



Adam Kucharski and Tim Hubbard

Tim Hubbard: Hello, and welcome to MyVLF and The Big Book Weekend. I'm Tim Hubbard. I'm a former BBC journalist and editor, based here in Cornwall, although I guess my bookshelves look pretty much like anyone else's. Our conversation today is organised by the Stratford-upon-Avon Literary Festival, which should have been running in its usual slot at the beginning of May. It's a terrific festival, I've had the privilege of hosting there for some years, now. Watch out for that in 2021 and also for some extra special additional events this autumn, as well as online. So, my guest today is Adam Kucharski. He's Associate Professor and Sir Henry Dale Fellow at the London School of Hygiene and Tropical Medicine, where he works on mathematical analysis of infectious disease outbreak. He also writes about science in publications such as *Wired*, *The Financial Times*, *Scientific American Times* and the *Observer*. His new book is *The Rules of Contagion*. This is the book we are going to be talking about today. Adam, hello, and welcome to this session.

Adam Kucharski: Hello, thank you.

Hubbard: Let's start at the beginning, Adam, because many people watching this might have assumed that this book is solely about the outbreak of COVID-19 that we are experiencing at the moment. Or at least solely about disease. That is not the case, is it? Financial boom and bust, misinformation, political thought, technology, all areas within it. Including [Hubbard yawns] the contagion of yawning. Can you explain how that works?

Kucharski: Yes. The book is really an attempt to look at contagion in its broadest sense. Even if you go back through the history of people studying contagious diseases, a lot of them turned their attention to other problems over the years. So you had STI researchers who thought about financial crises, malaria researchers who thought about contagious behaviours within populations. In many cases, a lot of these fields are tackling problems that have quite similar analogies. They certainly face similar challenges. So, the book outlines some of the approaches we can use to try and understand what is really driving a lot of that spread. And



whether there is potentially ways of avoiding mistakes by looking at what other fields are doing.

Hubbard: Many people would think that laughing and itching, the contagion of itching, for example, is a world away from the world that we are very interested in at the moment, the world of COVID-19. But there are some similarities, your book points out.

Kucharski: There are. One of the things with some of those behaviours, is there is evidence, if you take something like yawning, for example, relationships between people might play a role here. People who are more acquainted with each other might be more likely to catch a yawn, than you would from a stranger. The other thing with a lot of those behaviours that makes them easier to study is, you can run an experiment. You did it at the start there. You yawned and it caught on. When you're looking at something like smoking behaviour, violence, disease, you obviously can't run that experiment in the same way. You have to try and get at that question of how that contagion is happening with much sparser data. A lot of the principles around social interactions, how you interact with people, how close you are to them, relationships, that really drive everything from a lot of behaviours that we see, through to diseases.

Hubbard: You write in the book, your interest was sparked by you simply sending a tweet. What happened?

Kucharski: Yes! This was years ago. When I think a friend had sent me - we had this running joke about, when you read articles about computer hacking, you have this horrible stock photo of people in balaclavas - and he'd sent me a message with just a completely absurd photo that made no sense, really. So contrived. I stuck it on Twitter and said, "Look at this ridiculous stock photo." It started getting a lot of attention, journalists re-tweeting it and then some comments appeared below that it was from a documentary. I think I had misinterpreted it. First thing in the morning, hadn't had a coffee, hadn't really thought it through. Even in that short space, it got about 50,000 views and it just struck me, as somebody who works predominantly on diseases, why did that happen? Why were there factors about that that made it spread? Who were the people that I have contacts with that might have helped that



spread? If people hadn't corrected it, would it have slowed down? I think, for me, that opened up this range of questions. It's not just about saying, this has gone viral or not. It's much more interesting to think, why is it quick, why is it slow, why does it reach more people?

Hubbard: And why does it stop?

