#360: Can't give it up: The science behind addiction and the brain

VOICEOVER
This is Up Close, the research talk show from the University of Melbourne, Australia.

ANDI HORVATH
Hi. I'm Dr Andi Horvath. Today we bring you Up Close to the topic of addiction. We humans have used mind-altering substances from the earliest times in our history. Our addiction to these substances stretches back as far. These days we talk of drugs, recreational and pharmaceutical. Addiction to drugs is widespread globally, clearly harmful to health, society and is economically destructive. It's a brain disorder with the brain?'s altered metabolism amplifying drug cravings and making relapses back into drug dependence more likely.

Today on Up Close we explore the compulsions that drive drug addictions and the behaviours that surround drug taking and attempts to withdraw from addictive substances. The latest research into addiction does seem to hold some promise for helping, especially in the most intractable cases of addiction. Our guest is Professor Andrew Lawrence, a principal research fellow and associate director at the Florey Institute of Neuroscience and Mental Health, at the University of Melbourne. Andrew heads up the Addiction Neuroscience Laboratory there. Andrew, welcome to Up Close.

ANDREW LAWRENCE
Thank you.

ANDI HORVATH
Andrew first up, what is addiction?
ANDREW LAWRENCE

I guess at a simplistic level it could be operationally defined as a transition from a motivated behaviour into a habit and an abhorrent habit. Because it has that habit like nature it becomes essentially automatic and very difficult to cease.

ANDI HORVATH

Tell us about dependence and habit, because are the two the same?

ANDREW LAWRENCE

No, they're not the same but at some levels they're linked, so when we think about dependence people often think of a heroin addict who goes into cold turkey and has a very clearly identifiable withdrawal syndrome. That's due to a physical, physiological dependence. The person's withdrawing from the drug: their body has become dependent upon that drug and when the drug is no longer available or no longer within the body, the body reacts to that process. But in reality physical or physiological dependence can be managed quite quickly. People can be rendered no longer physically dependent upon a drug. But then there's another issue which we call psychological dependence, which is a much more complicated issue and that really is the key to long term relapse propensity, because the reality is people relapse many, many years when they're no longer dependent physically upon the drug. They could have been weaned off a drug and yet relapse two or three years later when they clearly are drug free.

ANDI HORVATH

Now we're going to revisit that particular concept, but I want to understand is addiction or substance abuse a brain thing?

ANDREW LAWRENCE

Absolutely. I mean there are identifiable changes in the brain when a person uses drugs, even just for recreational purposes. For those people that become addicted - which is the minority of people that use drugs I should add - but when those people do become addicted, there are clearly identifiable changes that occur within the brain. We can do similar experiments in experimental animals and demonstrate similar changes occurring in the brain that persist and that seem to equate to the behavioural manifestation of the addiction.
So even rats can get a bit drunk?

ANDREW LAWRENCE

Absolutely, rats and mice and other animals will willingly use pretty much any drug that you could imagine a human willingly using, whether it's tobacco - we use nicotine in animal experiments, but they will willingly use nicotine. They will willingly consume alcohol; they'll willingly consume opiates, cocaine, methamphetamine, cannabinoids, you name it.

ANDI HORVATH

From an evolutionary biology point of view what's going on there? How does this substance interact with our biology to maintain its power over us?

ANDREW LAWRENCE

Mm-hm. If we think from an evolutionary perspective our brain is not a series of neurones that are islands in isolation. They're in complex networks and within these networks there's a circuit that we commonly refer to as a rewards circuit. If you've had a pleasant meal you might think well that was really lovely, or you've got a partner and you've just had sex you might find that a very pleasurable experience. From an evolutionary perspective eating and procreating are necessary acts to ensure the survival of a species. If you perform a particular behaviour or particular act and it's a pleasant experience, the odds are that you're going to want to repeat that experience: i.e. you're going to want to repeat eating, or you're going to want to repeat procreating. It ensures survival. The problem is that drugs of abuse act upon that same network and therefore give this very highly reinforcing euphoric experience and their potency's very high. So taking drugs is typically a very salient experience and typically a relatively short-lived experience. So that high level of salience and pleasure and the short-lived nature typically develops that desire to repeat the experience.

ANDI HORVATH

So essentially drugs are harnessing that reward system of our brain?

