For linear polarizers, SHEL produces a purely imaginary weak value. Imaginary weak values do not shift the position, but rather the propagation angle of the light (*9*). Hosten and Kwiat developed a theory to describe the deflection of the optical beam under these conditions. Their theory predicts, and experimental results confirm, highly amplified shifts, about 10000 times δ (bottom right panel), a much larger effect than direct detection and larger amplification than even previously measured real weak values. This enhancement allowed the detection and characterization of SHEL over the full range of incident angles with angstrom precision.

SHEL is a very small effect in a standard glass-air interface, but it is predicted to be much more pronounced in photonic crystals (*1*). In such materials, SHEL may be a valuable tool for manipulating the angular momentum of photons in, for example, quantum information applications. Furthermore, by studying SHEL in clean optical systems, it may be possible to turn the tables and gain further insight into the spin Hall effect in semiconductors. In the first work on weak measurement, it was speculated that the technique could be useful in amplifying and measuring small effects (*6*). Now, 20 years later, this potential has finally been realized.

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10.1126/science.1154149

IMMUNOLOGY

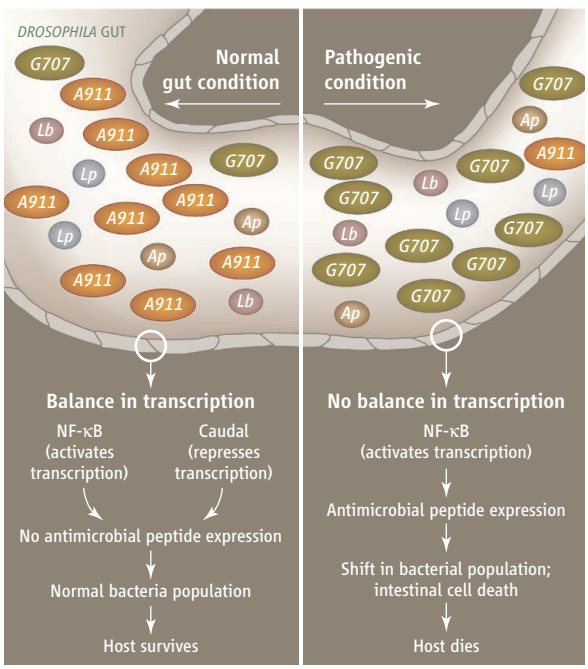
The Right Resident Bugs

A link between a transcription factor and control of immune responses in the fly gut opens the door to analyses of host-microbe mutalism.

Neal Silverman and Nicholas Paquette

The human gastrointestinal tract harbors ~500 distinct microbial taxa (*1*), comprising an estimated 10^{14} microbes. The proper maintenance of this microbial consortium is of great importance for health. Conversely, damage to the gut microbiota community is implicated in disease, such as inflammatory bowel disease (*2*). The immense complexity of gut flora and its complicated interactions with the immune system make the human gut a challenging experimental system. Recently, several groups have investigated the resident microbiota communities of insects, in particular the experimentally powerful fruit fly *Drosophila melanogaster* (*3*–*5*). These studies show that the insect intestinal microbiota, consisting of ~25 phylotypes with just a few dominant bacterial species, is much less complex than our own. On page 777 of this issue, Ryu *et al.* (*5*) exploit this limited microbial diversity and the genetic tools available in *Drosophila* to dissect the mutualistic relationship between the gut microbiota and their host.

Insects rely primarily on innate immune responses to control microbial infection. One of the best-studied mechanisms of immune protection in *Drosophila* is the inducible production of a battery of antimicrobial peptides. Production of these peptides is regulated by two signaling cascades—the Toll and immune deficiency pathways—that control transcription factor nuclear factor kappa B (NF-κB) homologs. In septic infection, the synthesis of antimicrobial peptides occurs primarily in the fat body (equivalent to the vertebrate liver). After oral infection with pathogenic microbes, expression of antimicrobial peptides can also occur in intestinal epithelial cells (*6*–*9*). However, under conventional culture conditions for flies, gut-specific expression of antimicrobial peptides is very low, even after ingesting nonpathogenic bacteria. Instead, reactive oxygen species are generated in intestinal epithelial cells to prevent growth of ingested, nonpathogenic microbes (*10*).



Gut microbes. Caudal inhibits the expression of antimicrobial peptide genes in the fly gut, even though the immune deficiency pathway is activated by resident gut microbes (**left**). In the absence of Caudal, antimicrobial peptides are produced, altering the composition of the bacterial population and resulting in apoptosis of the gut epithelium and increased mortality (**right**). *Lp, Lactobacillus plantarum; Lb, Lactobacillus brevis; Ap, Acetobacter pomorum.*

Ryu *et al.* investigated why antimicrobial peptide production is unaffected by resident intestinal microbiota in *Drosophila*, and the physiological consequence of this regulation. Surprisingly, bacteria normally resident in the gut activate the immune deficiency pathway in intestinal epithelial cells. Yet, this does not induce the expression of antimicrobial peptide genes.

