Condensed-matter physics

Electrons broken into pieces at crystal defects

Carmine Ortix

Fractional electric charges have been observed at crystal defects in artificial structures resembling materials called topological crystalline insulators. Such fractional charges could have various engineering applications. **See p.376 & p.381**

A fundamental physical constant known as the elementary charge, e, indicates the electric charge of a single electron. The constituents of matter have a charge that is an integer multiple of e. Nevertheless, solitary physical entities that carry a fractional charge can be formed in many-particle systems as end products of processes called emergent phenomena. Such entities typically arise as excitations (quasiparticles) in certain solid-state structures that have strong electron interactions[1]. In the past few years, researchers have predicted that the equilibrium state of peculiar materials known as topological crystalline insulators[2] could contain fractionally charged objects that stick to imperfections in the material structure[3,4]. On pages 376 and 381, respectively, Peterson *et al.*[5] and Liu *et al.*[6] bring this concept to reality by reporting experimental observations of fractional charges at structural defects called disclinations.

Crystalline solids consist of a spatially periodic array of atoms. Because the number of atoms in a solid is of the order of Avogadro's number (6×10^{23}), such an array can be considered infinite. Consequently, the structure looks the same from whichever position in the atomic lattice the solid is viewed – a property known as discrete translational symmetry. Crystals are further characterized by geometric transformations, such as rotations and reflections, that leave the structure unchanged. For example, a square lattice looks the same after consecutive rotations of 90°.

Crystal defects are regions in which this ideal symmetrical structure is distorted. And disclinations, in particular, are defects that disrupt the rotational symmetry in a certain area of the solid. A simple way to picture a disclination is to consider a process devised by the Italian mathematician Vito Volterra[7]: take, for instance, a square lattice, remove an entire quadrant from it, and then bend the structure to attach the lone edges (Fig. 1a).

Disclinations naturally occur in materials during their growth or as a response to mechanical deformation. These imperfections usually form in closely spaced pairs because it would require too much elastic energy (the energy stored when the structure is distorted) to form a single disclination. In each pair, one disclination has a negative value and the other a positive value for the Frank angle – a quantity that measures the wedge of material removed from or added to the ideal crystal to produce the defect. However, this feature represents a major roadblock to the direct observation of solitary trapped charges because the disclinations in each pair electrically neutralize each other.

The two research teams got around this problem by using synthetic structures called metamaterials. Peterson *et al.* constructed a metamaterial in which electric-circuit elements act as artificial atoms, and Liu *et al.* made one in which the artificial atoms are structures known as optical waveguides. Because metamaterials can be built one artificial atom at a time, producing a single disclination does not need elastic energy. Moreover, metamaterials can be engineered to mimic the properties of almost all materials, including the topological crystalline insulators in question.

But perhaps more central to the findings of both teams is that the distance between the artificial atoms can even be on the scale of a few centimetres – 100 million times larger than the distance between atoms in a material. Consequently, the teams could probe the local distribution of the synthetic electric charge with unprecedented precision, and show that a fractional charge accumulates at the disclination cores. This charge is precisely quantized (it has discrete values) and is a bulk quantity (it is independent of the specific details of how the disclination was engineered). Peterson *et al.* found that the charge is quantized in units of $e/4$ for a square lattice (Fig. 1b). By contrast, Liu *et al.* showed that it is quantized in units of $e/6$ for a hexagonal lattice (Fig. 1c).

The techniques developed by both teams to detect such a bulk quantity are not limited to metamaterials of topological crystalline insulators. For example, one might probe quantized charges in quantum materials called fragile topological insulators, which have been discovered in the past few years[8]. Unlike the topological insulators found in the mid-2000s[9,10], these materials lack any spectral signature, and their properties remain uncertain.

Fractional charges could also be explored at dislocations – defects in which it is the discrete translational symmetry of the crystalline structure that is locally broken. Unlike disclinations, these defects are (at least somewhat) mobile because they can move freely in certain directions. This property endows the fractional charges trapped by dislocations with a dynamic capability resembling those of fractionally charged quasiparticles.

Because the formation of dislocations requires less elastic energy than does that of disclinations, fractional charges trapped at these defects might even be observable in a conventional material, rather than in just metamaterials. But many challenges remain before this vision can become a reality. For instance, the number of materials hosting fractional charges is limited by an intrinsic property of electrons called spin that is absent

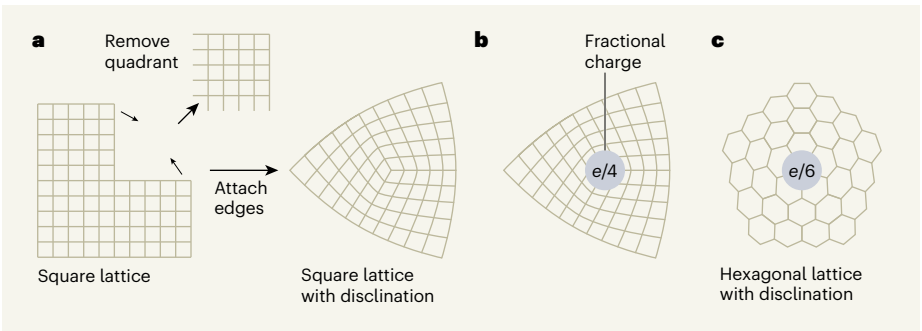

Figure 1 | Formation of fractional electric charges. a, A crystal defect called a disclination can be constructed using a mechanism known as the Volterra process[7]. In the example shown, a quadrant is removed from a square atomic lattice, and the structure is bent to attach the lone edges. **b**, Peterson *et al.*[5] show that such a disclination can trap a fractional electric charge that comes in units of $e/4$, where e is the charge of a single electron. **c**, Liu *et al.*[6] demonstrate that a disclination in a hexagonal lattice can instead host a fractional charge that comes in units of $e/6$.

in metamaterials. This property doubles the values of the trapped charges and can mask their quantized fractional nature, so material candidates should be carefully selected. Detecting fractional charges would also require local probe techniques, such as scanning tunnelling spectroscopy, to be pushed to their resolution limits. The efforts will be huge, but the successes of the current studies make the observation of fractional charges at material defects more likely in the foreseeable future.