ANDREW LAWRENCE

Absolutely, yes and that can be demonstrated in humans with imaging studies and with rodents in a range of different studies. It's quite clear that those drugs of abuse
all act one way or another upon that circuit.

ANDI HORVATH

Andrew, tell us about the spectrum of responses in humans. I know from my own group of friends I'm known as the "one-pot screamer" but then there are other people that can really hold their own.

ANDREW LAWRENCE

If you're talking about alcohol there's a whole range of factors that lead into that, so one is your metabolic capacity to metabolise alcohol through your liver. If you're a low metaboliser that's going to mean you're more likely to get drunk quicker and a good example of that, if we're thinking about genetic mutations, people often think about genetic mutations that confer additional risk to a disease or a disorder. Whereas there's a really good example of a genetic mutation that confers protection against alcoholism and that is typically in the Han Chinese population. It's a mutation in an enzyme called aldehyde dehydrogenase. So when you consume alcohol it goes into your liver and it's first broken down from alcohol into acetaldehyde by an enzyme called acetaldehyde dehydrogenase. The problem is that acetaldehyde is very toxic. It makes you feel unpleasant and it can make you feel nauseous. It can make your skin flush and get bright red cheeks and ears. It can make you sweat and it can make you feel dizzy and it's most unpleasant. So we have another enzyme called aldehyde dehydrogenase that rapidly breaks down the acetaldehyde and then it's ultimately excreted out through the urine in broken down forms. In the Han Chinese there's a mutation that basically means that this enzyme aldehyde dehydrogenase is a very low functioning enzyme, so when they drink alcohol there's a rapid build up of toxic acetaldehyde in their liver, makes them feel very unwell and therefore naturally limits the amount they consume. It's therefore a protective mechanism against the development of alcoholism.

ANDI HORVATH

Wow, so genetic predispositions do really matter when it comes to the biological response to alcohol. You mentioned the other end of the spectrum as well. Are there people who are quite immune to alcohol?

ANDREW LAWRENCE

Yes, there are. So you could imagine if someone's a high metaboliser, whether that's because they're a naturally high metaboliser or because over a period of time and consuming they've become tolerant and their metabolic capacity has increased. But equally there are people that may have a lower reaction to the centrally mediated
effects of alcohol, such as the alcohol induced ataxias, when people get a bit wobbly. People are less sensitive to that and they're less sensitive to the reinforcing effects. They may actually consume more to get the same effect to somebody else and that can instantiate that increased consumption.

ANDI HORVATH

What about the cultural elements in societies towards say alcohol since we're talking about alcohol? In Australian culture it seems to be quite a bit of bravado.

ANDREW LAWRENCE

Yeah, I think that's right. I mean if you look at Australia I think drinking is almost a national pastime. You look at major sporting events: typically they're sponsored by alcohol companies. There's nothing wrong with that per se, but then you see when a team has won a major tournament - often, not always - often they'll celebrate by consuming large amounts of alcohol. That message is given to the youngsters, that when they want to celebrate winning alcohol is a normal part of that celebration. Now I'm not saying that it doesn't have to be, but it doesn't have to be to excess either. There needs to be a message of responsible consumption. I'm not suggesting that we ban alcohol. I enjoy consuming alcohol myself. But the message to the youngsters has to be to consume it in a safe way and not to go on these binges which are very dangerous.

ANDI HORVATH

Sure. French culture for example has very much a normalised drinking with the meal, wine as part of the culture that seems to be a little bit healthier than binge drinking.

ANDREW LAWRENCE

Absolutely, a lot healthier and it's introducing children to a responsible use of alcohol. It's taking them away from the fact that alcohol is always equated to parties, getting out of control, rather than being responsible.

ANDI HORVATH

Now just to put the whole notion of addiction into context, we've been talking about the addiction of substance abuse. But are there similarities with say food addiction or even gambling addiction, sort of behavioural types of addiction?
ANDREW LAWRENCE

Absolutely. If you think about it simplistically at a behavioural level, they're all examples of compulsive type behaviour. So therefore at a behavioural level they all have a commonality. We also know now from neurobiological mechanisms that there are circuit similarities. There's not 100 per cent similar, but there are substantial similarities and there are treatment similarities. The fact that certain medications can be used and are used to treat opiate users, alcoholics, people with binge eating disorder and problem gamblers tells you that there's a chemical similarity in the brain underlying all of those different manifestations of a compulsive behaviour.

