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OPTICAL PHYSICS

How to hide in time

As if the idea of a device that makes an object seem invisible was not mind-boggling enough, researchers have now demonstrated a system that can conceal an event in time. SEE LETTER P.62

ROBERT W. BOYD & ZHIMIN SHI

An exciting development in optical physics has been the proposal[1,2] and subsequent demonstration[3–6] of a spatial cloak, a structure that can render invisible any object placed in a specific region of space. Writing in this issue, Fridman *et al.*[7] (page 62) extend this concept by demonstrating a temporal cloak — a device that hides events occurring during a specific time window.

Let us first describe the operation of a spatial cloak. One example of such a device consists of a shell that surrounds the object to be hidden[2]. Using a method known as transformation optics, the way in which the refractive index changes across the material that constitutes the shell is set such that any light ray incident on the shell is deflected so as to miss the object to be hidden. The ray is redirected so that, when it leaves the shell, it is travelling in the same direction as if both the shell and the object hidden inside had not been present at all.

The experimental realization of spatial cloaking is intimately related to the development of optical metamaterials[8]. These are artificial materials with highly controllable optical properties that can be very different from those of naturally occurring materials. A prime example is a metamaterial designed to have a negative refractive index so that it bends light rays in the opposite direction to that in which conventional materials do. So far, spatial cloaking has been realized in, for example, a cylindrical geometry at radio frequency[3] and 'carpet' geometries at infrared[4,5] and visible[6] wavelengths.

The concept of cloaking has been extended to cloaking in time by a recent theoretical treatment[9]. This work showed that a time gap can be opened in an optical wave by locally manipulating the speed of light such that the front and rear parts of the wave get accelerated and slowed down, respectively. Any event that occurs within the resulting time gap — in which no light is present — would be rendered invisible to someone monitoring the transmitted light wave. However, the presence of this time gap in the light intensity would be a clear indication that someone had tampered with the time history of the system. The gap can be closed by subsequently reversing the modification of the light's speed as it leaves the 'interrogation region' that is to be cloaked. In this way, the previously accelerated light gets slowed down and the previously slowed-down light gets accelerated. When the restored light reaches an observer, a continuous, uniform light field is observed, and there is no indication that some event has occurred.

In their experimental study of temporal cloaking, Fridman *et al.*[7] made use of time-lenses and dispersive media[10]. To understand the principle of a time-lens, we should recall that a conventional optical lens is a device that can cause an incident light beam to converge or diverge spatially. From a mathematical perspective, the spatial and temporal evolution of light are quite similar, and therefore the principle of a lens can be extended to a time-lens.

A time-lens modifies a light field's temporal, rather than spatial, distribution. An ideal time-lens changes the colour of the light field at different moments in time. This modified light field is then passed through a dispersive medium in which different colours of light travel at different velocities and therefore emerge from the medium with different time delays. When the system is properly designed, all the colours can be made to arrive at a given spatial point at the same time, or, by analogy with a conventional lens, they can all be 'focused' to the same point in time.

