

Giving a boost to atoms

Kristian Helmerson

Matter waves can now be amplified in the same way that a laser amplifies light. Such matter-wave amplifiers will be essential for future developments in atom optics.

It is difficult to imagine modern life without amplifiers. Whether your stereo is faithfully reproducing a Mozart concerto, or your cell phone is boosting a wireless call, amplifiers are used widely in daily life. Amplifiers, as the name suggests, amplify or enhance a signal. Typically, the signal is a wave, such as an oscillating electrical current, a radio wave or a beam of light. Quantum mechanically, matter can also be regarded as waves. Hence it seems reasonable to imagine that, like other waves, matter waves can be amplified. Such direct amplification of atomic waves has now been observed by a group at MIT. On page 641 of this issue, Inouye *et al.*¹ report phase-coherent matter-wave amplification using a Bose–Einstein condensate.

According to the quantum or wave-mechanics picture of matter, atoms have a wavelength that depends inversely on their speed. For atoms at room temperature, this wavelength is less than a nanometre. In addition, the atoms zip around in all directions and the probability that two atoms are in the same place, at the same time, with the same velocity, is very small. This makes it difficult to observe the wave properties of atoms in everyday experience. A Bose–Einstein condensate is a quantum gas in which many of the particles share the same quantum state²; that is, they are in the same place, at the same time, with the same velocity. So a Bose–Einstein condensate behaves as a macroscopic wave. At the ultracold temperatures (tens of nanokelvins) characteristic of a Bose–Einstein condensate, the atoms' wavelengths are tens of micrometres and can be observed with conventional optics. Such a large number of atoms having the same long wavelength makes a Bose–Einstein condensate the ideal system in which to demonstrate matter-wave amplification.

The basic function of an amplifier is to take a wave (the input) and then produce a more intense, but otherwise identical, wave (the output). In practice, an amplifier has a gain medium where there is a transfer of external energy (the pump source) to the output according to the input. The amount by which the output increases compared to the input is the gain of the amplifier. In an optical amplifier, or laser, the gain medium is a collection of atoms or molecules (gas, liquid or solid) with energy levels such that a transition between them is at the wavelength of light to be amplified. The pump source

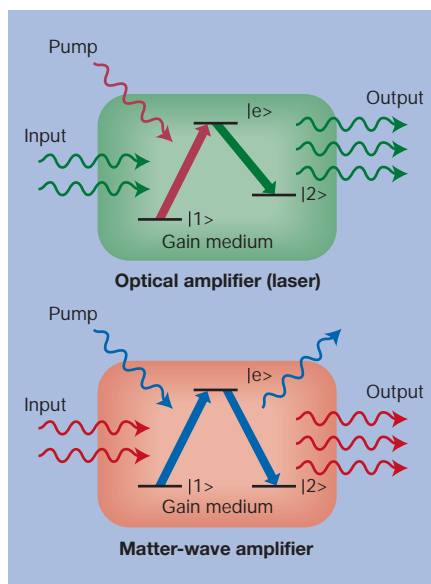


Figure 1 Optical waves or matter waves? In an optical amplifier or laser, the gain medium consists of atoms or molecules with at least three energy levels. Atoms in $|1\rangle$ are transferred to $|e\rangle$ by a pump source, which could be another laser. The input wave stimulates the atoms to transfer to $|2\rangle$ and emit a photon. The emitted photon is indistinguishable from the input photons. So a more intense, but otherwise identical, optical wave emerges from the output. In the matter-wave amplifier, three distinct energy levels or states of the atoms are required. Unlike light, the atoms in the matter-wave amplifier cannot simply be created, so the gain medium also serves as a reservoir of atoms. After absorbing a 'pump' photon, an atom in excited state $|e\rangle$ can emit a photon and end up in a different state (such as $|2\rangle$ with a random phase). But in the presence of the input matter wave the excited atom is more likely to end up in $|2\rangle$ with the same velocity and phase as the input atoms. So a more intense, but otherwise identical, matter wave emerges from the output. Amplification will end when all the atoms in the gain medium have been transferred to $|2\rangle$.

transfers some of the atoms into the higher of the two energy levels. The input wave then stimulates this population to transfer to the lower energy level, emitting radiation in the same direction and at the same wavelength as the input wave (Fig. 1). This process is called stimulated emission, and explains the name laser: light amplification by stimulated emission of radiation.

