APPLIED PHYSICS

Optical cooling without lasers

It is well established that solid objects can be cooled by harnessing the properties of laser light. A laser-free technique that attains such cooling by tuning thermal radiation could have many practical applications. SEE LETTER P.239

YANNICK DE WILDE & RIAD HAIDAR

When two bodies are at different temperatures and not in direct contact with each other, it is generally assumed that thermal radiation (heat) will be transferred from the hotter body to the colder one. If the distance between the bodies is larger than the dominant thermal wavelength (about 10 micrometres at room temperature[1]), there is a maximum heat-transfer rate, known as the black-body limit. On page 239, Zhu *et al.*[2] report that an electronic device called a photodiode can cool a solid that is colder than the photodiode when the two objects are in each other's near field — that is, when they are separated by a distance much smaller than the thermal wavelength. This demonstration could have a tremendous impact on the fields of cooling and heat management.

Until now, optical cooling of solids has been achieved only using laser light[3]. In this approach, a laser beam causes a solid to fluoresce, and the wavelength of the laser light is chosen so that it is longer than the average wavelength of the fluorescence. Because energy is inversely proportional to wavelength, the energy of absorbed photons is lower than the average energy of emitted photons. As a result, energy is removed from the solid, producing a net cooling effect.

Zhu and colleagues' experiment was motivated by a theoretical study in 2016 that proposed a completely different optical-cooling technique[4]. Instead of using laser light, which is coherent (its light waves are synchronized), this technique is based on the direct control of incoherent thermal radiation from a photodiode, combined with an enhanced transfer of thermal radiation in the near field (Fig. 1). A crucial step in the authors' experiment was tuning the photons' chemical potential — a quantity that characterizes the energy that can be absorbed or released by a change in the number of photons.

Photons are, in general, thought to have a chemical potential of zero. The reason is that, to maintain thermodynamic equilibrium, any change in the number of photons inside a container is immediately offset by absorption or emission of photons at the container's walls. However, a non-zero chemical potential can occur in a system in which emission or absorption of photons is associated with a change in the number of other particles that have non-zero chemical potentials.

This situation can arise, for instance, in a semiconductor-based photodiode when the energies of photons are larger than the semiconductor's bandgap (the energy required to generate conducting electrons). More specifically, when a negative (or positive) voltage is applied to such a photodiode, the absorption (or emission) of photons is correlated with the occupation (or vacancy) of electrons. The peculiarity of this correlation translates into a chemical potential of photons that equals the difference in the chemical potentials of occupied and vacant electronic states — a quantity that is proportional to the applied voltage[5].

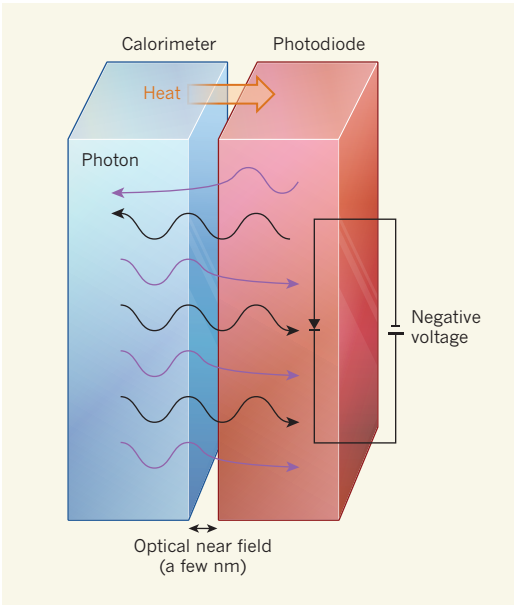

Figure 1 | A method for optical cooling. Zhu *et al.*[2] report an experiment involving a solid object (a calorimeter) and an electronic device called a photodiode, which has a higher temperature than that of the calorimeter. Photons are exchanged between the two objects. The authors show that the photodiode emits fewer photons when a negative voltage is applied to the device than when a voltage is not applied. Moreover, they find that photons can be transmitted between the calorimeter and the photodiode through 'evanescent' waves (purple) when the objects are in each other's near field — that is, when they are separated by a few nanometres. These evanescent waves greatly enhance the transfer of heat between the objects, compared with when they are separated by a larger gap. In this experimental set-up, the photodiode draws heat from the calorimeter, demonstrating an innovative technique for cooling solids.

