Casimir force changes sign

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This quantum attractive force induces measurable effects between ultrasmall mechanical components. New calculations indicate that systems could be engineered in which Casimir forces are repulsive.

In 1948, Hendrik Casimir calculated that the quantum fluctuations of an electromagnetic field, so-called zero-point fluctuations, give rise to an attractive force between objects[1]. This force is a particularly striking consequence of the quantum theory of electrodynamics (for a review, see ref. 2). Casimir's calculations were idealized — he considered two perfectly conducting parallel plates at absolute-zero temperature — but there are implications for more realistic objects. In *Physical Review Letters*, Kenneth *et al.*[3] have extended these considerations to real-world materials.

Their work follows that of Boyer in 1974, who also studied the case of parallel plates but with one plate perfectly conducting and the other having infinite magnetic permeability (permeability is a measure of the material's response to an applied magnetic field). For this special case Boyer found that quantum fluctuations induce a force with the opposite sign, causing the plates to repel each other[4]. Kenneth *et al.* now extend understanding of the Casimir force phenomenon to the more general situation of realistic 'dielectric' materials that are characterized by both their electrical permittivity (a measure of the material's response to an applied electric field) and their magnetic permeability. Their numerical results show that repulsive forces can arise in the general class of materials with high magnetic permeability.

Although the Casimir effect is deeply rooted in the quantum theory of electrodynamics, there are analogous effects in classical physics. A striking example was discussed in 1836, in P. C. Caussée's *L'Album du Marin* (*The Album of the Mariner*)[5]. Caussée reported a mysteriously strong attractive force that can arise between two ships floating side by side — a force that can lead to disastrous consequences (Fig. 1). A physical explanation for this force was, however, offered only recently, by Boersma[6], who suggested that it originates in the radiation pressure of water waves acting differently on the opposite sides of the ships.

His argument goes as follows: the spectrum of possible wave modes around the two ships forms a continuum (any arbitrary wave-vector is allowed); but between the vessels their opposing sides impose boundary conditions on the wave modes, restricting the allowed values of the component of the wave-vector that is normal to the ships' surfaces. This discreteness created in the spectrum of wave modes results in a local redistribution of modes in the region between the ships, with the consequence that there is a smaller radiation pressure between the ships than outside them.

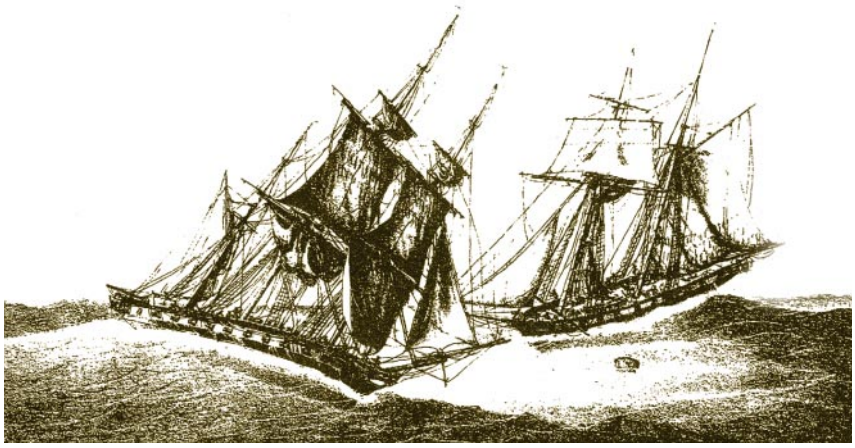

Figure 1 **A Casimir-like effect at sea. In the days of square-riggers, sailors noticed that, under certain conditions, ships lying close to one another would be mysteriously drawn together, with various unhappy outcomes. Only in the 1990s was the phenomenon explained as a maritime analogy of the Casimir force. (Illustration from ref. 5.)**

Analogous arguments had previously been employed by Milonni *et al.*[7] to explain the origin of the Casimir effect itself. In this case, the radiation pressure is due to electromagnetic waves rather than water waves. Casimir had considered a system at zero temperature in which, classically, no radiation pressure is expected. But the quantum theory of electrodynamics states that the electromagnetic field exhibits quantum fluctuations even at zero temperature, and these are the source of Casimir forces acting on macroscopic bodies. Another outcome of these quantum fluctuations is the van der Waals force[8], which, in essence, can be considered as the Casimir force at especially small separations.