Kucharski: And that is the key question. That's the one that were asking at the moment. When I first started outlining the book, a lot of the questions people would ask were basically variations of, how do I get my tweet to go viral? But I think now we are seeing a situation with the current COVID-19 outbreak, but equally things like the spread of violence, it is much more about, we know this is potentially contagious but what can we do about it?

Hubbard: Your research began at that point... If we can come onto that subject that everybody is talking about, obviously, which is the contagion of disease, in the book you write about the spread of malaria through mosquito infection. The work of a man called Ronald Ross, I think in the 19th century who worked on the maths to show that in the light against that disease, not every mosquito needed to be killed. That was a bit of a breakthrough moment, I think, wasn't it?

Kucharski: It was. This was in the 1880s and 1890s that he really first became interested in malaria. He was based in Bangalore and other parts of India. He was one of the first to demonstrate that it was spread through mosquito bite. Early hypotheses that you had were people were drinking water that had mosquitoes in. Eventually they pinned down that it was the bite that spread it and he got the Nobel prize for that. Then he was very interested in this question of control. At the time, the kind of obvious insight was, well, if mosquitoes spread malaria then as long as there are mosquitoes in the area, you have malaria. You can't get rid of it unless you eradicate every last mosquito in a particular location. He showed that actually, if you think about it as a dynamic process, you have somebody with malaria, they are infectious for a period of time, then they might recover. For transmission to happen, a mosquito has to bite that person. The mosquito has to survive long enough to become infectious, it has to bite someone else then that person has to become infectious. Actually, if you reduce mosquito numbers enough you can actually break this link of transmission, even



though there are still some mosquitoes out there. This idea that actually you didn't have to get rid of every last infection, you just have to break enough links in transmission for it not to be sustained is really now fundamental to disease control. It would evolve over time to what we now know as the reproduction number. This concept that you don't have to just stop every infection, as long as you stop enough transmission, your outbreak will decline.

Hubbard: Let's think a little bit about the terminology that we are hearing at the moment and that you refer to, in the book. Let's start with herd immunity. We've heard a lot about this. Could you explain about how that concept plays in calculations such as those of Ross, the man you've just mentioned, and Kermack and McKendrick, who you mention in the book.

Kucharski: This is an idea... you don't have to get rid of every mosquito to control an infection. In the 20s, Anderson McKendrick who was a researcher who built off Ross's work, looked at more general epidemics. Looked at things such as the plague which spread and had this very sharp peak. At the time, there were different theories about why epidemics peaked. Some people thought maybe the pathogen just becomes less transmissible over time. Others thought, maybe everybody becomes exposed eventually and that's why it ends. What this showed is that something subtly different happens. If you imagine people interacting early on. Each infectious person will come into contact... Anyone they come into contact with will be susceptible. As immunity builds within the population, they are more likely to come into contact with immune people than susceptible ones. Transmission will start to tail off, because... Not because everyone has been infected, but because in these interactions it becomes less and less likely that you will meet enough susceptible people. This idea of herd immunity in their case was for things where there wasn't vaccines, but... Vaccinologists realised that you could use this idea that if you can get enough immunity in the population through vaccination, you can actually protect the population, even though, for example newborns or people with certain conditions won't be able to get the vaccine.

Hubbard: So, herd immunity. The other term that we're hearing a lot about, particularly at the moment, is the curve of infections. Flattening the curve. In the book, you draw a comparison with the curve of home ownership, which is not an obvious comparison for many people, I



would imagine, when they come across that. Can you explain why those two terms can be used in both of these occasions?

