Cheating is a potential problem in any social system that depends on cooperation and in which actions that benefit a group are costly to individuals that perform them. Genetic mutants that fail to perform a group-beneficial function but that reap the benefits of belonging to the group should have a within-group selective advantage, provided that the mutants are not too common. Here we show that social cheating exists even among prokaryotes. The bacterium *Myxococcus xanthus* exhibits several social behaviours, including aggregation of cells into spore-producing fruiting bodies during starvation. We examined a number of *M. xanthus* genotypes that were defective for fruiting-body development, including several lines that evolved for 1,000 generations under asocial conditions and others carrying defined mutations in developmental pathways, to determine whether they behaved as cheaters when mixed with their developmentally proficient progenitor. Clones from several evolved lines and two defined mutants exhibited cheating during development, being over-represented among resulting spores relative to their initial frequency in the mixture. The ease of finding anti-social behaviours suggests that cheaters may be common in natural populations of *M. xanthus*.

The myxobacteria are soil-dwelling prokaryotes that exhibit a social life cycle similar to that of the eukaryotic slime mould *Dictyostelium discoideum*. Like the slime moulds, myxobacteria undergo multicellular development in response to starvation that yields fruiting bodies of remarkable morphological variety. This process involves multiple intercellular signals specific to distinct stages of development, and it results in a minority of the original population becoming stress-resistant spores that germinate under favourable conditions. Myxobacteria also exhibit social motility and social predation, swarming as a ‘wolf pack’ toward prey, which they kill and degrade by the secretion of extracellular compounds. A developmentally defective mutant of *M. xanthus*, when mixed with a developmentally proficient wild type, has five possible fates during development. First, the defective strain may sporulate with the same efficiency as it does in pure culture. This outcome corresponds to null hypothesis H1 here. Second, a partially defective genotype’s sporulation in the presence of wild type may be inhibited even below its efficiency in pure culture. The third and fourth potential fates are partial and complete rescue (relative to wild type), respectively, of the defective genotype by extracellular complementation in the presence of wild type. Complete rescue to the wild-type sporulation efficiency corresponds to null hypothesis H2 here. Fifth, a developmentally defective genotype may produce more spores in the presence of wild type than would a neutrally marked wild type introduced at the same initial frequency. This last outcome constitutes evolutionary cheating, because the defective mutant obtains disproportional reproductive success.

We first compared the developmental performance, in pure culture, of six experimentally evolved clones with that of their wild-type ancestor DK1622 (ref. 16). In pure culture, all six clones, to varying degrees, showed defects in spore production relative to DK1622 (Fig. 1). Thus, these evolved clones were all defective for this group function. We then measured spore production of these evolved defective clones when they were each mixed with their ancestor at an initial frequency of 0.01. The performance of each minority genotype was then contrasted with the two distinct hypothetical outcomes, H1 and H2 (Table 1). For five out of the six
defective clones, spore production was higher than predicted under H2, thus demonstrating at least partial complementation of their developmental defects. All five of these clones were rescued more than 10-fold above the expectation under H1, and in three cases the effect was greater than 1,000-fold. Sporulation of the sixth clone (GVB216.3) was hindered by the wild-type majority.

Out of the five clones exceeding the sporulation expected under H1, two showed partial complementation, because they performed worse than expected under H2; however, three clones exhibited cheating, as they performed significantly better than expected even under H1 (Table 1). Two of these cheaters (GVB208.3 and GVB214.3) showed moderate sporulation efficiencies as pure genotypes, but they had an advantage over wild type when they were rare. Clone GVB206.3 showed the most marked cheating: it was almost completely defective at sporulation in pure culture, but as a 1% minority it produced ~50-fold more spores than would a neutrally marked wild type.

To investigate more closely the frequency-dependent behaviour of these interactions, we measured the sporulation efficiencies of two evolved cheater clones (GVB206.3 and GVB208.3) in mixes with their wild-type progenitor at nine different initial frequencies (Fig. 2). Both clones cheated by performing better than expected for a neutrally marked wild type (H2) over a wide range of initial frequencies (Fig. 2a, b). Notably, as the initial frequency of each cheater genotype increased, the total spore production of the mixed culture fell below that of the pure wild type; this result indicates that cheaters indeed harm their group’s performance. Both cheaters sporulated much more efficiently than wild type at low initial frequencies, but their efficiencies dropped below the wild-type efficiency at high initial ratios (Fig. 2c, d). The relative sporulation efficiency dropped below 1 at a lower initial frequency for GVB206.3 than for GVB208.3.

