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Openings and Retrospectives

VIRAL SPEED: Infrastructure, Connectivity, Ontogeny; or, Notes on the Molecular Epidemiology of Epidemics

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An anthropology of speed is drawn to the experience of speed, as was noted in the introduction to this Openings collection, focusing on contemporary forms of acceleration and the foreclosures that result. In this article, I suggest that an anthropology of speed must attend to infrastructures of connection, and to speed as what transforms randomness into facticity. Taking a cue from the semantic clustering of acceleration, speed, and epidemics, and attendant notions of things "going viral," my proposal stems from a consideration of viral speed. At the most fundamental level, viral speed is expressed as the number of infections generated over time, such that a *fast* epidemic like Ebola infects many people in a short period of time, compared to a *slow* epidemic like HIV. Speed is a product of connectivity. Frequent users of urban transit, railway systems, or airlines know that the fastest route from point A to point B is not the shortest one, but the one that takes routes served at the highest frequency, ensuring shorter waiting times and mitigating the impact of missed connections. Molecular epidemiology shows that the speed of viral outbreaks also depends on network effects, suggesting that speed is a function of what connects us. Molecular epidemiology makes visible

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an ontology of speed, of how life is connection and connection makes space and time matter: how life is speed. Viral sociality foreshadows human sociality, at least in the age where human connection increasingly relies on technology. The molecular epidemiology of viral outbreaks illuminates speed as the product of infrastructures of connection.

Let me begin with what is by now the most notorious fast epidemic: the massive Ebola outbreak that struck West Africa in 2014 and continues to (slowly) sputter on. More than ten thousand died from the horrific epidemic of hemorrhagic fever caused by a highly infectious virus—so infectious that even fleeting contact with the bodily fluids of an infected person, or their cadaver, was enough to be contaminated. The virus, moreover, killed up to 90 percent of those infected (although actual figures may be lower). Remarkably, it now appears that this unprecedented, and unpredicted, epidemic, drawn out of a science-fiction scenario, stemmed from one single event: an event that led the virus to be transmitted from an infected bat to a young boy (Baize et al. 2014; Carroll et al. 2015; Mate et al. 2015). This remarkable assertion stems from molecular analyses of the epidemic, and points to how infrastructures of connection that enable speed also stabilize stochastic events.

Molecular epidemiology is a comparatively young science, but one that has come to play a growing role in understanding epidemics. As a science, molecular epidemiology became possible when the ability to sequence the genomes of microorganisms became harnessed to growing computational power from the mid-1990s onward. This allowed the genetic sequences of individual viruses and bacteria to be compared to each other. The degree of difference—or divergence—between genomes allows an analysis of viral kinship. Just as siblings share a hierarchically closer common parent than cousins, the more two organisms resemble each other, the more recently they diverged from a common ancestor. In this way family trees—or phylogenies—of organisms can be constructed, all the way back to a common ancestor or founder. Random mutations occurring as microorganisms replicate are the engine of genetic diversity. Mutations that compromise fitness are choked off, kept from reproducing, as hostile conditions—such as host immune systems—pick them off.

Molecular phylogenies are increasingly called upon to track the spread of epidemics, illuminate transmission dynamics, and pinpoint epidemic origins (see Georges-Courbot et al. 1997 for the earliest example with respect to Ebola). Yet the assumptions embedded in the construction of molecular family trees have been called into question. The first of these is the assumption of homogenous

evolutionary time, the so-called molecular clock, such that mutations occur at a steady rate over time. This stands at odds with theories of quantum evolution, which hold that rates of evolution can vary significantly, particularly in times of accelerated environmental change such as those often associated with the emergence of epidemics. Another assumption is that bursts of differentiation that lead to the founding of new family lines are associated with specific real-world events, such as instances of cross-species transmission known as zoonotic events. Most epidemics are believed to originate when a microorganism crosses the species barrier, leaving an animal host reservoir (such as the chimpanzee for HIV's simian ancestor, or bats for Ebola virus) to infect humans. Zoonotic events are sporadic, occur with variable frequency, and do not always trigger an epidemic. When human/animal interactions are dense and sustained, as in the case of poultry and pig farming in Southeast Asia, zoonotic events are regular occurrences and add a genetic froth to the genetic pool. Within this broth viruses can crystallize and sweep across the globe, as in the case of the annual influenza epidemic. Bursts of microbial evolution can be interpolated from the microbial family tree, and correlations can be attempted with actual real-world events such as a cross-species transmission, a climatic event, or a shift in host-population dynamics. Controversy over using molecular phylogenetic analyses to infer epidemic origins is best exemplified in the debates around the origins of HIV, which began when a British journalist, Edward Hooper, argued that contaminated batches of oral polio vaccine inadvertently spread the virus in the 1950s: a hypothesis that has since been largely discredited by molecular epidemiologists who instead point to rapid urbanization, railways, and sex-ratio imbalances that shifted sexual networks (e.g., Faria et al. 2014).