Instead, the homeobox transcription factor Caudal, well known for its role in the development of the gastrointestinal tract (*11*), represses antimicrobial peptide gene expression (see the figure). Blocking Caudal expression in intestinal cells by RNA interference (RNAi) increased production of antimicrobial peptides, causing profound changes in the gut's bacterial population, particularly in two species. The *A911* strain of *Acetobacteraceae*, a dominant member of the gut microbial community (more than 10^5 bacteria per gut) in wild-type flies, was greatly reduced (to less than 10^3 bacteria per gut) in flies where Caudal expression was disrupted by RNAi (*Caudal-RNAi*). By contrast, the *G707* strain of *Gluconobacter*, a minor constituent of the gut flora in wild-type animals, increased (to more than 10^4 per gut) in the *Caudal-RNAi* flies. *A911* bacteria were also sensitive to a

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synthetic antimicrobial peptide, Cecropin A, whereas *G707* was much less so. And expression of just one antimicrobial peptide (Diptericin or Cecropin A) in the gut of wild-type flies, by way of a transgene, caused a similar shift in the bacterial population.

Additionally, enhanced growth of *G707*, caused by production of antimicrobial peptides in the gut, was detrimental to the host. In the *Caudal-RNAi* flies, apoptosis of intestinal cells in the gut increased and fly survival decreased. These changes required the presence of *G707* bacteria, because apoptosis and survival returned to near-normal levels in germ-free animals (it is unclear how this change in bacterial population induces apoptosis). Whereas feeding germ-free animals *G707* bacteria induced cell death and mortality, feeding them "normal" microbiota (that are resident in wild-type animals, such as *A911*) did not induce cell death or changes in host survival. Moreover, *G707* fed to conventionally reared animals (with normal gut microbiota) did not induce any apoptosis. Indeed, germ-free animals first colonized with *A911* did not support the growth of *G707* and did not exhibit cell death after inoculation with *G707*.

The experiments by Ryu *et al.* elegantly demonstrate that the normal flora in the fly gut is sufficient to suppress the growth of pathogenic bacteria, a phenomenon referred to as colonization resistance. In humans, alterations in gut microbiota communities (such as that following antibiotic treatment) are theorized to lead to loss of colonization resistance and the expansion of minor gut microbial residents and other pathogens (*12*). This failure of colonization resistance has been linked to pathology induced by *Clostridium difficile* as well as infections in neutropenic patients. The data presented by Ryu *et al.* clearly establish the important role that microbiota play in their own proper maintenance, the ability of this microbial consortium to support and sustain health, and the critical role that properly regulated host immune responses play in supporting this microbial consortium.

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Published online 24 January 2008
10.1126/science.1154209
Include this information when citing this paper.

PHYSICS

From Complexity to Simplicity

A combination of positively and negatively charged current carriers may provide a key to understanding cuprate superconductors.

Sudip Chakravarty

HAMLET: Do you see yonder cloud that's almost in shape of a camel?

POLONIUS: By th' mass, and 'tis like a camel indeed.

HAMLET: Methinks it is like a weasel.

POLONIUS: It is backed like a weasel.

HAMLET: Or like a whale.

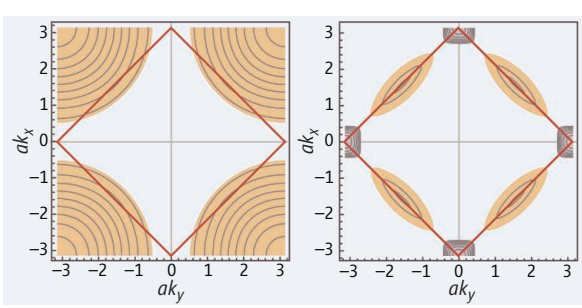
POLONIUS: Very like a whale.

—*William Shakespeare*

More than 20 years ago, Bednorz and Müller discovered superconductivity in copper oxides at remarkably high temperatures (*1*). Since then, physicists have struggled to understand the mechanisms at work. Recently, a set of experiments on cuprates in high magnetic fields (*2*–*6*) has completely changed the landscape of research in high-temperature superconductors (HTSs). In particular, the data suggest that the current carriers are both electrons and holes, when in fact the materials are "hole doped"—i.e., the current carriers should be positively charged. Moreover, the data cannot be reconciled with an important theorem about how electrons are organized in materials (*7*) unless one assumes that the signals arise from a combination of both holes and electrons. Until now, physicists have not been able to decide whether the cuprates, in Shakespeare's terms, are camels or whales; in fact, these experiments foreshadow a remarkable degree of simplicity in these complex materials.

The cuprates start out as insulators and become superconductors when doped with additional charge carriers. These so-called Mott insulators insulate by virtue of strong repulsive Coulomb interaction and need not break any symmetries in the lowest energy state, the ground state. A symmetry of a system is a transformation, such as a translation or a rotation, that keeps it unchanged. Such a symmetry is said to be broken, or spontaneously broken, if the system does not obey the symmetry of the underlying fundamental physical nature of the material; for example, a ferromagnet breaks the spin-rotational symmetry with its magnetization pointing in a definite direction. The notion of symmetry and broken symmetry finds many deep applications in physics.

Soon after the discovery of the cuprate superconductors, Anderson proposed (*8*) that their parent compounds begin as a featureless spin liquid that does not break any symmetries, called the resonating valence bond (RVB) state: "The preexisting magnetic singlet pairs of the insulating state become charged superconducting pairs when the insu-



In the pocket. Electron states in cuprates, with constant energy curves (black) plotted in momentum coordinates (ak_x, ak_y) in units of $\hbar/a$, where $\hbar$ is Planck's constant divided by 2π, and a is the lattice spacing. (**Left**) The Fermi surface separates the occupied states (light blue) from the unoccupied states (orange); the latter can act as positively charged carriers called holes. (**Right**) When the material is "underdoped," the Fermi surface may reconstruct, which forms two hole "pockets" (adding the four halves) (orange) and one electron "pocket" (adding the four quarters) (purple), as revealed by recent experiments.

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