Getting the Jump on Explosive Percolation

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effects [such as predation risk at flowers (*10*)], relapses after periods of abstinence, or withdrawal symptoms (*11*).

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MATHEMATICS

Getting the Jump on Explosive Percolation

An analytical approach to explosive dynamical percolation yields general conditions for the transition to be a continuous or discontinuous process.

Robert M. Ziff

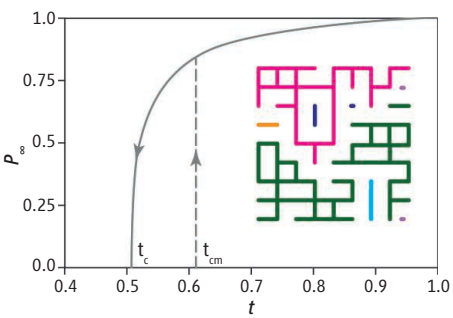
Percolation refers to the formation of long-range connectedness or conductivity in random systems. Simple models for percolation were independently devised in the areas of polymer science (*1*) and mathematics (*2*) in the 1940s and '50s, and have been both a persistent theoretical challenge and an enduring practical paradigm ever since. In the past decade, percolation has become a central problem in probability theory, and has figured in the work of two recent Fields medalists (*3*). A recent and somewhat controversial development concerns looking at the dynamics of percolation under various global bond selection rules and how percolating systems make the transition from being disconnected (or comprising a group of disconnected clusters) to being fully connected. It had been shown that the transition can proceed explosively, in which the transition is discontinuous (*4*), but that scenario was later challenged when it was shown that for some specific systems, such a transition is in fact continuous. On page 1185 of this issue, Cho *et al.* (*5*) show analytically and numerically that the explosive percolation transition can be either continuous or discontinuous, depending on the bias against certain "bridging" bonds and the dimensionality of the system.

In standard percolation, the probability P_∞ that a given point is part of a percolating cluster is a continuous function of the bond occupation probability p, the probability that a bond is conducting or open. P_∞ is zero for cases where $p < p_c$ but rapidly grows for $p > p_c$, where p_c is the percolation threshold that signals the onset of long-range connectivity. In an infinite system and for p slightly greater than p_c, P_∞ behaves as $P_\infty \approx (p - p_c)^\beta$, where β is a fractional power, such as $5/36 \approx 0.139$ in two dimensions (*6*), so the derivative dP_∞/dp is infinite at p_c. Being continuous, the percolation transition can be considered to be a kind of second-order phase transition.

Bonds can be added one at a time and clusters merged as they are added, leading to a dynamical percolation transition. Achlioptas *et al.* (*4*) introduced a new model of percolation in which two candidate unoccupied bonds are simultaneously considered, and only the one that joins the smaller cluster-size sum or product is added. Simulating this model, they found that the percolation transition is delayed to a higher value of p, but then occurs in an explosive and seemingly discontinuous manner, more akin to a first-order phase transition. Similar behavior is observed in many other systems, including regular lattices, when a variety of bond-addition rules are used (*7*–*10*).

Distinguishing a discontinuity from a sharp continuous transition in a finite system can be tricky, however. In 2010, da Costa *et al.* (*11*) showed theoretically that the random-graph model was in fact continuous under a modified product rule, but with a very small β (~0.056), which makes it nearly indistinguishable from a discontinuous transition. More investigation and proofs of the continuity followed for a variety of systems (*12*, *13*), and it is now generally accepted that the original two-choice product rule leads to a continuous transition, but it wasn't previously proven for a Euclidean lattice such as the simple square lattice.

Still, there remain some undoubtedly discontinuous explosive transitions out there. For example, a simple model of joining only the bond that gives the smallest product over all bonds in the system is discontinuous—the last bond joins two huge clusters together. A hierarchical long-range model also shows a discontinuity (*14*).



Joining the dots. Schematic of the probability P_∞ that a point belongs to the spanning cluster versus the occupation probability t, for regular percolation of $m = 1$ (solid line) and for a typical case with $m > 1$ (dashed line). A jump in P_∞ for $m > 1$ is evident. (**Inset**) A typical bond configuration showing a later stage where most of the sites belong to two distinct clusters. The algorithm preferentially puts new bonds along edges that do not connect the two clusters.

Cho *et al.* introduce a model of percolation with a new global constraint on a Euclidean lattice: If a candidate bond causes the system to percolate from one side to another, then the model biases against it. Specifically, m candidate bonds are chosen simultaneously, and if any are nonbridging, then one such bond is chosen randomly and occupied. If they are all bridge bonds, then one of those is chosen and occupied. Equivalently, just one unoccupied bond can be chosen and occupied (if it is a nonbridge bond) or occupied with an appropriate probability (if it is a bridge bond).