ANDI HORVATH

How widespread is this problem of addiction when we look at society's demographics and also globally?

ANDREW LAWRENCE

It is an enormous problem that costs a huge amount of money as well. It varies from drug to drug. Also your odds ratio of becoming an addict really depends a lot on how old you are when you start consuming those drugs. It could vary anywhere from probably five to maybe 20 per cent, depending upon what populations you look at, what drug you're talking about, age of initiation of drug use and all of those factors, but probably somewhere between five to 20 per cent for most drugs.

ANDI HORVATH

Give us some examples of the economic burden of substance abuse?

ANDREW LAWRENCE

Okay, so in Australia which is a pretty small country - it has a population of only 23 million - drug and alcohol abuse combined costs the economy $55 to $60 billion a year. That equates to somewhere between two and a half to three per cent of gross domestic product, GDP, and so if you crank the numbers up to the USA you're talking about $250 plus billion a year cost to the economy and society. It's enormous.

ANDI HORVATH

This is Up Close. We're delving into the addicted human brain today with Professor Andrew Lawrence, head of the Addiction Neuroscience Laboratory at Melbourne’s Florey Institute. Andrew, explain to us the different classes of substances. I know
there are stimulants and then there are depressants and then there are hallucinogens.

ANDREW LAWRENCE

Well if we start with the last group, hallucinogens, you can think of LSD is probably the most well known. But then obviously there are natural products as well such as magic mushrooms. Psilocybin is the ingredient there - and other natural products that have been used culturally by Indigenous communities for centuries, as parts of ceremonies and religious rites. More commonly now there are also synthetic hallucinogens. They tend to have a very different and distinct pharmacology to the other drugs of abuse and there's less of a dependence liability per se. They tend to act as what we call an agonist at a 5-HT2A receptor. The flipside of that is that antipsychotic medication that's used to treat people with psychosis and schizophrenia that may clearly be having hallucinations, are treated with drugs that block that receptor.

ANDI HORVATH

So there's a pattern there in the brain as to what hallucinogens do?

ANDREW LAWRENCE

Absolutely. You can think of them as a pro-psychotic drug at one level.

ANDI HORVATH

Alright. Let's explore the stimulants and depressants. Now I take coffee as a stimulant. What are the other stimulants?

ANDREW LAWRENCE

Yeah, well of course caffeine in coffee and tea for that matter - most people forget about caffeine being in tea, but it's the mostly widely used psychotropic drug in the world. Some people will have a withdrawal from caffeine on the weekend if they drink a lot of coffee during the week. On a Saturday they might feel irritated, a bit anxious, have a headache and often don't realise that they're undergoing a mild version of drug withdrawal, namely caffeine withdrawal. Other stimulants include cocaine, amphetamine type stimulants such as amphetamine or methamphetamine, ice. They're the most common psycho stimulants that are used recreationally. Cocaine was first used as a local anaesthetic and it has very good local anaesthetic properties, but it's also a highly potent and short-acting positive reinforcer. It blocks
the dopamine transporter in your brain and dopamine is the molecule that's released from part of that circuit that we called the reward circuit earlier on. When dopamine's released, if you've got cocaine in your system it will prevent the removal of dopamine from the synapse, so it prolongs the amount of time that dopamine is there and it elevates the amount of dopamine that's in the synapse relative to what it should be. The thing is with cocaine though it's a very short-acting drug. It's a simple molecule called an ester and in our bodies we have lots of enzymes called esterases which chop up esters. It's highly lipophilic which means it gets into the brain very quickly, but because it's a very simple ester and it's broken down by lots of enzymes, it means it has a very short what we call half life. It means the effect that you get with cocaine is a very short-lived effect. So if you imagine you've got a very salient experience that's highly euphorogenic but very short-lived, it's naturally going to develop that desire to want to go and repeat that.

ANDI HORVATH

Mm. An interesting opposition to this is that we also have depressants. We feel we need to stimulate ourselves, but we also need to wind down and bring down the anxiety or whatever it is that we crave the depressants for. Let's explore the depressants.

ANDREW LAWRENCE

Yeah, so I mean as you say they're used as a de facto way of calming down, of removing or reducing anxiety caused by the hurly-burly of day to day life and they're very effective acutely at that way. I guess alcohol is the archetypal depressant. The problem is of course if you take too much of any depressant you will die by respiratory depression. Opiates are a good example because opiates are depressants. When people die from an opiate overdose it's typically due to respiratory depression, because opiates act within the respiratory centres in your brain stem. If you overload that system you'll turn off and inhibit those breathing mechanisms and obviously die from respiratory depression.

