**Adam Guastella – What is love? Harnessing the power of oxytocin**

>> Welcome to the podcast series of Raising the Bar Sydney. Raising the Bar in 2017 saw 20 University of Sidney academics take their research out of the lecture theatre and into bars across Sydney all on one night. In this podcast you'll hear Adam Guastella's talk, What Is Love, Harnessing the Power of Oxytocin. Enjoy the talk.

[ Applause ]

>> Oh, man, this is so awesome. I've never given a talk in a pub before. It's usually boring academic environments. This is fantastic. Well, welcome, and hello loved up people. Thanks for turning out today. It's the love fest you've turned up for. And what I'm going to talk to you about is really how we're going to use love and these ideas of social bonding to advance mental health treatments. So let's get into the fun stuff first. I can feel the pheromones, the excitement, the adrenalin. I can feel it all in the room. And I just want to get that love out. I think it's really important we acknowledge the love in the room tonight. So what I'd like you to do, if it's not too much trouble, [inaudible] Harvey Weinstein and you've got the consent of the person beside you I want you to stand up and give that person a hug. Give them a hug. Come on. The hug is so important. Ah. Ah, this is wonderful. Okay, good. All right. Now, when we're giving a hug it's just an amazing chemical mixture that you've just activated in your body. When you do that tight hug, the release of dopamine, the release of oxytocin, it's a thrill, it's exciting. If you want to you can do a bit more of a rubbing there and just a bit of soothing. If you're tall you can tap on the top of the head and go there, there. It's really quite an amazing mix, and it really has an important role in social support. But that is not the goal of my talk. The goal of my talk today is a talk about social bonding, and I'm going to start with the initial phases of love, the pop science. Does anyone know about the initial phases of love? The people well versed in what happens when you first fall in love? Come on, you must, you must. You get mad. Your best friend all they talk about is that new partner. All they do is go on and on and on about how wonderful that person is. It's sickening. It's disgusting. But for that person it's all that matters. And you see what's happening in the brain in that initial phase of love is huge amounts of dopamine and oxytocin are being released. The reward centres of the brain are being lubricated. And in those moments attention is being placed on anything which reminds you that's awesome about your loved one. And we know what happens when we focus on everything that's awesome. My messiness becomes spontaneous and carefree. Everything that's possibly awful about that person becomes wonderful and great. It becomes sickening. Now, of course, that phase lasts only for a short period of time. And in humans we know six months is the average. But it can last for up to 18 months. Eighteen months of listening to your best friend talk about that partner. And when people have looked at the brains of these people that are so called in love, [inaudible], yup. The brains of people that are in love, the brains, the way they respond to this cue about their loved one is very similar to how they respond to drugs. It's like the brain is addicted, addicted to that person. It's amazing. And what happens when we break up? Well, we go through the drug of withdrawal. Oh my, it is so depressing. We have intrusions, nightmares, sickness. All we want is that person back in our lives for those weeks and those months to bring them back. It is a withdrawal system. It is a craving. And, again, imaging studies show that this is like the cravings of someone who cannot get their drug. Okay. Now, I could talk about this, and I could also talk to you about how to my people who don't necessarily like you to fall in love and you to be the most amazing person in terms of love and romance, but that's not the point of this talk. I'm going to talk about hard science. I'm going to talk about social bonds. Now, I want to [inaudible] briefly I'm going to do some serious un-learning of you based on the University of Sidney advertising campaign. Okay. So my name is Adam Guastella, and I'm a clinical psychologist. And in the 90s I was studying lots of things about psychology, about thoughts, emotions. But in the 2000s I got really interested in the biology of social behaviour. I've got a great team of researchers, Ph.D. students, staff, really energetic, excited to understand how we can improve treatment for social problems and so this is really [inaudible]. Okay. So let me tell you about why we're so interested, why do we care so much about social behaviour and the people who matter. I'm going to tell you about Rose [assumed spelling]. Rose she's a little eight year old girl. She's intelligent. She has lots of