As an HIV physician, I initially became familiar with molecular epidemiology in the early 2000s when analyses of HIV sequences were used to construct family trees of the virus in the debate over the origins of HIV. Phylogenies were instrumental in adjudicating disputes relating to the origin of this epidemic. Branch points in family trees identify moments of genetic exuberance (the equivalent to an epidemic of outmarriage in a previously endogamous village) that found new populations appearing as bristly clusters in the family trees. By the early 2000s, once genotyping of patients' viruses became routine to detect drug-resistance mutations, I would analyze viral genomes daily as part of my clinical job. Epidemiologists assembled genomes into phylogenies to inform public health efforts. They used these family trees to pinpoint clusters of transmission (visible in the

virtual space of the phylogenetic tree) and thereby provide clues as to where transmission might be occurring in real space and time.

Returning to Ebola, the ability to map the epidemic spread was of crucial significance in a rapidly spreading and lethal epidemic that threatened, at one point, to consume large swaths of West Africa. The tipping point was the epidemic in Liberia's capital city, Monrovia, in the summer of 2014. There, the epidemic spread out of control, hospitals were overrun, and patients died waiting to get in. Bodies were left on the street in scenes that shook the director of the Centers for Disease Control and Prevention, Thomas Frieden, when he came to visit in August 2014 (Frieden et al. 2014).

At the time, in the face of this terrifying, unforeseen, and out-of-control epidemic, urgent questions included those that could only be answered by molecular epidemiologists: namely, was the epidemic the result of an isolated incident, or had the virus somehow mutated and as a result become far more dangerous than previously thought? The need for molecular epidemiological data raised operational and epistemological issues from the get-go. Operationally, highly contagious blood samples had to be drawn from seriously ill patients stricken with Ebola and transported to laboratories in Europe with adequate biosecurity and molecular technological capacity. Epistemologically, molecular epidemiology offered clues—and raised new questions— about how a previously sporadic and rural epidemic, which had until then only ventured once outside of West Africa, had triggered a massive international epidemic that at one point burned uncontrollably in Monrovia.

All the viruses sequenced in this Ebola epidemic share a common ancestor. The phylogenies point back to a single event, perhaps a bat bite or a mango contaminated with an infected bat's saliva or urine and inadvertently consumed, which would have been responsible for an unprecedented epidemic. How could one random event trigger an epidemic? I have heard many explanations in the course of fieldwork in the region over the past two years. All three countries affected by the epidemic are among the poorest in Africa, and indeed the world; two have been ravaged by civil wars. Reconstruction in the wake of these wars added infrastructure—notably, in Sierra Leone, a network of roads that made previously treacherous journeys a thing of the past, and spurred trade. According to this line of reasoning, economic growth intensified travel and allowed the infection to spread along social and economic exchange routes. Other explanations, relating to deforestation and ecological changes due to climate change

(Leroy et al. 2004), seem less promising. Yet what each of these explanations pointed to was the articulation of infrastructure, connectedness, and (viral) speed.

Microbial phylogenies map space and time. Space is plotted out in terms of genetic distance, and it assumes some approximation of real-world geographic and biological distance. Distantly related microorganisms are unlikely to be found in the same hosts or in those from whom they acquired the infection; they are found in individuals separated by a longer chain of transmission than close relatives, whose more recent common ancestor was not too far back in time and not too far back in terms of transmission. In other words, as a microorganism moves from host to host, it mutates. Mutations give the bearer an advantage in response to changing hosts whose immune systems may be more efficient than those of other hosts (at killing certain microbial offspring or variants). Genetic sequences archive space *and* time. Or, alternatively, they are the materialization of speed, space over time. I argue that three ontologies of speed are revealed. The first expresses speed as change, as mutation. Faster transmission materializes as more mutations, more change. The second links change to infrastructures of connection—roads, commerce, and certainly the Internet. The third comprises the sedimentation of speed in viral genomes, snapshots of mutation that constitute stills of ceaseless viral change.

I have always been fascinated with these molecular diagrams and their ability to map previously invisible social relations. HIV is sexually transmitted, which makes it complicated to gather information about transmission networks. Reconstituting the social networks along which HIV spreads requires informants to disclose the identities of sexual partners, so that their sexual partners can in turn be identified—something people are often unwilling or unable to do. To research this, prior consent of sexual partners would be required. As a result, the information obtained through consent would be unrepresentative of the broader pool of sexual partners and of little scientific interest.

Molecular epidemiology provides another approach. As an epidemiologist once told me, "viruses don't lie," and molecular methods allow sexual networks to be elucidated as never before. The closer two viruses are (from two patients) the more "linked" they are—the viruses of two sexual partners are more alike than the viruses of their partners' partners' partners. Molecular epidemiology plots individual viruses sampled from patients by genetic distance, which corresponds to the likelihood that patients are part of the same chain of transmission. Phylogeny recapitulates kinship. Proximity translates into descent. Such studies

have been used to identify so-called clusters of transmission: individuals who share a nearly identical virus and therefore most likely acquired it from each other.