The evolved defective clones may have several mutations that distinguish them from their wild-type ancestor. It is therefore unclear whether the cheating phenotype can arise by a single mutation or whether it requires multiple mutations. To test whether a single mutation can produce cheating behaviour, we examined three genotypes that differ from their wild-type progenitor by a defined mutation. The production of M. xanthus fruiting bodies involves several extracellular signals that are expressed at specific stages of development, and the defined mutants that we examined are defective in production of signal molecules. Sporulation of these mutants is defective (at least partially) in pure culture, but can be rescued (at least partially) by extracellular complementation when mutants are mixed at a 1:1 ratio with wild type. Our re-analysis of published data from such mixes suggested that some mutants may even exhibit cheating behaviour. Therefore, we mixed a defined mutant that is defective in the production of C-signal with its wild-type progenitor at 1:1 and 1:99 ratios, and we carried out corresponding experiments with two different mutants defective in A-signal production.

One of the A-signal mutants (MS2021), which has a mutation in the asgE gene, showed partial complementation but no cheating (data not shown); that is, it fell between the expectations under H1 and H2. The C-signal mutant (LS523) and the other A-signal mutant (DK4312), which has a mutation in the asgB gene, both showed strong cheating behaviour when mixed with their respective wild-type progenitors. DK4312 was severely defective for sporulation in pure culture, but performed much better than expected for a neutrally marked wild type (H2) at an initial frequency of 0.01 (Fig. 3a). LS523, which also sporulated poorly in pure culture, showed a similar degree of cheating at an initial frequency of 0.01 (Fig. 3b). In 1:1 mixes, both mutants dominated the wild type and the total spore counts fell below wild-type levels, with LS523 having a greater negative effect on total spore yield than DK4312 (data not shown). These defined mutants failed to contribute normal amounts of a particular developmental signal to the group, yet they performed better than wild type when rare. Therefore, even single mutations can produce cheating behaviour in M. xanthus.

Our experiments with both the evolved clones and defined mutants indicate that developmental cheaters are readily obtained in M. xanthus. Cheater genotypes presumably also appear in nature. (Although cheaters have not been studied in natural populations of M. xanthus, developmental cheating has been reported in a natural isolate of the eukaryotic slime mould Dictyostelium mucoroides.)

One present, cheaters have the potential to invade social groups and persist indefinitely as a parasitic subpopulation. Our results therefore suggest that cheaters of myxobacteria are common in

Figure 2 Spore production of two evolved clones when mixed with their wild-type progenitor at nine different ratios, and the corresponding relative sporulation efficiencies. a, Mixtures of GVB206.3 with DK1622. b, Mixtures of GVB208.3 with DK1622. Squares, triangles and circles indicate total, DK1622 and evolved clones, respectively. The expected production of the evolved clones under H1 (solid lines), the expected production of evolved clones under H2 (dotted lines) and the spore production of DK1622 in independent pure cultures (dashed lines) are shown. Error bars indicate 95% confidence intervals. c, d, Sporulation efficiencies of GVB206.3 (c) and GVB208.3 (d), relative to that of DK1622 for these same initial mixing ratios. Dashed lines indicate a relative efficiency of 1.
nature, unless these bacteria have evolved strategies that oppose cheating. Such strategies may include reproductive cycles that are unfavourable to cheaters and policing functions that repress competition\(^\text{21–27}\).

In principle, the myxobacterial reproductive cycle could involve the filling of new colonies by either single or multiple spores. With single-spore colonization, the outcome of competition between cheaters and cooperators will depend on the performance of genetically homogeneous groups (assuming no migration or appearance of new mutants), a situation in which the developmentally defective cheaters are inferior. Alternatively, if colonies are founded by clumps of many spores, competition will occur within fruiting bodies, a situation in which cheaters are superior when rare and will therefore persist as a parasite population. Two features of the myxobacteria suggest they do not exclude cheaters by single-spore dispersal. First, \textit{M. xanthus} spores adhere tightly to one another and require vigorous sonication to separate them in the laboratory. Thus, new colonies in nature are probably founded by multiple clumped spores. Second, myxobacteria are highly motile, promoting migration that opposes the homogenizing effect of single-spore colonization. Barring some unknown policing mechanism that represses cheater genotypes, we therefore predict that cheaters are common in natural populations of \textit{M. xanthus}.