really fascinating interests. And she can talk your ear off. Man, she can talk your ear off about fantasy lands, warriors, princesses and dragons. Actually that's part of the problem. She talks your ear off about these things all the time. Interrupt and say something that you're interested in? Forget it. She'll tell you about the warriors and princesses all day. And while she'd love to attend school and she'd like to have lots of friends, it's an up and down day-to-day proposition. She feels overwhelmed with the amount of information the playground. The noise, the dirt, the way the kids play games, the predictability. She finally understands the rules of the game, and then a kid comes along and wants to change it. It's so frustrating. It's distressing. It's too much. No, it's just too hard. It's much easier to stay at home. And there's other problems. She goes to have a shower, and the feeling of the water on her skin is too much. She hasn't showered for days. She doesn't like to wear clothes based on the fabric of the clothing. And the school shoes they feel like pennies on her feet. Now, the family they recognise the potential of Rose. She's such a beautiful girl. And they've gone to doctor, to doctor, to occupational therapists, to psychologists, to doctor. Did I say doctor? And they've often waited months for their appointment. And then when they go to the appointment they say what can we do? And the doctor says, well, I don't know. There's not a lot of evidence. There's not much research that's been done. But maybe we could try this. And as the parents who have to drag their daughter into the car screaming forcing her to go to school the parents feel guilty. What are we doing to our beautiful child? The distress, the tears at night all the time. It doesn't stop. There's no break. What do you think that does? So for these families [inaudible]. So for these families it can be incredibly distressing. And it's not surprising that the parents it's high suicide rates. We often have families talking about suicide and parents. Distress, difficulties with work. This is a very serious issue. Let me tell you about Jack [assumed spelling]. Jack is a 14 year old boy. He is the fastest Rubik's Cuber you'll ever meet. He loves his Pokemon cards, and he gets right into computers. Unfortunately sometimes until four in the morning. He's never had friends to play over, though. He used to try, but he found it all too difficult. No one really liked Jack. He doesn't know how to start a conversation or how to follow the threads in conversation. It's that back and forth he has difficulty with. And while he loves to play handball, the risk that he might be being bullied is too much. He just goes to the library every day. That's his lunchtime in the library where it's safe and calm. And I can't tell you how many clients we have that spend their time in the library. So both of these people are described very broadly with not a lot of detail. But they're people that we would typically see who would be diagnosed with autism spectrum disorders. And before I go any further let me just clarify two points. If you've met one person with autism you've met one person with autism, okay? There's a lot of diversity in the spectrum. So when I give examples I'm just making generalisations about groups. But there's lots of diversity. And the second thing is Lorna Wing was one of the people responsible for the development of the diagnosis of autism. And she provided a wonderful description of what it's like to be involved with the diagnosis. It's like looking through a prism where at the other end a wonderful array of colours emerge at the other end. And those colours can't be defined by a diagnosis, can't be defined by a single person. There's a whole spectrum to understand. But these people have core difficulties, difficulties in interaction. And we often see difficulties in eye-to-eye gaze, reciprocation of social contact, understanding the nuances, the nuances, what's in between the hidden lines. It's that sort of stuff that people often miss. It can often be diagnosed by 18 months, and there's lots of research at the moment to really push the diagnosis at 18 months of age. Now, some of you will know that the rates of autism have really accelerated. I also just want to make this 100 percent clear. There are two main reasons why the rates of autism have increased. The first is diagnoses have changed over time, right? So there has not been an increase in autism. The people who have [inaudible] intellectual disability and for the really severe spectrum there's been no increase whatsoever. The rates do not say that at all. The increase has been entirely in the high functioning, what we call the high functioning group, the group that tends to go to schools and operate in other ways but have difficulties in social interaction. We're picking them up now, okay? The second thing is the