

PHYSICS

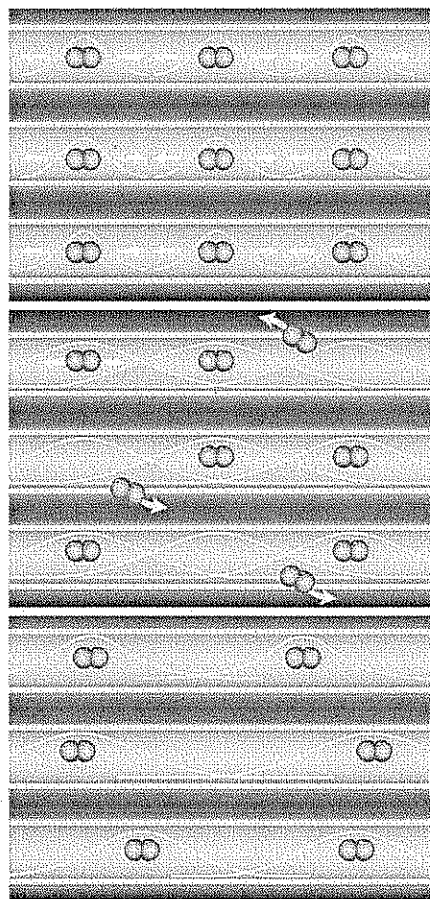
Improving Correlations Despite Particle Loss

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Correlations arise in systems where the behavior of any given particle depends strongly on all the other particles. Such correlations help to distinguish relatively simple gases (which are largely uncorrelated systems; over time, the average position of an atom in a gas does not depend much on the other atoms) from more complicated condensed matter systems. In traditional condensed materials, correlations arise from two effects: interactions (for example, the charge of electrons causes them to repel each other) and quantum statistics (for example, fermionic particles such as electrons cannot be in the same state, as stated in the Pauli exclusion principle). Yet, on page 1329 of this issue, Syassen *et al.* (1) show that in dilute quantum gases, correlations can arise through another mechanism: inelastic loss of particles from the system.

This approach uses the unusual properties of ultracold atoms that are held in optical lattices, standing waves of light created by interfering laser beams. Collisional loss allows pairs of colliding particles to escape the traps, leading to particle correlations. Despite being less dense than typical solids by a factor of 1 billion, gases of ultracold atoms provide a way to realize and study “condensed” matter physics (2). Trapped in a vacuum with magnetic or optical fields, these weakly interacting dilute gases can serve as nearly perfect realizations of some solid-state models and may help us understand phenomena such as high-temperature superconductivity or exotic quantum magnets. In addition, they present opportunities unavailable in traditional materials, such as the ability to precisely engineer lattice potentials to trap the atoms and to directly control the strength of atom-atom interactions.

Another important feature of trapped atom systems is that they can undergo inelastic atom loss, in which two or more particles collide to form a molecule, kicking those particles out of the trapped gas. Particles converging on the same point in space can undergo inelastic collisions and are effectively



annihilated—they disappear from the trap.

Collisional losses are normally considered undesirable for studying many-body physics because they can lead to heating and decay. Losses, however, depend on the positions of other particles, and loss rates have been used to measure two-particle correlations in a Bose gas of ^{87}Rb atoms (3). Syassen *et al.* point out that these losses can generate correlated states: The remaining atoms are more likely to be arranged to avoid each other. However, this effect alone would not be enough to generate stable correlated states, because the remaining separated particles could continue to adjust their positions, and would find and collide with other atoms until all atoms had decayed away.

Quantum mechanical systems can evolve in ways counter to our classical intuition, and

As a trapped quantum gas is slowly set free, pairs of colliding molecules escape, but these collisions actually help slow further losses.

Making gains out of losses. (Top) Molecules initially isolated from one another in a lattice are released along one direction and are allowed to spread out and interact with their neighbors. (Middle) When the molecular wave functions spread out enough for neighboring atoms to come in contact, strong inelastic loss of particles can occur. (Bottom) Counterintuitively, this strong loss can actually stabilize the system against further loss.

there is a quantum effect that can stabilize the system if the loss rate is much greater than the rate at which atoms can move from point to point in the gas. In the quantum Zeno effect, processes that strongly perturb a given state tend to inhibit the system from evolving into that state. It's as if the mere possibility of strong losses of colocated atoms tends to prevent separated atoms from moving together and annihilating. In the context of cold atoms, the quantum Zeno effect has been proposed as a means to stabilize a quantum “register” of atoms in an optical lattice (4).

Syassen *et al.* point toward the broader application of creating correlated many-body states. They first trapped an array of individual Rb_2 molecules into the separate sites of a three-dimensional (3D) optical lattice, which creates a correlated state (this state resembles a low-density crystal; see the figure, top panel). By decreasing the strength of the trapping lattice along just one direction, they converted the rubidium system into a 2D array of 1D gases (see the figure, middle panel). The previously isolated Rb_2 molecules were then free to move along the wires and annihilate each other (see the figure, bottom panel).

As these dynamic processes took place, Syassen *et al.* measured substantially lower particle loss than would be expected for an uncorrelated system, which shows that the particles are correlated—they avoid each other. More important, as particles disappeared, the loss rate slowed more than would be expected from the decreased number of atoms alone, which indicates that these loss processes further increase the correlations of the remaining atoms. This interpretation is supported by the authors' theoretical description of the system.

This inherently dynamic many-body problem presents some interesting theoretical

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questions. To what extent can temperature be defined, and how do the final states depend on the initial conditions? Is the approach taken here, in which the system starts in a correlated state, different from one starting with a completely uncorrelated system and then suddenly turning on loss processes? The authors' numerical calculations indicate that the system forgets any details of its initial state quickly, and it will be interesting to further elucidate the details.