Kucharski: Yes, so there is a lot of interest in visualising how these things spread. If you look, for example, at adoption of products... things like ownership, or particularly products that might be contagious, things like VCRs, or DVD players, or, more recently, things like Netflix, or something. What you will see is often exponential growth at first. More and more people will adopt it and then the number of people will tail off, as the laggards, the late adopters adopt it later on. So, if you actually look at the new people adopting something over time, it will grow very sharply and then it will peak and there will be fewer and fewer new people and, eventually, not that many new people will be adopting it over time. That is a similar principle to what we see with many disease epidemics. Initially, you have lots of people becoming infectious and then perhaps as control measures go in, that will tail off and decline. What is really crucial isn't just the fact that it happens, it is the shape of that. Are you having all of your infections right at the same point in this very sharp, large peak, or are you seeing this flatter, slower curve. And in the case of the measures we're putting in, they are really pushing the curve down to the point where you've got very few infections occurring and that is going to make a huge difference on the impact it has for the health service.

Hubbard: What about the curve that doesn't lessen? The curve that doesn't drop? This is the, I think you refer to it in the book, as the "zombie apocalypse..."

Kucharski: I think it goes back to this notion that things will grow exponentially until everyone gets exposed. Even if you do have something that can grow very quickly and you don't have a controlled measure, whether it's a new idea or whether it is, say, an infectious disease that can spread and can't be controlled... What almost always will happen is eventually you will get some accumulation of immunity, whether in the short term or much further down the line, which will start to slow transmission. The only way you get this exponential curve that keeps growing over time is if people who are infectious actively seek out people who are still susceptible. For example, I use this in the book, because it was the only one I could think of where people would change their behaviour this much, is something like if you had a zombie outbreak, where the zombies actively search out those few



susceptibles left. In reality, you almost never see an outbreak like that. You do see it slow down later on, for one reason or another.

Hubbard: Thinking about the rates of infection that you refer to there, you talk in the book about a reproduction number. Which I think is known in your business as an R number. You quote SARS as being 2-3, smallpox 4-6, chickenpox 6-8, measles 20. A figure which surprised me. How were those numbers calculated?

Kucharski: Yes, so this is a measure of basically each person who gets the disease, how many others they give it to. Those numbers you quoted are for fully susceptible populations. Measles, if you've got a susceptible group of people, it spreads incredibly quickly. That number is a combination of the interactions within a population and the biological characteristics of a virus. Measles spreads very quickly in the air, it hangs around for a long time. If somebody sneezes at an airport terminal and a whole bunch of people will get infected, because it is just so contagious. Diseases like SARS, for example, quite a lot of transmission happens, but because it's droplets, it is less able to go that far. We even see differences between countries, communities and more recently for coronavirus, people who have changed their behaviour. The virus is the same, but it's the interactions through which it is spread which causes the decline in the reproduction number.

Hubbard: With a certain degree of irony, you talk about the work of Florence Nightingale, and a statistician called William Farr, and their work in the field of contagion. Ironically, because we are seeing her name, now, on the doors of new UK field hospitals. What did their work show you? You write about it very interestingly in the book.

Kucharski: Obviously, Nightingale, renowned as the popular figure of the lady with the lamp, and her work on nursing, but she was also a remarkable statistician, highly respected researcher and an advocate for health. Her and William Farr, Farr came up with a lot of these ideas of normalising death rates. So rather than just looking at raw numbers of deaths in different populations, he would adjust for the population size, the age structure, to try and get a fair comparison of what was going on. Working with Farr, Nightingale adopted a lot of these ideas to look at the impact of healthcare on the Crimean War. A lot of soldiers were



dying of disease, more so than battles. And she wanted to be an advocate, to improve the conditions within hospitals. To do that, wanted to do it evidence-based, and came up with really innovative visualisations of data. Visualisations of data are commonplace now, but at the time, she really pioneered getting those health messages across and I think that idea that you build an evidence base of meaningful comparisons, you are not throwing together data in a way that doesn't make sense. You're getting meaningful data, you're visualising it and you're really advocating it. I think she was incredibly good at that and really set the way for how a lot of public health is done, in terms of building and presenting a clear evidence base.

Hubbard: You write in the book that we are talking about today, *The Rules of Contagion*, very entertainingly. It's a great read, we should say. Were you surprised by your research, for example into the work of Florence Nightingale, to see how contemporary it is?