increase is entirely in kids because they're the ones that are being assessed. We're not assessing adults. We're not going back 30 years and assessing the adults. It's entirely in kids. So this increase is leading to a recognition that autism is, in fact, the greater problem or affects a greater number of individuals than we previously thought. The rates are about one in 68. It also raises questions about what is social development. And let me just give you some examples. At six months of age we'd expect babies to be responding to their name, in some ways smiling and we can goo goo with them and tickle them when we say their name. At 12 months of age we'd expect them to respond to some gesturing and waving. At one and a half we might want to see some imaginative plays, some playing with toys and picking up the phone and pretending they're making a phone call. We might want to see them to be able to follow my pointing with my gaze to follow where I'm looking. At two we want them to start using phrases. And at four we'd want to see lots of creative play and turn taking. And when this goes wrong and there's lots of delay then we're starting to talk about autism, okay? So going back to our baby, baby infants, all right? One of the most important features of social development is the ability to read information, okay? It's the ability to know in this situation that I'm in right now that what I have to look at are all these wonderful faces interested in what I'm saying and these smiles. That's what I need to look at. Thank you very much. It's not the sun, it's not the lights, it's not the lights there, and it's certainly not the distraction of the noise behind us. It's the social keys. It's the stuff that matters in this situation right now. And it's thought that the ability to know, to look at the eyes and key parts of the face it tells us a lot about social situations. And if you don't have that basic capacity to look at the face and read the social cues you're missing a lot about social information. Now, Cline [phonetic] showed back in the early 2000s that in young infants, like we're talking nine month old infants, their ability to look at faces and just hone in on the right information it predicted social development capacity and autism years later, we're talking three, four years of age. More recently he showed that this was genetically controlled. So identical twins he showed the skein patterns of two year olds were 90 percent concordant which means the same, 90 percent. So if he presented social scenes, identical twins looked at the social scene in exactly the same way with 90 percent. Maybe ten percent would have been different. For those that were not identical twins it was 35 percent. So genetics plays a huge role in early development, 35 percent. Now, this is really important because the way that we pay attention is thought to be a fundamental building block of social development. First it's looking at mum and dad's face. Then it's learning to follow social rules and then teaching us social rules through gesturing. And if we don't get that imagine what it's like then to enter a playground and be faced with multiple kids saying multiple things to us all at once. This early development is critical. [Inaudible] bald head which is making this [inaudible]. And in autism we know there are clear brain developmental differences, not in everyone but in groups. And one of the most important developmental differences is brain overgrowth at the moment. That's the main, if you like, theme in the brain development literature. Brain overgrowth occurs in a large portion of individuals with autism and accelerates up until the age of ten. And what this means is there are synaptic connexions occurring in greater density with greater specialisation in specific regions. And you might think that's great, and it sort of is great. And it's thought that this higher density leads to specialisation of skill. But the problem with it is, and the current theory about it is it's not organised in the best way. The highways which join everything up don't organise the structures well enough. So when we have to do multitasking, complex tasks that require multiple different actions that's where we fall down with brain overgrowth. So we're going to talk about social development. And social development involves lots of things that we've just explained, language, planning, memory. And it could be a game of chess. You know, you could think of it as a game of chess except for one thing, it's spontaneous. You can't predict it, and it needs to be in sync with the other person. It's like a dance. You have to smile at the right time. You have to cry at the right time. You have to move at the right time. And there's lots of research to show that there's actually a physiological synchrony that our hearts in some ways move together with the person we're interacting with. It's a dance.