Zhu *et al.* applied a negative voltage to a photodiode, which meant that photons from the device that had energies larger than the device's bandgap had a negative chemical potential. This feature resulted in a reduction in the thermal radiation emitted by the photodiode compared with the case of thermodynamic equilibrium (zero voltage). Such an effect is known as negative luminescence and was used by Zhu and colleagues to mimic a photodiode that has an effective temperature lower than its actual temperature. Reducing this effective temperature below the temperature of a custom-made device (a calorimeter) naturally produced a flow of thermal radiation from the calorimeter to the photodiode.

However, Zhu *et al.* had to implement a second crucial step before they could detect a measurable cooling effect. To avoid the black-body limit, the photodiode and the calorimeter needed to be brought into each other's near field using a clever experimental set-up. It has already been observed that, at distances much smaller than the thermal wavelength, the transfer of thermal radiation can be greatly increased by the contribution of evanescent waves[6,7] — waves with amplitudes that decrease exponentially with the distance from the surface at which they are produced. The authors used this contribution, which can be thought of as energy tunnelling through the gap between two surfaces, to enhance the cooling by negative luminescence to a detectable level.

Now that Zhu *et al.* have shown that optical cooling can be achieved by controlling the chemical potential of photons, future studies should try to determine the fundamental limits of this technique. One way in which the cooling performance might be improved would be to cover the object to be cooled with a material, such as hexagonal boron nitride or silicon carbide, that supports surface phonon–polariton resonances — couplings of electromagnetic waves and atomic-lattice vibrations

that accumulate at a given energy and that are highly confined at the surfaces of these materials.

Surface phonon–polariton resonances give rise to enhanced near-field thermal radiation at almost a single wavelength[8], which greatly increases near-field heat transfer. Using a photodiode with a narrow bandgap that matches the energy of a resonance could lead to optical cooling that has extremely high efficiency. Such cooling would be faster than that of conventional coolers, with the advantage that the two objects would not need to be in contact with each other[4].

The authors' technique could also be improved using infrared plasmonic nanoantennas — devices that produce thermal radiation at well-defined frequencies and with electromagnetic energy that is highly concentrated in a region of space that is much smaller than the thermal wavelength[9]. Work published last year showed that such devices could be implemented in micrometre-sized, semiconductor-based infrared light detectors that can be operated at room temperature[10]. This suggests that, with some modification, Zhu and colleagues' set-up could be reduced in size and fabricated into semiconductor devices to carry out on-chip cooling of electronics. ■

Yannick De Wilde *is at the Langevin Institute, ESPCI Paris, CNRS, PSL University, 75005 Paris, France.* **Riad Haidar** *is at ONERA, the French Aerospace Lab, University of Paris-Saclay, 91120 Palaiseau, France.*
e-mails: yannick.dewilde@espci.fr; riad.haidar@onera.fr

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METABOLISM

Signalling a brake on heart disease

If blood flow from the heart is impeded, the pressure created causes tissue dysfunction. It emerges that different signals converge on the TSC2 and mTOR proteins to fine-tune the response of heart cells to stress. SEE LETTER P.264

BRENDAN D. MANNING

Chronic tissue stress causes the emission of molecular signals that propagate through cellular lines of communication known as signal-transduction pathways. These pathways trigger responses that can either exacerbate tissue damage or alleviate the harmful effects of the stress. Conditions that hinder the outflow of blood from the heart, such as high blood pressure (hypertension), can induce a type of chronic stress known as pressure overload. This can, in turn, cause abnormal overgrowth of the heart muscle (cardiac hypertrophy), which can precede heart failure (Fig. 1a). Ranek *et al.*[1] report on page 264 that, in heart muscle cells (cardiomyocytes) of mice, pressure overload activates two signal-transduction pathways, one involving an enzyme complex known as mTORC1, and the other involving the enzyme PKG1. The authors show that the two pathways converge to influence the adverse consequences of this stress.