Kucharski: It was... I had already known that she had done that statistical work, but I think it was the... Her way of thinking about it, actually realising just the depth of influence she had had amongst the field of what we do and, indeed, how many statisticians at the time were influenced by her work. I think she also really just had this fantastic passion for making change. She had this quote at one point, "Feeling is a waste of words, they should be converted into actions and actions that bring results." And although she certainly wrote up a lot of analysis, she had this very clear view that whatever you're doing, whatever you're writing should have an impact on a population, on its health. I think that is always worth bearing in mind, particularly people like me in academia and people who work in research. You need to have a focus on what beneficial impact your work might be having, rather than just abstractly talking about the things, many steps away from reality.

Hubbard: I was going to ask you about that. It seems to me, reading your book and reading about her generally, that she was able to convey to the population, something which politicians around the globe at the moment are doing less or more successfully. Which is actually engaging with the public and convincing them of the validity of their argument. She seemed to be able to do that very successfully.



Kucharski: Yes, and to the point where she persuaded Queen Victoria to commission a Royal investigation to investigate the situation in the Crimea and I think we see that echoed throughout public health. Even when you talk about things like violence, or other areas, that it is not just about building an evidence base, even if we have very good evidence. It's about having that... Ronald Ross had this line that an idea takes 10 years to take hold, no matter how good it is, and I think we often see that, particularly in those longer term health policies, even if something seems quite clear from the evidence, it might take a very long time – in some cases too long - to actually change what happens to policy.

Hubbard: Let's turn away from disease for a moment, now and turn to some of the other areas of contagion that you talk about. Let's begin with finance. You write about why certain markets financially grow and certain markets decline. The crash of 2008, the Southsea bubble, and even bitcoin. Can you talk about the mathematical modelling for that?

Kucharski: Yes, I think this refers to a quote by a couple of infectious disease researchers who looked at this and it is this idea in finance that if something is going up it's a good thing, and if something is going down, it's a bad thing. They pointed out if there's not a really good concept of why it is going up, that can lead you into problems. If you think of this idea of infection and susceptibility, I guess the simplest idea is something like a pyramid scheme. That one person joins a scheme, they have to recruit 10 others and they have to recruit 10 others and they have to recruit 10 others and you can very quickly work out that you are going to run out of susceptible people to try and recruit and that is when the scheme will burn out. If you look at some of these other slightly more subtle types of behaviours, it's what researchers refer to as the Greater Fool theory, you know something is overpriced, but you don't mind because you think there is someone else that is a greater fool that you can sell it on to. That is often why you get this huge inflation occurring within a bubble. Even bright people, Isaac Newton was sucked in in the South Sea Bubble and lost, by account, an absolute fortune, because he thought there was someone else to jump on. Often, that kind of growth and decline is for many of these things a reflection of it running out of susceptible people.



Hubbard: It is also an example of imagination running away with you, rather than the factual evidence. I was very surprised to read that about Isaac Newton.

Kucharski: Yeah, and in some cases, the attempts to debunk the myth can backfire. During the railway bubble where people poured huge amounts of money into this new technology, *The Times* actually warned against it being a bubble. And said, look, if people are pouring so much money into this, it could be devastating for the economy and people reading that thought, “Wow, people are pouring money into this, I need to get onto that.” It had the kind of opposite effect than it was intended.

Hubbard: One of the other intriguing chapters in the book is the contagion of violence. You give interesting examples here about the contagion of violence, largely from the United States. What modelling has taken place there?

