

How the Persians Were Saved by Lightning

Causation and the collapse of possibilities

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The events in history can have the most bizarre causes and the most dramatic consequences. In 283 AD, the fearsome Roman army, under Emperor Carus (Fig. 1), was camped on the frontier of Persia. They had recently booted the Persian forces out of Roman territory, and Carus hungered for more. As told by Edward Gibbon in *The Decline and Fall of the Roman Empire*, for the Romans “Flattery and hope painted, in the most lively colors, the fall of Persia, the conquest of Arabia, the submission of Egypt.”^{1:191}

But before their assault on Persia, the Roman camp was deluged with fierce thunderstorms. The Emperor was ill with some unspecified disorder, and to add insult to injury, his pavilion was struck by lightning. Afterwards, when the scorched tent was entered, Carus was found dead. “The power of opinion is irresistible,” wrote Gibbon. “Places or persons struck with lightning were considered...with pious horror as singularly devoted to the wrath of Heaven.”^{1:190} The terrified army felt they were being instructed by the gods to stay behind the River Tigris as the proper Roman border, “and the Persians wondered at the unexpected retreat of a victorious enemy.”^{1:191}

Gibbon and history have assumed that the Roman army was superior to the Persian army, and that had the war ensued, booty and splendor aplenty would have been paraded in a glorious triumph through the streets of Rome. But we’ll never know because the possibilities were made moot by a stroke of luck, and what actually happened was that the Persians were not, in fact, defeated.

There’s no doubt about the importance of lightning strikes in nature. We understand the basic principles by which electrical charges build up in the atmosphere, but we can only talk probabilistically about their discharges (Fig. 2): their specifics, whether they will hit a tree near you—or you—are unpredictable. Does this reflect a defect in our theory of physics, or are probabilistic statements *themselves* the proper theory of such events?

Chance is fundamentally important in life, too. Genetic mutations and the Mendelian transmission of alleles (genetic variants) from parent to offspring are fundamentally “random” occurrences, meaning that the chromosomal location of a mutation, or the allele that is transmitted to a given child, can only be predicted probabilistically.

Genetic variation has important effects on the life history of individuals, who start out as single fertilized egg cells. In each of the subsequent billions of cell divisions that occur, a few new mutations are scattered across the copies of the genome in the new cells. These changes are inherited by the cell’s lineage of descendant cells during the individual’s life. They thus accumulate and proliferate through somatic (body) cellular

history and, along with environmental exposures, affect the person’s traits. You do not have a single genotype but instead are a cellular mosaic that is not predictable from the genotype with which your life started. There is no turning back, and each individual’s unique biological nature is affected by the functional effects of their unique patchwork of randomly occurring genetic variation.

The same is true in evolutionary terms. Mutations are inherited and proliferate through generational history. Since each is a random occurrence, the variation in the next generation, much less thousands of generations from now, is not specifically predictable. So chance seems deeply built into life—or is it?

DOES CHANCE ACTUALLY EXIST?

In a bizarre way of ignoring knowledge and trusting to fate, we often flip coins to help us through life. Taking a chance on life in this way reflects one’s view—belief, really—about the degree to which random events truly occur in nature.

A coin may be declared “fair” if it comes up heads roughly half the time. However, a coin-flipping machine built by Stanford statistician Persi Diaconis showed that if the coin always starts out facing the same way in the machine’s controlled environment, the result is the same every time (<http://www-stat.stanford.edu/~cgates/PERSI/>). So a coinflip is not an inherently random event at all. Presumably, there is so much imperfection in our knowledge of the factors that affect the outcome that thumb-based flips only *seem* random.

How general is this? Mendelian inheritance appears to be random at

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Figure 1. Image of the Roman Emperor Marcus Aurelius Carus, ca AD 283. Public domain.

the molecular level: whether a given sperm or egg receives, say, a parent's **A** or **a** allele seems, like a coinflip, entirely due to chance. But if we could peer into each cell before it starts dividing, would the transmitted allele be perfectly predicable? Could we make the same perfect prediction many cell divisions earlier? If so, could we get this information without disturbing the system and altering what would have happened were we not looking? How could we tell?

Coinflips and Mendelian transmission are repeatable phenomena, which is really what "probability" and "random" refer to, and they also are causally independent events in that the outcome of one instance seems not to affect the outcome of subsequent instances. It may be an illusion, but each trial appears to have the same underlying probability, so that if enough coins are flipped or enough children are born to an individual; their outcomes will *converge* on that value (50% for fair coins and genes). Even if the world is purely deterministic, our amount of uncertainty somehow evens out.

