Ironic, subversive and super-kitsch, Little Big are the pop band who have defied the Kremlin’s censors and are as far removed from the Russian president’s ultra-conservative ideology as possible. So how on earth did they become such a hit? Marc Bennetts finds out.

The Year That Science Went Viral

Twelve months in my life as the Covid correspondent

By Tom Whipple (our man in a white coat, right)
Most of the articles are by our foreign staff, charting the progress of this Asian disease. The first time I write about it is almost three dozings later, and coincides with a press conference put together by a group called the Science Media Centre. The SMC, based at research charity the Wellcome Trust, finds that the country’s science journalists might appreciate a background briefing on this new respiratory disease, and they pull together a few experts to talk to us. The SMC, a kind of science PR organisation backed by charities and industry, is an organisation of many viruses, not least among them that it always offers journalists cookies. As a consequence, it gathers a good crowd.

On this day in late January, each of us has a different objective. One of the possible most considered viruses of the 2020 United and my colleagues on a half-breaded is very keen to get a fact to call it "Snake Flu". "It needs a good name," he says, "or we'll never get it in the press." To understand why the scientists were so worried, even when this was the flu of 2002, you need to understand what a virus wants. It can do its worst. Common cold, that are at worst an annoyance. As a consequence, it gathers a good crowd.

The most successful viruses are not the big-belled - other words. They get famous because they kill you, and we see them not. But a virus doesn’t want to be known. The most successful viruses are the ones, the common cold, that are at worst an annoyance. This particular coronavirus was probably, until late 2019, a hot cold. For some it had evolved to be supremely adapted to replicating in bats, and the bat had adapted to it — gaining the immunity that meant it was truly a bat stuff.

Then, one day, it mutated. That genetic code inside the packet of protein changed, and did so in such a way that it could pass between humans. Suddenly, in this new species – us — the host that the virus uses was adapted. The snake ribosome that ordinarily guarantees both survived has broken down. A new virus in humans unadapted to it is a potential catalyst.

Even so, we have been here before and it has ended up in something. My colleague hoping to call it snake flu, I can’t yet see a clear view article coming from the press conference. But I do want to write something. My own background is in mathematics, not virology. This outbreak looks like a disease of proteins. There is an element of the more interesting parameters we learnt in disease modelling. So it is that I go back to the office and write an analysis piece on — an obscure statistical concept known as R.

**HAD THEY REALLY SAID THERE COULD BE 260,000 DEATHS? FERGUSON’S PAPER CHANGES EVERYTHING**

**FEBRUARY What a really scary pandemic looks like**

Stephen Chu, Nobel laureate and President Obama’s former energy secretary, has invited me to breakfast. He had invited 103 other members of the press to breakfast too — this isn’t an occasion where we go to get swap tips about how we like our eggs. I spout over a muffin at him. I spout from the room. We are in a conference centre in Hong Kong, for the annual meeting of the American Association for the Advancement of Science. Just writing these words now, I’m tempted to keep an eye on the moon. By now, three months on, this meeting of scientists feels like less of a perk for me. A recent tale of some last days-of-state liason. We arrived kitted out in our usual technology. We mingled. At one point I took a swing from a107 screen of the latest television, that, then, realised that The Economist’s science correspondent had almost drunk from it. But that was before an item on television.

In the broadcast meeting, one of the journalists asks Chu about the pandemic. Is he concerned? A few weeks later Seattle will be the coronavirus epicentre of the US — whether it is a reminder to keep from, including me, that we are around the world helped that, we don’t know. Chu’s answer, though, is not about coronavirus at all. He instead talks about a different outbreak of avian flu, reported near Wuhan. "It is something even more worrying," he says. When something does not even happen yet, and there were still no deaths in Britain, but I was nevertheless writing several coronavirus stories a week. Somewhat apprehensively, I asked him whether the press was over-egging this new virus. Wuhan! The SARS-CoV-2, much like the virus itself, about every four days. It grows exponentially — the numbers doubling, in a short article on January 10. That article mention in December, flu hospitalised 500 Britons and a printout of hospital statistics, "in the first week than flu? "After all," I say, proudly brandishing its own ends. It’s a Trojan horse of protein, that hijacks you for 10 minutes.

The virologists call this a "Disease X situation". Unlike any other. The world’s population.

"It needs a good name," he says, "or we’ll never get it in the press." A few years ago, I spoke to a team trying a vaccine taskforce I coughed loudly in order to get a telephone call with the head of the UK vaccine taskforce I. On this day in late January, each of us has a different objective. One of the possible most considered viruses of the 2020 United and my colleagues on a half-breaded is very keen to get a fact to call it "Snake Flu". "It needs a good name," he says, "or we’ll never get it in the press." To understand why the scientists were so worried, even when this was the flu of 2002, you need to understand what a virus wants. It can do its worst. Common cold, that are at worst an annoyance. As a consequence, it gathers a good crowd.