Kucharski: I think there has been increasing work thinking of violence from a public health point of view, and the analogy I draw in the book is with a lot of the early study of germ theory in the 1800s, particularly in the 1850s and this idea that, at the time a lot of the people believed in the miasma theory, that diseases were caused by bad air. Obviously, over time we realised it was pathogens and that was what was causing the transmission and that was what we needed to control. I think there's a lot of people now, thinking of violence in a similar way. Violence isn't just this unpredictable event, this thing that we can't do anything about. It's just bad people. In many cases, there is a clear structure to that violence and one event will spark another one, will spark another one. If you can understand those network links as much as you would for an infectious disease, if an event happens that gives you an opportunity to intervene. Particularly, for violence, the contagion occurs over weeks or months. If you imagine for an infection if you're talking about contact tracing, a case appears and you've got a few days to prevent another infection, whereas for violence, you've got a longer window. That means that these measures that really try and go in and intervene with people who are at risk, people who have local community workers who know those communities and have seen quite a lot of success, not just in the US, but Glasgow have adopted quite a lot of these measures, as well. Really thinking of it not as this random



process, but as a dynamic, often contagious set of events that you can try and target as much as you would an infectious disease.

Hubbard: In this chapter, you also talk about suicide and media guidelines for reporting suicide. What data is there to suggest that people are vulnerable to that sort of contagion? They hear about someone's suicide on the radio or television and they are swept, engaged, convinced, moved to commit the same sort of self-violence?

Kucharski: I think there are increasing examples of that. One of the challenges of looking at a general population with any form of contagion is that it is quite hard to distinguish what is a clustering of behaviour from what's actually contagion. Media reports are often a clear example, because transmission can only go in one way, if that makes sense. If you have a celebrity death that is widely reported and you later see subsequent suicides in similar groups with similar methods and with other kind of analogies, then often that is very suggestive. Many examples now out there even in recent years, of a celebrity death, where they have broken these guidelines. In particular, there's a lot of guidance against not describing particular methods in detail and not attributing it to a clear causal factor. So not saying, they committed suicide because of this one thing or this one event. And also, trying to draw associations with the media saying, the sort of person, this sort of behaviour. That is why they turned to this. In situations where outlets have broken these guidelines, you have seen following incidents. We've also seen that happen in other acts of violence. Things like mass shootings. These kind of copycat things.

Hubbard: We'll come on to politics, the contagion of politics, an interesting subject, in a minute. If we could just first think about online and the process and the impact of technology. Here we are, you and I today, having this conversation through technology. Talk to us, if you will, about the term influencer and also the term troll. Because, reading the book, I understand that both meanings have changed over time. Both have played a part in online contagion. How is that?

Kucharski: Yeah, they've played a huge part. The challenge with influencer is, it has evolved in its meaning over time and if you are thinking about sparking contagion, often influencers



will appear in that conversation. Early on, this idea emerged with the six degrees of separation. This idea that, for example, if you want to get a message to a certain person somewhere else, in the early days it was sending a letter. Often, in those early experiments certain people would crop up more often than others in that network. It suggested that there were these little-known people who, at first glance, this seemed important, but actually seemed to connect societies more than others. I think they sold this idea to marketers, thinking we don't have to put loads of money on celebrities and big TV budgets, if we can target these influential local people, we can actually spread the word much more than it would otherwise. I think the challenge is, this has kind of evolved as a lot of research has now shown that those people don't really exist in that same way. One of the reasons is, if you want something to spread, you need people to be both susceptible and influential. Say you've got a high profile and I want you to spread a message, you've got to be susceptible to my message first and then you've got to spread it. The problem is, a lot of people are either influential or susceptible. There's not many people who are a combination of both. I think often what happens is when people talk about influencers, they talk about celebrities or high-profile journalists. Really, if you're paying a celebrity a lot of money to promote your product, that's not really clever social influencer marketing policy. What you're doing is just paying a mass media brand to promote it. Which is fine, but I think that message is being kind of blurred, a bit. Someone that I talk to who works a lot in this area, says there is an interesting version of this and there's a boring version. The boring version is the one that I described, where some people are a bit more influential. People will typically conflate them to make it sound like they're doing something clever. To your second point, to the trolls, I think in the early days of the Internet it was almost kind of mischievous. If you think of the kind of classic storytelling version of the troll, what would often happen is that people would just do something really absurd to trigger a response. I think we've all seen it, those examples... A letter to a complaints department that is just so ridiculous no one could take it seriously. And then somebody does reply, taking it seriously and that is the kind of trolls trying to get a response. I think it has evolved into essentially anyone who's nasty online. The usage has transitioned. There's been a lot of work, now looking at what makes a troll. I think, again, it is this idea that a lot of people have that there's just bad people out there. Actually, a lot of research has shown that many of us can become trolls and behave quite unpleasantly online. A lot of it depends partly on what other people are doing in that online conversation,



