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ATOPHYSICS

Tunnel vision

Jonathan P. Marangos

The tunnelling of a bound electron out of an atom in a laser field is a well-known quantum-mechanical process. But it happens very quickly, and it takes some fast work with X-rays and lasers to see it in action.

Advances towards making measurements with attosecond time resolution have caused much excitement in recent years. An attosecond is 10^{-18} s, and this is the timescale over which electron states in matter evolve. Such evolution drives many processes that are of interest to quantum scientists, among them chemical reactions, changes in material structures, optical responses and quantum coherence. Elsewhere in this issue, Ferenc Krausz and colleagues (Uiberacker *et al.*)¹ extend a technique that brings us within sight of these fast-moving phenomena.

Measurements in the attosecond domain are based on the use of high-intensity laser pulses that comprise just a few wave-cycles of electrical field and have a precisely defined form². This strong, oscillating electrical field can be used to extract an electron from an atom, in a quantum effect termed tunnel ionization, and then drive it back in with increased energy. This process is signalled by a short burst of coherent X-rays, typically a few hundred attoseconds long³, that is perfectly synchronized with the laser field's cycle. Such X-ray bursts have been used in various ways to investigate, for example, the movements of protons in small molecules⁴, and the decay of states in atoms where several electrons have been excited⁵.

Krausz and his colleagues at the Max Planck Institute for Quantum Optics in Garching, Germany, were pioneers of these methods. They use a precisely controlled laser field that has a cycle time of around 2,700 attoseconds to produce, in a gas jet, a synchronous X-ray burst some 250 attoseconds long and with a photon energy of around 90 electronvolts. This X-ray burst is delivered to the sample to be measured, followed, after a carefully controlled delay, by the original laser pulse. In previous experiments⁵, the researchers had clocked the emission time of X-ray-excited electrons with a precision of around 100 attoseconds by measuring the effect of the delayed laser field on the energy distribution. This set-up, termed attosecond streaking spectroscopy, worked well for excitation processes in which an electron is eventually ejected. But in many cases,

the electron does not leave the sample. A new technique was needed to measure the dynamics of these more deeply bound states.

In their latest paper (page 627)¹, the researchers describe how they have used the same basic tools to look into such deep states in neon (Ne) atoms (Fig. 1). The 90-eV photons of the X-ray burst had enough energy to free one electron from the atom immediately and excite a second to a higher, but still bound, energy level, creating an excited ionic state, Ne^{+*}

(Fig. 1a). When the subsequent laser electrical field is near a maximum, it can strongly distort the potential that binds the electron. The excited electron encounters a finite barrier through which, with high probability, it can tunnel to freedom⁶. An appropriate maximum of the laser field occurs twice per optical cycle (Fig. 1b). The signal of Ne^{2+} ions thus accumulates over time in steps.

The accumulated yield of Ne^{2+} ions depends on the delay between the X-ray burst and the laser pulse. The number of field maxima that can contribute to the Ne^{2+} signal, and so the Ne^{2+} yield, changes in steps as the delay is varied. The steepness of the steps indicates the duration of the tunnel ionization. This behaviour shows that the X-ray excitation and ionization processes of these excited states are indeed very rapid, and must occur in a combined time of less than 300 attoseconds. Although this temporal confinement of the laser-induced ionization had long been suspected, it had not been directly confirmed before. The measurements also show that tunnel ionization becomes the principal ionization process at surprisingly low intensities.

Because the tunnelling from the bound states is so rapid and so exactly synchronized to the X-ray burst, the yield of ions from this process is a suitable probe of their electron dynamics.