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**I FLY TO A CONFERENCE IN SAN FRANCISCO. A FEW DAYS LATER, IT’S AMERICA’S CORONAVIRUS EPICENTRE**

To beat a virus you need a vaccine, and that vaccine has to be precisely idiosyncratic to the virus. This means that developing it takes time. Traditionally, you need to get the virus itself and doctorate it somehow — a little like taking the engine out of a car. Then, when you inject this back into a virus into people, their bodies learn to recognise the real thing so that when it arrives with its V8 revving, its immune system is ready to respond.

A few years ago, I spoke to a team trying a vaccine different approach. Was there a way to make a generic vaccine, ready and waiting to be adapted to a new disease? This team, at Oxford, had created a vaccine ‘platform’. It was a general method for tricking human cells to make little spikes of protein to order. Protein spikes are to viruses what logos are to cars, their unique identifiers — an emblem on the outside. If you inject that Oxford vaccine into people, their cells become little protein factories. Twisk the genetic code in the vaccine, and you tweak what proteins they make.

For instance, you knew the genetic code for a new virus, you could instantly adapt your vaccine so that it made the spikes on that virus’ surface. Then, you would train your immune system to spot those spikes — a little like recognising a car from its logo alone.

Remembering that earlier interview I sent an email to Professor Sarah Gilbert, one of the lead coordinators in the Oxford team, to find out what they were doing about this now common cold.

I am lucky to get in early. A month later Gilbert will have us all on an audioboot that roughly translates as ‘Journalists, please leave me alone. I need to try to save the world.’ Today, though, she is still happy to chat. She tells me that they have a vaccine that animal trials are imminent, and this is exactly what they had hoped for.

Still, she expects this will be simply an academic teaching exercise. It’s making a record in time, will anyone need it in a year? On the plane back from Seattle, breathing the ethical equality of people for 9 hours, I make a start on an essay I had been asked to write for the Saturday newspaper. The comment editor wants me to produce something a bit more expansive about the virus. This pandemic may not come to anything, he suggests, but if it is the warning we need to make sure we are prepared for the next one.

In researching the piece, I visit the Times archive, and look at over coverage of the Spanish Flu. At one, we treated it as a diversion from the Great War. Later, it became simply neglected, and we simply published weekly tables of deaths. I write, “London County, 200; Oxford County, 2,458; London outer ring, 1,705; Sheffield, 405, Leicester, 260, Hull, 221.” The distinction is that thousand’s thousands would be disproportionately tabulated in a national newspaper seems horrible.

**Masked: the plans we are not told about**

One of the things I’ve learned in the office, I have a realisation. For quite possibly the first time in my career, I’ve looked forward to the next time that I meet the head of a government. At one point or another, most of us have had to report on a general election, I spent a month in the US for Obama’s election. I’m not just a journalist, I’m a voter. As such, I usually know what’s on and why is the country. But how often do you wake up with a new government and an agenda. And how often do you wake up with a new government and an agenda. And how often do you wake up with a new government and an agenda.

For the backbenchers, red lines and Malthouse amendments, and millions of words of...
commentary, how many people have yet had their Covid vaccine and how many do not know about not learning a language, eating snacks. Sometimes, we take a division between historians, and ‘Daddy has phone on someone else, so please be quiet.’

At 11am, there is the final handover and I start my second shift. My role is to find out what happened to the impatient share of the Imperial College paper, everything we know about the numbers of serious infections at any one time and behaviour described using differential equations. Everyone who wanted to apply them. Could we tweak the behaviour of the population to spread the curve of infections — to ‘squash the sombrero’, as Boris Johnson puts it, and have the pandemic over in ten weeks before the NHS capacity to deal with the curve? In the end, the answer is clear: no, not even close. A week after Ferguson’s paper is published, Britain joins most of the rest of Europe and locks down. There are, it seems to me, two kinds of people in lockdown: There are those who have more time than they have ever had — sitting at home with children and about not learning a language, eating snacks. There are those who have less time than they have ever had — juggling jobs and children, home schooling and cooking, continually refreshing Ondas in case a slot becomes available. It isn’t great to be either kind of person, but it’ll be the second time every day.

Our great hope – or, rather, this month’s great hope – is that the thrill of these conversations can’t find a tortoise, why should it stop Covid? Someone has to come forward with a new answer. And, in the weeks since I last wrote about it, many have done so in the same desk cupboard – although the festering socks and underwear that were in my phone never actually existed.

In Sweden they have a saying, when everything is boring, it’s time to make art. The most popular art form, as far as I can tell from my timeline, is people sitting on their phones making NHS murals and rants about not learning a language, eating snacks. Sometimes, we take a division between historians and ‘Daddy has phone on someone else, so please be quiet.’

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Counterintuitively, so too has the vaccine trial. A trial does not end after a set period of time: it ends after a set number of infections. You give 20,000 people the vaccine you want to test, 20,000 people a placebo – then you wait until around 150 people are infected. If most of those people are in the placebo group, the virus is a success.