but also just things like mood. A lot of people can behave very differently online to what they would in normal circumstances, just because of the nature of online life is very different.

Hubbard: The contagion, though, of online life is what you write about here in the book and let's just think, for a moment, of the craze – me term- of things like the ice bucket challenge, or videos... you mention in the book, drinking goldfish. You make the point that cascades don't quite spread as pandemics do. Although, yet again, there are similarities.

Kucharski: Something like the ice bucket challenge is probably the closest we would see to a pandemic in terms of online contagion. If you remember that, someone would douse themselves in ice water, they would nominate a couple of others and they would nominate a couple of others and you would get a somewhat clockwork outbreak, until eventually you just ran out of people to nominate or there weren't enough. It was kind of herd immunity, in effect, that caused outbreak to slow down. That is a very rare type of online popular trend that does that. What would often happen is that most people would share something and nobody else will share it and a handful of people will share a video or a piece of content, and most people will have seen it from a handful of sources. In infectious disease outbreaks, we would call these super spreaders, or super spreading events, rather. The super spreader kind of puts a bit too much narrative on the individual, whereas it might just be a random event that happens. In diseases, a super spreading event might involve 10, 50, 100 people. Online, one of those really influential people, one of those mass celebrities, could spread to millions. So you get this incredible variety in contagion that a lot of people... Something like 95% of tweets aren't shared by anyone. They just go absolutely nowhere. Then you get this tiny fraction that actually drives most of these outbreaks.

Hubbard: We also want to think about the inherent dangers online in terms of data collecting. You talk about a data broker now being a recognised career path. It certainly wasn't when I was at school. That's another story! The scandal of the Cambridge Analytica broke in 2018, which seems to be a general warning to many people... Do you think that that message is actually, even now, not getting home, about the dangers of personal data being available to contagious outbreak, as it were?



Kucharski: I think it's been a really interesting few years for this. A lot of this data collection has been happening over a longer period of time. The idea of a data broker comes from this fact that a lot of information about you on its own isn't that valuable. For example, if I just know that some given number of people have visited a website, that's not particularly helpful. But if then I start to know their ages, who they are, what they've bought previously, what are their interests - then you start to get enough links between these pieces of information to say something useful. I think that's what a lot of... The idea behind Cambridge Analytica, the methods have been debated about actually how much you can persuade people given that information, but it is this principle if you could link what they are doing on Facebook to what their political behaviour was, then maybe you could make some predictions and change people's behaviour from this. I think what's surreal, I found, certainly within the last few weeks, is in the book I talk about the spread of information, information about you ends up in all sorts of hands, and you're probably not aware of it. Particularly things like GPS data. You've seen over the last year or two of companies that track people to remarkable detail and sell that information onto other organisations. Now with the current coronavirus outbreak, we're seeing a lot of discussion about the way we're going to hopefully resume some normal elements of life is to have more detailed tracking. One thing that has been proposed are phone apps that track you and your interactions in detail. I think there are ways of doing that more carefully and more privacy focused, but we have seen a lot of debate and one of the things that I mentioned is that this data can be helpful for health, but when I was writing it there were perhaps fewer tangible examples, but now we've got a very clear one, where if we can find a way of tracking quite detailed information about people but in a way that protects privacy, that could be a very powerful tool for understanding how a very real biological infection spreads.