Now, $m = 1$ corresponds to standard percolation with critical bond occupancy t_c. For $m > 1$, Cho *et al.* observe that percolation is delayed until a later occupancy $t_m > t_c$, but

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then, once the boundary between the two large clusters has been breached, bridging bonds cease to exist, and the system follows essentially the behavior for $m = 1$. Thus, there is a jump in P_∞ and the system shows a discontinuity (see the figure).

However, when going to larger systems, some telling behavior is found. For m less than a critical value m_c, t_m decreases to the standard percolation value t_c and the discontinuity disappears. Likewise, when, for $m > m_c$, t_m increases to 1, the explosive percolation occurs but only at full occupancy. Cho *et al*. relate m_c theoretically to the backbone fractal dimension and predict $m_c \approx 2.55$, which is consistent with their extensive simulations.

The work of Cho *et al*. shows the importance of taking the infinite-size limit and explains some of the confusing and contradictory behavior seen in this field previously. Exactly at $m = m_c$, the discontinuity in the transition persists with a critical occupancy t_{cm} less than 1, so in this special case a nontrivial discontinuous transition on an infinite Euclidean lattice evidently exists. Investigating the nature of this transition and finding other models that can be tuned to a similar tricritical state is a rich area for future research.

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IMMUNOLOGY

Guilty by Association

Fingerprinting regulatory T cells in tumors reveals that they are specific for organ-expressed antigens.

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Developing tumors face a multifaceted immune response that can ultimately "edit" the tumor by eliminating cells that cannot evade antitumor responses. Consequentially, tumors employ many mechanisms to evade immune responses, including expression of inhibitory chemokines or cytokines and recruitment of immunosuppressive cells such as regulatory T cells (T_{regs}) (*1*, *2*). T_{regs} are known for suppressing immune responses and maintaining immune homeostasis, but are also often found in the tumor microenvironment where their presence correlates with poorer patient outcomes. Moreover, acute T_{reg} depletion in transplant models of cancer results in more potent antitumor immune responses. Thus, T_{regs} are considered important players in tumor development. However, relatively little is known about why T_{regs} infiltrate tumors or how they are formed. On page 1219 of this issue, Malchow *et al.* (*3*) address these questions and discover that developing tumors do not elicit novel T_{reg} responses, but rather they recruit and/or expand T_{reg} populations naturally found within the tissue from which the tumor arises.

Tumors can elicit T cell responses because they have distinct protein expression relative to normal cells (*4*). T cells bearing antigen-specific surface T cell receptors (TCRs) recognize tumor antigens displayed on the cell surface, which results in T cell activation, proliferation, and destruction of target cells. Multiple layers of regulation exist to avoid T cells that recognize "self antigens" and target healthy cells. This regulation starts in the thymus, where developing T cells with TCRs that recognize self antigens are deleted or become immunosuppressive T_{regs} (*5*). Although the complete pathway for developing these "natural" T_{regs} (nT_{regs}) is unclear, it involves a molecule called autoimmune regulator (Aire), which is expressed by specialized thymic epithelial cells and helps them express a variety of tissue-restricted self antigens. In this way, developing T cells are first exposed to these antigens before leaving the thymus (*5*). Tissue-specific self antigens are thought to be critical for the function and localization of some nT_{regs}, but how they affect tumor-infiltrating T_{regs} is less well understood. Complicating matters, T_{regs} can also be generated post-thymically from conventional T cells, when they are exposed to antigen in the presence of immunosuppressive molecules such as transforming growth factor–β. Induced T_{regs} (iT_{regs}) are critical to maintaining immune tolerance to environmental antigens (e.g., commensal bacteria), and it has been suggested that immunosuppressive factors within the tumor microenvironment could convert tumor-specific T cells into iT_{regs}. Furthermore, because nT_{regs} and iT_{regs} can differ in plasticity (*6*), it is plausible that one population could be more amenable to therapeutic modulation. Thus, it is important to identify the antigens recognized by tumor-associated T_{regs} and to determine the role of antigen recognition in their development and function.

To address the role of antigen recognition, Malchow *et al.* used an elegant system for sequencing the TCRs expressed by tumor-infiltrating T_{regs} and conventional T cells. Like fingerprints, these TCR sequences distinguished different T cell clones present within the tumor microenvironment. Using a genetically engineered mouse model of prostate tumor development, they found that tumors were consistently infiltrated by T_{reg} populations bearing a single TCR (called MJ23), which differed from TCRs present on conventional tumor-infiltrating T cells. Furthermore, this TCR was specific for an antigen expressed in normal prostate tissue. Thus, this T_{reg} population was not selected for on the basis of expression of a novel antigen by a developing tumor, but was instead capable of recognizing an antigen expressed by the tissue from which the tumor arose.

The authors went on to show that the MJ23 TCR was sufficient to drive T_{reg} development within the thymus and that this process required the function of Aire. This led to the accumulation of T_{regs} within prostate-draining lymph nodes of male mice, regardless of whether they had a tumor. MJ23 TCR expression also caused nT_{reg} development in female mice, which lack prostate tissues but likely carry the gene that encodes the rel-

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