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>> So let me just give you a glimpse of how complicated this might be in just one story. We have Dean [assumed spelling]. We'll call him for argument sake Dean Jones. And he's an audacious young cricketer. He is sweating profusely from his brow. It is the 48th over. He fidgets, he moves with every ball, he pats his trousers. He taps his bat on the pitch, and he looks at the scoreboard every five seconds. A [inaudible] tosses the ball, and without a second thought he dances down the pitch to hit [inaudible] for a six over cover. But, atlas, it's the death rattle. He falls to his knees, his head in his hands, and he remains motionless for two minutes. Well, the baller is thrilled. He runs over, taps him on the head and says what a great shot. Now, if you don't know anything about cricket I'm suspecting that paragraph was a little difficult to understand. And that was the point. At the start he was very anxious. I never said that. He was very anxious because it was a 48th over, there's 50 overs in a match, he's trying to score the winning runs. There's a death rattle. He's been balled, I didn't say that. Who knows what a death rattle is? He didn't take his head off and put it in his hands. He leant over and put his head crouching in his hand. And the baller certainly wasn't congratulating him for a great shot. He was excited he got him out. I never said that. It was implicit in the story. And if you can't read in between the lines then you get the story wrong. And that is the key feature of social interaction. You need to be able to read in between the lines. Because then you can respond appropriately in social situations. Does that make sense? So this is a really complex thing. How do we teach this, how do we do this? So complex that, in fact, when I was training as a psychologist in the 90s there wasn't really any intervention. I don't recall every being taught anything about how to improve social development as a psychologist. I don't recall being taught much about autism in general. And, in fact, most of the work was done on schedules about reinforcements and punishments to manage behaviour, all right? We were just trying to manage the situation, not actually change trajectories or improve skill. And the literature, the science, the evidence will back me up on that. So we conducted a review just a couple of years ago on every single study that had used a medical or dietary supplement in child autism, every single study, okay? We had some really, really important selection criteria. They had to have more than ten kids in an arm, right? So more than ten kids in a placebo arm, and then whatever the treatment was, the active arm, okay? There needed to be some diagnostic instrument. And there needed to be a scale that was recognised as reliable as an outcome, right? So just to give you -- if we had that inclusion criteria in schizophrenia, depression, anxiety we'd have hundred of studies, hundreds. I came out, well, I, my students and me a team came up with 37 studies in total, 37. The diagnosis has been around since 1918, 37 studies. Only one of these studies had used more than 100 children, one, one study. Most of the studies at 10 or 20 kids. Six studies in total had any biological measure. I mean we're talking about a neurodevelopmental disorder. It's a brain developmental disorder. Six studies had included any measure of biology. And this is the catch cry here, 40 percent of autism is [inaudible] with intellectual disability. None of the studies had any child with intellectual disability. It stunned me, I was like this is terrible. No wonder when families go to the GP and ask for a treatment the GP goes I don't know. We could try this, we could try that maybe. Have you seen that news article on the website [inaudible] London said maybe we could try that. It's an absolute disgrace. And this is partly why the dominant treatments medically for autism are repurposed drugs that are used for other conditions. So the only approved medication are antipsychotics. They're used for schizophrenia. But we don't even use it as an antipsychotic. We use it to reduce irritability and aggression and to sort of slow things down a bit. There's lots of studies looking at antidepressants to try and reduce anxiety. But there's nothing, social development forget it. And these studies, 37 studies, none of them showed benefits in social development. Now fast forward to 2017, the year right now, and things are looking a bit more promising. We're a bit more excited, and there's a couple of reasons. Has anyone heard of the Brain That Changes by Norman Doidge? Yes, we've got some hands up, right? That came out in the mid 2000s. And it was a lay book, but it was really a great book demonstrating where the field was at in the 2000s. What we learnt about the brain was that it has capacity for change, for new learning. And the brain wasn't hard wired. It wasn't set for life. The environment, what you did, what you learnt impacted on growth. And there's great capability for new learning. We learnt about different features of the brain. So we learnt about glial cells which we generate, they repair and they establish new connexions. Even in adult brains, even in older brains. And we also learnt that the environment plays a huge role. So things like exercise, diet, let me say it, alcohol all plays an important role in these processes. But all this work was driven largely by the other end of life, the dementia alzheimer space. We know, for example, there's lots of studies around cognitive training, exercise. There's a real big push at the moment for how we can look after our brains to reduce the impact of alzheimers and dementia. But it's filtered back to autism. And the reason why it's filtered back is