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trinsically dim ones. A program scheduled for the Hubble Space Telescope will obtain color photometry of the stars in the center of both remnants (*13*), and strong constraints will be placed on the companion properties, even if nothing is detected.

If the material accreted by the white dwarf is neither H nor He, C + O from a disrupted C + O white-dwarf companion is another possibility. This alternative, known as the double-degenerate scenario, involves the progressive approach of two white dwarfs orbiting around the center of mass of the system while they emit gravitational wave radiation. The less massive white dwarf is disrupted in the process, forming a torus of material around the most massive one. The accretion of this mass by the surviving white dwarf could cause its explosion. The lack of detection of any surviving companion could eventually confirm that it is destroyed in the course of the binary evolution, as expected in the merging of C + O white dwarfs. Such a confirmation would establish this scenario as the right evolutionary path. Some objections have been raised, however: fine tuning in the accretion process might be required to avoid the burning of C into Ne and Mg, which would lead to a collapse event instead of an explosion (*14*). The final accretion resulting from merging still needs careful numerical evaluation to ascertain the final result of the double-degenerate scenario.

Transfer of H or He to the white dwarfs in the single-degenerate scenario differs from the C + O transfer in that it opens up more possibilities of explosion: if the H-accreting white dwarfs were the right evolutionary path to type Ia supernovae, they could undergo explosions starting at the edge and propagating toward the center well before the white dwarf could reach the Chandrasekhar mass. The explosion of Chandrasekhar-mass white dwarfs gives a successful account for the common type Ia supernovae, as shown by a model proposed by Nomoto, Thielemann, and Yokoi (*15*). But explosions that happen before reaching such a mass are found in the calculations where a He detonation is triggered in the external layers (*16*). Although they might not respond to the common type Ia phenomenon, they could correspond to very dim ones (*17*). A mixture of almost standard Chandrasekhar explosions with some very faint "peculiar" sub-Chandrasekhar explosions could exist. A few extremely faint type Ia explosions have been identified, in any case: The last supernova of type Ia that exploded in the Andromeda galaxy, in 1885, was of such a type. On the other hand, the evolutionary path toward explosion will not be directly reflected in the spectrum of the exploded white dwarf itself.

Whatever the companions to those supernovae might be, their emission has been obscured by the overwhelming luminosity of the exploded white dwarf. Despite the difficulties, however, important physical understanding will eventually arise: Pairs of white dwarfs should be merging in the universe, and one wonders what kind of object results. If compact objects can arise in this way (*13*), they should contribute to the population of neutron stars. If an explosion is obtained, many things will be explained to the satisfaction of those who searched and saw nothing: The merging of two white dwarfs will not give any H signatures nor a strong radio or x-ray emission after the explosion. It will not leave behind any star that can be observed afterward. These reasons tempt people to bet on them for type Ia supernovae. On the other hand, there are many loose ends that need tying up, and the outcome of the single-degenerate pairs and their possible relation with type Ia supernovae will also eventually be clarified. Perhaps nature chooses more than a single evolutionary path toward stellar explosions.

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NUCLEAR TRANSPORT

Whose Finger Is on the Switch?

David S. Goldfarb

Having successfully infected a cell, viruses co-opt the cell's own intracellular processes to replicate. Often key cellular reactions, these co-opted functions are also of interest to molecular biologists. One such process is nuclear transport—the mechanism by which cells move macromolecules into and out of the nucleus. In a report on page 1842 of this issue, Richards *et al.* (*1*) illuminate the mechanism of nuclear transport, describing its control by the small guanosine triphosphatase (GTPase) Ran. And in a related paper on page 1845, Her *et al.* (*2*) report that vesicular stomatitis virus (VSV) shuts off host transport with a flick of the Ran switch. Ironically, an efficient switching mechanism, evolved to control nuclear transport, creates an Achilles' heal susceptible to viral attack.