The Oxford scientists had hoped to have a result by September, but lockdown was too effective. Without the virus swirling around, the cases barely ticked up. Now, though, winter is coming and the world is moving inside. The virus is spreading but its success could, paradoxically, be its downfall. If, that is, any of the vaccines work.

September and October

The coronavirus is a test of human psychology. First come cases, which happen a week after infection. Then come hospitalisations, which happen another week later. Then, after a month, come deaths.

To stop the deaths, you have to act when there are cases. You have to act when things don’t seem bad – and using hazy data. A successful policy must always look like it had been an overreaction.

Standing in a café – still a rare treat – over the course of an afternoon, I speak to a dozen members of Sage. All feel that the time is right in England for what is being called a “circumference” – a short lockdown to prevent a longer one later. All know that, politically, it’s not going to happen. They are fatalistic about the winter ahead. Coronavirus is a test of human psychology, they tell me, and we are failing.

Lockdown 2: the darkest hour

The clocks have changed, the sky is grey, my children are not yet up, and I am having a minor meltdown.

It is 6am, and a desultory dawn is revealing another grim day of a lockdown. The day before, 492 deaths were reported. As the rain drums against my kitchen window, I find I am furious.

Over the summer there was a strange mobilisation on the internet. Climate sceptics and assorted contrarians shifted discipline, like a footballer during a loan move, to apply their skills to coronavirus. An orthodoxy developed that the pandemic was all over, and the experts were lying.

Then cases started rising, and – with a growing audience, including many Tory backbenchers – the sceptics needed an explanation. First, we were told, the test didn’t work and it was all false positives.

Then, when there were too many, they were “casedemic” – asymptomatic infections in people who weren’t ill. Then when all the people receiving false positives – like big hypochondriacs – started dying that wasn’t cause for concern either. They were dying not of covid but with covid.

It’s exhausting.

What excuse will the coronasceptics come up with to minimise this next? “I think you’ll find Liverpool’s crematoriums are still not at capacity”?

My job, in peacetime, involves writing about all kinds of science. I chat to interesting, clever people about interesting, clever things. I am used to dinosaurs and asteroids, not contrarians and columnists. Suddenly, though, science is political – and the political people don’t understand it. Now, as deaths accumulate, it’s too late.

The whole summer and autumn has been a failure of science communication.

Amateur epidemiologists with graphs made in Excel briefly became blogosphere celebrities. Moon-landing-hoax-level complexity was deployed in baroque Twitter threads to reassure us it was all fine. Academics nitpicked around the edges, allowed themselves to be misinterpreted, and in doing so undermined the core. Columnists with humanities degrees started throwing around recherche accusations – “You don’t understand Bayes’ probability theorem.” No, you don’t understand Bayes.

We have – to give just one example – Desmond Swayne MP claiming the tests can’t be trusted as they give a false positive rate of 2.4 per cent. He was speaking at a time when the total positive rate was far less than half that. I don’t want to blind you with science, Sir Desmond, but how can you get fewer total positives than false positives?

We needed a debate about policy; instead, we had a debate about reality. Rather than discussing what to do in a mature fashion, we have played whack-a-mole in the carnival of bullshit. And all the while the virus, unperturbed by conspiracies, has spread. More people have died, more businesses have been harmed, because we never got to discuss the pandemic like grown-ups.

It is a comment online below one of my pieces, accusing me, again, of ignorance of false positives, that finally tips me over the edge. In one furious hour as the sun rises I write a 1,000-word comment piece. The last paragraph is, “A hailstorm of bollocks landed in a sea of filth, and the ripples of excrement washed over all of us.”

Just as I finish, a child pushes open the kitchen door and the morning begins. My mouse hovers over “send”. Is it too strong? I save to drafts. Later.

Ugur Sahin remembers when Albert Bourla, the CEO of Pfizer, rang to tell him they had trial results on the vaccine they had developed together.

“There was an elongation of time,” the BioNTech chief executive officer tells me. Bourla was about to pass on the most important number in the world. Holding the phone to his ear, “the anxiety grew and grew”. Then came the result: 90 per cent efficacy, far better than anyone dared hope. “It was an extreme relief. It just means so much.”

A week later Tal Zaks, chief medical officer of Moderna, heard a very similar number, relating instead to his vaccine. It was, he said, “one of the greatest moments of my life”.

A week after that Sarah Gilbert, not a woman given to hyperbole, was at home when it was her turn. It was, she tells me with some difficulty, “one of the greatest moments of my life”.

The very sensible scientists were wrong.

Most vaccines, we know, fail. Most that succeed take years. Very sensible scientists very sensibly warned that we could not rely on a vaccine as our exit strategy.

The very sensible scientists were wrong. They were gloriously, joyously wrong. “We have a vaccine for the world,” says Professor Andrew Pollard, who ran the Oxford trial. “This is an incredibly exciting moment for human health.” You wait a year for a vaccine, then three come along all at once.

A long winter is ahead, but in the hard and frosty ground of lockdown we can already see the green shoots of spring.