Ohm's Law in a Quantum World

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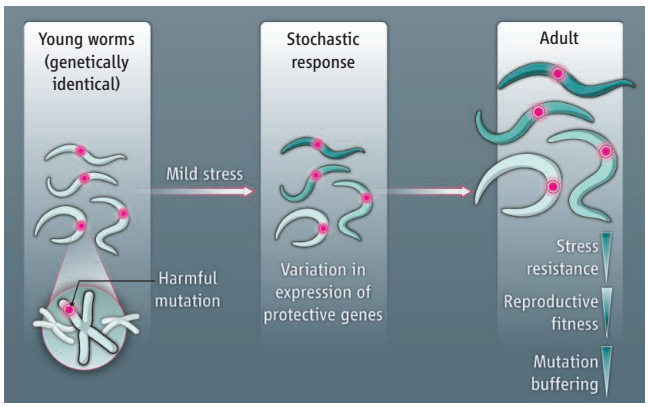

The final effect. A mild stress response protects genetically identical young worms against deleterious mutations. However, because of individual variation in stress signaling, the extent of this protection varies. When such worms carry a potentially harmful mutation, individuals with high stochastic expression of certain stress-protective genes (dark green) show better survival than individuals with lower expression (light green), but at a cost of reduced fecundity. Although genetically identical, the starting population already has variable expression of stress-protective genes, which also affects the outcome of mutations (not shown).

viduals with greater chaperone expression were more resistant to deleterious mutations. Even early worm embryos differ in their chaperone expression, which may influence the outcome of mutations in each individual (*9*). Moreover, higher stochastic chaperone levels are correlated with extended life span (*10*), and the inverse also holds true: A mutation can increase the stochastic variation in the expression of certain genes, which can in turn determine mutation outcome (*11*).

Given the positive effects of the chaperones, why don't all worms exhibit high chaperone expression levels? Casanueva *et al*. found that individuals with high chaperone expression produced fewer offspring (reduced fecundity). Stochastic variation in chaperone expression may therefore represent a bet-hedging strategy, allowing populations with varying chaperone expression to have a better chance of surviving in both benign and stress-inducing conditions than a population exhibiting uniform gene expression.

Although genetic individuality is emerging as a central theme of research, underlying promising initiatives in personalized medicine, Casanueva *et al*. caution that "nongenetic individuality" (*12*) extends beyond unicellular organisms and has important functional consequences. Even though a person's genome sequence may be available, with all genetic interactions completely unraveled, and life history fully known, other, non-genetic differences may constitute an elusive determinant of risk for disease. It is unclear whether the findings of Casanueva *et al*. can be simply extrapolated to humans, especially because such a risk diversification strategy may prove particularly useful for self-fertilizing organisms such as *C. elegans*. Moreover, only temperature-sensitive mutations were affected by differences in chaperone expression, suggesting that this phenomenon may be restricted to a subset of mutations. Nevertheless, given the conservation of stress response mechanisms and chaperones from yeast to humans, we may have to consider that destiny is not wholly determined by DNA, and that sheer luck—in the form of stochastic variation in gene expression—also has a say.

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APPLIED PHYSICS

Ohm's Law in a Quantum World

Conductivity measurements reveal unexpected classical electronic behavior at the quantum level.

David K. Ferry

Ohm's law is an empirical law based on the observation that the electrical transport properties of materials exhibit linear behavior. The principle is that the voltage that develops across a piece of conducting material is linearly proportional to the current flowing through it; the constant of proportionality being termed the resistance, $V = IR$ (*1*). Such behavior is the backbone of classical conduction in these materials. In the 1920s and 1930s, it was expected that classical behavior would operate at macroscopic scales but would break down at the microscopic scale, where it would be replaced by the new quantum mechanics. The pointlike electron motion of the classical world would be replaced by the spread out quantum waves. These quantum waves would lead to very different behavior. On page 64 of this issue, Weber *et al*. (*2*) have constructed atomic-scale nanowires in Si and have observed that Ohm's law remains valid, even at very low temperatures, a surprising result that reveals classical behavior in the quantum regime.

Transport in the quantum world is characterized by coherence, where the phase behavior of the waves remains undisturbed over sizable distances (typically on the scale of the sample size). This coherence leads to wave interference, such as the Aharonov-Bohm effect in conducting rings (*3*), where waves traveling around different paths (an impurity or defect, for example) can interfere with each other. Coherent transport in nanowires is determined by the number of transverse wave modes that propagate in the constrained geometry (*4*, *5*) (analogous to the different waves on a plucked string). In this regime, the resistance of the nanowire is given by the Landauer formula (*6*), $R = h/(2e^2N)$, where N is the number of the modes, e is the electronic charge, and h is Planck's constant. Note that the conductance, and hence the resistance, is independent of the length of the sample of material being investigated (see the figure, red curve). In the classical world, the resistance depends linearly upon the length—a