In their work, Fridman and colleagues used a split time-lens, which is a slight modification of a time-lens. This lens is composed of two half time-lenses, which are connected at their tips. The light passing through the first half of the split time-lens experiences a colour change in the opposite direction to that passing through the second half: the first half makes the light bluer and the second half makes it redder. Then, after passing through a dispersive medium — an optical fibre in the
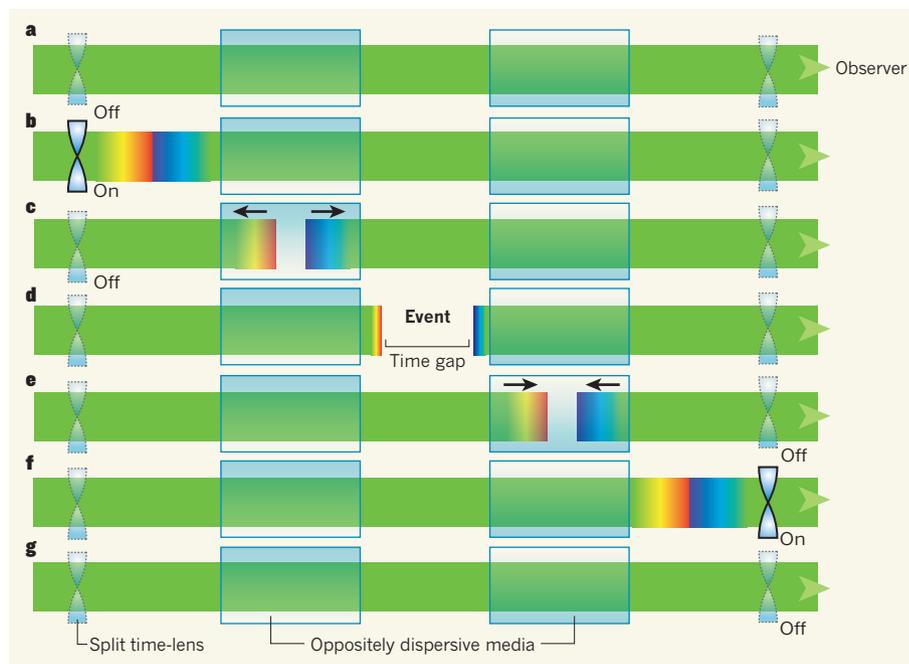

Figure 1 | Schematics of the temporal cloaking system of Fridman and colleagues[7] at different times. a, A continuous stream of green light passes through the system from left to right. **b,** The first time-lens is turned on and the light's colour changes as a function of time. **c,** The modified light travels through a dispersive medium. Because in this medium the blue-shifted light travels faster than the green, and the red-shifted light slower, a time-gap gradually opens up. **d,** The time gap is maximally open, and in its centre an event occurs in the form of a light pulse (not shown). **e,** The time gap gradually closes as the light passes through an oppositely dispersive medium from the first one. **f,** After the time gap is completely closed, a second time-lens is turned on such that all the colours are changed back to green. **g,** All the observer sees is basically a continuous green light as if the event in **d** never occurred.

authors' study — the light from the first half experiences a negative time delay (it accelerates) compared with the original green light, whereas that from the second half experiences a positive time delay (it slows down). This opens up a time gap of approximately 50 picoseconds in the transmitted light intensity. Afterwards, the time gap is closed seamlessly using similar techniques involving an oppositely dispersive medium from the first one and a second split time-lens (Fig. 1).

To demonstrate temporal cloaking in this system, Fridman *et al.*[7] created an 'event' in the form of a light pulse, at the centre of the time gap, that has a different frequency from that of the light passing through the system. The temporal cloaking is turned on or off by controlling the operation of the split time-lenses using additional laser light. The authors found that the detected signal associated with this event becomes more than tenfold weaker than the event's original signal. This result demonstrates that the event has been cloaked.

The distinction between temporal and spatial cloaking can be understood in terms of a metaphor involving automobile traffic. A spatial cloak acts like a junction in the form of a 'cloverleaf' interchange or flyover, in which the traffic is guided (by slip roads) to bend around a certain region of space. After passing through the junction, the traffic continues in the same direction as if the junction did not exist. By contrast, a temporal cloak behaves like a railway crossing. Traffic is stopped when a train passes, forming a gap in the traffic. After the train has passed the crossing, the stopped cars speed up until they catch up with the traffic in front of them, and the fact that a train has crossed the intersection cannot be deduced by observing the traffic flow.

