time-dependent frequency  $\omega(t)$ , with a rise or fall characterized by the timescale  $t_c = \omega |d\omega/dt|^{-1}$ . Representative  $t_c$  (orange boxes in Fig. 1) span a dynamic range of many decades. Manipulation of plasmons with gating electrodes or transient gratings typically yield  $t_c$  no faster than nanoseconds, whereas optical pulses enable access to femtosecond timescales.

The magnitude of  $t_c$  is governed by either the excitation or relaxation mechanisms of the electronic system for rising or falling  $\omega(t)$ , respectively. An example of rising  $\omega(t)$  is the creation of the population inversion or hot carriers, which occurs on sub-picosecond timescales; an example of falling  $\omega(t)$  is the cooling of hot carriers by emission of acoustic phonons, which leads to  $t_c$  of the order of a few picoseconds<sup>5</sup>. In some cases, emission of optical phonons or tunnelling of hot carriers across atomically thin barriers can yield to  $t_c$  as short as 10–30 fs (ref. 10).

Another critically important timescale is the plasmon dephasing time  $\tau$  (yellow boxes in Fig. 1). The dephasing time measures plasmonic losses originating from three principal factors: (1) carrier relaxation (including tunnelling processes<sup>10</sup>); (2) plasmon scattering by defects and inhomogeneities; and (3) dielectric losses in the layers proximal to the plasmonic medium. The propagation of nonequilibrium plasmonic waves can only be observed provided  $t_c > \tau$ . For highly confined plasmons in graphene,  $\tau$  can approach a fraction of a picosecond at room temperature<sup>5</sup> and the propagation lengths reported by Vitiello, Cocker and colleagues seem to imply still longer  $\tau$  in black phosphorous (the orange triangles and bars, respectively, in Fig. 1).

Plasmon losses in semiconductors can in principle be compensated using optical gain<sup>9,11</sup> or parametric amplification<sup>12</sup>, thus increasing  $\tau$ . Achieving long plasmon lifetimes in combination with rapid control times will eventually enable ultrafast plasmonic circuits, modulators and switches for a variety of nanophotonic applications. Dmitri N. Basov is in the Department of Physics, Columbia University, 538 West 120th Street, New York, New York 10027, USA. Michael M. Fogler is at the Department of Physics, University of California San Diego, 9500 Gilman Drive, La Jolla, California 92093-0315, USA.

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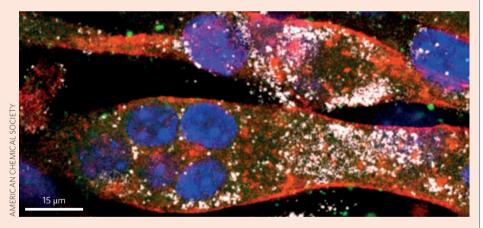
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## GOLD NANOPARTICLES

# A warm-up for muscle cells

To grow artificial muscles or mend natural tissues it is necessary to stimulate muscle cell contraction and differentiation. *In vitro*, this is typically achieved by electrical stimulation, mechanical stretching or light pulses. Each method, however, presents challenges in terms of generation of toxic by-products, invasiveness or limited applicability. A. Marino *et al.* now show that mild photothermal stimulation can also activate myotube contraction (*ACS Nano* http://doi.org/bznk; 2017).

To do so they use silica-gold core-shell nanoparticles that absorb in the nearinfrared (the ability to penetrate through deep layers of tissues makes the nearinfrared the spectral region of choice for nanomedical applications). When they incubate the myotubes with the nanoparticles and irradiate with nearinfrared light pulses, the authors measure a 5 °C increase in the local temperature. Concomitantly, they observe a large increase in myotube contraction compared with control cells subjected to irradiation but with no nanoparticles. In line with previous reports, they demonstrate that the enhanced contraction is related to an increase in the interaction between the two major protein filaments in the myotubes,



actin and myosin. This is a potential advantage over alternative treatments such as electro-stimulation, which works by modulating the cytosolic calcium levels and thus is associated with an increased risk of cell death due to an elevated intracellular concentration of this ion.

Additionally, under an extended heat treatment, during which cells are repeatedly irradiated with near-infrared pulses for five days, Marino *et al.* also observe the overexpression of heat shock proteins, which have been shown to protect tissues from apoptotic damage and to promote mitochondria biogenesis. The authors suggest that these observations could be used to design a wireless system for switching on muscle contraction *in vivo* while boosting the defences against apoptosis and skeletal muscle loss.

The confocal fluorescence image depicts the cytoplasmic localization of the core-shell nanoparticles (white) in C2C12 muscle cells; cell nuclei are shown in blue, and the actin and myosin filaments in red and green, respectively.

### CHIARA PASTORE