By contrast, mutation and lightning strikes are not really repeatable events of this kind, and it is not clear whether they *have* underlying probabilities—or even what it would mean for a unique event in history to have a "probability." Instead, we use some indirect criteria for declaring them to be "random." We look at many mutations and find that they have

occurred roughly as often everywhere in the genome as would be expected if the causes were blind to the place on a chromosome they strike. But that is very different from repeatedly subjecting the *same* copy of a DNA sequence to multiple hits.

In fact, mutation is not uniform across the genome, and Mendelian segregation is not always a 50–50 proposition. Such deviations from an assumed theory draw a lot of attention, and we now know that for chemical reasons, certain areas of the genome are more likely to be mutated than are others; examples are changes in the length of consecutive runs of the same nucleotide, and **C**'s that are chemically modified to affect gene expression are vulnerable to chemical conversion to **T**'s (see Wikipedia "mutation"). Similarly, some genes experience *meiotic drive* (or *segregation distortion*), in which transmission of a heterozygous parent's alternative alleles to its offspring is unequal; one example whose mechanism is becoming known affects sex ratios in flies and may have to do with sex-chromosome competition.^{2,3} However, even when we understand these special situations, their outcomes are still probabilistic: the exceptions show that our theory was too simple, but do not address the determinism question.

When what we care about is not individual events but the *aggregate* his-

tory of outcomes such as mutations or lightning strikes over an army encampment, we can treat unique random events as if they were repeatable, reflecting the probability of a mutation *somewhere* on the genome per generation, for example. That is what we do in assessing phylogenies by using molecular clocks, relating the accumulated variation in DNA sequences to the time since they diverged from a common ancestor. Molecular clocks work because of the independence assumption that one mutational event does not affect subsequent ones.

But the same is *not* true in important areas of life where DNA *does* "remember" specifically what happened to it.

THE LEGACY OF CHANCE

Mutations are transmitted across generations and generate divergence among lineages of descendant DNA sequences. It can be shown mathematically that if transmission is probabilistic, as it appears to be, then because populations are finite, every mutation's descent lineage will eventually either become fixed, displacing all others, or go extinct. This is variation forever lost to nature, an irretrievable legacy of chance.

One often hears that if a genetic variant has no effect on the orga-

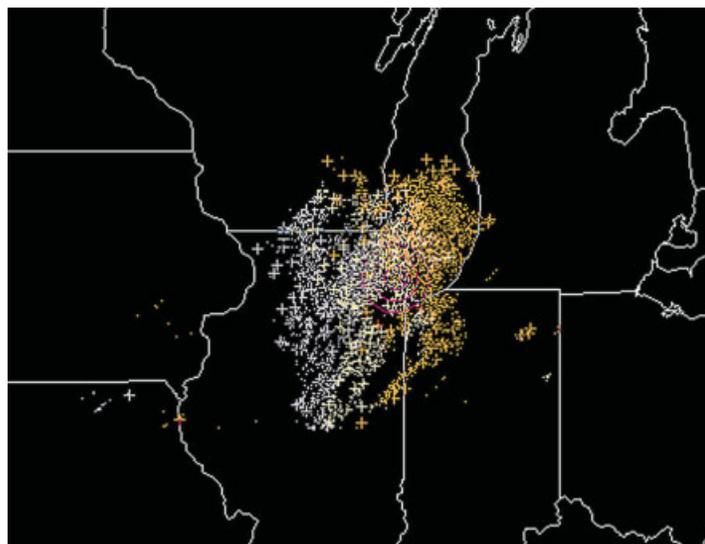


Figure 2. Lightning in a storm area around Chicago. The specific location of individual strikes can only be predicted probabilistically. Satellite image courtesy Nels Shirer, from the NOAA National Severe Storms Laboratory.

nism, it cannot be “seen” by natural selection, and although it will change frequency by chance (genetic drift), it cannot affect the functional nature of life. But that is a serious mistake. It is reproductive “Darwinian” *fitness*, not phenotypes, that count in evolution, and a genetic variant *can* have a functional effect that will drift in frequency if it does not affect fitness. Traits vary and change over time due to phenotypic drift, not just selection, and examples include anthropologically important traits such as skeletal shape.⁴⁻⁹ The shapes of tomorrow depend on what is here today, regardless of how it got here.