ANDI HORVATH

Now I've heard you describe addicts as people who aren't so much trying to get high, but instead trying to not get a low. Can you explain what's going on in that statement?

ANDREW LAWRENCE

If you think about the transition to addiction and the reasons why people do things repeatedly, as we said before if an experience is a pleasant experience the chances
are you're going to repeat it. So if that experience is repeated a number of times, that pleasant experience continues and you develop that habit of wanting to go back to do that experience. But the reality is every time we use drugs, whether it's alcohol or just as I said a moment ago with the caffeine withdrawal on the weekend: the trade-off with using drugs is the relative size of the acute high or the pleasurable side of things, with an acute withdrawal effect which occurs every time we use drugs. So with alcohol, an example of an acute withdrawal effect would be a hangover the following day if people had consumed too much alcohol. They might have a headache, they might be irritable, they might feel nauseous and that is an example of acute withdrawal. So there's always this trade-off between the pleasant experience when you're using the drug and the unpleasant side of the come down afterwards, or the acute withdrawal. That's even without being an addict. People have withdrawal acutely from a drug every time they use it. It's just more often than not they don't notice it. The problem is when drug use becomes longer and longer and more and more established, relatively the size of the high is diminished compared to the size of the low. So in the early days the scale is tipped in the favour of the high: you have a very big high and a very small low. So you think gee, that wasn't so great afterwards but that's worthwhile coping that so that I can experience the high part again. When people become addicts often you seem to find that they're in this enormous low that it's very hard to get out of and so the best tried and tested way of getting out of the low is to re-use drugs, because it doesn't necessarily get them back up as high as where they were, but it gets them up out of the low.

ANDI HORVATH

Andrew, animals have been used to obviously study this effect: the highs, the lows, the various neurotransmitters in our brain and the various pathways that connect all this. How much insight have they really given us?

ANDREW LAWRENCE

I'd say a huge amount - although you have to remember the very important caveat, that they are animals and it is a model and any model is only as strong as its weakest link. But a lot of the animal studies match up very well with the human imaging studies and clearly there are things that we can do in animals invasively that are ethically not possible to do in humans. Let's just say you have a thought experiment and hypothetically we take 100 rats and we give 100 rats the opportunity to use cocaine. The vast majority will voluntarily choose to use cocaine. If that cocaine use continues for a period of time, say two or three weeks and then you look at that circuit in their brain, all of those animals will typically show some form of modification and dysfunction within that circuit. However if you let animals continue to consume cocaine for longer periods of time you basically end up with two groups of animals. The majority, probably 80, 85 per cent of the animals will just essentially titrate their drug use. They'll have the same amount of drug each day and if you give them an unpleasant consequence of that drug use, they will reduce their drug use or
even stop taking drugs. Whereas the minority, this kind of 15 or so per cent of animals, you typically find they start to gradually escalate their drug use. They're willing to work harder for their drugs and they become resistant to punishment, or resistant to an adverse consequence of that drug use, because if you equate back to humans, humans always have a consequence of their drug use. Not all animal experiments impose a consequence to the animal of their drug use, but when you do impose a consequence the vast majority will cease that drug use. Yet there's still this hardcore minority if you like that are willing to take the punishment because they still want to get hold of the drug. They're the ones that we call the addict-like animals. We can't say they're addicted truly as a human would be, but we call them the addict-like animals.

ANDI HORVATH

This starts to explain things like withdrawal, cravings and relapse for these individuals as well. Tell us more about that?

ANDREW LAWRENCE

Yeah, absolutely because the animals that are the addict-like animals are far more prone to relapse in a number of ways. One is that they're resistant to extinction and extinction is a form of behavioural therapy. It's a new form of inhibitory learning where we try and dissociate the task the animal performs from the delivery of the drug and so they learn a new contingency if you like, that the task that previously gave them drug no longer gives them drug. The addict-like animals find it harder to undergo that new form of learning, but even when they have they're more susceptible to a relapse subsequently if for example, a cue that was previously paired with the drug is made available to them again, they would be more prone to relapse. If we go back to the circuit level function, if you remember I said all of those animals after a relatively short period of drug use would show some circuit level dysfunction. Well what researchers in France have shown, if you look at these animals when you get them at a longer point in time where they've split into these non-addicted and addict-like animals, that that circuit level dysfunction in the majority of the non-addict animals if you like, has been restored by some as yet to be defined endogenous restorative mechanism. Whereas those that are the addict-like rats still have this persistent dysfunction which is contributing towards that relapse propensity.