\section*{Methods}

\subsection*{Strains}

Two wild-type clones of \textit{M. xanthus} were used in this study, along with mutant genotypes derived from each of them. Wild-type strain DK1622 (ref. 16) is sensitive to the antibiotics used to distinguish competing genotypes. DK1622 is ancestral to the evolved genotypes GVB206.3, GVB208.3, GVB210.3, GVB212.3, GVB214.3 and GVB216.3, all of which are genetically marked by resistance to rifampicin\(^\text{28–30}\). These six clones were isolated from six populations that had evolved independently from DK1622 for 1,000 generations in a nutrient-rich, liquid habitat\(^\text{6}\). The C-signal defective mutant LS523 (csgA mutation) and MS2021 (ref. 10) (asgB mutation) are derived from DK1622 through genetic manipulation and are resistant to oxytetracycline\(^\text{1}\). DK101 is another developmentally wild-type strain that carries a pilQ mutation that hinders S motility\(^\text{29–30}\); it is sensitive to kanamycin. DK101 is the progenitor of the A-signal defective mutants DK4312 (ref. 7) (asgB mutation) and MS2021 (ref. 10) (asgF mutation), both of which are resistant to kanamycin. All strains are stored as clones in an ultralow freezer.

\subsection*{Sporulation assay}

Cultures growing vegetatively in CTT liquid\(^\text{1}\) were harvested by centrifugation at 4,900g for 15 min at room temperature and resuspended in 0.5 ml TPL liquid\(^\text{1}\) at \(~\sim 5 \times 10^6\) cells ml\(^{-1}\). Resuspended cultures were mixed at appropriate ratios, and 100 ml of the mixtures were spotted on TPL agar plates. Development progressed at 32°C to 68 h, when cells were collected into 0.5 ml of sterile H\(_2\)O, heated for 2 h at 50°C, sonicated by microtip, diluted and plated by mixing samples with 10 ml CTT soft agar (0.5% agar). Collected samples containing rifampicin-resistant evolved clones were also plated in CTT soft agar containing 5 mg ml\(^{-1}\) rifampicin. Spore counts of the rifampicin-resistant genotypes were obtained from plates containing rifampicin, and total spore counts from mixed populations were obtained from plates without antibiotic; spore counts of the rifampicin-sensitive wild-type strains were then calculated as the difference between these plate counts. The plating efficiencies of the rifampicin-resistant genotypes were similar on selective and non-selective agar. The kanamycin- and oxytetracycline-resistant genotypes, however, showed decreased plating efficiency during germination on selective relative to non-selective plates. Therefore, to estimate the marked genotype frequency for mixed cultures containing DK4312, MS2021 or LS523, numerous single colonies were transferred (4–5 days after plating) from the non-selective plates to CTT plates containing the appropriate antibiotic (40 mg ml\(^{-1}\) kanamycin or 12.5 mg ml\(^{-1}\) oxytetracycline) and the frequency of colonies growing on selective plates was determined after three additional days. Sporulation estimates are the mean of log$_2$-transformed spore counts for at least three replicate assays. A marker-control experiment was run for the rifampicin resistance marker using DK1622 and a developmentally proficient, rifampicin-resistant derivative of DK1622. (The resistant clone is the proximate ancestor of the evolved genotypes used in this study\(^\text{22}\).) This marker-control experiment indicated that rifampicin resistance caused a slight disadvantage during development; however, any handicap associated with this or other resistance markers is conservative with respect to the inferences of complementation and cheating among the evolved clones. Sporulation efficiency is calculated as the ratio of a genotype’s spore production to its initial vegetative population size. In Fig. 2c, d, a relative sporulation efficiency of 1 means that an evolved clone and its progenitor produce spores with equal efficiency; values higher than 1 indicate that the evolved clone has a higher sporulation efficiency, whereas values less than 1 indicate higher efficiency of the wild-type progenitor, DK1622.