To some very deterministic biologists, reproduction is *not* left to chance! They refuse to concede that a trait—or even an aspect of DNA sequence—is here unless it had a selective function,¹⁰ and determined searches are made to find it. Some see in life a kind of molecular inevitability, in which the properties of its molecules, including genes, are such that function can be predicted from their necessary interactions.¹¹⁻¹⁵ For example, a signaling molecule and its receptor will bind to each other, or Cs will bind to Gs in DNA—they do not have any choice. In a similar vein, because development is a complex, hierarchically timed trellis of interactions among countless molecules, animal form is said to be *canalized* (channeled) the way water flows downhill in river beds, restricting the types of change that can evolve.¹⁶⁻¹⁸ Traits that may seem unrelated, such as the parts of a skull, are sometimes correlated by *genetic integration*, if their development is affected by some of the same genes.¹⁹⁻²²

At least in the short run, genetic integration seems to constrain what natural selection can do to a trait. In a kind of evolutionary momentum, once a basic body plan is established, it delimits its descendants, which is why, some argue, no new body plans have arisen since the Burgess Shale some 500 million years ago. If that is true, then it also would have been true of the conditions that led to the Burgess Shale, and molecular inevitability can—must?—be extended, in an endless extrapolation, back to the first bubbling of molecular life in the primordial soup.

Such constraints leave little room for chance, but if we follow that road, it will take us to some ironic turns.

WHAT IF THE FAULT, DEAR BRUTUS, IS IN OUR STARS?

Suppose the universe was *entirely* deterministic, with nothing whatever truly based on chance. A nature driven by deterministic laws propels everything in clockwork fashion. Only ignorance leads us to think that by flipping coins we can decide “by chance” who kicks off in a football game.

From this point of view, there is no such thing as drift, genetic or otherwise—only its appearance. Nothing falls to chance, not even an army deterred by lightning: the storms were building as the Romans confidently pitched their tents on the Persian frontier. The tape of life was prerecorded in the factory of the Big Bang. With God’s computer, everything would be predictable. Indeed, the gearing of a wholly clockwork universe connects everything uniquely to everything else. If that is how things are we will have to concede that astrologers are right after all: the position of the stars is precisely related to what will happen to you today or in your future. And forget about privacy:

I could use *my* astrology chart (Fig. 3) to explain *your* life!

If everything is determined, not only is there no genetic drift but the ironic twist is that there is no natural selection either, because selection is a competitive phenomenon, and if everything is prewired, there is only the illusion of competition. Adaptation would then be like professional wrestling, a rigged show with no true struggle to see who wins. The appearance of adaptive order is just the unfolding of biochemical inevitability. Evolutionary biology is the pursuit of illusions.

However, if to the contrary the universe is *not* just clockwork and true chance does exist, then even natural selection, so often assumed to be life’s archetypal “force”, must rest on non-determinism, and not be a force at all. There must be some true, not just illusionary, chance that the hare escapes the hound or that the Big Bruiser will be pinned by Mr. Wimp. Chance is an essential component of selection, and statistical estimation is our best and only hope of identifying and assessing its role. But then what is the nature of these chance competitive events? Since each hare chase is a singular event, is there really some underlying “probability” involved?

A similar irony can be turned around and said of genetic drift.

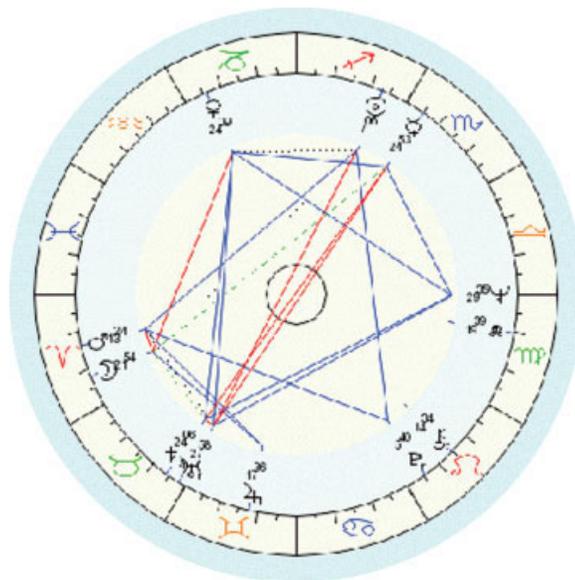


Figure 3. Astrology chart. From www.astgro.com.

Genetic drift is purported to be what happens to selectively neutral variation. But what does it mean to say that variation is selectively neutral? It means, in essence, that each competing genotype has the same chance of reproducing—*exactly* the same chance. Since it is literally impossible to show that two things have exactly the same properties, which would require infinitely precise measurement, pan-selectionism is completely right! No two genotypes have exactly the same fitness. There can be no true drift: even if across a particular generation exactly the same proportion of alleles are transmitted, this is only a chance outcome giving the illusion of fitness equality. That is easy to see, for example, in that 10 coin flips coming up 50% heads does not prove a coin is fair.