ANDI HORVATH

You're listening to Up Close. Our guest is Professor Andrew Lawrence from the Florey Institute's Brain Centre where his research into drug addiction may offer some better ways to deal with the human brain’s response to drugs that cause addiction. Andrew, let's look at the current therapies. How do they work, things like naltrexone
and these types of drugs?

ANDREW LAWRENCE

Naltrexone is what we call a non-selective opioid receptor antagonist and what it does is it blocks the ability of either endogenous opioids such as enkephalins or endorphins to activate that receptor, but it also prevents synthetic opiates or natural opiates such as morphine or heroine from activating that receptor. So it's not only used though in the treatment of heroin addiction which may seem obvious from what I've just said, but it's also used in the treatment of alcoholism and also has been used successfully for some people at least, in the treatment of binge eating and in gambling disorders as well. So this kind of tells us that a lot of those different forms of compulsive behaviour are maintained at some level by the release of endogenous opioids within the brain. What we're doing with the patient that's being treated with naltrexone is it's preventing those endogenous opioids from acting on their receptor and continuing that behaviour; and that actually tells us at least that those peptides, those endogenous peptides must be being released during the consumption of the alcohol, or during the gambling for example, because otherwise the antagonist would fail to have an effect on changing the behaviour.

ANDI HORVATH

Let's talk about your research. You've been on the hunt for some other receptors in the brain as possible new drug targets in the future.

ANDREW LAWRENCE

Absolutely, so we've been investigating the circuits in the brain, particularly that drive relapse to alcohol seeking. We've identified a couple of other peptide systems in the brain that are strongly implicated in that behaviour and act within that circuit, one of which is a peptide called orexin. The Americans call it hypocretin, but it's the same peptide. We've demonstrated as have others that that's inextricably linked to cue-mediated relapse to alcohol seeking. In fact we were the first to demonstrate that almost 10 years ago. We've subsequently gone on to pinpoint the areas in the brain where these receptors are being activated to drive this relapse and there is a large pharmaceutical company interest in orexin peptides and antagonists of orexin receptors, not only for addiction type treatment but also for sleep disorders and a range of other factors. Another peptide system that we've come across that is implicated in this circuit as well is a peptide called Relaxin-3, which was actually identified at the Florey many years ago as a brain peptide, but no one knew what it did. In the last few years we've demonstrated that the neurones that produce this peptide interact with this reward circuit. No one knew they did up until our experiments and we've demonstrated a really key role for receptors that this Relaxin-3 peptide acts upon. They're called RXFP3 receptors - but we've demonstrated that
as opposed to the cue-mediated relapse that we spoke about with orexin, we know that these Relaxin peptides are very, very strongly implicated in relapses mediated by a stressful experience. So we've got these two systems, one of which we know is more highly tuned in to a stress type relapse and the other system, the orexin system which is tuned into a cue relapse where it is also tuned into a stress relapse, but we still have this dichotomy if you like of different precipitants of relapse may actually invoke different subcomponents within that circuit and therefore may be targeted in a differential manner.

ANDI HORVATH

What's the Relaxin molecule actually do?

ANDREW LAWRENCE

The Relaxin-3 is a neuropeptide, which means it's a peptide made within the brain in a certain group of neurones. It's implicated in a number of functions: arousal, stress reactivity, spacial navigation, emotional memories and for all of these reasons we hypothesised that it's likely to be implicated in drug seeking behaviour as well, because also we knew it was implicated in altering feeding behaviour.

ANDI HORVATH

So this will take a while to get drugs that respond to these receptors that you've identified. We're looking at a long period of clinical studies as well to get these on the market.