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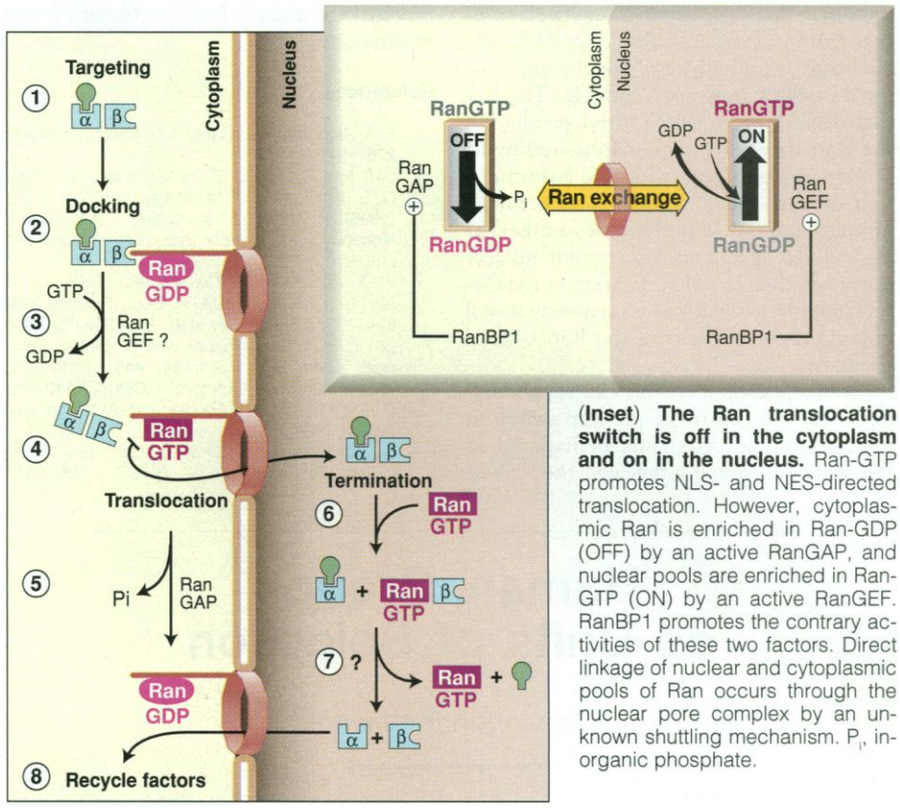
Pores arrayed in the nuclear membrane form multiple gateways in and out of the nucleus. Various cargo are delivered from the nucleoplasm and cytoplasm to the nuclear pore complex by distinct targeting pathways (*3*). The best understood targeting pathway is nuclear localization signal (NLS)–directed protein import (see the figure). Cargo with NLSs is targeted to the nucleus by soluble factors (α and β) that promote docking at sites adjacent to the pore complex and subsequent translocation through the pore. Targeting pathways for different classes of cargo use both common and distinct transport factors (*4*). Export from the nucleus is also mediated by multiple pathways. Nuclear export signal (NES)–directed protein export is distinct from the export of mRNA (as heterogeneous nuclear ribonucleoprotein), tRNA, and rRNA (as ribosomes), each of which apparently occurs by a distinct pathway (*3*). Few components of any export apparatus are

known. Now Richards *et al.* (*1*) show that the GTPase Ran is a common factor required for the translocation of cargo in both directions.

Like other GTPases that control protein targeting Ran is a molecular switch. Although Ran-GDP is likely to have its own activities (for example, to promote docking) Ran-GTP is considered the active or "on" form because it promotes translocation. Initially, Ran-GDP binds at or near NLS-cargo docking sites situated on filaments that protrude from the pore complex into the cytoplasm (*5*). A possible mediator of Ran-GDP function is the small Ran-GDP binding factor NTF2/P10 (*6*). The conversion of (docked) Ran-GDP to Ran-GTP by an unidentified cytoplasmic guanine nucleotide exchange factor (RanGEF) likely throws the Ran-GTP conformational switch and triggers translocation. (Identification of this RanGEF will tell us much about the switch control mechanism.) So the Ran switch performs a general gatekeeping function, determining when NLS cargo is admitted to the translocation channel. The timing of the switch is crucial, and docked Ran must initially be in the GDP form. In fact, high cytoplasmic levels of Ran-GTP prevent the formation of the NLS cargo targeting complex and abort transport before docking. On the other side of the nuclear envelope, Ran-GTP terminates import in the nucleus by triggering the dissociation of the targeting complex and releasing NLS cargo to the nucleoplasm (*5*). The cytoplasmic pool of Ran is kept in the translocation off position by a cytoplasmic Ran GTPase activating protein (RanGAP) and a Ran-GTP binding protein (RanBP1), which work together to purge the cytoplasm of free Ran-GTP. Indeed, a Ran-GTP–free docking zone is so important that in mammalian cells RanGAP is specifically targeted to the pore complex by a novel ubiquitinlike covalent modification that probably recycles docked Ran-GTP back to Ran-GDP (*7*).

