

Particle physics

Precision precession

Frank Wilczek

The most accurate measurement yet of the way an elementary particle wobbles — precesses — in a magnetic field is getting physicists excited. If it is right, we may be on the threshold of a new era of particle discoveries.

A large collaboration of scientists, known as experiment E821 at Brookhaven National Laboratory, announced on 9 February the latest result from their study of the behaviour of fundamental particles (specifically muons) in a magnetic field¹. Their measurement of the magnetic moment of the positive muon has an uncertainty of just over one part per billion, three times better than the existing value. Moreover, it deviates in a small but important way from the prediction of the theory of matter (also known as the standard model — a woefully inadequate description). At this level of precision, the behaviour of the muon becomes sensitive to the existence of new heavy particles, such as those required by supersymmetry, a very popular but still hypothetical extension of the standard model. Although not yet statistically airtight, the E821 result could be a glimpse into a new world of physical phenomena.

The story of magnetic moments is brief, but glorious. Evidence that the electron had a magnetic moment was first gleaned in 1925 by two graduate students, Samuel Goudsmit and George Uhlenbeck. By carefully studying atomic energy levels (spectra), they discovered discrepancies with prevailing theories that could be resolved if it were assumed that all electrons act as tiny bar magnets. The strength of these elementary magnets is commonly given in terms of the gyromagnetic ratio, g , the relationship between the magnetization of the particle and its rotation. In a magnetic field the axis of a particle's intrinsic rotation, or spin, precesses in the same way that a toy top wobbles as it spins. The frequency of the particle's precession is μB , where B is the size of the magnetic field and μ is its magnetic moment given by gsq/mc , where s , q , m are the spin, charge and mass of the particle, respectively, and c is the speed of light.

Classical physics predicts $g = 1$ for a spinning charged particle, but to fit their observations Goudsmit and Uhlenbeck required $g \sim 2$ (where g_e is g for the electron). In 1928 Paul Dirac proposed a modification of the Schrödinger equation describing the quantum behaviour of electrons, to make it consistent with the special theory of relativity. Though it was motivated from unrelated considerations, Dirac's equation predicts $g_e = 2$ for simple, point-like electrons, providing a neat agreement between experiment and theory that lasted two decades. Then, in 1947, Polykarp Kusch and collaborators

determined that the experimental value of g_e is actually slightly bigger than Dirac's value, by approximately $g_e - 2 = 0.00236$.

Theorists quickly appreciated the significance of this discrepancy. The Dirac theory applies to an idealized electron, existing in isolation in empty space. But real observable electrons are surrounded by virtual particles that briefly pop in and out of existence in the quantum 'vacuum'. In one sense virtual particles are purely theoretical constructs, because they are by definition too short-lived to be observed directly, but to physicists they are quite real and have a tangible influence on the particles we observe.

In 1948 Julian Schwinger calculated that the interaction of electrons with virtual photons modifies their magnetic moment in a way that quantitatively explains Kusch's result. Richard Feynman did the calculation in a different and simpler way that could be generalized. Using these techniques, and the modern theory of matter, magnetic moments can be calculated with extraordinary precision. For this work, Dirac won the Nobel Prize for physics in 1933, Kusch in 1955, and Schwinger, Tomonaga and Feynman in 1965.

The same calculations hold for muons, which are elementary particles that resemble electrons in every way except that they are about 200 times as heavy. They have a half-life of about a microsecond, and so are not found on Earth in ordinary matter, although they do appear in cosmic rays. They are more con-

venient to study than electrons because the muon's magnetic moment is considerably more sensitive to the effects of heavy particles. The strength of the muon's bar magnet, g_μ , can be measured in a high-quality magnetic field by monitoring how rapidly it precesses. But to reach the precision of the Brookhaven experiment requires the world's largest superconducting magnet (Fig. 1) and measurements from billions of muons. The experiment is a *tour de force* of modern experimental technique, despite being relatively cheap by high-energy physics standards. The character of the result from E821 is best conveyed through its accurate quotation: $g_\mu - 2 = 0.0023318319 (\pm 0.0000000013)$.