Hubbard: You mention the change there since you wrote the book, the timing of the book is extraordinary, to be published in February just before the coronavirus outbreak really became big globally. And yet, some things are constant here. You talk about the world of politics and the idea of disinformation in politics, not just incorrect information being spread through contagion, but also information which makes us doubt the very nature of truth here. A very interesting chapter within the book about politics.



Kucharski: Yes, I think we've seen a huge amount of coverage of fake news over the last few years. I think there's been some really nice work to try and untangle what is driving that and what we talk about when we do that and some of it is referred to as misinformation. For example, if you see a story about a particular fact or particular report of something that isn't true, but you don't know that it isn't true, but you share it and think it's interesting, you are spreading misinformation. You may not be aware of it, but you're sharing it as if it is true. Disinformation is something that can be far more dangerous. This is something that basically undermines your confidence in what is a fact and what is not. Often, what you'll see is, particularly in some of the political campaigns of recent years and some of the interference that has happened, is organisations just throwing out huge numbers of stories, often on both sides of the argument, just to completely blur who's to blame, who's right and who's wrong. In many cases the aim isn't to get anyone to believe the stories, it is to kind of say, "How can we know for sure?" We're seeing with the current epidemic and in politics of recent years, a lot of people get to this point of, "Well, there's no way I can know. They're all bad. What can I do about it?" It's this almost resignation that I can't have any faith in the truth and I think that's what a lot of disinformation is trying to do. Undermine your confidence that you can have any sense of what is really happening. In my view, that's far more dangerous than you have shared a news story that you didn't realise was false and when someone corrects you, you may well change your mind.

Hubbard: Just on a lighter note, as this is My Virtual Literary Festival, through Stratford Literary Festival, just a nod to Evelyn Waugh and *Scoop*, which you pay tribute in the book, in which a journalist goes to a foreign country and he's too bored, drunk to bother. He writes a totally fictitious story which then becomes the truth, somehow.

Kucharski: Yeah, and I think in the book I use that as an example of this feedback cycle you can get. I think often what happens is with online content is people throw information at high profile figures, journalists, politicians to try and influence the behaviour. In *Scoop*, Wenlock Jakes is this journalist who ends up in the wrong city, but writes about war and chaos and economic crises. Other journalists arrive and realise they have to submit stories too, because the star reporter is submitting all this drama. So they do it and it has the effect of tanking that country's economy and actually making that prediction happen. That's an extreme example



from the 30s, but I think we do have that feedback effect now, where one little-known outlet can jump on a story and within a day you've got something as a mainstream media talking point which, in many cases, we've got no idea where that information, that suggestion first came from.

Hubbard: In our final five minutes, let's just think a little about statistics. You used a very neat quote in the book that statisticians, like artists, have the bad habit of falling in love with their models, which I thought was a great quote. How do I, as a layperson, simply know what to believe?

Kucharski: I think that's a really good question. In my experience of communicating statistics and models, you often get two extreme reactions. Either people think, "This is really complicated and I can't possibly trust it." Which to some extent does make sense, if it is a bit unusual, people just don't... The other extreme is people are like, "This is complicated, it must be right. These people must know what they're talking about." I think in many cases when we're talking about contagion or data in populations, there is just some quite simple things you can do to watch out for kind of key issues. That's what I tried to do in the book. Just, for example, assumptions that quite clearly go wrong. One thing I mentioned in the book was a disease model prediction that predicted 77 trillion cases. You don't need any background in maths to realise that if you have an epidemic that grows indefinitely and you don't put any cap on your population size, you're very quickly going to get an unrealistic epidemic. In many ways, I think that is what's important, not to think so much about the kind of complexity or the jargon that people use, but actually a basic sense check. If someone is proposing a value or a conclusion, actually just to take one step back and to strip away the jargon. I think good science and good evidence doesn't try to bludgeon people with complex words and terminology. It should outline an idea, which once you see it, you realise it's kind of obvious. Hopefully, there are elements in the book that once people think about contagion and think about those issues, a lot of them will be kind of obvious. Because once you think about contagion in that way, you realise that it doesn't take too much to spot what is plausible and what isn't.



