

The Rules of Contagion

Why Things Spread –
and Why They Stop

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Introduction

A FEW YEARS AGO, I accidentally caused a small outbreak of misinformation. On my commute to work, a friend who works in tech had sent me a stock photo of a group hunched over a table wearing balaclavas. We had a running joke about how news articles on computer hacking would often include staged pictures of people looking sinister. But this photo, below a headline about illicit online markets, had taken things much further: as well as balaclavas, there was a pile of drugs, and a man who apparently wasn't wearing any trousers. It seemed so surreal, so inexplicable.

I decided to tweet it. 'This stock photo is fascinating in so many ways,' I wrote,¹ pointing out all the quirks in the image. Twitter users seemed to agree, and within minutes dozens of people had shared and liked my post, including several journalists. Then, just as I was starting to wonder how far it might spread, some users pointed out that I'd made a mistake. It wasn't a stock photo at all; it was a still image from a documentary about drug dealing on social media. Which, in retrospect, made a lot more sense (apart from the lack of trousers).

Somewhat embarrassed, I posted a correction, and interest soon faded. But even in that short space of time, almost fifty thousand people had seen my tweet. Given that my job involves

analysing disease outbreaks, I was curious about what had just happened. Why did my tweet spread so quickly at first? Did that correction really slow it down? What if people had taken longer to spot the mistake?

Questions like these crop up in a whole range of fields. When we think of contagion, we tend to think about things like infectious diseases or viral online content. But outbreaks can come in many forms. They might involve things that bring harm – like malware, violence or financial crises – or benefits, like innovations and culture. Some will start with tangible infections such as biological pathogens and computer viruses, others with abstract ideas and beliefs. Outbreaks will sometimes rise quickly; on other occasions they will take a while to grow. Some will create unexpected patterns and, as we wait to see what happens next, these patterns will fuel excitement, curiosity, or even fear. So why do outbreaks take off – and decline – in the way they do?

THREE AND A HALF YEARS into the First World War, a new threat to life appeared. While the German army was launching its Spring Offensive in France, across the Atlantic people had started dying at Camp Funston, a busy military base in Kansas. The cause was a new type of influenza virus, which had potentially jumped from animals into humans at a nearby farm. During 1918 and 1919, the infection would become a global epidemic – otherwise known as a pandemic – and would kill over fifty million people. The final death toll was twice as many as the entire First World War.²

Over the following century, there would be four more flu pandemics. This raises the obvious question: what will the next one look like? Unfortunately it's difficult to say, because previous flu pandemics were all slightly different. There were different strains of the virus, and outbreaks hit some places harder than

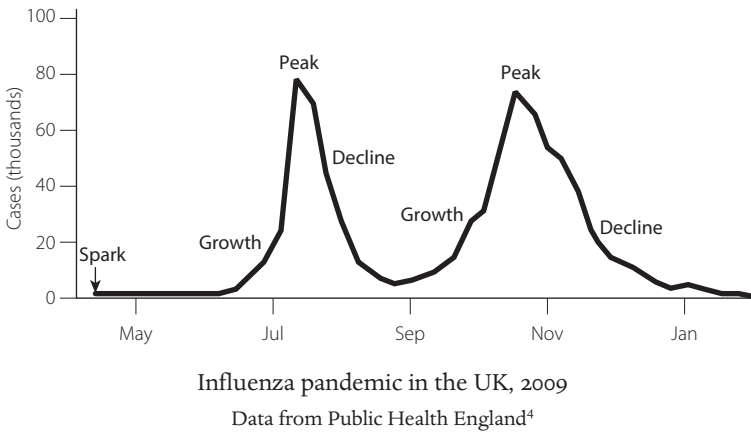
others. In fact, there's a saying in my field: 'if you've seen one pandemic, you've seen ... one pandemic.'³

We face the same problem whether we're studying the spread of a disease, an online trend, or something else; one outbreak won't necessarily look like another. What we need is a way to separate features that are specific to a particular outbreak from the underlying principles that drive contagion. A way to look beyond simplistic explanations, and uncover what is really behind the outbreak patterns we observe.

That's the aim of this book. By exploring contagion across different areas of life, we'll find out what makes things spread and why outbreaks look like they do. Along the way, we'll see the connections that are emerging between seemingly unrelated problems: from banking crises, gun violence and fake news to disease evolution, opioid addiction and social inequality. As well as covering the ideas that can help us to tackle outbreaks, we'll look at the unusual situations that are changing how we think about patterns of infections, beliefs, and behaviour.