Theorists have also worked hard to reach this level of accuracy. Their calculations must include not only the effects of virtual photons, but also more complex effects that arise because the properties of the virtual photons are themselves modified by interactions with virtual electrons, muons, tau leptons and quarks. Moreover, the small effects of virtual versions of much heavier particles, such as W and Z bosons, come into play. When all this is taken into account, there is a mismatch between theory and experiment of about four parts per billion. The discrepancy is about 2.6 standard deviations — highly suggestive, but not yet definitive.

When assessing loose talk about shaking the foundations of physics or overthrowing the standard model, which has already appeared in the popular press, we should keep in mind the size of the reported effect. Ordinarily, a theory whose rigorous application predicts the results of subtle experimental results with part-per-billion accuracy might be considered to be doing pretty well! So it is here. For this reason, the most plausible explanations of the discrepancy — barring computational or experimental error, or a statistical fluke — involve adding to the accepted theory



Figure 1 The 14-metre-diameter superconducting magnet used by the E821 experiment at Brookhaven National Laboratory, Long Island, New York. It provides a magnetic field of extremely high quality, allowing physicists to measure the magnetic behaviour of the muon with unprecedented accuracy.

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of matter, rather than overthrowing it. Indeed, the simplest explanation is that there is some new species of virtual particle that has inadvertently been left out of the theoretical calculation, because its analogue in the real world is very heavy and unstable and has so far escaped detection in particle accelerators.

Particularly attractive, because it is also suggested by several other pieces of indirect evidence, is the possibility that some of these particles are supersymmetric². Supersymmetry theory predicts that every fundamental particle in the standard model has a heavier superpartner — the selectron for the electron and smuon for the muon. If supersymmetry is true, it provides new virtual particles galore, with masses of 100–1,000 times the mass of a proton. Our knowledge of the correct model of supersymmetry to use — if any — is limited, so no precise prediction is possible. But the observed E821 discrepancy

sits quite comfortably in the range preferred by models of low-energy supersymmetry³.

The reported E821 results are based on analysis of data taken in 1999. A data sample from 2000, containing four times as many positive muons, awaits analysis. Similar data are now being taken for negative muons, which should be able to confirm whether particles and antiparticles behave symmetrically. Elsewhere, work currently underway will narrow down residual uncertainties in the predictions of the standard model. A year from now we'll either be celebrating at the oasis — or mourning the mirage. ■

Frank Wilczek is at the Center for Theoretical Physics, Massachusetts Institute of Technology, Cambridge, Massachusetts 02139-4307, USA. e-mail: wilczek@mit.edu

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Molecular biology

A big development for a small RNA

David A. Clayton

The RNA-processing enzyme MRP contains an RNA component that is essential for its activity. Unexpectedly, it seems that mutations in the gene encoding this RNA cause a multifaceted human disease.

Over 35 years ago, some members of the Amish families in Pennsylvania were found to be suffering from an unusual inherited disease. Later, the disorder was also discovered in a small fraction of the Finnish population. People with the disease are unusually short, have changes in the structure and abundance of their hair, and show abnormalities in the development and function of their bone and cartilage, so the disease was given the name cartilage–hair hypoplasia (CHH)¹. Affected people may also have a compromised immune system, making them more susceptible to infection and predisposing them to cancer, particularly tumours of the lymphatic organs.

Which gene, or genes, might be mutated in people with CHH? In earlier genetic studies, the culprit was tracked down to one of the non-sex chromosomes, and was mapped to a region on chromosome 9 (ref. 2). It was also discovered that the disorder is a recessive condition¹, meaning that, for the disease to occur, both copies of chromosome 9 — one from each parent — must bear mutations in that chromosomal region. Yet, despite these crucial discoveries, attempts to pinpoint the underlying gene have been unsuccessful — until now. The answer, revealed by Ridanpää and colleagues³ in a recent issue of *Cell*, is unexpected.