Hubbard: From your point of view, as a mathematician, as a statistician, how aware are you of the emotional impact that your statistics have on me as a layperson?

Kucharski: I think certainly statistics and anything that captures situations affects people. I think that's why people have a duty to try and communicate them. That's why I've written the book and I've tried to make an effort to get those measures out there. I think whether we are talking about things in disease, epidemiology or we're talking about financial models, or violence, or algorithms which categorise people for crimes and whether or not they're going to be sentenced. These are having a real impact on people. We need to put the evidence out there of what is driving those and where the flaws might be, so all of us, even if we're not mathematicians or scientists, we have enough understanding of what might be going on so that we can make criticisms and judgements on whether we think what's being done is appropriate.

Hubbard: And, finally on this, what about the notion of "Maybe" in your book? As a mathematician, again, you deal in definites, in black and white. What credence do you give to the unknown?

Kucharski: I think one of the transitions I've had in my career, my background is in pure maths, where you have a proof and that proof is going to stand for good. It's transitioned, because I work more on things like social behaviours and epidemiology, now, and it's much more about strength of evidence and there are a few epidemiologists that I mentioned in the book, one of them is Alice Stewart, that mention don't use X-rays on pregnant women, or have them in shoe shops so people can see their feet, these are not good ideas. At the time, there wasn't overwhelming completely conclusive evidence, but there was enough that suggested there was a problem. There was uncertainty, but there was, in her view, enough indication. I think that's what we have to do, whether we're talking about the current situation, with some of the data that we put together on how severe COVID-19 is, whether you're talking about online information in the role of certain groups and spreading harmful content. In many cases, you're not going to have a definitive, this is 100% this exact value. I think it is the question of, have you got enough evidence to conclude that it is a problem worth taking seriously? I think that's really the balance we've got to strike and obviously if



we get new information we've got to update. I think we have to be comfortable in many ways not having a precise answer, but having the confidence that we can do something useful.

Hubbard: The final question, let's return to COVID-19. You mentioned it there, how do you judge the global spread of the disease as we see it at the moment? How is that behaving mathematically speaking? Is it behaving as you would have imagined, as the targets suggested? And, also, are there already lessons that we're learning, as we anticipate future pandemics, from the one that we are in at the moment?

Kucharski: Yeah, I think there is a lot that we are still learning about this in terms of exactly how it transmits. I think the broad conclusions that we saw early on have held up. This is something that spreads easily amongst people. We estimated quite early on that there was this transmission without clear symptoms, which from a control point of view just puts you in something that is very difficult indeed. We also had evidence that these kind of lockdown measures could reduce transmission. The challenge is it doesn't seem like there is... Once you get a lot of transmission happening, you either have to do this contact tracing very targeted measures, with a really high level of effectiveness, which a few countries are managing, many aren't. Or you end up in this kind of lockdown situation. I think the question that every country is now facing is where are the solutions in the middle? If you can't get contact tracing to be at this huge, huge level, and you need other measures in place, what's the least disruptive combination? I think that's what we're going to see over the coming weeks, coming months. As countries start to loosen things, some will probably get some transmission increasing, some will manage to keep it down and really we've got to work out and learn what is that combination that is most effective and least disruptive, essentially, going forward.

Hubbard: The contagion of COVID-19, *The Rules of Contagion* is the book we have been discussing, why things spread and why they stop. Hugely enjoyable read, my thanks to Adam Kucharski and also to the Stratford-upon-Avon Literary Festival and The Big Book Weekend. From us, it's goodbye.

Kucharski: Thank you.