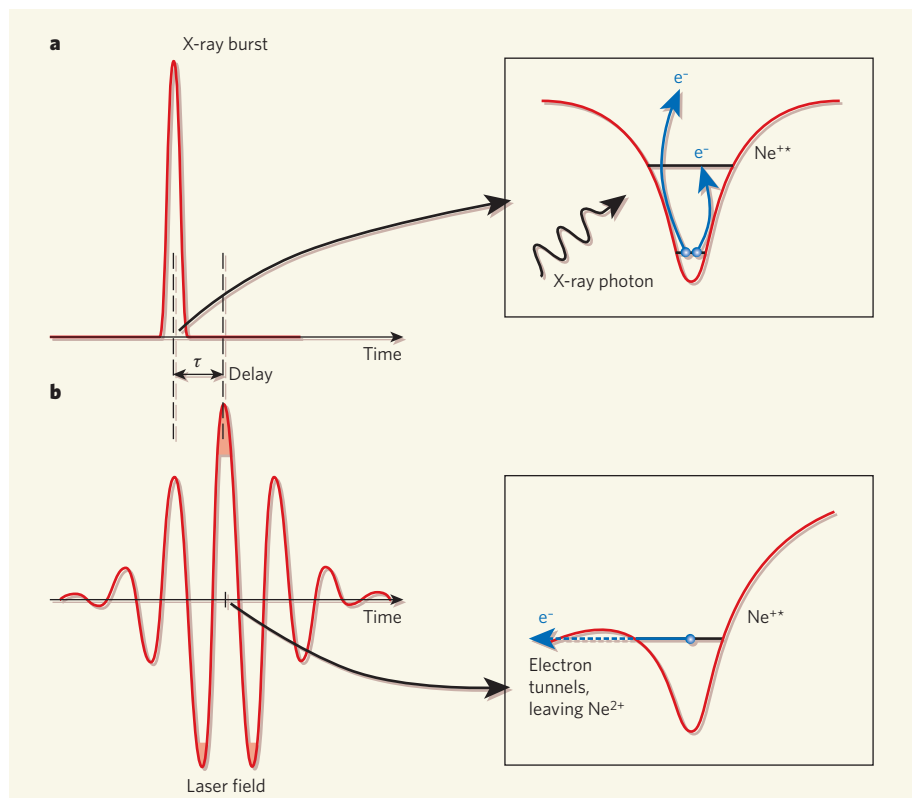


Figure 1 | Attosecond probe. In the experiments of Uiberacker *et al.*¹, the X-ray burst and the laser electrical field have a relative delay, τ , between their centres that can be varied. **a**, The X-ray burst excites two electrons in a neon (Ne) atom. One of these leaves directly, and the other is excited into a bound state of the Ne^+ ion, Ne^{+*} . **b**, The following laser field can, when near one of its maxima (in one of the shaded regions), significantly perturb the Ne^+ ion potential, allowing the second electron to tunnel away quantum-mechanically, resulting in a significant degree of ionization to Ne^{2+} . As the delay τ is changed, the X-ray burst scans across the laser-field profile. The number of field maxima that can contribute to the Ne^{2+} signal, and so the Ne^{2+} yield, changes in steps as τ is varied.

The authors argue that further measurements with improved precision will allow tunnelling to be characterized more fully. Such measurements will require X-ray pulses significantly shorter than those of a few hundred attoseconds that are presently available. Once this hurdle has been surmounted, the tunnelling method could be used to probe how electrons behave in a wide range of atomic and molecular systems with attosecond precision.

In the meantime, Uiberacker *et al.* turn their attention¹ to the significantly slower processes associated with the decay of excited states in the xenon ions Xe²⁺ and Xe³⁺. Using their laser-field tunnelling method to produce Xe⁴⁺ ions, they have been able to measure cascaded

population transfer processes among the electron states of ions that have lifetimes of as little as 6 femtoseconds (6×10^{-15} s). They thus already show the potential of their method to extend and increase the precision of findings established in earlier spectroscopic studies. ■

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PLANT BIOLOGY

Sticking with auxin

Tom Guilfoyle

Auxin is one of the main agents that regulate plant growth and development. Intricate crystallographic studies reveal how this hormone acts as a 'molecular glue' in mediating substrate–receptor interactions.

Research into plant hormones has been coming on apace in the past few years. The receptors for these hormones that have been identified, including that for auxin, have turned out to be surprisingly different from the receptors for animal hormones¹. On page 640 of this issue, Tan *et al.*² take the story further — their first-of-a-kind crystallographic studies provide

more revelations about auxin perception and the auxin receptor.

In 2005, a receptor for auxin (or indole-3-acetic acid, IAA) was identified as the F-box protein TIR1, short for 'transport inhibitor response 1'; TIR1 is a component of a cellular protein complex known as SCF^{TIR1} (refs 3, 4). The substrates for TIR1, Aux/IAA

repressors, are recruited to the receptor in an auxin-dependent manner and, after binding to TIR1, are degraded. Identification of the TIR1 receptor suggested that auxin perception and the signalling pathway to auxin-regulated gene expression was direct and simple, but it left various questions. Tan *et al.*² now describe crystal structures of a TIR1 complex that reveal how auxins fit into a surface pocket of TIR1 and enhance the binding of Aux/IAA repressors to TIR1.

Auxin-regulated gene expression triggers most of the processes controlled by this plant hormone. Many auxin-induced genes are regulated by the interplay of two classes of gene-transcription factors, auxin-response factors (ARFs) and the Aux/IAA repressors⁵ (Fig. 1). ARFs bind to auxin-response promoter elements in auxin-response genes. When auxin concentrations are below a threshold level, Aux/IAA repressors associate with ARF activators and repress the expression of these genes. Conversely, higher levels of auxin lead to destruction of the Aux/IAA repressors, and to activation of the genes^{6,7}.