because we think, well, early on when we're trying to lay the foundations, the important foundations of social development, if we expose young children to the right environments we can improve outcomes. And that's certainly the focus now. So we've got lots of training programmes, really important training programmes for early development, engaging joint attention, eye-to-eye gaze sort of work, understanding social rules, lots of play, lots of imaginative, imaginative play. And there's no doubt that these procedures produce a lot of benefit. But there's a couple of problems. Some of them require 40 hours per week, that's right, 40 hours per week with families. That costs a lot of money. So the one we're using at the moment you have to do it for two years. And the evidence is we think, we [inaudible] think it's doing a good job. The evidence is okay for a year or two. But there's been no study. I mean it's a really hard study to run. But there's been no study which is to track development for many, many years to show changes in development as a result of this intervention. But it's something that all children with autism should have access to. So let me get back to the point of this talk which is also to discuss the neurobiology. And there was something going on in the nursing wards which was really, really important. And Vincent de Vigneaud, the organic chemist, was critical in understanding what that was. It was a thing called oxytocin. And what he identified was the structure of oxytocin. He was able to characterise this nonapeptide, this nine amino acid peptide in the body. And that was wonderful. I mean for organic chemistry, for identifying natural substances and the structure of it, for organic chemistry it opened up a whole new wonderful world of peptide research. And he won the Nobel Prize two years later. But what it also did was it gave a wonderful opportunity for new interventions. So oxytocin we know in the human body when it's naturally released it has very important effects. So in labour it is released naturally and facilitates contractions and allows babies to be born quickly and smoothly. It facilitates milk let down for breast feeding. It's involved in sexual reproduction. It's involved in eating, and we know it's also involved in cardiovascular regulation. And we also know is has immune activation properties to have protective effects in terms of wound healing. Okay. But once he was able to identify the structure he developed what we call the synthetic version of oxytocin. It can be administered as an injection. And it changed, if you like, the nursing wards across the world. For any woman here who's given birth 50 percent of you, I think it's 50 percent, don't quote me on that although it's been recorded, 50 percent I think receive oxytocin that facilitates contractions. If there's any problems, if there's any holdup in the birthing process, again, oxytocin facilitates contractions saving thousands of lives of babies and their mothers where problems emerge. It also results in milk let down. So when mother's are having difficulty feeding their children it's often used to facilitate outcomes. So it already back in the 60s and 70s and today had a huge impact on society. But it also as associated with lots of other observations. Nurses started noticing, wow, when we give this oxytocin people seem to get this warm, fuzzy feeling. They start to cuddle a lot more. They seem to be able to relax. And we started measuring oxytocin in the blood. And what did we find? Well, when we touch, when we cuddle, when we kiss oxytocin is released, okay? We started to think, wow, this may be used as a cuddle hormone. This is a hormone that's important for social bonding. At times it can act inversely to cortisol, the stress hormone. So we think that in some ways oxytocin might have a double effect of reducing anxiety as well as increasing approach behaviour and social behaviour. But all this stuff is correlational. We don't believe that correlation means causation. That's a very sciencey sort of statement but it's true. None of this means that oxytocin does anything. And where we needed to go was unfortunately, I hate to say it, I've never done it, I don't do it, to animal science, okay? We had to start injecting oxytocin directly into the brain. We had to start looking at the brains of animals to see what happens to the oxytocin receptors. Now, how many of you have wondered if rats fall in love? Yeah, how many of you have wondered about the maternal behaviour of sheep? Okay. Well, if you're not interested because it's developed careers of scientists. There are so many scientists, lots of really great scientists, some of my favourite scientists have developed their careers around such questions. And they've developed amazing answers. So we'll start going back to 1979. And in 1979 there's this great researcher, Cort Pederson. And Cort Pederson he wanted to study maternal behaviour in rats. So what he did is he got virgin female rats. Virgin, got that, female, got that, and they're rats, right? So what happens with virgin female rats is that if they get baby rats, baby pups and it's not their rats in the animal kingdom it's every man for himself or a woman. They attack and kill. It's all [inaudible], sorry. If we administered oxytocin in to the brain of these virgin female rats they exhibited 30 minutes of lovely, beautiful, enticing maternal behaviour. If they then got something which blocked the oxytocin there was no maternal behaviour. This was fascinating. And Cort just went, wow, this is great. Is this the first evidence of the biological foundation to social behaviour? Well, Keith Kendrick in the 80s and 90s thought, well, rats, it's just rats, let's try some sheep. Let's see what happens in the sheep in terms of maternal