Quantum effects of gravity

Thomas J. Bowles

The effects of gravity and quantum mechanics rarely overlap because of the different scales involved. An experiment with ultracold neutrons has now been able to probe both simultaneously.

The visible effects of gravity usually occur at large scales: gravity governs the path of projectiles, and the motion of stars and planets. By contrast, the effects of quantum mechanics — one of the great successes of twentieth-century physics — are usually only observable at the atomic scale. In the quantum realm, the gravitational force is so weak that it is difficult to observe quantum effects caused by gravity. But on page 297 of this issue, Nesvizhevsky and collaborators[1] report an experiment in which they observed quantum effects of gravity on the behaviour of ultracold neutrons (UCNs). These neutrons have kinetic energies so low that they can be trapped by gravity above a reflecting surface.

Neutrons can be reflected from surfaces when the repulsive force arising from the potential barrier at the surface is greater than the downward velocity component of the neutron perpendicular to the surface. Because the potential barrier is so small, normally only those neutrons arriving at a shallow angle to the surface will be reflected, whereas those that are dropped vertically on to the surface will be absorbed, or transmitted to the other side. But the total velocity of UCNs is so small (less than 8 m s^{-1}) that they are always reflected, regardless of the angle of incidence. Another important characteristic of UCNs is that their gravitational interaction is about as strong as their kinetic energy. A UCN that leaves the surface in the vertical direction is slowed down and eventually turned around by gravity. These two properties allowed Nesvizhevsky *et al.*[1] to construct a trap in which the UCNs are limited in their motion by a reflecting surface from below and by gravity from above.

The trap built by Nesvizhevsky and colleagues can be described as a potential-energy well. A particle in a potential well is trapped because it doesn't have enough energy to escape the well. Classically, a particle inside the well can have any energy as long as it is less than the escape energy. But in quantum mechanics, particles in a potential well are only allowed discrete energy values. In the case of electrons inside an electromagnetic potential well, the discrete (quantum) states are directly responsible for the structure of atoms. The gravitational potential well created by Nesvizhevsky *et al.* also has discrete energy states, the lowest ($n = 1$ state) being at 1.41 peV (1 peV is 10^{-15} electron volts), corresponding to a vertical UCN velocity of 1.7 cm s^{-1}. A UCN with this velocity can travel only 15 μm in the vertical direction before it is turned around by gravity.

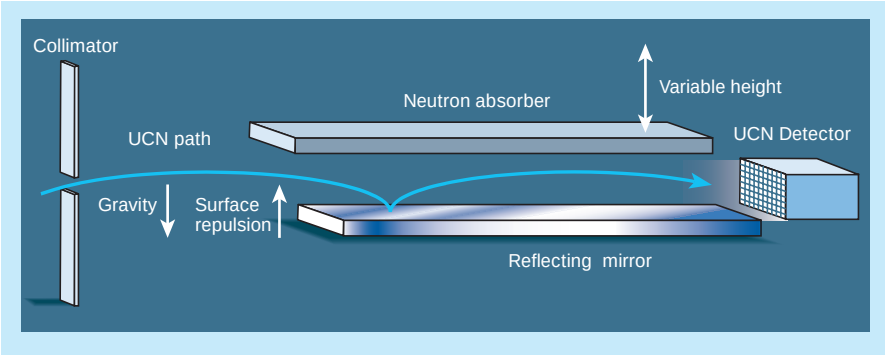

Figure 1 **A system for analysing the flight path of ultracold neutrons (UCNs). In their experiment, Nesvizhevsky *et al.*[1] take a beam of UCNs and let them fly horizontally above a reflecting mirror. Providing that all other forces (excluding gravity and repulsion by the mirror) are eliminated, the UCNs will follow parabolic trajectories through the system. According to quantum mechanics, if one measures the vertical velocity component of a UCN in the trap, there are expected to be discrete values corresponding to the quantum energy levels of the trapped neutrons. By adjusting the height of a neutron-absorbing layer above the mirror, Nesvizhevsky *et al.* identify the lowest-energy UCNs transmitted through the system, and therefore the first quantum state of UCNs allowed in the trap. This is a first indication of quantum effects caused by gravity.**

So in this experiment, quantum mechanics requires that a UCN inside the trap cannot have a vertical velocity component of less than 1.7 cm s^{-1}. It can have higher values, but a UCN with greater energy must have a vertical velocity that corresponds exactly to one of the higher energy states ($n = 2, 3, \ldots$). Because the gravitational force acts on the vertical velocity only, there is no potential well in the horizontal direction in the trap, and the neutron's horizontal velocity can have any value.