The matter-wave amplifier works on the same principle of stimulated emission, but with stimulated emission of atoms, not radiation. Unlike light, atoms cannot be created out of a vacuum, so you need a reservoir of atoms to be emitted. Inouye *et al.*¹ prepared their input matter wave using optically induced Bragg diffraction³ of a Bose–Einstein condensate. This is a process whereby atoms are exposed to two laser beams with slightly different frequencies. The atoms, initially at rest, absorb a photon from the higher frequency beam and emit into the lower frequency beam. The result is that the atoms get a momentum kick equal to the difference in momentum of the two beams, which is given by their wavelength and relative directions. Hence an input matter wave is produced which recoils in a chosen direction.

Amplification of the input wave is achieved by 'pumping' the Bose–Einstein condensate with an intense pulse from one of the lasers used for Bragg diffraction; here the condensate is behaving as the gain medium (Fig. 1). Some of the condensate atoms absorb a photon from the laser beam, and if they 'see' the input matter wave, before they can radiate the absorbed photon by spontaneous emission, they will be induced to emit photons along the direction of the other laser beam. As a result, the recoiling atoms will have the same momentum (the same direction and wavelength) as the input wave. In other words, the input wave has been amplified by stimulated emission, producing a larger output wave from the pumped gain medium. Inouye *et al.* observe typical gains of 10 to 100, which means there are 10–100 times more atoms in the input pulse after amplification.

An amplifier that can produce an output wave that is more intense than, but otherwise identical to, the input wave is called phase coherent. This distinguishes such amplifiers from those that simply increase the strength of the output. This might be acceptable if you simply wanted a high-power light beam to cut material. But phase-coherent amplification is important in applications that are phase sensitive, such as interferometry or phase-modulated signal transmission. Inouye *et al.* used interferometry to check the phase coherence of their amplifier. They used optically induced Bragg diffraction to produce a reference-

matter pulse identical to the input pulse. By looking at the interference of this reference pulse with the amplified pulse, the researchers verified that the process was phase coherent.

Previous studies have observed stimulated emission without seeing phase coherence. The ability of optically pumped condensate atoms to serve as a gain medium for matter-wave amplification was first observed by the MIT group⁴. They showed that a pumped condensate, initially radiating photons by spontaneous emission, had sufficient gain to amplify emission in a random direction. This process is analogous to the amplified spontaneous emission of light in atomic vapours. Stimulated emission of atoms is also demonstrated by four-wave mixing of matter waves⁵. In this case the stimulated emission of atoms is mediated by the mean-field interaction of the atoms in a Bose–Einstein condensate, and not by pumping from an optical field.

The demonstration by Inouye *et al.*¹ that matter waves can be amplified and maintain their phase is an exciting development in the rapidly growing field of matter-wave optics. Already, there are reports of similar experiments from researchers at the University of Tokyo (M. Kozuma, personal communication). But the task is not easy. Inouye *et al.* estimate a gain of four for phase-coherent amplification (compared to a number gain of 10–100). Atoms, unlike photons, can interact with each other directly, leading to distortion of the amplified matter wave. Nonetheless, such experiments are perhaps the closest matter-wave equivalent of the optical laser. The first generation of ‘atom lasers’⁶ was based on the coherent extraction of a beam of atoms from a Bose–Einstein condensate. But some argued that the analogy with optical lasers is incomplete because you cannot really amplify atoms. The

demonstration of phase-coherent matter-wave amplification by stimulated emission of atoms is much closer to a true atom laser.