Historically, the Casimir effect has been considered to be an exotic quantum phenomenon, but now it is starting to take on technological importance. Because of its relatively short range, it has only a very small effect on the dynamics of macroscopic mechanical systems. But the Casimir force has a major role in modern micro- and nanoelectromechanical systems (MEMS and NEMS), where the distances between neighbouring surfaces are typically far less than 1 μm (ref. 9). This new branch of microelectronics uses the methodology of integrated-circuits manufacturing for the fabrication of on-chip, fully integrated, miniature sensors and actuators, with a rapidly growing range of applications.

In tiny devices such as these, the Casimir force can cause mechanical elements to collapse onto nearby surfaces, resulting in permanent adhesion — an effect called 'stiction', which often proves to be an important factor in the malfunction of NEMS. Follow-up theoretical and experimental studies to the work of Kenneth *et al.*[3] might uncover ways to engineer NEMS in which the Casimir forces are repulsive. They may even open the way for new applications of NEMS that are, in effect, immune to stiction.

Kenneth *et al.* also emphasize that the Casimir force is non-additive. For additive forces, the total force acting on a body is simply the sum of the pairwise contributions between bodies — for example, the total electrostatic force on an element A, interacting with elements B and C, is found by adding the force between elements B and A and the force between elements C and A. For

the Casimir force, however, the additive approach can break down completely. For the case they considered, Kenneth *et al.* show that the additive pairwise approach can even erroneously predict attractive forces, although the exact calculation proves that the forces are repulsive.

The development of vital tools, such as scanning probe microscopy and MEMS and NEMS technology, has made possible a new generation of experiments[2], and the importance of the Casimir phenomenon for both fundamental physics and practical applications is now becoming more widely appreciated. The interplay between basic science and technology is certain to motivate the study of Casimir forces in the years to come. ■

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DNA repair

Right on target with ubiquitin

Cecile M. Pickart

Cellular DNA-repair mechanisms prevent mutations from accumulating, thereby averting defects in cell function. A molecule best known for its role in protein degradation is now shown to have a specific task in DNA repair.

The activity of many of the proteins in our cells depends on the chemical 'labels' that are attached to them. A commonly used label is the ubiquitin molecule, which regulates numerous cellular processes[1]. Many of ubiquitin's regulatory functions reflect its role as a tag that singles out proteins to be degraded[1]. Yet 'ubiquitination' can also signal other fates[2]. The idea that cells can interpret ubiquitin signals in diverse ways was first suggested more than a decade ago, when a protein called Rad6 — which is needed for cells to repair damaged DNA[3] — was found to be an enzyme that helps to join ubiquitin to target proteins[4]. Despite this early hint, further evidence of a role for ubiquitin in protecting DNA was elusive: the relevant target proteins, and the consequences of their modification, remained unknown. But, on page 135 of this issue, Hoege and co-workers[5] identify the first functionally relevant target of ubiquitination during DNA repair — a protein called PCNA.

DNA is highly susceptible to environmental insults that can alter its sequence (causing mutations), or prevent it from being copied altogether (causing cell death)[6]. For instance, DNA damage caused by chemicals or ultraviolet light can block the progress of the enzymes that copy DNA — DNA polymerases — creating gaps in one of the two strands of a newly produced DNA molecule. Ubiquitination is a positive signal in a pathway that permits DNA replication to be completed despite such damage[7]. This pathway, which requires the Rad6 protein, does not remove the original lesions, but instead uses 'post-replicative' DNA synthesis to fill in the gaps, using an undamaged strand as a template. The synthesis can follow either of two routes, one error-prone and the other error-free. The error-free mechanism is especially important — if it fails, the error-prone mechanism takes over, generating mutations in the newly made DNA. Post-replicative repair is the least well understood of the several DNA-repair mechanisms in higher organisms[6].

