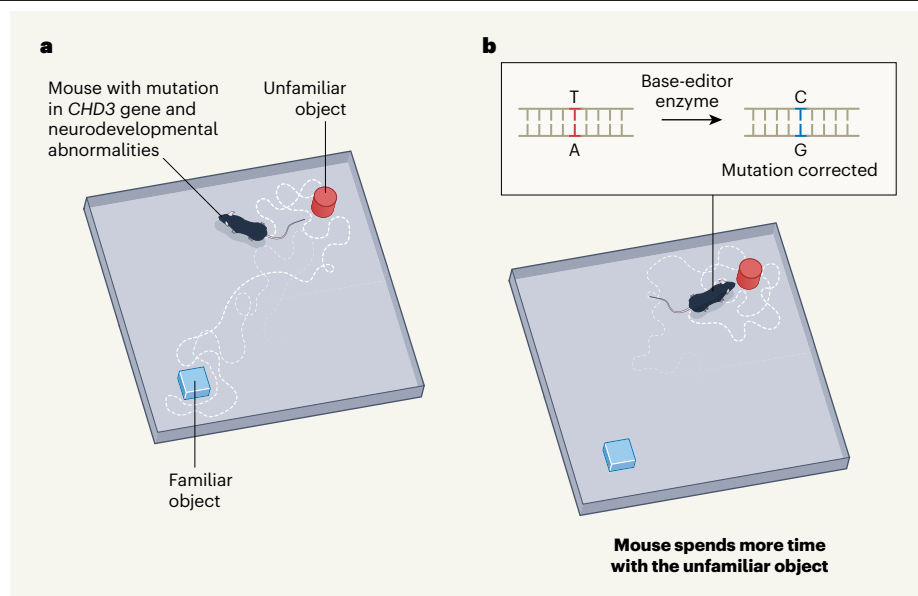


Figure 1 | A system to treat a neurodevelopmental disorder. Yang *et al.*¹ present a gene-editing approach tested in mice, which uses a technique called base editing to correct a mutation associated with Snijders Blok–Campeau syndrome. The syndrome can arise from a mutation of a single thymine (T) nucleotide, which pairs with an adenine (A) nucleotide, replacing a cytosine (C) nucleotide, which pairs with a guanine (G) nucleotide, in the *CHD3* gene. This can cause a variety of developmental and behavioural problems. The authors showed that the base-editor enzyme that they used could correct the mutation in adolescent mice. **a**, Animals with the mutation have abnormal behaviour when examined in tests, such as spending less time with a previously unknown object than they do with a familiar object. **b**, Mice in which the mutation had been corrected spent more time with an unfamiliar object than they did with a familiar object. (Adapted from Fig. 4 of ref. 1.)

commonly identified genetic change to *CHD3*. This mutation is also a dominant mutation, meaning that it only needs to arise in one copy of the *CHD3* gene to cause the disorder. The mutation changes one of the amino acids that is encoded by the DNA. The authors engineered a mouse model to have this mutation and found that the amino-acid substitution interferes with CHD3 protein stability, reducing the levels of this protein by 50%. Thus, this missense mutation functionally mimics other mutations that lead to a smaller-than-usual version of the protein being expressed (protein truncation), resulting in insufficient full-length functional CHD3.

The authors found that mice with the *CHD3* mutation showed some symptoms that are similar to those associated with the human condition. Newborn mice separated from their mothers vocalized less than their non-mutant littermates. Adults had alterations in their movement, in their spatial memory, in their ability to carry out motor tasks, difficulties recognizing new objects and a reduced preference for interacting with previously unencountered mice. However, their overall brain anatomy and the patterning of cell types in cortical regions of the brain was mostly unaffected by the mutation, suggesting that some features that are present in the human condition are not found in mice.

Dominant missense mutations are strongly linked to neurological disorders, from

neurodevelopmental to neurodegenerative conditions^{5,6}. These types of mutation lead to alterations of a single amino-acid residue of a protein. Although the proteins can still be made, their structure and function can, nevertheless, be affected by the alteration of this single amino acid. Methods to correct such mutations might be transformative for future clinical treatment options.

Yang and colleagues developed a method to edit this particular type of mutation using a base-editing technique. This approach uses what is called CRISPR technology to guide a DNA-modifying enzyme, called a deaminase, which performs a precise change at a specified base pair, without cutting the DNA backbone. Although base editing is limited to correcting single bases, this is typically all that is needed to tackle missense mutations. Moreover, problems that can arise from other types of gene editing, such as the risk of incorrect DNA repair, are eliminated with base editing, making it an attractive therapeutic approach.

To test whether their base-editing approach worked in mice, Yang and colleagues developed a pair of viral delivery vectors, each carrying part of the sequence that is needed for neurons to assemble the base editors. This was necessary because the base-editor sequence is too big to fit into a single viral vector. Therefore, a neuron must be infected with both viral vectors to assemble the base-editing machinery, which can reduce the

overall editing efficiency. Nevertheless, Yang and colleagues show clear evidence of editing, with CHD3 protein levels returning to near normal in mice with the mutated gene. Moreover, mouse behaviour mostly returned to normal across all of the behavioural functions that were tested (Fig. 1). This was particularly impressive because the base editors were administered to the mice at the beginning of their adolescent developmental period, rather than just after birth.

The results suggest that CHD3 has a role in chromatin regulation after birth, and that neuronal function is responsive to CHD3 protein levels being restored. This is promising for future studies because it suggests that it might be possible to improve symptoms even if treatment occurs at a relatively late stage of development. However, it is worth remembering that mice lack some of the anatomical changes that are often observed in children with the syndrome. Some of these changes stem from events that occur before birth, so early intervention will probably be crucial for the treatment of Snijders Blok–Campeau syndrome in people.

Although this base-editing approach shows promise, both for Snijders Blok–Campeau syndrome and for related dominant missense mutations, some challenges remain. For such gene-editing approaches to progress to clinical use, a suitable method for safely delivering the base editor to the brain must be developed. Base editors have already successfully treated a life-threatening genetic liver disorder in the first-known bespoke gene-editing therapy for a baby⁷. That approach used a lipid nanoparticle for delivery, which is similar to how some COVID-19 vaccines are delivered. Progress in generating similar nanoparticles that can cross the blood–brain barrier is under way⁸. Alternatively, viruses remain promising vehicles and these can be delivered either directly to the brain or can enter through receptors on the blood–brain barrier. Clearing these remaining hurdles will be transformative for rare-disease treatments, for which progress cannot come soon enough.

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