A similar problem arises in Taylor–Couette flows. Here, the flow occurs in the space between two concentric cylinders, and it is driven by spinning either the inner cylinder, the outer cylinder or both. This flow finds numerous applications in bioreactors, fractionation columns, heat transfer and filtration devices, but it has been studied most extensively because it exhibits a highly ordered route from laminar to turbulent flow[6]. The flow state is again determined by the Reynolds number, and we also have the notion of an ultimate state.

A related system is Rayleigh–Bénard flow, which is the flow in a box that is heated from below. Kraichnan made an important prediction regarding turbulent heat transfer in this kind of flow[2], relating the Nusselt number, Nu, the non-dimensional heat transfer rate, to the Rayleigh number, Ra, the non-dimensional temperature difference. In the limit of large Ra, Kraichnan's theory holds that $Nu = Ra^{1/2}$, but the search for this limit has proven unsuccessful[7,8], primarily due to practical difficulties. However, the Rayleigh–Bénard problem has been an inspiration to the Taylor–Couette problem, in that it is possible to cast the latter in terms of an 'equivalent' Nusselt number Nu_w, and a number that is similar to the Reynolds number — the Taylor number Ta. By using these numbers in place of a friction coefficient and the Reynolds number[9], we arrive at the prediction of an ultimate regime for Taylor–Couette flow where we expect to find $Nu_w = Ta^{1/2}$.

To establish this regime, it is obviously necessary to achieve an extremely high Reynolds (or Taylor) number. This is challenging to accomplish in experimental work, as it requires a very large and certainly expensive apparatus. But it is similarly difficult in numerical work, as it would require the resolution of an exorbitantly large range of time and spatial scales. However, Zhu and co-workers achieved this goal, numerically and experimentally, by taking inspiration from turbulent pipe flow.

When the surface of a pipe is rough, and the asperities are much larger than the smallest eddies in the flow, it is well known that the friction factor becomes independent of the Reynolds number[9]. In a sense, this is the ultimate regime for pipe flow. By making the surfaces of their rotating cylinders sufficiently rough, Zhu et al. effectively achieved much higher Reynolds numbers than were previously possible, and they were then able to observe the ultimate regime for Taylor–Couette flows.

An immediate implication of this work is that in probing the ultimate regime, it also supports and validates the understanding of the sub-ultimate regimes. That is, the Reynolds number and Taylor number are both better understood as a result of this study. In addition, it now seems obvious that Rayleigh–Bénard flows can be studied in this way, by introducing roughness to generate an ultimate regime, and some work has already appeared along these lines[10].

So far, however, the work has concentrated on describing the bulk properties of the ultimate regime, such as heat transfer and the friction factor. This does not imply that we understand the details of the turbulence itself, such as its length-scale distribution, the interactions among scales, its generation and dissipation. As a salutary example, rough pipe flows have been studied for a long time, and its ultimate regime was established as early as 1933 (ref. [3]). Yet we are not that much closer to understanding the turbulence in the fully rough regime, nor can we yet make the leap from the fully rough case to the smooth case. As always in turbulence research, much work still needs to be done. ❐

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EPIDEMIC SPREADING

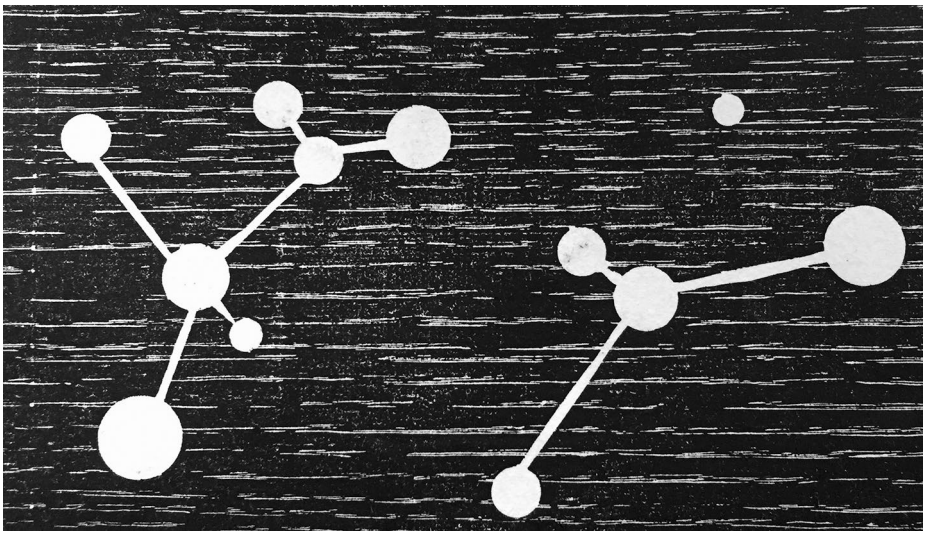
Don't close the gates

It seems obvious that restricting travel should help prevent the surge of epidemics. But a new mathematical analysis now demonstrates that mobility often reduces the heterogeneity in population distributions, thereby lowering the epidemic risk.

