

with traits providing protection from another (such as predation)—then an evolutionary stalemate could prevent adaptation at the rate necessary for species to cope with predicted climatic changes.

In the study by Harmon *et al.*, predator density dependence plays a key role in determining the effects of climate change on the growth of the prey population. This raises a complex issue for ecologists, because environmental disturbances strongly affect prey density. Quantitative food webs depict links between species as having a certain strength (the frequency of the interaction is often used as a surrogate), yet the interaction strength may be a nonlinear function of prey density. Altered interaction frequencies in food webs after environmental perturbations (8) could be driven by relative shifts in the abundance of different species and/or by nonlinear responses of different predators to altered prey densities. Environmental

changes could even alter the response of predators to prey density—for example, if habitat simplification alters search efficiency (9). Metrics of food web structure that are advocated for their insensitivity to differing relative abundances of prey species (10) may not detect these ecologically important changes.

When Earth undergoes climatic change, species either adapt or go extinct (11). However, during each period of change, the spatial rearrangement of extant species and the emergence of new lineages will provide a novel context within which evolution takes place. Thus, even if the genetic variation necessary for selection exists in a population, food web interactions may impose constraints on the rate or direction of evolutionary change. The work of Harmon *et al.* suggests that the difficulties inherent in studying the combined complexities of ecological and evolutionary forces can be overcome. This will be neces-

sary if we are to predict the survival of species in an uncertain future.

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PLANT SCIENCE

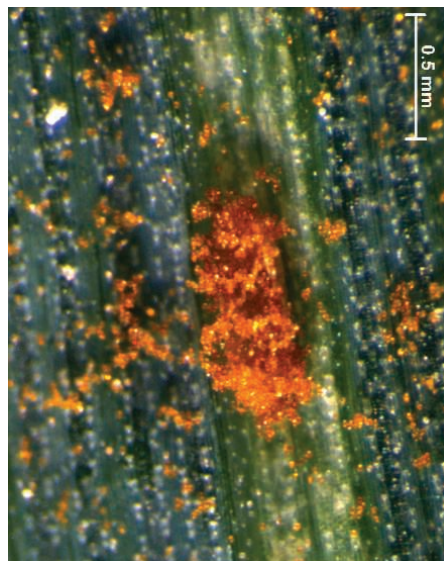
Anti-Rust Antitrust

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Human civilization rests upon the products of agriculture, but numerous fungi, bacteria, and viruses compete with us for the crops that we cultivate. Given the growing human population and demand for increased food production from less land, minimizing crop loss from pathogen infection is critical. Two reports in this issue, by Fu *et al.* on page 1357 (1) and by Krattinger *et al.* on page 1360 (2), identify molecular mechanisms that control durable, broad-spectrum, disease resistance in wheat, the second-most-produced cereal crop in the world.

Understanding the molecular basis of specific plant resistances has enabled the development of crop varieties that resist common diseases. These resistance mechanisms follow a “gene-for-gene” model, whereby specific plant recognition of a pathogen gene product yields near-absolute resistance to pathogens containing the recognized gene. However, this form of resistance is vulnerable to rapid counter-evolution by the pathogen (3). This generates a continuous and resource-intensive cycle of plant resistance gene discovery, crop breeding, and eventual resistance gene

“defeat” by the pathogen, which has driven interest in identifying durable forms of resistance to pathogens. In most plant-pathogen systems, durable resistance has been identified through quantitative trait locus (QTL) mapping. Although identified QTL provide quantitative resistance to a range of pathogen



Wheat rust. Close-up of a wheat leaf rust pustule releasing orange urediospores (orange spots) on a leaf of the susceptible spring wheat cultivar Thatcher.

The identification of genes that confer durable, multipathogen resistance may help breeders overcome devastating wheat fungal diseases.

species and genotypes therein, the molecular bases of these broad quantitative resistances have remained largely unknown (4).

Krattinger, Fu, and their colleagues begin to show that mechanisms underlying quantitative resistance are not simply weak alleles of genes involved in specific gene-for-gene resistance, as has been proposed. Rather, each plant species may contain multiple, possibly independent, mechanisms of quantitative resistance. The latter may reveal similar processes in other plants or may be taxonomically limited.

Krattinger *et al.* describe the cloning of the wheat *Lr34* QTL that has been used to confer resistance to multiple rusts (see the figure) and a mildew in the field for nearly 50 years (5). The locus harbors the gene *Lr34*, which encodes an ATP-binding cassette (ABC) transporter (4). ABC transporters, or pleiotropic drug resistance transporters, move diverse chemical compounds, including plant natural products, across membranes. The *LR34* transporter is weakly homologous to *PEN3*, an ABC transporter from the plant *Arabidopsis thaliana*, which facilitates resistance to the same mildew via the targeted export of plant metabolites (6, 7). The mechanistic similarity between *LR34* and *PEN3* reinvigorates hypotheses that specific and

diverse plant natural products may play essential roles in plant resistance to pathogens (8).