One player lurking in the shadows is RanBP1 (*8*), a conserved protein comprised of a Ran binding domain (RBD) and a COOH-terminal NES (Lys^{178}-Val-Ala-Glu-Lys-Leu-Glu-Ala-Leu-Ser-Val-Arg^{189}) that is responsible for its steady-state cytoplasmic localization. When expressed alone, the RBD localizes to the nucleus, indicating that RanBP1 probably shuttles between cytoplasm and nucleus. Other activities ascribed to RanBP1 suggest that it links nuclear and cytoplasmic pools of Ran (*7*).

Richards *et al.* (*1*) now demonstrate that NES-directed export of RanBP1 requires Ran-GTP but not GTP hydrolysis, and that NLS-directed import and NES-directed export can be functionally uncoupled, at least temporarily. The requirement for nucleotide triphosphate hydrolysis in nuclear transport has long been a confusing issue (*3*). Although GTP hydrolysis converts Ran to a conformation that promotes translocation, vectorial translocation through the pore complex per se has not been directly shown to require energy. This remains a significant biochemical problem in the field.



(Inset) The Ran translocation switch is off in the cytoplasm and on in the nucleus. Ran-GTP promotes NLS- and NES-directed translocation. However, cytoplasmic Ran is enriched in Ran-GDP (OFF) by an active RanGAP, and nuclear pools are enriched in Ran-GTP (ON) by an active RanGEF. RanBP1 promotes the contrary activities of these two factors. Direct linkage of nuclear and cytoplasmic pools of Ran occurs through the nuclear pore complex by an unknown shuttling mechanism. P_i, inorganic phosphate.

Proposed role of Ran in the import of NLS cargo to the nucleus. (**1**) The targeting complex forms when the NLS receptor (α) binds NLS cargo and the docking factor (β). (**2**) Docking occurs at filamentous sites that protrude from the pore complex. Ran-GDP, presumably initially bound to NTF2, docks independently. (**3**) Transfer to the translocation channel is triggered when an unidentified RanGEF converts Ran-GDP to Ran-GTP. (**4**) The nuclear pore complex catalyzes translocation of the targeting complex. (**5**) Ran-GTP is recycled to Ran-GDP by docked ubiquitin-RanGAP. (**6**) Ran-GTP disrupts the targeting complex by binding to a site on β that overlaps with α binding site. (**7**) NLS cargo dissociates from α, and Ran-GTP may dissociate from β. (**8**) α and β factors are recycled to the cytoplasm.

Because NLS-directed import is triggered by the carefully orchestrated conversion of Ran-GDP to Ran-GTP, the conclusion that NES-directed export also requires Ran-GTP suggests that both pathways are controlled by the same mechanism. But a critical difference between nuclear and cytoplasmic pools of Ran destroys this apparent symmetry. Whereas a cytoplasmic RanGAP favors high cytoplasmic Ran-GDP levels, an abundant nuclear RanGEF favors high nuclear Ran-GTP levels. It appears that in the nucleus the Ran switch is stuck in the on position, explaining why docking complexes are not known to accumulate about the nuclear face of the pores.

Does Ran retain control of export in the Ran-GTP–rich environment of the nucleus? NES-directed export may occur constitutively and without control, or control may be relegated to a step before Ran-GTP action. Alternatively, the pool of available nuclear Ran may not actually be bound to GTP. Full understanding of the Ran switch in the nucleus will probably have to await the characterization of other key export factors, including the still unidentified NES receptor.