When we think of how chance makes, breaks, savors, or favors variation in evolution, we are in a kind of never-never land between determinism and probability. Things may be so similar that we have no ability to discern their difference when chance is involved. Things might be deterministic, but our knowledge too limited to know it. Apparent randomness can arise if the causal connections between separate events are so distant that we cannot detect them. This even might be the case in a fully deterministic universe, say between the astrological position of the stars and whether it is a good day to propose marriage. A cosmic ray may be making a beeline to mutate a particular nucleotide in the DNA in some particular cell in your brain. But in a clockwork universe, their most recent causal connection may have been before the beginning of life on Earth.

In the end, natural selection and drift are not different phenomena but instead are aspects of a single reproductive probability spectrum, reflecting events that may be so singular that they do not actually have underlying probabilities.²³ Despite its intensity, the selection-neutralist controversy is at its core not about incompatible facts but rather about how to assess that spectrum: what criteria, like cutoff *p* values (statistical significance levels), do we use—do we *choose*—to declare that some-

thing is due to selection whose “cause” we might identify? This is not a trivial point since most natural selection appears, on average at least, to be so weak that evolutionary outcomes are barely different from those that would be due to chance alone—differences that might at any (or every) given time be undetectable with achievable sample sizes.^{23,24}

The contingent nature of life-as-we-see-it is such that once a “chance” event occurs, things can go on a very different tangent, and change in the state of today, whatever its true origin, affects the state of *all* tomorrows. Afterwards, those contingencies are usually unknown, and therein lie many problems of deep practical importance in understanding the history of life on both developmental and evolutionary time scales.²³

THE COLLAPSE OF POSSIBILITIES TO A UNIQUE TRUTH

An important objective of genetics is to predict an individual’s phenotype—say, disease or stature—from his or her genotype. In evolutionary biology, we would like to predict the future directions of adaptive selection given the genetic and environmental situation at any given time. We usually confess that we cannot do this except probabilistically in the biomedical case, and most evolutionary biologists (though not, perhaps, the most deterministic) would say that beyond general aspects of canalization, we cannot predict the adaptive future of life at all.

Nonetheless, to the extent that determinism exists, or equivalently in practical terms that underlying probabilities are stable, we regularly try to “retrodict” causation by knowing the outcome—a phenotype or adaptive trait—and inferring its underlying developmental or evolutionary history. We see members of dairying populations who, for genetic reasons, produce the milk-digesting enzyme lactase into adulthood and infer that dairying gave their ancestors a slightly higher probability of producing offspring than their peers without the favored genotype.^{25,26} The past may be prologue, but we do not have direct access to the past, so

such reconstructions should properly be couched in circumspect terms. It is a longstanding problem in evolutionary biology, with few unambiguous answers, and separates the probabilists from the determinists, those who see contingency more than inevitability in life.

A good illustration of the issues is the genetic risk of breast cancer in women who carry specific known variants in the BRCA1 and BRCA2 genes. These are among the most high-risk genotypes for multifactorial traits, and nobody doubts their causal role. What one wants to do is provide *future* risk estimates for young individuals with those genotypes today so that appropriate prevention or therapy can be applied. But this is based on risks that were estimated retrospectively, and future risk estimates inherently assume that the myriad causal factors, mainly unknown, that affected past risk will persist into the future. But we know that is often dramatically not the case. For example, the risk of cancer by age 60 associated with the known BRCA1/2 alleles rises from around 45 to 90%, depending on whether a woman was born before or after 1940.²⁷ The situation is actually even more problematic: Is everyone with a BRCA mutation at the same average risk as her peers, or are some at very high and others at minimal risk? There are many other similar examples. Diabetes is pandemic in Native Americans and appears to have a genetic component, yet was a rare disease before about 1950.²⁸ A specific allele in a gene called HFE is present in most northern Europeans with the iron-storage disease hemochromatosis, but in a random sample of the population, this common allele generally has little or no effect.^{29,30}

We use words like “probability” for these kinds of biological phenomena, but that is rather misleading. The situation is not like coinflipping, where there at least seems to be a common probability underlying repeatable trials. Every individual may be too different for that to be so. This is a deep epistemological problem; the connections between genotype and phenotype, or between phenotype and fitness, are problematic regard-

less of whether the universe is truly deterministic or truly probabilistic because we usually do not know what are the risk factors, and hence the risk, even if deterministic.