Because spatial and temporal cloaking work in different physical dimensions — space and time, respectively — there is no fundamental reason why the two techniques cannot be combined so that full spatial–temporal cloaking could be turned on or off at will. Nonetheless, what Fridman *et al.* have demonstrated as a first unidirectional temporal cloaking device could already be useful in some applications, such as enhancing the security of communication in fibre-optic systems. Future directions may include increasing the cloaking time towards the order of microseconds to milliseconds, and building a device that can work simultaneously for incident light coming from different directions. ■

Robert W. Boyd *and* **Zhimin Shi** *are at the Institute of Optics, University of Rochester, Rochester, New York 14627, USA.* **R.W.B.** *is also in the Department of Physics and the School of Electrical Engineering and Computer Science, University of Ottawa, Ottawa, Ontario K2P 1A3, Canada.*
e-mails: boyd@optics.rochester.edu; zshi@optics.rochester.edu

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NEUROSCIENCE

Behavioural effects of cocaine reversed

Cocaine use causes lasting changes in behaviour by altering the strength of connections between neurons. The finding that these changes can be reversed in mice suggests strategies that could be used to treat drug addiction. SEE LETTER P.71

MARINA E. WOLF

Synaptic plasticity — the process by which connections (synapses) between nerve cells grow stronger or weaker depending on their activity level — is essential to normal development and learning. But synaptic plasticity also has a role in brain disease, including that resulting from drug abuse. Understanding this role is a challenging problem. Over the past decade, drug-addiction researchers have made progress towards this goal, aided by the fact that different facets of addiction can be modelled in animals and involve well-characterized brain circuits. From these studies, we know that drugs of abuse produce synaptic plasticity in the brain's 'reward circuitry' and that this contributes to addiction-related behaviours. On page 71 of this issue, Pascoli *et al.*[1] report that reversal of cocaine-induced synaptic plasticity in mice resets such behaviours to the pre-cocaine baseline.

Pascoli *et al.* studied synaptic plasticity associated with cocaine-induced behavioural sensitization — the increased behavioural response to a drug that occurs over the course of repeated administration and which persists long after drug exposure is discontinued. Even a single cocaine exposure in mice can cause sensitization to the drug's locomotor stimulatory effects (hyperactivity), thereby enhancing the locomotor response to a subsequent 'challenge' injection of cocaine. Opinions differ about the clinical relevance of sensitization, but according to one influential addiction theory, cocaine's incentive motivational properties (which make users want it) undergo sensitization[2].

The authors focused on part of the brain known as the nucleus accumbens and its major cell type, the medium spiny neuron (MSN). These neurons receive and integrate input signals — in the form of glutamate molecules — from cortical and limbic brain regions that control motivated behaviours, and then signal the motor circuitry to trigger a behavioural response (Fig. 1). There is evidence that glutamate synapses to MSNs are strengthened in cocaine-sensitized rodents[3], but exactly which synapses are strengthened, and how this relates to sensitization, is controversial.

To address these fundamental questions, Pascoli *et al.* gave mice a single injection of saline or cocaine, using enough of the drug to produce 'one-shot' locomotor sensitization. They took brain slices from the mice 7 days later, and used high-frequency stimulation (HFS; a series of electrical pulses) to produce long-term potentiation of glutamate synapses onto MSNs of the nucleus accumbens in the slices. Long-term potentiation (LTP) is a form of synaptic plasticity in which increased activity strengthens glutamate synapses, often through insertion into the neuronal membrane of additional glutamate receptors known as AMPA receptors. The authors found that the magnitude of HFS-induced LTP produced in cocaine-exposed neurons was approximately half of that observed in saline-treated controls. This could be explained if cocaine selectively eliminates HFS-induced LTP in a subpopulation of MSNs. But which one?

MSNs can be classified according to whether they express the D1 or D2 subtype of dopamine receptor (D1R or D2R). These subpopulations generally have distinct projection targets and different functions, although the distinctions are less clear in the nucleus accumbens than in the neighbouring dorsal striatum[4]. To distinguish between these subpopulations, Pascoli *et al.*[1] used transgenic mice that express green fluorescent protein in either D1R- or D2R-expressing MSNs. They thus observed that a single exposure to cocaine abolished HFS-induced LTP selectively in D1R neurons.

Crucially, the authors found that the abolition was not caused by impairment of mechanisms