# *Science* Magazine Podcast

## Transcript, 20 September 2013

http://podcasts.aaas.org/science_news/SciencePodcast_130920_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, Adrian Cho, a staff writer for *Science Magazine* and *Science*NOW, is here to talk about some of the recent stories from our daily news site. I'm Sarah Crespi. First up, we have a story on a potential treatment for a growth disorder. Researchers have nailed down the mechanism behind a kind of dwarfism, achondroplasia, but not how to treat it. This latest study looks at one such method. Adrian, can you describe how this disorder works?

**Interviewee – Adrian Cho**

Sure. When you grow as a child your bones grow at the ends in a growth plate. And what happens in those growth plates is that cartilage cells die and then are replaced by bone. And the disease achondroplasia is caused by the fact that the cells will have receptors on their surfaces – cartilage cells – and they get hyperstimulated by a substance called fibroblast growth factor, and that prevents the cells from dying so that they can be replaced with bone, and as a result the bones don't grow to their proper length. People who have achondroplasia tend to be shorter than about 1.5 meters, or a little less than five feet tall. They can also have problems such as the passage in the spinal cord can be narrow and they can have problems with their breathing and also the collection of fluid around the brain. So it's a serious condition.

**Interviewer – Sarah Crespi**

So with this mechanism in hand, what do the researchers do to try to interfere with the development of this disorder?

**Interviewee – Adrian Cho**

Well, first of all it must be said that this is a study in mice, so this is not a study in people. But what researchers at the French National Institute of Health and Medical Research did was to try to tie up this fibroblast growth factor by putting in extra copies of this receptor into the mice. And what they found is that the extra free-floating copies of the receptor just soaked up the growth factor and that kept it from binding to the cartilage cells and interfering with the normal growth. And quite remarkably, they found that when they did this in mice the mice developed normally. The incidence in these mice of curvature of the spine dropped from 80% to as little as 6% depending on how much of these receptors they injected into the mice. So it appears to essentially block the condition.

**Interviewer – Sarah Crespi**
So what are the next steps for studying this as a treatment for this disorder?

**Interviewee – Adrian Cho**
Well, this is just a mouse study so clearly this sort of thing would have to be tested for safety in humans and the like. It's notable that there are actually four different targets that are being investigated. This is just one of four molecular targets that's being investigated to treat achondroplasia, so there may be similar strategies involving different molecules. But this is just sort of the first step for this molecule.

**Interviewer – Sarah Crespi**
So next up we have a story on salty cichlids. The seemingly omnipresent cichlids – freshwater fish that live in lakes and ponds in South America, India, and Africa – have long been kind of a sandbox for biologists who study evolutionary systems. But there's still a big debate about how these fish got onto all these different continents. What's the hypothesis so far?

**Interviewee – Adrian Cho**
The debate centers around when the continents that we know and love today formed. About 135 million years ago the continents we have today were all glommed together in one supercontinent called Gondwana. And Gondwana broke apart and the different parts migrated across the globe to give us the world as we know it. The hypothesis had been that cichlids actually just went along for the ride, that they evolved before Gondwana broke up and that as the different parts moved they basically just went along like Huck Finn riding his raft on top of the different continents.

**Interviewer – Sarah Crespi**
So what made researchers decide that maybe this wasn't quite the right thing, that this wasn't what had happened?

**Interviewee – Adrian Cho**
Well, the problem is the breakup of Gondwana took place about 135 million years ago. The oldest cichlid fossils are only about 45 million years old. So that leaves about 90 million years of time unaccounted for. And one possible explanation was that the cichlids were there but they just hadn't been found yet. So to test that, a paleobiologist at Oxford University looked at the known fossil record of cichlids and also looked at sedimentary rocks that could plausibly contain cichlids. His analysis suggests that that scenario just doesn't really hold water. If that were the case, then for some unknown reason the probability of preserving cichlids would have had to have been 10-30 times worse in that 90 million years they're not accounted for. The researchers also looked at the genetics of cichlids to see when the species emerged and how it related to other types of fish. And what they found is that cichlids seemed to have originated about 65-57 million years ago, so long after Gondwana broke up and spread apart in pieces.

**Interviewer – Sarah Crespi**
So these two different methods found that it's pretty unlikely the fish stayed in the lakes as these land masses broke apart and traveled away. What's the alternative then?

**Interviewee – Adrian Cho**
Well, the alternative is obviously that they had to spread afterwards, right, that after the continents had been established that they had to make the big jumps across the ocean to spread to different continents. That's a little tricky because they're freshwater fish and the ocean is a big body of saltwater, so it's a little hard to see how that could happen. Although it's possible that cichlids were carried from Africa to South America on east-west currents, it's even possible that the outflow from the Congo River carried them across the ocean, which was narrower when all this was happening.