The work of Syassen *et al.* represents a first step in solving the experimental chal-

lenges to making this collision process generally useful. This technique could be used to produce other interesting states, such as fractional quantum Hall states (5) and systems that exhibit anyonic statistics (6). Here, the particle correlations create composite particles that have fractional electron charge or obey "fractional" quantum statistics somewhere between those of a boson and a fermion. Even in the absence of these applications, the present work opens up the experimental and theo-

retical study of a new class of interesting dynamic problems.

References

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MOLECULAR BIOLOGY

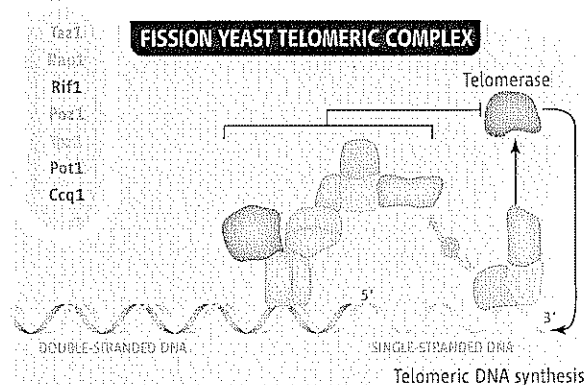
Refined View of the Ends

Alessandro Bianchi and David Shore

The linear genomes of eukaryotes pose specific problems for the protection and replication of DNA ends, or telomeres. These challenges were overcome by the evolution of a specialized protein apparatus that, together with the short DNA repeats that recruit these proteins to chromosome ends, forms the telomeric complex (1, 2). On page 1341 of this issue (3), Miyoshi *et al.* identify new protein components of the fission yeast telomeric complex and offer new insights into telomere function and evolution.

In yeasts and mammals, the short but highly repeated DNA motif synthesized by the telomerase enzyme is partly single-stranded, terminating in a 3'-OH overhang (enriched with the nucleotides thymine and guanine) at the tip of the telomere. Consequently, both double-stranded and single-stranded telomeric DNA binding proteins have evolved. Both sets of proteins protect chromosomes and regulate telomerase activity. The emerging picture is that the double-stranded binding factors repress telomerase through a mechanism that depends on the number of bound molecules, which is linked to telomere length; overhang-binding proteins participate in telomerase activation.

The core mammalian telomeric complex is called shelterin, for its role in telomere protection (4). It is delivered to telomeres by two of its components, telomeric repeat binding factor 1 (TRF1) and TRF2. TRF2 recruits an additional factor, repressor activator protein 1 (RAP1). The mammalian overhang-binding



Fission yeast telomeric complex. In this model, the Pot1-Tpz1-Ccq1 complex promotes telomerase activity when bound directly to the 3'-OH overhang, but not when recruited to the Taz1-Rap1 complex by Poz1. The extent of sequestration of the Pot1-Tpz1-Ccq1 complex depends on the number of telomere-bound Taz1-Rap1 molecules, which in turn is dictated by telomere length.

unit is a heterodimer of the protein protection of telomeres 1 (POT1) and TPP1 (previously known as TINT1/PTOP/PIP1). A sixth factor, TRF1-interacting nuclear factor 2 (TIN2), completes the core complex. Of the two well-studied yeast model systems, *Saccharomyces cerevisiae* and *Schizosaccharomyces pombe*, only the latter has orthologs of TRF1/TRF2 (called Taz1) and POT1.

Using a biochemical approach, Miyoshi *et al.* identify the Pot1-interacting partner in *S. pombe* (the TPP1 ortholog, called Tpz1). They also reveal two additional components of a Pot1 complex localized at telomeres: Poz1, a small protein of unknown function and with no obvious homologs, and coiled-coil quantitatively enriched protein 1 (Ccq1), whose multiple functions include heterochromatin formation (5), meiosis (6), and, as now reported, telomerase regulation (3). Tpz1, like its partner

Studies in fission yeast have expanded the known repertoire of proteins that assemble at chromosomal ends to control telomere function.

Pot1, is essential for telomere protection, and for Poz1 and Ccq1 association with the telomere. Poz1 in turn interacts with both Rap1 and Tpz1, and thus has an important architectural role in bridging the Taz1-Rap1 complex with Pot1-Tpz1 (and Ccq1). The set of mapped interactions is reminiscent of those described for the human shelterin complex, raising the question of whether Poz1 might be a TIN2 homolog. One important difference is that Poz1 in *S. pombe*, unlike TIN2 in mammals, interacts with Rap1.

Although loss of Pot1 or Tpz1 in fission yeast results in telomere deprotection and cell death, mutant cells lacking either Poz1 or Ccq1 grow normally. However, Poz1 and Ccq1 do protect telomeres, as the double mutant is inviable and loses telomere sequences rapidly. Both proteins also regulate telomerase, but in opposite ways: Poz1- and Ccq1-deficient cells have elongated and shortened telomeres, respectively. One attractive hypothesis is that the Pot1-Tpz1-Ccq1 complex (possibly together with Poz1) forms a subcomplex at telomere overhangs that recruits and activates telomerase (see the figure). This agrees with recent findings that human POT1-TPP1 interacts with telomerase *in vivo* and stimulates its activity *in vitro* when bound to its DNA substrate (7, 8). Importantly, Miyoshi *et al.*, demonstrate that Tpz1 immunoprecipitates telomerase activity from yeast cells, in a Ccq1-dependent manner.