Samuel V. Scarpino

Public health authorities — and indeed human instinct — have long taken for granted that restricting mobility can prevent a localized outbreak from growing into an epidemic. This belief has led to calls for international border closure during all of the recent outbreaks, including Zika, Ebola, H1N1 and SARS. Writing in *Nature Physics*, Gómez-Gardeñes et al.[1] have now challenged the assumption that movement spreads pathogens and thus facilitates epidemics. Surprisingly, their work suggests that mobility might actually lower the risk of widespread epidemics.

Although it is true that isolating infectious individuals can slow disease transmission — by, say, closing live bird markets during avian influenza outbreaks[2] — it remains controversial, both in the public health and scientific communities, whether the wholesale restriction of movement can prevent epidemics[3]. For example, mathematical and computational models of Ebola[4] and



Credit: *Reticulations*. Woodcut print by Betty Scarpino and Samuel Scarpino.

influenza transmission[5] found that closing international borders might only delay the epidemic peak by a few weeks and would not reduce the total number of cases.

Despite their colloquial use, the distinction between outbreaks and epidemics has a specific definition, which is grounded in the physics of spreading processes[6]. The mathematical model used by Gómez-Gardeñes et al. categorizes individuals as either susceptible to infection or infectious and, critically, as susceptible again after recovering from their infection. Within this framework, known as a susceptible–infected–susceptible (SIS) model, the transition from an outbreak to an epidemic is characterized by the system entering a stable equilibrium with a non-zero fraction of infectious individuals present in the population.

It is this mathematical distinction — between small outbreaks and large epidemics — that Gómez-Gardeñes et al. studied in the context of mobility. Their model considers epidemics as a reaction–diffusion process, where individuals are infected via local interactions with infectious individuals (reactions) and can move throughout a larger metapopulation (diffusion). If you are unfamiliar with the concept of a metapopulation, think of a collection of cities with movement between them for work. Reaction–diffusion models, although most common in chemistry, have also seen widespread application across the physical and natural sciences. In these models, it is often possible, after making a few simplifying assumptions, to construct an analytical approximation for when the sputtering transmission chains of an outbreak will transition into the stability of an epidemic.

Gómez-Gardeñes et al. studied how the epidemic threshold, the critical point where outbreaks transition to epidemics, is affected by mobility. Specifically, the authors provided an analytical approximation to the eigenvalues of a matrix encoding how individuals are connected in the metapopulation. For a collection of cities, this matrix would record the probability that an individual in city i would contact an individual in another city j. The epidemic threshold is related to the largest eigenvalue of this matrix — similar to using the eigenvalues of the Jacobian to study critical transitions. For low values of mobility — namely, when individuals in city/population i are much more likely to interact with each other than with individuals in population j — the authors found that the epidemic threshold increases, which implies a lower probability of an outbreak growing into an epidemic.

The interesting physics in the paper by Gómez-Gardeñes et al. — that mobility hinders epidemic spread — depends on two key assumptions. First, the individuals must recover and again become susceptible to infection. There are many human, animal and plant diseases like this — seasonal influenza and gonorrhoea being two examples in humans[7]. However, it would be interesting to study how mobility affects epidemic risk for diseases where individuals have lifetime immunity after infection — a so-called susceptible–infected–recovered (SIR) model. Second, the population sizes in the metapopulation must be different. In general, greater asymmetry in population sizes leads to higher epidemic risk and, because mobility reduces population size asymmetry, it lowers epidemic risk. Lastly, it is worth remembering that a set of standard assumptions will always apply to using such an eigenvector approach to study critical transitions.

Despite the surprising discovery of Gómez-Gardeñes et al., such a counterintuitive pattern is not completely unknown. For example, it was found that dynamically exchanging social contacts (a process also thought to facilitate disease spread) can actually reduce epidemic potential[8]. More intriguingly, a similar phenomenon was discovered by evolutionary biologists in the early 1990s and was expanded on in recent years[9,10]. Although a more restricted model was considered, there it was shown that mobility can impede the evolution of beneficial mutations, a process whose physics are closely related to disease spread[11].

Gómez-Gardeñes et al. also performed an empirical analysis of high-resolution mobility data from Cali, Colombia, a city of 2.4 million people, and found mobility rates between neighbourhoods are in the regime where epidemic risk could be lowered by movement. Their finding, that urban metapopulations can have a lower epidemic risk due to mobility, might inform ongoing debates about how urbanization affects the emergence and establishment of disease[12]. There are of course broad parameter ranges of their model where mobility does exacerbate epidemic spreading and, rightly, the authors end with a call to action for advancing our empirical understanding of human mobility. Indeed, the epidemiological modelling community is coming to appreciate how complex mobility patterns (for example, in dengue outbreaks in Pakistan[13]) challenge conventional wisdom.