Nesvizhevsky and colleagues used the intense UCN source at the Institut Laue-Langevin reactor in Grenoble, France, to produce a highly focused beam of UCNs. The authors control the vertical velocity component of UCNs entering the trap by adjusting the height of a neutron-absorbing material above a reflecting surface (the mirror in Fig. 1). This system serves to analyse the vertical velocity component of the UCN in the gravity–mirror trap by measuring the transmission of UCNs through the system as a function of the height of the neutron absorber above the mirror.

The authors found that no UCNs were transmitted at all until the absorber was more than 15 μm above the mirror. In classical mechanics, neutrons with any value of vertical velocity could be transmitted, so one would expect to see the number of UCNs increase as the absorber is raised. In the quantum picture, until the vertical velocity component of the UCNs coincides exactly with the energy of the first quantum state, no UCNs can exist in the trap, so no UCN can be transmitted. As the height of the absorber increases further, sudden increases in the number of transmitted UCNs are expected whenever their vertical velocity component matches the higher-energy quantum states.

The data show some hint of stepped increases at the values corresponding to higher energy states, consistent with the existence of these states, but they are not yet conclusive. Nonetheless, the evidence for the existence of the first energy state is convincing and confirms that a quantum effect occurs in the gravitational trap. The difficulty of this measurement should not be underestimated. The researchers are measuring a quantum effect caused by gravity that requires a resolution of 10^{-15} eV. Interactions of the neutrons with other fields would normally obscure such a tiny effect, but the neutron's lack of electric charge and the low kinetic energy of the UCNs make such observations possible.

The authors are planning further studies of gravitationally trapped neutrons. The

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energy resolution of the system is defined by the quantum uncertainty principle and is determined by the time the UCNs spend in the trap. If this time can be substantially increased, an energy resolution as high as 10^{-18} eV could be achieved. More intense sources of UCNs (which are under development) are required for such precision, and could lead to new studies of fundamental physics. For example, improved tests of the equivalence principle are needed to investigate the interplay of quantum mechanics and gravity. The equivalence principle says that in a uniform gravitational field all particles, regardless of their mass or composition, fall with the same acceleration (9.8 m s^{-2} near the surface of the Earth). This means that the inertial and gravitational masses of the neutron have to be equivalent. Until now, such assumptions have been hard to check systematically, but the work of Nesvizhevsky and colleagues could provide physicists with a new probe of the fundamental properties of matter. ■

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1. Nesvizhevsky, V. V. *et al. Nature* **415,** 297–299 (2002).

Diabetes

Fat in all the wrong places

Jeffrey Friedman

Obesity and a rare, congenital absence of fat cells are associated with damaging levels of fat in various tissues, and diabetes. Leptin helps to remedy these problems by causing oxidation of fatty acids in mitochondria.

Obesity is often associated with insulin-resistant diabetes, and the health consequences of excessive fat can largely be attributed to this connection[1]. The cause of the association is not known. But from studies using the hormone leptin to treat severe insulin-resistant (type II) diabetes in experimental animals, it seems that the likely culprit is fat, not in specialized adipose tissue, but elsewhere. Writing on page 339 of this issue[2], Minokoshi *et al.* add to this picture by showing that leptin can cause fat to be cleared from skeletal muscle and by describing the cellular mechanism involved.

Diabetes is characterized by increased levels of glucose in the blood stream, which results in organ damage. Blood glucose is controlled by insulin, which is secreted by beta cells in the pancreas. Insulin stimulates the uptake and use of glucose by muscle and fat cells (adipocytes), and reduces its output by the liver, thus lowering glucose levels in the blood. Insulin also stimulates lipid biosynthesis in fat and liver cells.

Obesity is generally associated with resistance to insulin-stimulated glucose transport in muscle and fat, and to insulin-stimulated suppression of glucose production in the liver. When insulin secretion cannot meet the increased demand in obese subjects, a rise in blood glucose levels ensues. These events can, to some extent, be reversed by even modest weight loss, which improves insulin signalling and protects pancreatic beta cells. But why is obesity associated with insulin resistance, and weight loss with improved insulin signalling? Various reasons have been proposed, one of which is that excess lipids, particularly lipids in the 'wrong places', can inhibit insulin signalling and also impair beta-cell function[3–6].