In refining the map position of the chromosomal region that is associated with CHH, Ridanpää *et al.*³ disclosed 11 candidate genes

that might underlie the disease. Ten of these, as expected, encode proteins. But, when the authors examined these genes from CHH patients, they did not find any mutations that were likely to have caused the disease. The eleventh gene from these patients, however, did have potentially relevant mutations. This gene is different because it is the only known nuclear gene that encodes the RNA component of an RNA-cleaving enzyme, a ribonuclease known as MRP. The RNA encoded by the gene is an essential part of the enzyme, but it is not translated into protein.

This unanticipated finding, that alterations in the gene encoding the RNA component of an RNA-processing enzyme are at least partly responsible for the complex physical hallmarks of CHH, immediately shifts our attention to the enzyme itself (Fig. 1). MRP is a large protein–RNA complex that was first characterized for its ability to cleave the ‘primer’ RNAs needed for copying mitochondrial DNA⁴ — hence the name MRP, for ‘mitochondrial RNA processing’. But most MRP is localized to a subsection of the nucleus (the nucleolus), where it participates in a late stage in the processing of the so-called 5.8S ribosomal RNA in yeast, and presumably in other organisms as well⁵.

The CHH-associated mutations that Ridanpää *et al.* found in the gene encoding MRP’s RNA component were insertions or duplications of sequence in the promoter (a

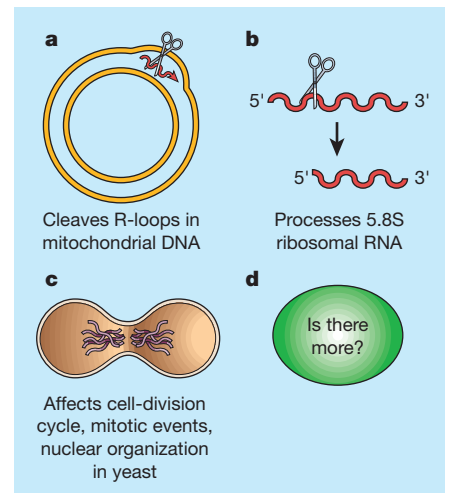


Figure 1 Functions of the RNA-cleaving enzyme (ribonuclease) MRP. Ridanpää *et al.*³ show that mutations in the gene encoding the RNA component of MRP underlie an inherited disorder, cartilage–hair hypoplasia. These mutations could affect several cellular processes. a, Processing of mitochondrial ‘primer’ RNA (wavy line) during the replication of mitochondrial DNA. The mitochondrial DNA is shown as a double ring. The primer RNA must first associate with the DNA template, forming an RNA–DNA hybrid. The MRP (shown as scissors) then cleaves the primer RNA, leaving it with free 3' ends, to which bases can be added by the mitochondrial DNA-copying machinery (DNA polymerase; not shown). Loss of MRP activity in mitochondria might lead to a depletion of mitochondrial DNA. b, A late step in the processing of precursor RNA to mature forms of 5.8S ribosomal RNA. The ribosome is required for protein translation, so loss of MRP activity might hamper translation of key proteins. c, In yeasts, mutations in the RNA¹¹ or protein¹⁰ components of the MRP result in alterations in the mitotic cell-division cycle and cell morphology. The exact role of MRP RNA here is unknown. d, MRP might have yet more functions, either as a site-specific ribonuclease, or together with other activities.

control region), or principally single base changes in the RNA-coding part. The authors looked at cells from two patients who had both types of mutation: one copy of the gene had mutations only in the promoter region; the other copy had mutations only in the coding sequence. These cells contained less than half the amount of the MRP RNA found in cells from controls. And this RNA came only from the genes that had mutations in the coding sequence, implying that the reduced RNA levels were caused by alterations in the structure of the promoters of the other genes, and hence in the control of gene expression. Cells from patients with mutations only in the RNA-coding part expressed the RNA at normal levels. Interestingly, these coding-sequence mutations