Science Magazine Podcast

Transcript, 3 May 2013

http://podcasts.aaas.org/science_news/SciencePodcast_130503_ScienceNOW.mp3

Promo

The following is an excerpt from the *Science* Podcast. To hear the whole show, visit www.sciencemag.org and click on "*Science* Podcast."

Music

Interviewer – Sarah Crespi

Finally today, we have David Grimm, online news editor for *Science*. He's here to give us a rundown of some of the recent stories from our daily news site. I'm Sarah Crespi. So Dave, first we have a story about the first people of Australia. Where do they come from? How did they get there?

Interviewee – David Grimm

And how many were there? That's really been a big question, and some scientists have speculated that it was a very small, what they call, founder population – maybe just a few hundred people. And that over the millennia, that population of Aborigines grew to more than a million. Today, it's about half a million. This new study suggests that it was a much large founding population, perhaps on the order of about 3,000 people, that originally founded Australia.

Interviewer – Sarah Crespi

And how long ago are we talking – 3,000 people going to a new continent?

Interviewee – David Grimm

We're talking about 40,000 to 50,000 years ago. These people would have come from Southeast Asia, and the new date comes from looking at a number of artifacts from these aboriginal people. The researchers looked at 5,000 cooking pits, human burials, shell heaps, charcoal deposits – any sort of evidence of very ancient human habitation. They dated these materials with radiocarbon dating, and they were able, with the radiocarbon dating the amount of material that they were finding, to sort of backdate a population.

Interviewer – Sarah Crespi

That's kind of an unexpected method for determining population structure. A lot of people would think you'd go to the genes of the current population and look back. How did those kinds of results compare with this radiocarbon dating of artifacts?

Interviewee – David Grimm

So the genetic evidence was suggesting that this was a smaller founder population, but the advantage of the new method is it's not just looking at genes, it's actually looking at actual physical evidence of occupation. And when you look at things like burial pits and cooking pits, you're really looking at things that would suggest what people were actually doing and how many people may have actually done it. Now again, this is still an open

debate, because we really can't go back in time and figure out how many people were there. What's interesting about this new number though is if this 3,000 number is actually accurate, what it suggests is it wasn't just maybe a family that got lost in the sea or maybe a few families that got lost in the sea and ended up in Australia. It really suggests there was a purposeful migration to this continent. It could have been because of scarce resources in Southeast Asia or some other factors, but it really shed some light on the motivations of why people came to Australia in the first place.

Interviewer – Sarah Crespi
We have, maybe, a motivation here, but what about the how? How did large-sized populations like this get over to Australia?

Interviewee – David Grimm
Well, to do that, they'd really need some pretty big powerful boats and that's one, sort of, criticism of this study is that when Europeans first began to colonize Australia in the 18th century, what they saw with the Aborigines is that their boats were actually kind of flimsy. They were small, fragile rafts made of reeds or branches. That's not the kind of boat you would need to transport hundreds or thousands of people. It's possible that 50,000 years ago these people actually had a much more sophisticated watercraft and they became less sophisticated over time, but that's certainly one of the problems with assuming that a very large founder population.

Interviewer – Sarah Crespi
Really interesting. Next up, we have real life *Pinky and the Brain*.

Interviewee – David Grimm
Well for anybody who remembers Animaniacs, the series in the 1990's, there's a couple of characters named Pinky and the Brain. They were mice, and every episode, they tried to take over the world. Well, one of the problems with mice, why we don't think they are as intelligent as us and why they probably won't be taking over the world anytime soon, is that their brains don't look like ours. It's not just a size issue; it's actually a structural issue. Our brains, specifically our cerebral cortex (this is the outermost layer of the brain that's associated with high level functions such as memory and decision making) – it's very folded in our brains. You can actually – if you even conjured an image of our brain, you can sort of see those folds in your mind. The mouse brain, it doesn't really look like that. It's a much smoother structure. The advantage of the folds is it gives our brains a lot more surface area. That's a lot more processing power, if you will, to do a lot of higher level functions – things like language and complex thought – that mice aren't capable of, or at least we don't think they are capable of. And the question is, how did we get those folds in the first place?

Interviewer – Sarah Crespi
Right and how do we make mice smarter – a very important scientific question.

Interviewee – David Grimm

That's the other question. In terms of the fold question, which is a little bit easier to answer, it has to do, at least according to this new study, has to do with a gene called TRNP1. Researchers have found that when they mess with this gene in fetal mice, the mice brains start to look a lot more like human brains. They tend to have a lot more what are called cortical folds. Now, the researchers didn't test that the mice were actually any smarter, but the brains actually looked a lot more like ours. When they looked in human brains, they found lower levels of this TRNP1 protein in areas that were destined to form folds versus in areas that did not form folds, which suggests that the presence of this protein actually inhibits folding. Counter intuitively, the less of this protein you have, the more cortical folding that you have.

Interviewer – Sarah Crespi
So mice have the same gene that we have that makes our brain fold so much differently than theirs. What does this say about the difference between humans and mice?