Aux/IAA repressors contain four conserved domains, and one of these, domain II, is responsible for the instability of these proteins (Fig. 2a, overleaf). Domain II has a hallmark GWPPV amino-acid motif that is recognized by TIR1 in the SCF^{TIR1} complex^{8,9}. IAA binds to TIR1 to enhance the recruitment of the motif to TIR1, as do two synthetic auxins — 1-naphthalene acetic acid (1-NAA) and 2,4-dichlorophenoxyacetic acid (2,4-D, which is used as a herbicide) — but with different affinities (Fig. 2b).

Tan *et al.*² expressed TIR1 complexed with ASK1 (a SCF^{TIR1} adaptor) in a baculovirus–insect system. After demonstrating that auxin enhanced the binding of an Aux/IAA protein to the complex, they obtained crystal structures for the complex alone and for complexes bound to IAA and the two synthetic auxins along with an Aux/IAA peptide containing the GWPPV motif (Fig. 2). The crystal structures showed that the TIR1–ASK1 complex had a mushroom shape, with the leucine-rich-repeat domain of TIR1 forming the cap, and the F-box of TIR1 along with ASK1 forming the stem. A pocket on the top of the TIR1 leucine-rich-repeat domain functions in both auxin binding and substrate recruitment.

It turns out that auxin binds to the bottom of the pocket in a 'promiscuous' binding site that tolerates moderately different planar ring structures (that is, natural and synthetic auxins). The Aux/IAA peptide binds in close proximity to the auxin-binding site in the upper part of the pocket. The GWPPV motif is packed directly against auxin and covers the auxin-binding site. This is thought to trap auxin in the binding pocket until the Aux/IAA peptide is released and moved along the degradation pathway. The crystal structure also revealed an unexpected moiety in TIR1, which turned out to be a tightly bound inositol hexakisphosphate (InsP₆) molecule. InsP₆ functions in many

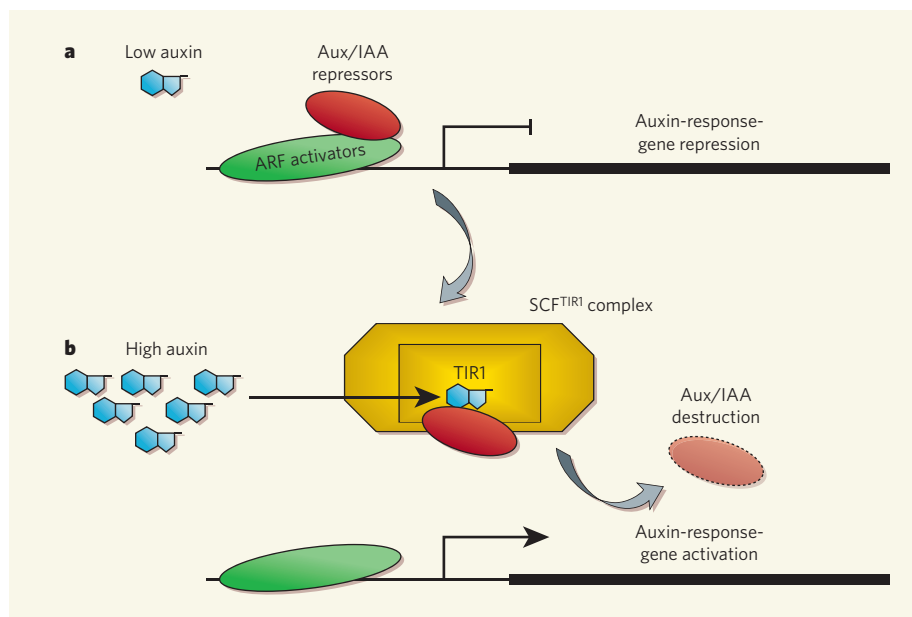


Figure 1 | The auxin signalling pathway. ARF activators bind to auxin-response elements in promoters of auxin-response genes. **a**, When auxin concentrations are low, Aux/IAA repressors associate with the ARF activators (via domains III and IV, see Fig. 2) and repress expression of the genes. **b**, When auxin concentrations increase, auxin binds to the TIR1 receptor in the SCF^{TIR1} complex, leading to recruitment of the Aux/IAA repressors to TIR1. Once recruited to the SCF^{TIR1} complex, the repressors enter a pathway that leads to their destruction and the subsequent activation of the auxin-response genes.