It is difficult to imagine the future impact of such studies — few would have predicted the success of the modern laser. The earliest optical lasers were clunky devices that languished in labs for years before anyone found a use for them. But a coherent matter wave is inherently more difficult to work with than a beam of light. For example, there is no material equivalent to glass that a beam of atoms can readily pass through, and the atoms need to be maintained in an ultrahigh vacuum. But there may be times when matter waves are better than laser light. For instance, phase-coherent optical amplifiers are used in optical interferometer gyroscopes to measure gravity accurately. Matter-wave interferometer gyroscopes are already more sensitive than their optical counterparts⁷, but could be further improved by incorporating a matter-wave amplifier. Phase-coherent optical amplifiers are also used to boost the output of a laser, as in fibre-optic communications. Similarly, a phase-coherent matter-wave amplifier could be used to boost the output of an atom laser, for example in atom lithography. Such amplification of matter waves may be the future of better and brighter atom lasers. ■

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Alzheimer’s disease

The tangled tale of tau

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Alzheimer’s disease is the main form of dementia in today’s ageing population. It is characterized by abnormal protein deposits in the brain, such as the extracellular amyloid plaques and the intracellular neurofibrillary tangles. The tangles are made up of a protein called tau, which is modified by phosphorylation and aggregated into so-called paired helical filaments. Because hyperphosphorylation seems to occur before aggregation, it is considered one of the earliest signs of neuronal degeneration. But which protein kinases are responsible for it?

Two papers in this issue go some way

towards answering this question. Tsai and colleagues¹ (page 615) report that the cyclin-dependent kinase 5 (Cdk5) is deregulated in the brains of people with Alzheimer’s disease. Not only that, but Cdk5 also hyperphosphorylates tau and accumulates in tangle-bearing neurons. And Greengard and colleagues² (page 669) show that Cdk5 can alter signalling processes in neurons by altering the balance between its kinase and phosphatase activities.

The tau protein is associated with neuronal microtubules. It stabilizes them and regulates the transport of vesicles or

organelles along them, supports the outgrowth of axons and serves as an anchor for enzymes³. The human central nervous system contains six isoforms of tau, generated from a single *tau* gene by alternative splicing. Embryos contain only one (the smallest) isoform; the larger isoforms are induced only when neurons differentiate and cell processes (axons and dendrites) are generated. Tau is a basic protein, rich in serine and threonine residues, and it can be phosphorylated by many kinases. A common hypothesis is that phosphorylation weakens the affinity of tau for microtubules. As a result tau detaches and then aggregates, eventually leading to neuronal degeneration (Fig. 1).

Which kinase initiates this chain of events? Proteins related to the cell-cycle kinase Cdc2 came under suspicion early on for several reasons. First, tau from the brains of patients with Alzheimer’s disease is phosphorylated at serine or threonine residues followed by a proline. This points to the activity of a ‘proline-directed’ kinase, which includes members of the Cdc2 family, but also other kinases such as the mitogen-activated protein kinases or GSK-3. Second, one Cdc2-family kinase, Cdk5 (also known as NCLK, for neuronal Cdc2-like kinase), is one of two main tau-protein kinases isolated from brain tissue (the other being GSK-3b⁴). Third, proline-directed phosphorylation of tau increases strongly during mitosis, and correlates with other markers of mitosis⁵. This is consistent with the idea that degeneration could be the response of a differentiated neuron to inappropriate mitotic signals, which forces that neuron to commit suicide (apoptosis). Indeed, inhibitors of Cdc2-like kinases can prevent an apoptotic response⁶.

Arguments against this circumstantial evidence include the fact that proline-directed phosphorylation of tau does not strongly influence its functions (binding to microtubules, for example) compared with the effects of other kinases such as the micro-

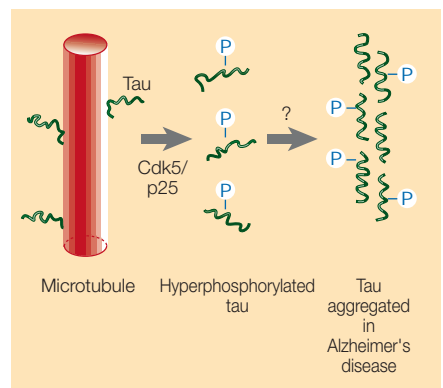


Figure 1 Hyperphosphorylation and aggregation of tau. Phosphorylation of tau is thought to weaken its affinity for the microtubules to which it normally binds. Tau then detaches and forms the intracellular aggregates characteristic of Alzheimer’s disease.