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Ageing

An anti-ageing mechanism for protein restriction

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Two animal studies show that restricting the dietary intake of branched-chain amino acids can extend lifespan by modulating the mTOR signalling pathway. But more research is needed before this diet should be recommended in people.

The idea that dietary restriction can be used as a tool to increase lifespan has been a centre-piece of ageing research for decades. But the mechanisms by which dietary restriction might act, and the specific nutritional components involved, remain unclear. Writing in *Nature Aging*, two groups demonstrate the importance of one type of nutrient, branched-chain amino acids (BCAAs), in the ageing of fruit flies[1] and mice[2]. Their work adds to our understanding of the effects of specific dietary components on ageing and connects previous observations into a coherent model.

BCAAs are the three essential amino acids leucine, isoleucine and valine, which cannot be synthesized by humans and so must come solely from dietary protein. Leucine is a potent activator of the 'mechanistic target of rapamycin' (mTOR) protein, which serves as a key regulator of cell growth and differentiation. Both dietary protein restriction (which results in low levels of leucine and other BCAAs) and inhibition of mTOR can extend lifespan in animals[3,4].

In the first of the current papers, Lu and colleagues[1] set out to better understand the mechanism by which dietary protein restriction extends lifespan in fruit flies. They focused on sestrin, an evolutionarily conserved stress-inducible protein that links amino-acid abundance to mTOR signalling[5]. Sestrin acts as part of a complex that inhibits mTOR — a brake that is released when sestrin senses and binds to leucine[5]. The authors found that flies deficient in sestrin fail to respond normally to dietary protein restriction. They identified a specific amino-acid residue in sestrin

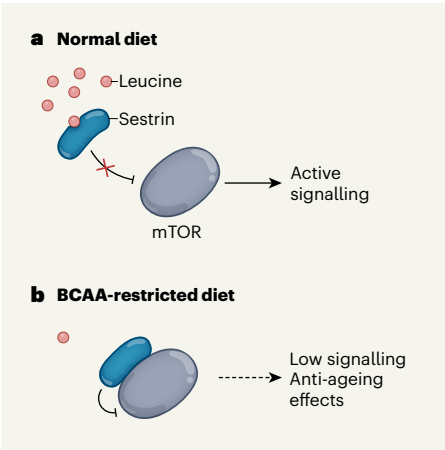

Figure 1 | Branched-chain amino acids (BCAAs) in ageing. a, The protein sestrin is thought to bind to the BCAA leucine. When sequestered by leucine, sestrin cannot exert its effects as part of a complex (not shown) that inhibits the protein mechanistic target of rapamycin (mTOR). **b**, Lu *et al.*[1] report that restricting dietary BCAAs in flies increases the inhibition of mTOR by sestrin. Reduced mTOR signalling produces anti-ageing effects. Richardson *et al.*[2] find that BCAA restriction also has anti-ageing effects in mice, supporting the idea that this pathway is common to multiple species.

(arginine 407) that senses amino acids. Flies that carry a mutation in this residue have lower mTOR activity than do controls. They are also longer-lived, and are protected against the negative lifespan-shortening effects of a high-protein diet.

In the second study, Richardson and colleagues[2] provide complementary data on the effects of a BCAA-restricted diet in mice. In contrast to a previous study[6], they observed a robust lifespan extension in male mice fed a BCAA-restricted diet throughout life, equal to the benefits of dietary protein restriction. Interestingly, female mice showed no lifespan extension from BCAA or dietary protein restriction, and if BCAA restriction was started during middle age, the benefits on males were greatly reduced. Thus, both studies collectively point to mTOR as a primary mediator of the benefits associated with BCAA restriction (Fig. 1).

Lu *et al.* go on to provide evidence that — at least in fruit flies — sestrin-mediated inhibition of mTOR improves gut function by activating an intracellular recycling process called autophagy in intestinal stem cells. These findings dovetail nicely with a large body of literature indicating that direct pharmacological inhibition of mTOR with the drug rapamycin can enhance autophagy and increase lifespan and the period of life spent in relatively good health (healthspan) in model organisms[3]. In mice, however, the effects of rapamycin seem to be more robust and less sex-dependent than those of BCAA restriction[7], suggesting that there are key differences between these interventions. There are several possible explanations for these differences. For instance, mTOR-independent effects of BCAA restriction might counteract some of the benefits of this dietary intervention, or the more-potent mTOR inhibition caused by rapamycin could affect downstream pathways differently[3].

Low-carbohydrate ketogenic diets, which are often lower in protein than control diets, can also increase lifespan and healthspan in mice[8,9]. The hormone fibroblast growth factor 21 (FGF21) is upregulated in animals subjected to either ketogenic diets or dietary protein restriction, and it has been implicated in the effects of dietary protein restriction on longevity[10]. Interestingly, Richardson *et al.* found preliminary evidence that lifelong BCAA restriction leads to FGF21 upregulation in the longer-lived male mice, but not in the shorter-lived females. The impact of FGF21 on mTOR signalling is complex, however, with evidence for both activating and inhibitory effects in different tissues. Future studies should address the overlapping and distinct mechanisms of action for these two dietary interventions, as well as for direct inhibition of mTOR by rapamycin.

Together, the current studies provide key insights into the mechanistic basis by