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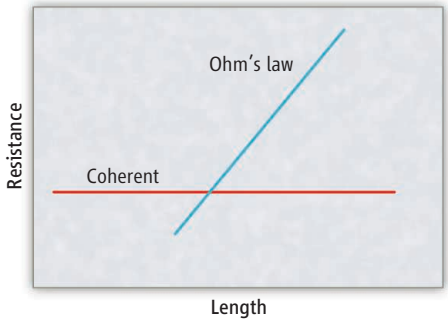
longer piece of material requires more voltage and hence has higher resistance (blue curve). Here, the resistance is given by $R = L/\sigma A$, where L is the length of the sample, A is the cross-sectional area, and σ is the conductivity. Weber *et al*. observe linear dependence of the resistance upon the length down to ~10 nm, which implies that classical behavior was maintained, even at low temperature. What breaks up the coherence, and leads to the classical behavior, is dissipation arising from collisional processes in the material, and this is expressed within the conductivity.

In a given material, variations in temperature or in electron scattering can move the blue curve up or down. The resistance from various mechanisms are additive, so that the total resistance can follow the quantum curve until it intersects with the Ohm's law curve and will then follow the latter. Hence, there is a crossover region where the two curves meet. In materials that are more conducting than silicon, such as nanowires in indium arsenide, experiments (*7*) have shown that this crossover occurs for lengths of almost 200 nm, even at room temperature, and this has been confirmed by calculations (*8*). It is clear that the onset of Ohm's law depends on the strength of the scattering of the electrons within the nanowire. In the work of Weber *et al*., the number of phosphorus atoms, and hence the number of electrons, is on the order of 10^{21} cm^{-3}. This very high doping means that the scattering, and the resistance are much larger, which will lead to a shorter coherence length, and they found that this crossover had to be below 10 nm even at their low temperature.



Ideal behavior. In systems where quantum coherence governs the transport, the resistance is independent of the length of the sample of material (red curve). Classical Ohm's law requires that the resistance be linearly proportional to the length (blue curve). Hence, for a given sample, a crossover from quantum to classical behavior where the two curves meet is expected. The measured resistance would be a sum of these two curves. Weber *et al*. (*2*) report classical behavior under conditions where quantum effects would be expected.

The implications of having Ohm's law satisfied at such low temperatures are interesting. Normally, we would expect to study pure quantum effects at low temperatures, but the results here suggest that more care will be needed to sort quantum effects from classical ones. This is also not good for concepts that use phosphorus atoms as qubits in quantum computing (*9*, *10*), because the coherence needed becomes questionable. On the other hand, this may well pave "the way for ultra-scaled classical as well as quantum electronic components…" (*2*). This is good news for the semiconductor industry, which seeks to extend Moore's law [see, e.g., (*11*)] for several more years, a process enabled by scaling individual devices to smaller sizes. It has been thought that quantum effects would limit this in the near future, but the results presented by Weber *et al*. suggest that several generations are still possible.

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PLANT SCIENCE

Controlling Hormone Action by Subversion and Deception

Molecular mimicry between a plant hormone receptor and a kinase allows them to swap binding to a phosphatase to control a signaling pathway.

Jeffrey Leung

The Nobel chemist Roald Hoffmann described molecular mimicry as a mechanism founded on subversion and deception. On page 85 of this issue, Soon *et al*. (*1*) report intriguing evidence for such mimicry in the transmission of signals elicited by the plant hormone abscisic acid (ABA). Using the model plant *Arabidopsis thaliana*, the authors show that the ABA-bound receptor and kinase alternately bind to a phosphatase, turning on and off this stress-responsive pathway, respectively. Not surprisingly, ABA is behind this "subversive" dealing in partner swapping.

ABA is a terpenoid that promotes seed maturation, prevents germination, and curtails excessive water loss during vegetative growth. In the face of climate warming and water shortages, modifying ABA synthesis or perception as possible solutions to sustain crop yield for food and fuel has attracted the interests of policy planners and scientists.

The core ABA signaling module involves three different types of proteins (*2*): the PYR/PYL/RCAR family of ABA receptors; clade A type 2C protein phosphatases (PP2Cs, including ABI1, ABI2, and HAB1); and group III SUCROSE NONFERMENTING1 RELATED kinases (SnRK2.2, 2.3, and 2.6). These three protein types are necessary and sufficient to mediate an ABA-triggered model signaling cascade in vitro (*3*).

ABA is cradled deep inside the receptor and is enclosed by conformational changes of the two substructures called "gate" and "latch" (*4*). The "gate" creates a new surface on the receptor that can tether the PP2C, freeing the SnRK2 to autophosphorylate (Ser^{175} in the activation loop of SnRK2.6) and subsequently phosphorylate downstream targets. In the absence of the hormone-receptor complex, the PP2C turns off the signaling pathway by dephosphorylating the SnRK2. Although ABI1, ABI2, and HAB1 interact interchangeably with SnRK2.2, SnRK2.3, and SnRK2.6 (*5*–*10*), the functional importance had not been clear.

Soon *et al*. show that besides the catalytic removal of the phosphate from the phospho-

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