**Interviewer – Sarah Crespi**
Finally, we have a story on cork-tainted wine. Okay, I've tasted not quite right wine, but to tell you the truth I've never actually sent it back. What makes wine taste off or have a taste of a cork in it?

**Interviewee – Adrian Cho**
Well, so corked wine doesn't exactly taste like cork. The reason it's called corked is because it often occurs that the wine goes bad because there's a leak around the cork and it doesn't remain airtight, and then you can get this off taste, this sort of musty, acrid taste of corked wine. And, in fact, this so-called corked taint is produced largely by a molecule known as 2,4,6-trichloroanisole – or TCA – and researchers knew that, they knew that TCA was the main culprit in corked wine. What they didn't know was exactly what it did. The presumption was that when you taste corked wine you're either tasting the TCA or smelling the TCA, and that it's unpleasant and that was what was making the wine taste bad. New research by a team at Osaka University in Japan actually shows that it's kind of the opposite; the TCA actually interferes with your sense of smell and basically messes up your ability to perceive the taste of the wine properly.

**Interviewer – Sarah Crespi**
Okay. So why did they think that TCA wasn't acting as a normal odorant molecule – something that you would just inhale, it interacts with your senses, and then you have a different smell?

**Interviewee – Adrian Cho**
Well, so they didn't go in assuming that they had to rewrite the book on TCA, they just didn't know what the mechanism was and so they set out to find out. They assumed that TCA activated nerve cells in your nose called olfactory receptor cells. So the first thing they did was to look at the effect of TCA on these cells, and they actually used cells from newts which are three times as large as those from humans and so they're easier to study. So the first thing they did is they put little electrodes into these cells and they exposed them to TCA, and the presumption was that TCA would make these nerve cells fire. And, in fact, what happened is that TCA suppressed these nerve cells and made them fire less.

**Interviewer – Sarah Crespi**
So how exactly do the researchers think that TCA suppresses this reaction in the cells?

**Interviewee – Adrian Cho**
Okay. So the nerve cells fire and create electrical impulses when calcium ions enter through the cells through little passages called ion channels. And what this study shows is that TCA blocks up these ion channels so that the calcium can't get in. And it turns out that TCA is a thousand times more effective in blocking these ion channels than other odor blockers that are sometimes used in the manufacturing of perfume.

**Interviewer – Sarah Crespi**
Okay, so that's newts. What does it do for people?

**Interviewee – Adrian Cho**
Okay. So obviously you can't do exactly the same experiment with people, so what they did next was to turn to tasters. And so they recruited 20 folks from the Daiwa Can Company in Tokyo, which produces food containers, and these were people who were experts in tasting for off flavors but not experts in tasting wine in particular. What they did is they ran double-blind taste tests with red and white wine to which the researchers added variable amounts of TCA, and they then measured how much of the TCA was required to detect it and they found out that people could detect this substance at a level of about 10 parts per trillion which is pretty low. So the assumption is, combining the taste-testing data and the data from the newts, is that the TCA somehow gums up your sense of smell, and because it's blocking your sense of smell you just don't taste the wine correctly.

**Interviewer – Sarah Crespi**
So this is blocking the ability of these cells to respond to smell, but is it just a general odor blocker when these wine tasters have TCA in their systems, are they only doing tongue tasting and not nose tasting?

**Interviewee – Adrian Cho**
It's not entirely clear exactly what's going on with the TCA because it doesn't render the wine completely tasteless. So it's possible that the TCA, for example, blocks only some of the ion channels in these neurons, not all of them. There's also a very curious effect here. The TCA just seems incredibly potent, because it turns out that when the researchers add just enough TCA so that there's about 600 molecules per cell, they get this big effect in the newt studies even though each of these cells has about 100,000 ion channels. So it's clearly not that the TCA just blocks the ion channels, it must be doing something more complicated. And the researchers hypothesize that this stuff settles into the cell membrane in some way that just messes up all the ion channels at once. So it's not exactly clear exactly how TCA does this.

**Interviewer – Sarah Crespi**
Okay. Well what else is on the site this week, Adrian?

**Interviewee – Adrian Cho**
Well, this week researchers with the Curiosity rover on Mars looked for and did not find methane, so that's interesting because methane could be produced by biological activity. Researchers also came up with a way to reprogram mature cells back into so-called pluripotent stem cells with roughly 100% efficiency, which is much better than they've been able to do in the past. And then on the policy side of things, for the first time in 82 years Australia does not have a Science Minister because the new Prime Minister of Australia, Tony Abbott, did not appoint one.

**Interviewer – Sarah Crespi**
Thanks, Adrian.

**Interviewee – Adrian Cho**
My pleasure, Sarah.

**Interviewer – Sarah Crespi**
Adrian Cho is a staff writer for *Science Magazine* and for *Science's* daily news site, *Science*NOW. I'm Sarah Crespi. You can check out the latest news and the policy blog at *Science*Insider at news.sciencemag.org.