Let's start with the shape of an outbreak. When disease researchers hear about a new threat, one of the first things we do is draw what we call an outbreak curve – a graph showing how many cases have appeared over time. Although the shape can vary a lot, it will typically include four main stages: the spark, growth, peak, and decline. In some cases, these stages will appear multiple times; when the 'swine flu' pandemic arrived in the UK in April 2009, it grew rapidly during early summer, peaking in July, then grew and peaked again in late October (we'll find out why later in the book).

Despite the different stages of an outbreak, the focus will often fall on the spark. People want to know why it took off, how it started, and who was responsible. In hindsight, it's tempting to conjure up explanations and narratives, as if the outbreak was inevitable and could happen the same way again. But if we simply list the characteristics of successful infections or trends,



we end up with an incomplete picture of how outbreaks actually work. Most things don't spark: for every influenza virus that jumps from animals to humans and spreads worldwide as a pandemic, there are millions that fail to infect any people at all. For every tweet that goes viral, there are many more that don't.

Even if an outbreak does spark, it's only the start. Try and picture the shape of a particular outbreak. It might be a disease epidemic, or the spread of a new idea. How quickly does it grow? Why does it grow that quickly? When does it peak? Is there only one peak? How long does the decline phase last?

Rather than just viewing outbreaks in terms of whether they take off or not, we need to think about how to measure them and how to predict them. Take the Ebola epidemic in West Africa back in 2014. After spreading to Sierra Leone and Liberia from Guinea, cases began to rise sharply. Our team's early analysis suggested that the epidemic was doubling every two weeks in the worst affected areas.⁵ It meant that if there were currently 100 cases, there could be 200 more in a fortnight and another 400 after a month. Health agencies therefore needed to respond quickly: the longer it took them to tackle the epidemic, the larger their control efforts would need to be. In essence,

opening one new treatment centre immediately was equivalent to opening four in a month's time.

Some outbreaks grow on even faster timescales. In May 2017, the WannaCry computer virus hit machines around the world, including crucial NHS systems. In its early stages, the attack was doubling in size almost every hour, eventually affecting more than 200,000 computers in 150 countries.⁶ Other types of technology have taken much longer to spread. When VCRs became popular in the early 1980s, the number of owners was doubling only every 480 days or so.⁷

As well as speed, there's also the question of size: contagion that spreads quickly won't necessarily cause a larger overall outbreak. So what causes an outbreak to peak? And what happens after the peak? It's an issue that's relevant to many industries, from finance and politics to technology and health. However, not everyone has the same attitude to outbreaks. My wife works in advertising; while my research aims to stop disease transmission, she wants ideas and messages to spread. Although these outlooks seem very different, it's increasingly possible to measure and compare contagion across industries, using ideas from one area of life to help us understand another. Over the coming chapters, we will see why financial crises are similar to sexually transmitted infections, why disease researchers found it so easy to predict games like the ice bucket challenge, and how ideas used to eradicate smallpox are helping to stop gun violence. We will also look at the techniques we can use to slow down transmission or – in the case of marketing – keep it going.

Our understanding of contagion has advanced dramatically in recent years, and not just in my field of disease research. With detailed data on social interactions, researchers are discovering how information can evolve to become more persuasive and shareable, why some outbreaks keep peaking – like the 2009 flu pandemic did – and how 'small-world' connections between distant friends can help certain ideas spread widely (and yet

hinder others). At the same time, we're learning more about how rumours emerge and spread, why some outbreaks are harder to explain than others, and how online algorithms are influencing our lives and infringing on our privacy.

As a result, ideas from outbreak science are now helping to tackle threats in other fields. Central banks are using these methods to prevent future financial crises, while technology firms are building new defences against harmful software. In the process, researchers are challenging long-held ideas about how outbreaks work. When it comes to contagion, history has shown that ideas about how things spread don't always match reality. Medieval communities, for example, blamed the sporadic nature of outbreaks on astrological influences; influenza means 'influence' in Italian.⁸

Popular explanations for outbreaks continue to be overturned by scientific discoveries. This research is unravelling the mysteries of contagion, showing us how to avoid simplistic anecdotes and ineffective solutions. But despite this progress, coverage of outbreaks still tends to be vague: we simply hear that something is contagious or that it's gone viral. We rarely learn why it grew so quickly (or slowly), what made it peak, or what we should expect next time. Whether we're interested in spreading ideas and innovations, or stopping viruses and violence, we need to identify what's really driving contagion. And sometimes, that means rethinking everything we thought we knew about an infection.