In a fascinating and seminal study, a group of sociologists used another method to reveal sexual networks without requiring informants' disclosure of the names of their sexual partners (Bearman, Moody, and Stovel 2004). In a suburban Colorado school, investigators used a computerized questionnaire to ask students to identify other students they had had sex with from a list of those attending the high school. The computer program anonymized their choices, and generated a map of the sexual network. The map looks a bit like one would imagine an airline network map: spokes branch out from a series of linked hubs. In the article, the authors refer to this as a *spanning tree network*, referring to how tree branches and roots can entangle while trunks line the side of a road. (In this case, the trunks are the interhub links.) Three findings emerge from this remarkable study. The first, which confirms other studies of sexual networks but also of other types of linkage such as Internet searches, suggests that a lucky few are hyperconnected (i.e., have huge numbers of partners), the majority have a much smaller number, and an unlucky few have very little. This corresponds to common sense and, indeed, the normal distribution found with many naturally occurring phenomena. When translated into a network, however, this logarithmically increases the degree of connection and potential exposure of individuals the closer they get to the hubs of the sexual network. The second relates to the structure of the network. Since choice of sexual partners is not random, the authors tried to identify rules that would predict the structure they found. They found one, which they called *the yuck factor*: partners did not hook up with ex-partners' current partners' former partners. What is striking here is that this is a kinship rule. In Lévi-Straussian terms, this is a rule that generates exogamy. The third is a practical consequence: just by diminishing by a small number the amount of sexual partners, the sexual network disaggregates, isolating hubs from each other. Small changes in a network's connectivity can transform its overall structure. Molecular epidemiology reveals a similar picture, with bursts of viral evolution suggesting highly efficient transmission hubs (or "super-spreaders") that push out large numbers of viruses, which then end up languishing in the evolutionary dead ends of sexual monogamy.

For many years, I presented these studies to make a case for social structure, in the sense first advocated by Émile Durkheim. Contrary to Claude Lévi-Strauss's view, the formal properties of social structure are neither linguistic nor a property of mind; they reflect the ontology of connection. Even in what would seem to

be the most intimate and individualized sphere—our choice of sexual partners—we obey algorithmic rules, a powerful argument that the social is structured independently of individual volition or agency. In this article, however, I have explored another angle. I have argued that speed is also an ontology of change, of viral mutation that functions as the irreducible element of speed. This is what enables speed to be mapped in molecular epidemiologies, which constitute sedimented residues of ceaseless mutation, archived in viral genomes. Rather than the inability to imagine a future, in this case speed is instead the ceaseless possibility offered by the random mutation of life.

REFERENCES

Baize, Sylvain, et al.
2014 "Emergence of Zaire Ebola Virus Disease in Guinea." *New England Journal of Medicine* 371: 1418–25. https://doi.org/10.1056/NEJMoa1404505.

Bearman, Peter S., James Moody, and Katherine Stovel
2004 "Chains of Affection: The Structure of Adolescent Romantic and Sexual Networks." *American Journal of Sociology* 110, no. 1: 44–91. https://doi.org/10.1086/386272.

Carroll, Miles W., et al.
2015 "Temporal and Spatial Analysis of the 2014–2015 Ebola Virus Outbreak in West Africa." *Nature* 524: 97–101. https://doi.org/10.1038/nature14594.

Faria, Nuno R., et al.
2014 "The Early Spread and Epidemic Ignition of HIV-1 in Human Populations." *Science* 346, no. 6205: 56–61. https://doi.org/10.1126/science.1256739.

Frieden, Thomas R., Inger Damon, Beth P. Bell, Thomas Kenyon, and Stuart Nichol
2014 "Ebola 2014—New Challenges, New Global Response and Responsibility." *New England Journal of Medicine* 371: 1177–80. https://doi.org/10.1056/NEJMp1409903.

Georges-Courbot, Marie-Claude, et al.
1997 "Isolation and Phylogenetic Characterization of Ebola Viruses Causing Different Outbreaks in Gabon." *Emerging Infectious Diseases* 3, no. 1: 59–62. https://doi.org/10.3201/eid0301.970107.

Leroy, Eric M., et al.
2004 "Multiple Ebola Virus Transmission Events and Rapid Decline of Central African Wildlife." *Science* 303, no. 5656: 387–90. https://doi.org/10.1126/science.1092528.

Mate, Suzanne E., et al.
2015 "Molecular Evidence of Sexual Transmission of Ebola Virus." *New England Journal of Medicine* 373: 2448–54. https://doi.org/10.1056/NEJMoa1509773.