Interviewee – David Grimm
Well, researchers have often thought that the reason that we are so much smarter than mice and other animals is because we have a whole bunch of genes that they don't have. Now that may still be the case, but what this study shows is that it's not a question of having a gene or not having a gene. It's really the expression of that gene – how much protein that gene produces. And in this case, both us and mice have TRNP1. It's just that we're producing a lot less of the protein from this gene than mice are – that's making our brains more folded – and that's giving us an advantage over the non-Pinky and the Brain rodents out there.

Interviewer – Sarah Crespi
Last up, we have a new way of treating the flu.

Interviewee – David Grimm
That's right, Sarah. So flus aren't just annoying, they can be very, very deadly. And researchers have been looking for ways not just to prevent flus from happening in the first place but when somebody gets the flu or influenza, as scientists call it, what are some of the ways to prevent it from actually killing you.

Interviewer – Sarah Crespi
We already do have a few flu drugs out there. Tamiflu is the one that most people are familiar with.

Interviewee – David Grimm
Right. There's also a drug called Relenza. And these drugs work okay. They block a surface protein on the influenza viruses, and that prevents them from leaving a cell after reproduction. They are pretty effective, but scientists have sort of questioned the safety of some of these drugs. You also ideally also want more than one way to combat the flu, especially if you're going to have something very deadly like the Spanish flu or the Swine flu. So in this new study, researchers decided not to go after the virus but to go after our own immune systems.

Interviewer – Sarah Crespi
Are they trying to boost the immune system or what kind of mechanism are they tackling?

Interviewee – David Grimm
It's actually the opposite. What they are trying to do is actually calm down the immune system. One of the reasons flu is thought to kill us is because it unleashes what's called a cytokine storm. This is a catastrophic over activation of the immune system that leads to numerous inflammatory substances being released into the whole body. It can result in multiple organ failures. So the question is can we not necessarily stop the flu but stop the flu from over activating our immune system, which eventually is what kills us.

Interviewer – Sarah Crespi
So are they able to show that they could do this?

Interviewee – David Grimm
They did. They focused on a molecule called Toll-like receptor 4 or TLR4. And TLR4 normally alerts the immune system to certain bacteria in the body, and it can be responsible for these cytokine storm-like events. So the question is, can we inhibit TLR4 and prevent the flu virus from over activating our immune system? What's cool about focusing on TLR4 is there's already a drug in clinical trials that blocks TLR4. It's called eritoran, and it's been used to treat sepsis. And sepsis, kind of like flu, is a problem which results from an infection in the blood, which also causes the body to overreact and can lead to death. What the researchers found was when they blocked TLR4 in mice that had been infected with a lab strain of influenza, 90% of the untreated mice die compared to only 10% of those given the drug. This drug was given two days after infection. When they gave the drug six days after infection, 33% of the animals survived, which isn't great, but it's a whole lot better than every animal dying and again, a lot better than the controls.

Interviewer – Sarah Crespi
So this is, as you say, in clinical trials for sepsis. Is the next step to do clinical trials for flu?

Interviewee – David Grimm
That would be the case. The first one tested in ferrets, which have been sort of a workhouse animal for testing flu therapies. And then ostensibly, they would go onto humans if it were successful in ferrets. One of the cool things, again, about this drug is because it's already in clinical trials, there's already safety information being compiled on it. That may speed its transition to market assuming it's as effective in humans as it is in mice.

Interviewer – Sarah Crespi
Great. So Dave, what else is on the site this week?

Interviewee – David Grimm
Well Sarah, for *Science*NOW, we've got a story about researchers developing cameras that see a lot like insects do. Also a story about why sharks cannibalize each other in the womb. For *Science*Insider, our policy blog, we've got a story about a pesticide ban in Europe. Also a story which has been generating a lot of attention about a Congressional proposal in the United States to add an additional layer of oversight to NSF grants. This would potentially replace the peer review process with something like a Congressional review. It's generating a lot of comments as might be expected from our readership and from the community.

Interviewer – Sarah Crespi
And you can also find a related editorial in this week's *Science* by Kenneth Prewitt.

Interviewee – David Grimm
And finally for *Science*Live, our weekly chat on the hottest topics in science, this week's *Science*Live is about, speaking of flu, the H7N9 bird flu. What are scientists learning about how dangerous it is, and what can be done to prevent it. Next week's *Science*Live is about the search for exoplanets. What are we learning about worlds like ours outside of our solar system. So be sure to check out all these stories on the site.

Interviewer – Sarah Crespi
Great. Thanks, Dave.

Interviewee – David Grimm
Thanks, Sarah.

Interviewer – Sarah Crespi
David Grimm is the editor for *Science's* online daily news site. You can check out the latest news and the policy blog, *Science*Insider, at news.sciencemag.org where you could also join a live chat, *Science*Live, on the hottest science topics every Thursday at 3 p.m. U.S. Eastern time.