ANDREW LAWRENCE

Yes. With the orexin molecules there's already pharmaceutical company interest in those compounds that act [at] orexin receptors. With the Relaxin-3 side of things it's much more embryonic. There is interest in it, but one of the problems that we have on that side of things is all of the compounds at the moment we have to use are peptides. Peptides aren't so good as therapeutics because you want to be able to take a tablet that is orally bioavailable and that accesses the brain. Peptides often are not good at accessing the brain because they're relatively large molecules, but also because peptides are found naturally within the brain they typically have a short half-life because the body is very well set up to chop up peptides from a metabolic perspective. So we really need some small molecules that act on the Relaxin-3 receptors to help us go forwards.
Andrew, I'd like to explore more about the issue of relapse. Now nicotine is a very addictive substance. I'm not sure if it's one of the most addictive substances on the planet, but there seem to be biological and social triggers that make people relapse?

ANDREW LAWRENCE

It is very addictive and the primary reinforcing strength of nicotine is not necessarily as high as some other drugs, but it's a very strong secondary reinforcer, a very strong conditioned reinforcer. People will associate a particular time of day as being a time they have a cigarette, or if they have a coffee: I always have a cigarette with a coffee. Or if they're having a drink in the evening: I always have a cigarette with a drink, or always have a cigarette after a meal, or stuck in a traffic jam: oh, I'll light up. I'll always have a cigarette - so you get these contextual associations that become ingrained with the use of the cigarette. That's why it's hard to break, because then if you decide you want to stop smoking, you get yourself back into one of those situations, you're telling yourself oh, this is time to have a cigarette. So now you've got to tell yourself this is no longer time to have a cigarette - and that's a very difficult conversation to have with yourself. It's a combination of biological triggers, psychological triggers through these cue associations and social triggers, because the social and the contextual influences go hand in hand, because if you always equate being with a certain group of people to smoking, when you're with that group of people you're going to think about smoking.

ANDI HORVATH

Andrew, what about diet and exercise?

ANDREW LAWRENCE

So they're going to be protective measures. Obviously diet, you assume it's a healthy diet, not an unhealthy diet because an unhealthy diet could cause a compulsive binge eating disorder by itself. But if we assume it's a healthy diet, that is part of a healthy lifestyle, part of a recovery process, as is exercise. Exercise is actually a very efficacious antidepressant and some analyses have suggested its good as SSRIs, your Prozac type medications for treatment of depression. Exercise stimulates the growth of new neurones in your brain. In animal experiments if you do what's called environmental enrichment, where you provide them with running wheels and things of that nature to explore and to interact with, that is very protective against addictive type behaviours in animals. There is good neurobiological evidence that it's a worthwhile thing to introduce into a treatment program.

ANDI HORVATH
Andrew, I have to ask can you become addicted to exercise?

ANDREW LAWRENCE

There are certainly people that compulsively exercise and there are animals that will show obsessive or compulsive exercise. If you give them running wheels they will run kilometres a night, just as some humans will go and run kilometres every day. If you think about the definition of addiction as a clinical disorder though it's that it's doing you harm. Even if someone is obsessively or compulsively exercising, if it's not doing them or anyone else any harm you can't operationalise it as an addiction. It's just a - may be an abhorrent behaviour.

ANDI HORVATH

Is that similar to the adrenalin junkies, the people who like to do sort of high risk type behaviours?

ANDREW LAWRENCE

To some extent, yes. Your adrenalin junkies so-called, your people that like to skydive or whatever it is they like to do would typically score very highly on psychological tests of thrill seeking behaviour. Now that in itself is a risk factor for drug abuse and for transition to addiction in drug abuse, because people that are high thrill seekers or risk takers may be more likely to use a drug of abuse than someone who scores very low on that scale, because someone that scores very low on that scale may be more worried about the consequence of the drug use, rather than the nice experience of using the drug in the first place, so it can be a risk factor for drug use.

ANDI HORVATH

Andrew, thank you for being with us today on Up Close.

ANDREW LAWRENCE

You're welcome, my pleasure.

ANDI HORVATH

Professor Andrew Lawrence is a principal research fellow and associate director at the Florey Institute of Neuroscience and Mental Health, based at the University of
Melbourne. You can find links to his work, research and publications on the Up Close website, plus a full transcript of this and all our other podcasts.

Up Close is a production of the University of Melbourne, Australia, created by Eric van Bemmel and Kelvin Param. This episode was recorded on 30 November 2015, produced by myself with studio production by Peter Clarke and audio engineering by Gavin Nebauer. I'm Doctor Andi Horvath. Cheers.

[Music plays].

VOICEOVER

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