The mTORC1 pathway operates in all cells, detecting changes in their local environment and controlling cell and tissue growth accordingly[2]. On activation, mTORC1 promotes protein synthesis and the formation of cellular components, while suppressing autophagy, a process in which cellular constituents are broken down and recycled[3]. Genetic and dietary factors can lead to chronic mTORC1 signalling in various tissues, a feature shared by diverse disease states, including cancer, obesity and immunological and neurological disorders[2]. Studies in mice show that abnormal loss or gain of mTORC1 signalling can both result in cardiac dysfunction[4–6], underscoring the importance of precise regulation of this pathway in the heart.

Ranek *et al.* found that mTORC1 is chronically activated in the hearts of mice subjected to sustained pressure overload, and that treatment with everolimus, a pharmacological inhibitor of mTORC1, can prevent the development of cardiac hypertrophy and dysfunction resulting from this stress. Interestingly, the authors observed that the drug sildenafil (Viagra), which strongly activates the PKG1 pathway, also blocks the development of cardiac hypertrophy resulting from pressure overload. Ranek *et al.* went on to identify a previously unknown mechanism whereby PKG1 inhibits mTORC1 signalling.

The protein TSC2 is a key regulator of mTORC1 activity in all tissues. TSC2 forms a complex with another protein, TSC1, to

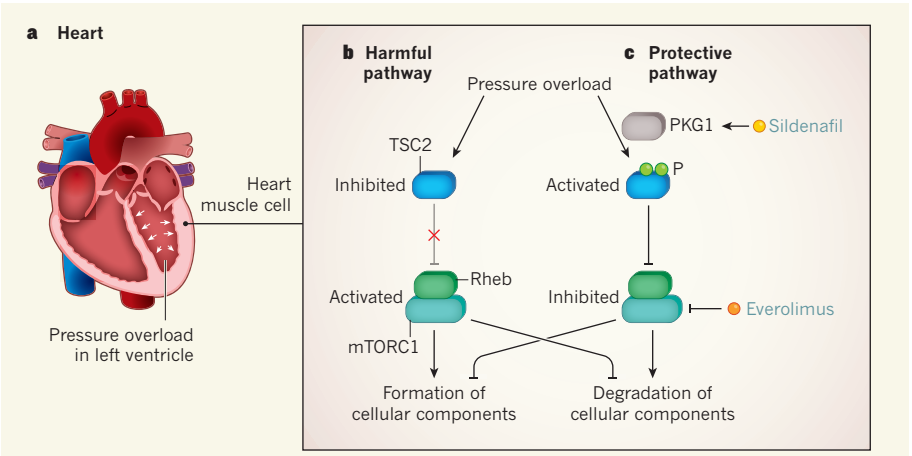

Figure 1 | A key regulatory pathway in the response of heart cells to pressure overload. **a**, Hampered outflow of blood from one of the heart's chambers (left ventricle pictured as an example) leads to sustained pressure on the heart muscle, an effect called pressure overload. **b**, Heart muscle cells sense pressure overload through molecular signalling pathways that regulate the TSC2 protein. Sustained pressure overload inhibits TSC2 (possibly through phosphorylation), preventing the protein from inhibiting the Rheb–mTORC1 protein complex. Chronic mTORC1 activation stimulates the synthesis and inhibits the degradation of cellular components, leading to abnormal tissue growth and heart disease. Ranek *et al.*[1] find that pressure overload also modestly activates the PKG1 protein, which phosphorylates (P indicates a phosphate group) and activates TSC2. This activation attenuates Rheb–mTORC1 signalling and mitigates the harmful effects of stress. Sildenafil strongly activates PKG1, blocking mTORC1 activation and protecting the heart. The mTORC1 inhibitor everolimus also protects the heart by inhibiting the Rheb–mTORC1 complex.