According to this 'lipotoxicity hypothesis', insulin resistance develops when excess lipids are deposited in insulin-sensitive cell types other than adipocytes (which are uniquely designed to store fat for use as energy during hard times). The other cell types include those of liver and skeletal muscle. The result of excess lipid is an inhibition of insulin action. The precise identity of the lipid factor responsible is not known, although fatty acyl CoA (a biochemically active fatty acid) and/or diacyl glycerol molecules (one glycerol molecule bound to two fatty acids), acting through a form of protein kinase C, are likely suspects[5]. By activating this kinase, the lipid molecules seem to reduce the activity of a molecule known as IRS-1, a key component of the insulin signalling pathway.

The latest instalment of this story begins with an apparent paradox. Although obesity is associated with insulin resistance and lipotoxicity, so too is a rare human disorder, lipodystrophy, in which fat tissue is absent. As a result, excess lipid accumulates in tissues such as the liver and skeletal muscle, and patients often suffer from a virtually untreatable resistance to insulin. Genetically engineered experimental animals show the same symptoms[7]. In both obesity and lipodystrophy, then, excess lipid in cell types other than adipocytes is associated with insulin resistance. If the lipotoxicity theory is true, then depletion of this intracellular lipid should improve sensitivity to insulin.

Leptin is produced by adipose cells and 'reports' nutritional information to regulatory centres, in the hypothalamus and elsewhere, to regulate the amount of adipose tissue. Treatment of mice with a genetically engineered form of the hormone indeed reduces the amount of adipose tissue. But it also reduces the levels of intracellular lipid in, for example, skeletal muscle, liver and pancreatic beta cells[1,8,9], and — as predicted by theory — improves sensitivity to insulin[6,7]. Treatment of one strain of lipodystrophic mice with leptin depletes the massive fat deposits in the liver and elsewhere, and corrects the animals' diabetes[7]. In a second lipodystrophic strain, fat-cell transplants from normal mice correct the diabetes, but transplants of fat cells that do not produce leptin (from *ob/ob*

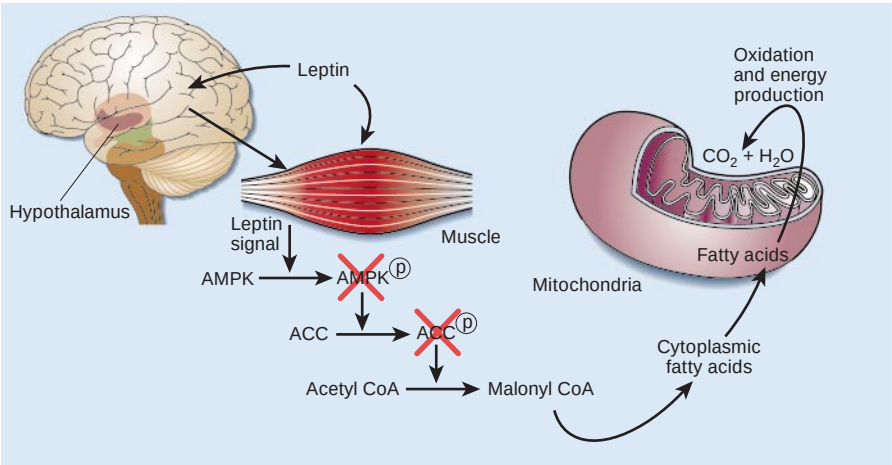

Figure 1 **Leptin's control of fat in skeletal muscle[2]. In cells, there is a balance between transport of fatty acids into mitochondria and their subsequent oxidation, and storage of these compounds as triglycerides in the cytoplasm. This balance is regulated mainly by malonyl CoA, a fatty acid that is generated by the enzyme acetyl CoA carboxylase (ACC). Malonyl CoA inhibits transport of fatty acids into mitochondria, thereby preventing their oxidation[12]. Leptin causes the phosphorylation of AMP-activated protein kinase (AMPK), which in turn phosphorylates ACC, inactivating it[13]. Leptin thus inhibits malonyl CoA synthesis, leading to greater mitochondrial import and consumption of fatty acids. These events seem to result both from the direct action of leptin on skeletal muscle and from its indirect influence that operates through the hypothalamus.**

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