Viewed prospectively, many different things might happen when some or all of the causal components are probabilistic (or, if deterministic, unknown). From today forward, we usually have little ability to predict with confidence which will happen. History is the collapse of today's many *possibilities* into a single tomorrow—what actually *did* happen. Viewed retrospectively, this collapse generates an illusion of causality that is all too tempting to extrapolate into the future as if the past were deterministic.

That is what Gibbon did as he surveyed Roman history. He offered causal explanations that can be endlessly disputed because with complex causation, there is as yet no method of resolving the collapse of possibilities in contingent histories—of society, organisms, or species. Gibbon blamed the eventual fall of Rome on the passive acceptance of Christians who, unlike the pagan Romans, expected their rewards to come at the end of their lives, not their swords. Christian historians, feeling the thrust of this view, parried vehemently against Gibbon's heresy. The same kinds of issues and arguments pertain to what genes do from conception onward in the history of individuals, and from mutation onward in the history of species.

Whether the universe is a mechanical or a probabilistic clock, there seems no prospect that we will understand it enough to predict the contingent, nonrepeatable evolutionary future with precision. In some ways, this is a liberating fact: we are freed from having to address the ultimate philosophical question whether anything in life is truly due to chance. Indeed, we are more than freed from it: that truth is barred from us. We will never know if we wend our professional lives chasing illusion.

SO HOW WERE THE PERSIANS SAVED BY LIGHTNING?

Is this all just mind games, or is it important? To some of us, a desire

to know the truth about nature makes it matter. But if we cannot accurately predict phenotypes from genotypes, our ability to solve practical problems such as individualized genetic prognosis and treatment will be correspondingly limited. We also will be kept at arm's length from evolution, too. If genotypes have weak or unstable predictive value, that reduces the potential impact or strength of natural selection and our ability to reconstruct the true reasons for speciation and adaptive evolution.

Replication sometimes helps. We can estimate the probability of breast cancer in different samples of BRCA mutation carriers or the selective advantage of black, peppered moths on sooty trees.³¹ If we get similar results, we begin to accept our explanations. But few selective scenarios are solidly established. Only a few malaria-associated mutations have been proven truly to be protective, though the geographic distribution of many others, in relation to malarial environments, suggests that they are. Adult lactase persistence seems convincing because it has evolved independently in different populations; however, the associated chromosome region is very large and the phenotypic effect rather modest, with little if any bearing on fitness today, so the actual selective cause in the past might have been something else entirely.²⁶ There is a panoply of measures available to detect evidence of selection from DNA sequence comparisons,^{32–34} but they are fundamentally statistical. Important practical applications, such as disease risk estimates based on chance events improperly understood, can be highly inaccurate. This is why the legitimacy of the many new services offering genetic risk estimates is controversial.^{35–37} At the moment, at least, we usually have to be happy with statistical, and hence subjective, confidence in our explanations.

The idea of randomness may itself seem dodgy, as perhaps the coin-flipping machine shows. Is chance an inevitable product of measurement error, or are we thinking about nature in the wrong way? Science is uneasy with claims that aspects of nature have no cause we could ever understand. Declaring that the world

is truly deterministic but will unavoidably appear probabilistic could get you accused of being a scientific defeatist (or worse, a post-modernist). And it is not very satisfying, if you seek genotype-based predictions or evolutionary scenarios that you want to know are true. On the other hand, declaring the world to be inherently random could seem mystical. After all, does randomness have some sort of "cause", and if so, how can a cause yield probabilities?

In an army, as with the loss of an allele in genetics, death is forever, whether its cause be the chance arc of a flying arrow, a stumbling horse, some systematic force—or a lightning bolt. The Romans had no satellite images or theory of meteorology by which to judge the capricious location of lightning strikes. Gibbon acknowledges the uncertainties by citing a letter by Emperor Carus' secretary to the Roman Praefect, who calmly wrote that rather than lightning (or the will of the gods), "as far as we have been able to investigate the truth, his death was the natural effect of his disorder."^{1:190} If so, a chance event, *improperly* perceived as deterministic, saved the Persian empire.

NOTES

I welcome comments on this column: kenweiss@psu.edu. I have a feedback and supplemental material page at http://www.anthro.psu.edu/weiss_lab/index.shtml and blog on similar topics, The Mermaid's Tale, at <http://ecodevoevo.blogspot.com>. Thanks to Anne Buchanan, Ellen Quillen, Sam Sholtis, and John Fleagle for critically reading this manuscript. This column was written with financial assistance from funds provided to Penn State Evan Pugh professors.

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