Fu *et al.* find that another QTL, *Yr36*, which provides quantitative resistance to stripe rust, harbors the gene *WHEAT KINASE START 1* (*WKS1*). The *WKS1* protein contains a steroidogenic acute regulatory protein–related lipid transfer domain (*START*) and a functional enzymatic (kinase) domain, suggesting a role for plant lipids in cell signaling mechanisms that confer disease resistance. This specific combination of domains, unique among plants examined to date, appears to have evolved just before the divergence of wheat and its closest relatives. However, although *WKS1* provides resistance to diverse stripe rusts, the resistant allele is present in only a minority of the species tested, and even within the species in which *WKS1* occurs, it is not ubiquitous across all genotypes. This suggests that the gene evolved early in the wheat lineage and was then repeatedly lost. Natural variation of gene function is also a hallmark of gene-for-gene resistance loci, where continual shifts in pathogen populations are believed to drive rapid evolution of these genes (9, 10). Given the dearth of cloned quantitative resistance loci, it remains to be seen if these loci show enhanced levels of polymorphism and evolutionary dynamics similar to those for gene-for-gene resistance.

Why don't pathogens evolve counter-resistance to durable plant resistance? Perhaps these quantitative resistance loci target components of pathogens that are so critical for pathogen success that their encoding genes are evolutionarily constrained relative to similar pathogen genes involved in gene-for-gene interactions. Alternatively, these quantitative genes may provide resistance to the plant at less detriment to pathogen fitness than do gene-for-gene resistances. In both scenarios, decreasing the selective pressure for pathogens to evolve counter-resistance promises greater long-term success for improving crop yields using durable quantitative plant defenses. Additionally, for both cloned QTL described by Krattinger *et al.* and Fu *et al.*, alleles associated with loss of plant resistance to the tested pathogens would be predicted to produce modified, rather than nonfunctional proteins. Future inquiry into possible alternative functions of these genes or fitness costs of possessing resistant alleles at these loci may provide further insight into the evolution and durability of these mechanisms.

The studies by Fu *et al.* and by Krattinger *et al.* provide a first glimpse into the molecular mechanisms controlling quantitative resistance. Together, they demonstrate that multiple mechanisms can contribute to quantitative

resistance within a given plant and that these mechanisms are not obviously associated with the well-studied and taxonomically widespread specific gene-for-gene resistance mechanisms. Mechanisms of quantitative resistance may be unique to particular plant lineages. Thus, while providing valuable tools to develop durable resistance against important wheat pathogens, these studies also argue that the molecular mechanisms of quantitative resistance need to be studied in a wide range of crop and model plants to fully explain the phenomenon.

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ENGINEERING

Infrastructure Design Issues in Disaster-Prone Regions

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If designed and managed well, infrastructure—the networks that transport people and goods, distribute energy, and maintain communications and the buildings in which people live, work, and play—contributes to societal sustainability and resilience in areas at substantial risk from catastrophic events such as hurricanes. The extent to which infrastructure functions after such events depends on design choices that trade off, at least implicitly, current construction costs for future repair and replacement costs. These choices are based on assumptions that may not reflect all of the relevant factors.

Recent advances in assessment and design allow the economic and environmental trade-

offs in both design and postdisaster restoration to be explicitly considered and managed proactively during the design process. Similarly, infrastructure design can take advantage of the interactions between the natural and built environments in disaster-prone regions.

For example, we have an extensive record of hurricane impacts and can design structures to withstand those impacts in many cases. Despite advances in our knowledge of structural design for hurricane-prone areas, economic damage to buildings from major hurricanes in the United States has remained largely steady over the past four decades when adjusted for population growth and inflation (1, 2) (see the figure, panel A).

Consider as an example a new commercial development to be built in a hurricane-prone area. Traditional design practice would be to design the individual buildings, utility poles,

Advances in infrastructure assessment and design should help designers and builders support sustainability and resilience goals.

water distribution pipes, and flood protection systems to meet the design wind speeds and flooding levels specified in the relevant standards (3) or occurring with a preselected yearly probability. Current standards (3) specify that most structures (except for critical structures such as emergency response facilities) should be designed to withstand the stresses imposed by a hurricane that would occur, on average, every 50 years. This approach makes three fundamental assumptions that should be questioned.

First, it implicitly assumes that the design standards strike the proper balance between the benefits and costs of different design alternatives. Design standards are intended to apply across many different buildings, each with location-specific costs of reinforcement and failure. For example, two neighboring low-rise industrial buildings may have been

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