A theory of happenings

WHEN I WAS THREE YEARS OLD, I lost the ability to walk. It happened gradually at first: a struggle to stand up here, a lack of balance there. But things soon deteriorated. Short distances became tricky, while slopes and stairs were near impossible. One Friday afternoon in April 1990, my parents took me and my failing legs to the Royal United Hospital in Bath. By the next morning I was seeing a neurological specialist. The initial suspect was a spinal tumour. Several days of tests followed; there were X-rays, blood samples, nerve stimulation, and a lumbar puncture to extract spinal fluid. As the results came in, the diagnosis shifted towards a rare condition known as Guillain-Barré syndrome (GBS). Named after French neurologists Georges Guillain and Jean Alexandre Barré, GBS is the result of a malfunctioning immune system. Rather than protecting my body, it had started attacking nerves, spreading paralysis.

Sometimes the sum of human wisdom is to be found, as writer Alexandre Dumas put it, within the words ‘wait and hope’.¹ And that was to be my treatment, to wait and to hope. My parents were given a multicoloured party horn to check the strength of my breathing (there was no home equipment small enough for a toddler). If the horn failed to unroll when I blew, it meant the paralysis had reached the muscles that pumped air into my lungs.

There is a photo of me sitting on my grandfather's lap around this time. He is in a wheelchair. He'd caught polio in India aged twenty-five, and had been unable to walk since. I'd only ever known him like that, his strong arms wheeling uncooperative legs. In a way, it brought familiarity to this unfamiliar situation. Yet what linked us was also what separated us. We shared a symptom, but the mark of his polio was permanent; GBS, for all its misery, was usually a temporary condition.

So we waited and we hoped. The party horn never failed to unroll, and a lengthy recovery began. My parents told me GBS stood for 'Getting Better Slowly'. It was twelve months before I could walk, and another twelve before I could manage anything resembling a run. My balance would suffer for years to come.

As my symptoms faded, so did my memories. Events became distant, left behind to another life. I can no longer remember my parents giving me chocolate buttons before the needles. Or how I subsequently refused to eat them – even on a normal day – fearing what would come next. The memories of games of tag at primary school have faded too, with me spending all of lunchtime as 'it', my legs still too weak to catch the others. For the twenty-five years that followed my illness, I never really spoke about GBS. I left school, went to university, completed a PhD. GBS seemed too rare, too meaningless to bring up. Guilt-lain-what? Barré who? The story, which I never told anyway, was over for me.

Except it wasn't quite. In 2015, I was in the Fijian capital Suva when I encountered GBS again, this time professionally. I'd been in the city to help investigate a recent dengue fever epidemic.² Transmitted by mosquitoes, the dengue virus causes sporadic outbreaks on islands like Fiji. Although symptoms are often mild, dengue can come with a severe fever, potentially leading to hospitalisation. During the first few months of 2014, over 25,000 people showed up at health centres in Fiji with a suspected dengue infection, putting a huge burden on the health system.

If you're imagining an office perched on a sunny beach, you're not picturing Suva. Unlike Fiji's resort-laden Western division, the capital is a port city in the southeast of the main island, Viti Levu. The two main roads of the city loop down into a peninsula, forming the horseshoe shape of a magnet, with the area in the middle attracting plenty of rain. Locals who were familiar with British weather told me that I'd feel right at home.

Another, much older, reminder of home was to follow soon after. During an introductory meeting, a colleague at the World Health Organization (WHO) mentioned that clusters of GBS had been appearing on Pacific Islands. Unusual clusters. The annual par for the disease was 1 or 2 cases per 100,000 people, but in some places they'd seen double figures.³

Nobody ever worked out why I got GBS. Sometimes it follows an infection – GBS has been linked to flu and pneumonia, as well as other diseases⁴ – but sometimes there's no clear trigger. In my case, the syndrome was just noise, a random blip in the grand scheme of human health. But in the Pacific during 2014/15, GBS represented a signal, just like birth defects would soon do in Latin America.

Behind these new signals lay the Zika virus, named after the Zika Forest in southern Uganda. A close relative of the dengue virus, Zika was first identified in the forest's mosquitoes in 1947. In the local language, Zika means 'overgrown'⁵ and grow it would, from Uganda to Tahiti to Rio de Janeiro and beyond. Those signals in the Pacific and Latin America in 2014 and 2015 would gradually become clearer. Researchers found increasing evidence of a link between Zika infection and neurological conditions: as well as GBS, Zika seemed to lead to pregnancy complications. The main concern was microcephaly, where babies develop a smaller brain than usual, resulting in a smaller skull.⁶ This can cause a host of serious health issues, including seizures and intellectual disabilities.