Isolating and caring for infectious individuals is a vitally important public health strategy, which both slows disease spread and reduces morbidity/mortality. However, the paper here provides both analytical and empirical evidence that — as previous studies have suggested about individual diseases — broader mobility patterns can have a range of effects on outbreaks. Indeed, as Gómez-Gardeñes et al. and others have found, challenging long-held assumptions in epidemiology can uncover a richer physics and may ultimately advance our capacity to prevent epidemics. ❐

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ACTIVE GALACTIC NUCLEI

Cosmic-ray tracing

Active galactic nuclei are firm favourites to be revealed as the source of cosmic rays, but solid evidence has proven elusive. A model taking both local and global nuclei propagation into account may help to close the deal.

Julia Becker Tjus

Where do cosmic rays come from? This question has been a topic of research ever since their first detection in 1912. Despite searching for over a century, we have come significantly closer to an answer only in the past decade, by combining observational results with a proper theoretical treatment of particle propagation and interaction in the Universe. Writing in *Nature Physics*, Ke Fang and Kohta Murase[1] have taken us a step further, by presenting a model that properly takes into account the full propagation from the acceleration site of active galaxies to Earth — capable of describing cosmic-ray, photon and neutrino data at the same time.

Active galactic nuclei are among the most luminous objects in the Universe. This class of objects is defined by the presence of a supermassive black hole in the centre of a galaxy, which is surrounded by an accretion disk. Often, these objects emit giant radio jets that extend far beyond the host galaxy itself, in which cosmic rays could be accelerated. The electromagnetic power of the accretion disk can reach up to 10^{41} W, outshining the entire light of the galaxy as produced by hundreds of billions of stars by many orders of magnitude.

This property is one of several that make active galactic nuclei prime candidates for the acceleration of cosmic rays to extreme energies of 10^{20} eV. However, due to cosmic-ray scattering off cosmic magnetic field inhomogeneities, in combination with a large number of sources emitting cosmic rays, the flux detected at Earth is isotropic. The task of quantitatively verifying or falsifying the hypothesis of active galactic nuclei as cosmic-ray sources is a highly challenging one that scientists have been pursuing ever since the first detection of a cosmic-ray event at 10^{20} eV in the 1960s[2].

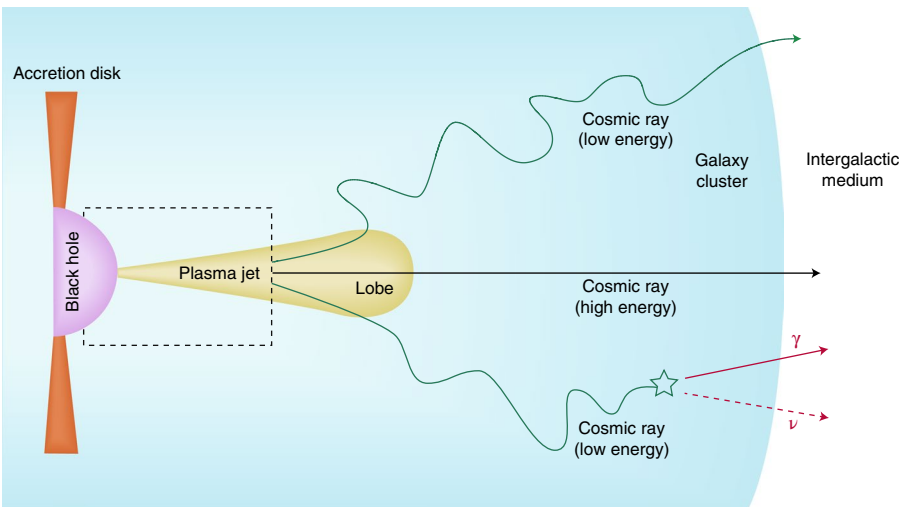

Fig. 1 | The two-step propagation scenario for the generation of cosmic rays from an active galactic nucleus located in a cluster (not to scale). In this scenario, particles are accelerated in the region close to the black hole (dashed box), move along the plasma jet and exit at the lobe region where a shock front is formed. If their energies exceed 2×10^{19} eV, these cosmic rays form a straight path (black) through the cluster. Lower-energy cosmic rays (green) diffuse through the cluster (blue) and may undergo interactions with infrared photons or hydrogen, producing neutrinos and gamma rays (red) that travel straight to Earth. Once they exit the cluster, the cosmic rays are further deflected by magnetic fields and interact with the extragalactic background light to produce more neutrinos and gamma rays. The model[1] is consistent with all observations of cosmic rays, gamma rays and neutrinos on Earth and predicts a dominant contribution to all three messengers.

Progress in the measurement of cosmic-ray observables has advanced with unprecedented success in the past decade: cosmic-ray observatories like the Telescope Array and the Pierre Auger Observatory have been built to quantify energy budget, composition and arrival direction patterns. At the same time, high-energy gamma-ray telescopes such as Fermi, MAGIC, VERITAS and H.E.S.S. have succeeded in determining the properties of high-energy sources in the Universe, to which the interaction of cosmic rays