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A family of candidate taste receptors in human and mouse

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The gustatory system of mammals can sense four basic taste qualities, bitter, sweet, salty and sour, as well as umami, the taste of glutamate1±6. Previous studies suggested that the detection of bitter and sweet tastants by taste receptor cells in the mouth is likely to involve G-protein-coupled receptors7,8. Although two putative G-protein-coupled bitter/sweet taste receptors have been identified9, the chemical diversity of bitter and sweet compounds leads one to expect that there is a larger number of different receptors10,11. Here we report the identification of a family of candidate taste receptors (the TRBs) that are members of the G-protein-coupled receptor superfamily and that are specifically expressed by taste receptor cells. A cluster of genes encoding human TRBs is located adjacent to a Prp gene locus12, which in mouse is tightly linked to the SOA genetic locus that is involved in detecting the bitter compound sucrose octateacetate13±15. Another TRB gene is found on a human contig assigned to chromosome 5p15, the location of a genetic locus (PROP) that controls the detection of the bitter compound 6-n-propyl-2-thiouracil in humans16,17.

To search for taste receptors, we devised a strategy that was based on four ideas: first, taste receptors would be encoded by a family of related genes; second, some taste receptor genes would be found at genetic loci associated with the ability to taste specific compounds in mouse or human; third, taste receptors would be G-protein-coupled receptors (GPCRs) that have limited sequence similarity to other members of the GPCR superfamily; and last, taste receptor genes might be found by using the resources of the Human Genome Project to look for GPCR-encoding genes in genomic regions implicated in taste perception.

We first asked whether there are genes encoding new GPCRs in the region of the human genome corresponding to the mouse SOA locus, which is tightly linked to a Prp gene13,14. Using the Jackson Laboratory Mouse Genome Informatics website (http://www.informatics.jax.org), we determined that the Prp gene and the SOA locus are located on mouse chromosome 6 (63.6 centimorgans (cM)) and that the syntenic region in human is on chromosome 12p13. We then determined whether any of the genes that mapped close to the mouse SOA locus had been cloned in human and were deposited in the National Center for Biotechnology Information (NCBI) nr database (http://www.ncbi.nlm.nih.gov); we used the sequences of those genes to search the NCBI Human Genome Sequence (HGS) database, focusing on human chromosome 12. Among the genes we used for this search was a Prp gene, which we found on the chromosome-12 contig NT_001856. By examining the contig map of chromosome 12 in the NCBI HGS database, we were able to identify contigs that flanked NT_001856. This provided us with a focus set of contigs that might contain taste receptor genes.

To find genes encoding GPCRs in this focus region, we first searched the human chromosome 12 database with large sets of GPCR protein sequences that we compiled from a GPCR database (http://www.gpcr.org/7tm). Although we identified a few genes on human chromosome 12 that appeared to encode GPCRs, none was located in the focus set of contigs or their vicinity. We then searched the database with a member of the V1R family of candidate pheromone receptors (V1R5)18,19, because members of this family are not in the GPCR database and therefore had not been included in our GPCR sequence sets. We identified two sequence stretches in contig NT_001856 (which contains the Prp gene) encoding protein sequences distantly related to V1R5. When we retrieved the DNA sequence in and around one of these DNA regions and translated it, we determined that it contained an intronless gene encoding a putative receptor protein of 311 amino acids (hTRB2 for human taste receptor, family B, no. 2) with weak homology to V1R5 (Fig. 1).

We then asked whether hTRB2 belongs to a family of related receptors, as we expected would be the case for taste receptors. Using hTRB2 to search the chromosome 12 database, we identified eight related genes, all in the NT_001856 contig. Of the eight TRB genes, six encode receptors related to hTRB2 (Fig. 1) and two are pseudogenes. Using these TRBs, we were unable to find any members of this family in either the NCBI nr or expressed sequence tag databases, consistent with the idea that these receptors might be expressed exclusively in taste tissue. However, we did identify a gene encoding a TRB family member (hTRB7, Fig. 1) in a contig assigned to human chromosome 5p15, the location of PROP, the genetic locus that governs the ability of humans to taste 6-n-propyl-2-thiouracil, a bitter compound16. We also found a total of five TRB genes (one a pseudogene) on three chromosome-7 contigs, two of which are assigned to 7q31-32 (data not shown).

The candidate receptors encoded by the TRB genes on chromosomes 12 and 5 (and 7) share sequence motifs with one another, uniting them as members of the same receptor family. Although they have the seven-transmembrane domain structure characteristic of GPCRs, they are unrelated in sequence to both mGluR4, which detects glutamate10,21, and the candidate taste receptors TR1 and TR2 (ref. 9). In addition, mGluR4, TR1 and TR2 have long extracellular amino-terminal domains that are proposed to bind ligand, whereas TRBs have very short N termini, suggesting that they use a different mode of ligand binding. Although TRBs are distantly related to V1Rs, TR1 and TR2 resemble V2Rs (refs 22–24), candidate pheromone receptors that are expressed, with V1Rs, in the vomeronasal organ. The TRBs that we have identified show high variability in protein sequence, suggesting that, like odorant receptors in the nose25, different family members may recognize chemicals with very different structures, such as chemically diverse bitter tastants.