Because of its essential role in most import and export pathways, Ran is an attractive target for viruses wanting to inhibit the gene functions of their hosts. Vesicular stomatitis virus very efficiently co-opts host functions such as protein synthesis for its cytoplasmic replication. Having no obvious requirements for host nuclear functions, VSV is content to economize by dispensing with nuclear DNA and RNA synthesis. The new results of Her *et al.* (*2*) suggest that this is achieved by inhibiting nuclear transport. In a classic Trojan horse strategy, 1600 to 1800 copies of a molecule called the M protein are released from the incoming viral RNP core to wreack havoc on the nuclear transport apparatus. The expression of M

protein in *Xenopus* oocytes causes the coordinate inhibition of most Ran-dependent import and export pathways, thereby implicating Ran as the target of M protein. The fact that tRNA export, which is independent of the Ran system (9), is not inhibited by M protein is consistent with the hypothesis that the Ran gatekeeping system is the M protein target. If M protein uses a preexisting cellular mechanism to inhibit nuclear transport, then the identification of the factors that interact with M protein may reveal important components of the Ran control apparatus.

Both reports in this issue leave us wondering about the control of the Ran switch in the nucleus and cytoplasm. Perhaps, when the control apparatus is revealed, the VSV M protein will already have its finger on the mechanism.

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EVOLUTION

Human Groups as Units of Selection

David Sloan Wilson

Holistic thinkers throughout history have compared human societies to single organisms. Modern scientists have tended to dismiss the organismic view of society as a misleading metaphor, but a recent article by anthropologist C. Boehm, director of the Jane Goodall Research Center at the University of Southern California, suggests that it may contain an element of truth (*1*).

Boehm's article appears in a supplemental issue of *The American Naturalist* devoted to the subject of multilevel selection (*2*). Natural selection within a single population can explain the functional design of individuals, which causes them to survive and reproduce more successfully than their neighbors. However, this process cannot explain the evolution of altruistic behaviors, which are good for the group but, nevertheless, decrease the relative fitness of the altruistic individual within the group. Even behaviors that benefit the group as a collective, at no cost to the individual, are merely neutral from the standpoint of within-group selection. Darwin was aware of this problem and proposed that natural selection can operate at more than one level of the biological hierarchy. Altruists may be less fit than nonaltruists within a single group, but groups of altruists are more fit than groups of nonaltruists. Groups can evolve into adaptive units if the process of group selection is sufficiently strong, relative to the process of individual selection.

One for all... The !Kung tribespeople, a hunter-gatherer society in Africa, foster an egalitarian society by using group decision-making. [Courtesy of Irven DeVore/Anthro-photo]

The organismic view of human society can therefore be scientifically justified, but only if group selection has been a significant force in human evolution. Most evolutionary biologists have dismissed this possibility, because they believe that group selection requires extreme genetic variation among groups. Boehm's article suggests that other factors caused group selection to be important in human evolution, despite the fact that human social groups are genetically diverse.

According to the new work, virtually all hunter-gatherer societies have an egalitarian ethic that makes it difficult for individuals to increase their fitness at the expense of other individuals in the same group. The impulse to dominate and surpass one's neighbors is not absent, but it is successfully resisted by pressure from other members of the group in most cases, resulting in what Boehm calls a reverse dominance hierarchy. The egalitarian ethic causes meat and other important resources to be shared among the entire group, circumscribes the power of leaders, punishes free-riders, and causes virtually all important decisions to be made by a consensus process. As a result, the egalitarian ethic accomplishes a degree of behavioral uniformity within groups, and differences between groups, that could never be predicted from their genetic structure.

Boehm focuses on three implications of egalitarianism for multilevel selection theory: thwarting the ambitions of would-be dominators, making decisions as a group, and punishing free-riders. The egalitarian ethic includes a set of social norms that define the dos and don'ts of the society. Striving to achieve at the expense of other members of the group ranks high among the don'ts, and few individuals are powerful enough to resist the collective moral outrage of their neighbors. Mild forms of social control, such as gossip and withholding social benefits, are usually sufficient to control would-be dominators, but more extreme measures, such as ostracism and execution, are recorded in the ethnographic

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