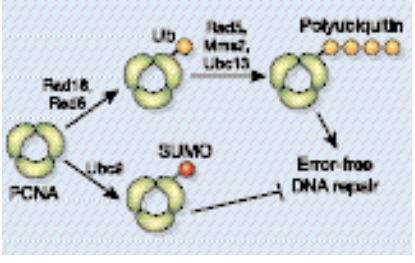

Figure 1 **Connecting DNA repair and ubiquitin, through the PCNA protein. The PCNA trimer, shown at the left, encircles DNA and binds to DNA-replicating enzymes (polymerases). Hoege *et al.*[5] have found that one complex of ubiquitin-conjugating proteins (Rad18 and Rad6) attaches a single ubiquitin (Ub) to a specific lysine amino acid in PCNA. A second conjugating complex (consisting of Rad5, Mms2 and Ubc13) extends a polyubiquitin chain from this first ubiquitin. The modified PCNA then promotes error-free post-replicative DNA repair. Modification of this same lysine amino acid by SUMO, another member of the ubiquitin family, depends on a distinct conjugating factor (Ubc9) and inhibits such DNA repair.**

The main type of error-free post-replicative repair requires five enzymes, including Rad6, that conjugate ubiquitin to target proteins (Fig. 1)[7]. The pathway also relies on a specialized signal in which several ubiquitins are chained together in a particular way[8]. These properties bespeak an intimate involvement of ubiquitin in DNA repair, but several questions remained unanswered until now. Which proteins are modified with ubiquitin by the dedicated conjugating enzymes? Which polymerase (or polymerases) carries out the bulk of error-free post-replicative synthesis? And what is the consequence of labelling the target proteins with ubiquitin? In providing an answer to the first question — that PCNA is ubiquitinated during post-replicative repair — the work of Hoege *et al.*[5] sets the stage for answering the second and third.

PCNA was already known to work with one DNA polymerase in DNA replication[9], and with others in DNA repair. How did Hoege *et al.* discover that PCNA is also a target for ubiquitination? As is often the case, serendipity was important. The researchers actually set out to study SUMO, one of several ubiquitin-like proteins (the name stands for 'small ubiquitin-related modifier') that act through a similar conjugation mechanism[10]. Hoping to gain insight into SUMO-mediated signalling, the authors purified 'sumoylated' target proteins from yeast cells, and found that PCNA was among them.

While confirming this finding, Hoege *et al.* also noticed other modified forms of PCNA, which proved to be attached to the special chain of ubiquitins that is the signature of error-free repair. This 'polyubiquitination', but not sumoylation, required the Rad6 pathway to be active and was induced by DNA damage. Moreover, DNA repair was inhibited when the authors mutated the lysine amino acid in PCNA to which polyubiquitin becomes attached, and genetic analysis showed that this effect resulted from blocking the conjugation of PCNA to ubiquitin. So PCNA is truly a repair-relevant substrate for ubiquitination. But the effect on DNA repair of mutating PCNA's ubiquitin-attachment site is weaker than the effect of blocking ubiquitin conjugation altogether, implying that further targets of polyubiquitination remain to be discovered.

So the polyubiquitination of PCNA is needed for DNA repair. What about the sumoylation of this protein? Ubiquitin and SUMO are attached to target proteins by different conjugating factors. Nonetheless, they can modify the same lysine residue of PCNA. Hoege *et al.* present several lines of evidence that suggest that occupation of this lysine by SUMO inhibits DNA repair. Such antagonism between ubiquitin and SUMO was known in protein degradation[11], but it seems that the strategy may apply more broadly.

With a known substrate in hand, Hoege