Are TRBs expressed in taste receptor cells, as they must be if they are truly taste receptors? To address this question, we turned to the mouse. We first asked whether we could isolate sequences encoding TRBs from either mouse genomic DNA or complementary DNA prepared from mouse taste tissue. We used polymerase chain reaction (PCR) with degenerate primers matching conserved sequences in TRBs to amplify related sequences, and then cloned
Urban climate

The water cooler

Ever thought about escaping the heat of the city by getting down to the lakeside? According to Arin Jazwilevich and colleagues (Climatic Change 44, 515–536; 2000), having a lake on your doorstep may not only provide a waterside retreat but help keep the city’s climate appreciably cooler.

Mexico City, it seems, was such an example. Earlier this century, the relatively small city (86 km²) was bordered by Lake Texcoco (120 km²), and the ‘urban heat island’ effect — where city temperatures exceed those in surrounding rural areas — was only 1.5 °C. Urban expansion has dramatically altered the balance, so that the sprawling city (pictured), at 1,200 km², now dominates the Basin of Mexico. The lake has been reduced to a mere 10 km² or so in area, and the heat of the city can exceed that in neighbouring countryside by 8–10 °C. This commonly observed effect is usually explained by the surface geometry and thermal properties of all that extra concrete and asphalt.

But what part might the lake play? Jazwilevich et al. developed a model to reproduce urban climate under present-day conditions and those of 1921, using the respective city and lake areas. Then, taking the situation in 1921, and reducing lake area to today’s extent, they found that the expected temperature field was almost identical to that of today. From this the authors infer that the increase in city temperatures is largely due to the reduction in lake area and the evaporative cooling it provided, as well as the massive urbanization. Perhaps the finding has come too late for Mexico City. As urban areas continue to swallow up land around the world, however, town planners would be wise to keep a little bit of Venice in their designs for the comfort and health of inhabitants.

Jim Gillon

Evolution

Bacterial cheaters

Joan E. Strassmann

Cooperative groups of higher organisms are vulnerable to cheaters that reap the benefits of cooperation without paying the costs. For example, an unrelated male lion may take a gazelle away from the females that killed it. On page 598 of this issue¹, Velicer and colleagues show that similarly selfish behaviour is also seen in bacteria — specifically, in groups of Myxococcus xanthus.

As they move through the soil, preying upon other microorganisms, M. xanthus individuals behave in group-coordinated ways that are more usually associated with complex social animals than with bacteria². Individuals move and feed as a cooperative, tight group reminiscent of teams of hunting army ants or wolves, but in this case secreting enzymes that kill and degrade (lyse) the prey before them³–⁴.

But M. xanthus display their most dramatic social act when they are starving. Individuals aggregate even more densely than when feeding and form a raised ‘fruited body’ (Fig. 1, overleaf), in which a minority of cells convert from vegetative rods to hardy, spherical spores⁵–⁶. During this process, many cells commit suicide and lyse, releasing their contents¹. The contents of lysed cells might enable spore formation by other cells — the contents may be eaten, or might otherwise influence the sporulating cells⁷. The development of a fruited body involves cell-to-cell signalling, the formation of a multicellular structure, and morphological changes in individual cells. Myxobacteriologists therefore consider the fruited body to be a simple model system in developmental biology⁸, with processes at least partly analogous to those that form structures as complex and integrated as a fruitfly’s body.

Standing in marked contrast to this cooperative, and often altruistic, behaviour, however, are the social cheaters described by Velicer et al.¹. The authors isolated M. xanthus mutants showing developmental defects from six populations that had evolved in a liquid medium in which fruiting-body formation could not occur. The populations had evolved for 1,000 generations — a short time in evolutionary terms, but still a much longer period than would be feasible in experiments on most organisms. After this period, clones from all six mutant lines showed a decreased ability to form spores when starved on a solid substrate, a loss of function that is not surprising given that the bacteria had adapted to an environment in which the function is not needed.