In February 2016, triggered by the possibility that Zika was

causing microcephaly,⁷ who announced that the infection was a Public Health Emergency of International Concern, or PHEIC (pronounced 'fake'). Early studies had suggested that for every 100 Zika infections during pregnancy, there could be between 1 and 20 babies with microcephaly.⁸ Although microcephaly would become the primary concern about Zika, it was GBS that first brought the infection into health agencies' focus, as well as into mine. Sitting in my temporary office in Suva in 2015, I realised that this syndrome, which had shaped so much of my childhood, was one I knew almost nothing about. My ignorance was mostly self-inflicted, with some (entirely understandable) assistance from my parents: it was years before they told me GBS could be fatal.

At the same time, the health world was facing a much deeper ignorance. Zika was generating a huge volume of questions, few of which could yet be answered. 'Rarely have scientists engaged with a new research agenda with such a sense of urgency and from such a small knowledge base,' wrote epidemiologist Laura Rodrigues in early 2016.⁹ For me, the first challenge was to understand the dynamics of these Zika outbreaks. How easily did the infection spread? Were the outbreaks similar to dengue ones? How many cases should we expect?

To answer these questions, our research group started to develop mathematical models of the outbreaks. Such approaches are now commonly used in public health, as well as appearing in several other fields of research. But where do these models originally come from? And how do they actually work? It's a story that starts in 1883 with a young army surgeon, a water tank and an angry staff officer.

RONALD ROSS HAD WANTED to be a writer, but his father pushed him into medical school. His studies at St Bartholomew's in London struggled to compete with his poems, plays and music,

and when Ross took his two qualifying exams in 1879, he passed only the surgery one. This meant he could not join the colonial Indian Medical Service, his father's preferred career path.¹⁰

Unable to practice general medicine, Ross spent the next year sailing the Atlantic as a ship's surgeon. Eventually he passed his remaining medical exam and scraped into the Indian Medical Service in 1881. After two years in Madras, Ross moved to Bangalore to take up a post as Garrison Surgeon in September 1883. From his comfortable colonial viewpoint, he claimed it was a 'picture of pleasure', a city of sun, gardens and pillared villas. The only problem, as he saw it, was the mosquitoes. His new bungalow seemed to attract far more than the other army rooms. He suspected it was something to do with the water barrel sitting outside his window, which was surrounded by the insects.

Ross's solution was to tip over the tank, destroying the mosquitoes' breeding ground. It seemed to work: without the stagnant water, the insects left him alone. Spurred on by his successful experiment, he asked his staff officer if they could remove the other water tanks too. And while they were at it, why not also get rid of the vases and tins that lay scattered around the mess? If the mosquitoes had nowhere to breed, they would have little option but to move on. The officer wasn't interested. 'He was very scornful and refused to allow men to deal with them,' Ross later wrote, 'for he said it would be upsetting to the order of nature, and as mosquitoes were created for some purpose it was our duty to bear with them.'

The experiment would turn out to be the first in a lifelong analysis of mosquitoes. The second study would come over a decade later, inspired by a conversation in London. In 1894, Ross had travelled back to England for a one-year sabbatical. The city had changed a lot since his last visit: Tower Bridge had been completed, Prime Minister William Gladstone had just resigned, and the country was about to get its first film parlour.¹¹ When Ross

arrived, though, his mind was focused elsewhere. He wanted to catch up on the latest malaria research. In India, people regularly fell ill with the disease, which could lead to fever, vomiting, and sometimes death.

Malaria is one of the oldest diseases known to humanity. In fact, it may have been with us for our entire history as a species.¹² However, its name comes from Medieval Italy. Those who caught a fever would often blame ‘mala aria’: bad air.¹³ The name stuck, as did the blame. Although the disease was eventually traced to a parasite called *Plasmodium*, when Ross arrived back in England the cause of its spread was still a mystery.

In London, Ross called on biologist Alfredo Kanthack at St Bartholomew’s, hoping to learn about developments he may have missed while in India. Kanthack said that if Ross wanted to know more about parasites like malaria, he should go and speak to a doctor called Patrick Manson. For several years, Manson had researched parasites in southeastern China. While there, he had discovered how people get infected with a particularly nasty family of microscopic worms called *filariae*. These parasites were small enough to get into a person’s bloodstream and infect their lymph nodes, causing fluid to accumulate within the body. In severe cases, a person’s limbs could swell to many times their natural size, a condition known as elephantiasis. As well as identifying how the *filariae* caused disease, Manson had shown that when mosquitoes fed on infected humans, they could also suck up the worms.¹⁴

Manson invited Ross into his lab, teaching him how to find parasites like malaria in infected patients. He also pointed Ross to recent academic papers he’d missed while out in India. ‘I visited him often and learnt all he had to tell me,’ Ross later recalled. One winter afternoon, they were walking down Oxford Street, when Manson made a comment that would transform Ross’s career. ‘Do you know,’ he said, ‘I have formed the theory that mosquitoes carry malaria just as they carry *filariae*.’