Velicer et al. then mixed the mutants with ancestral (wild-type) populations, at a ratio of 1% evolved clone to 99% ancestral clone. Under these competitive conditions, five of the six defective clones produced more spores, relative to the number of input cells, than they had when in isolation. Three of these clones did even better in such mixtures than the wild-type clones, and thus qualified as ‘cheaters’.

Velicer and colleagues also studied three other, previously known, developmental mutants that were defective in either of two signalling systems and, consequently, in spore production when in pure culture. In mixtures of 1% mutant to 99% wild-type clones, two of these mutant clones generated spores at a higher frequency than did the wild-type clones. So, some developmental mutants not only revert to spore production when in a mixture with wild-type cells, but can even be over-represented in the spores in comparison with their original frequency.

But exactly how these mutants cheat is not obvious. There is no clear functional class of altruists, analogous to honeybee workers or to stalk cells in cellular slime moulds, that the cheating M. xanthus avoid joining. The M. xanthus individuals that are the best candidates for altruists are the cells that lyse during development, although it is not even certain that these lysed cells are actually eaten by the sporulating cells. Moreover, we do not know whether the cheating clones cheat specifically by avoiding lysis. The mutants might simply be able to continue growing while some of the wild-type cells lyse, because the mutants do not pay all the metabolic costs of producing the signalling chemicals that contribute to the formation of the fruited body¹. If the mutants’ advantage is indeed that they can simply keep on growing, they would need to be able to continue dividing after being plated on starvation medium. This might be possible, as they could eat lysed cells.

Cheating M. xanthus clearly evolve easily, but their fate in nature is uncertain. Rare cheaters exploit the wild-type bacteria to increase in frequency within groups. But when they become common, they disrupt sporulation, and the whole group suffers⁹. So, an understanding of population struc-
A new spin on magnets

Thomas F. Rosenbaum

Just like photons propagating through space, electrons flowing through a crystalline metal travel in simple plane waves. Add sufficient disorder and those electrons are no longer free to fly. Rather they will diffuse from scatterer to scatterer, performing a random walk through the ‘dirty’ metal. At very low temperatures, near absolute zero, the phase of the electron becomes important and quantum interference effects manifest themselves. At the quantum level both the amplitude and phase of the electron interactions in the presence of electron–electron interactions in the presence of magnetic field is the primary means of information storage in the hard disk on a computer. Magnetoresistive sensors are also found at the business end of a car’s speedometer. New materials continue to emerge as potential candidates for magnetic recording. Many different materials have shown magnetoresistance that involves scattering of conduction electrons by magnetic fields. For example, when a magnetic field is applied the scattering may be reduced, leading to negative magnetoresistance.

In the ferromagnet studied by Manyala et al., the unusual square-root dependence of the electrical resistance on magnetic field, and the positive nature of the magnetoresistance, point to a different underlying mechanism. In fact, the magnetoresistance of a classic semiconductor such as phosphorus-doped silicon, in which the effect was first observed, switches from negative to positive when quantum interference effects emerge as the temperature drops below 1 K. This means that the resistance switches from shrinking with magnetic field to growing with magnetic field as the temperature is lowered. Remarkably, Manyala et al. show that the disordered, metallic ferromagnet Fe$_{1-y}$Co$_y$Si behaves just like a doped semiconductor, but at a temperature two orders of magnitude higher. It is the large (megagauss) internal fields of the ferromagnet that allow this behaviour to occur at higher temperatures. The same electrons, with intertwined electronic spin and charge, are responsible for both the magnetic and electrical properties of Fe$_{1-y}$Co$_y$Si. This is in contrast to what happens in most magnetoresistive materials, where the electrons involved in electrical conduction are different from those responsible for the magnetism. In Fe$_{1-y}$Co$_y$Si, the effects of quantum coherence have been amplified by the magnetic nature of the doped FeSi host, in the same way that shrinking electronic devices to the nanometre scale reveals the subtleties of quantum processes at room temperature in quantum dots and magnetic heterostructures.

The consequences of magnetoresistance in Fe$_{1-y}$Co$_y$Si extend beyond the new insights that it provides into the fundamental quantum nature of charge transport in magnets. An electrical resistance that changes with magnetic field is the primary means of information storage in the hard disk on a computer. Magnetoresistive sensors are also found at the business end of a car’s speedometer. New materials continue to emerge as potential candidates for magnetic recording. Many different materials have shown magnetoresistance that involves scattering of conduction electrons by magnetic fields. For example, when a magnetic field is applied the scattering may be reduced, leading to negative magnetoresistance.

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