bonding. And he showed the same results. And not only did he show the same results, but he was able to show the density of the oxytocin receptors in the brain. So the degree of receptors in the brain predicted how well the mothers looked after their lambs. It was really quite cool. Really very interesting. And Keith was also able to show that oxytocin didn't have an effect once the bond was formed. So it did have some effect but not a huge effect. It was in the formation of that initial bond with that cute lamby that was really important. I'm going to talk about one more animal model. It's the most famous animal model. It is the love boat of all animal models. Has anyone heard of a prairie vole? No prairie vole lovers here? One person, okay. Now, if you haven't heard of the prairie vole you need to know about them. Because Sue Carter in the 90s and 80s and Larry Young after her became very, very interested in prairie voles because they show [inaudible] monogamous bonding, right? They mate with a partner for life. Well, there's a few indiscretions here and there. We won't talk about those. But it's as close as we can get, right, that they basically mate for life. And what Larry and Sue were able to show was that if they gave oxytocin directly into the brain and then they asked them to cuddle up to their partner they developed a monogamous bond for life with that partner. If we blocked oxytocin the bond was not formed. And they've also got this what we call brother or sister vole. We'll call them the Leonardo DiCaprio of voles. They're the montane voles who are polygamists. I mean they just run around doing what they want all the time. Okay. So, anyway, we inject oxytocin into the brain of these Leonardo DiCaprios, right? And what happens? They demonstrate monogamous bonding for life. It was really fascinating, really cool stuff. So and then Larry has since published huge numbers of nature and science papers. If you want to get to the specifics about how the bonding works and the interaction in particular with dopamine, the reward chemical that makes it not only bonding but also a rewarding thing, it's a wonderful read, a really wonderful read. And I don't want to say that oxytocin is the hormone on its own. It's not just oxytocin. It's the interaction of dopamine. Maybe some lubrication with oestrogen. Maybe some reduction in corticosteroids, but there is no other hormone that has the effect on its own as oxytocin does. Now, that's all good news. But there are times and there are some inconsistencies in the literature. I don't want to say this is all fantastic. The importance of context matters. So I'm all for this one, if the animals are in an environment where they need to defend and protect their offspring oxytocin increases aggression, okay, increases defensive behaviours. So in different contexts it behaves differently, it results in different outcomes, and that's really, really important. And in recognition of this view, Jennifer Barnes, a great Canadian researcher, provided a wonderful review on the importance of the environment, what's happening in that moment to the response for oxytocin. And so she referred to the contextually specific effects of oxytocin. It's just that that doesn't happen everywhere. I can't give you all oxytocin. You'll all fall in love with me, it's not going to happen. We have to be at least having dinner, okay? So now we're showing you the critical importance of oxytocin, the causal effects of oxytocin in animals which is really nice, really interesting. But does that mean anything for humans? Well, we didn't really know. So in 2005 I was reading this stuff and thinking, well, you know, when I get people with social problems coming along we've got no great treatments that people get excited about. So I wonder if we could somehow use this to facilitate learning, to help bring the pieces of the puzzle together? And so back in 2005 that's what I did. And what I did is I went to a professional drug development pharmacy if you like, a clinical trial specialist and asked them to develop oxytocin nasal spray. Now, why a nasal spray? Anyone got any ideas? Well, firstly an injection is far less palatable, right? That's one thing. How many university students are going to let me inject them with a drug? Probably not as many, no. Hang on, we're in Sydney [inaudible], okay, maybe I could revise that. The second thing is that the nasal passageways, so when we breathe stuff in, it's the only part that has direct access to the brain through things like the olfactory bulb and the trigeminal nerve. So when you inject something it goes into the bloodstream and you're hoping it will eventually pass through the blood brain barrier, eventually through the blood and get into the brain. But it's a very indirect way. There's a possibility of going straight to the brain from the nasal passageway. I'll get to the specifics about that later. So what happened is I though, well, let's just give it to some university students. There had been one study over in Germany that showed it increased trust. Wow, that's pretty cool, increasing trust. But it's not close enough to the autism difficulties we see. So then we measured eye gaze, so the ability to look at the eyes or faces. And what we found was that oxytocin doubled the amount of time that students looked at the eyes and faces. And I thought, wow, that's really cool. There had never been any form of anything administered which has doubled the amount of time of looking at the eyes and faces. And we did lots of other experiments showing it improved the encoding and memory for social faces. And it improved detection specifically for words that have social themes. We're really excited. And lots of other studies emerged showing it improved emotion recognition. And there's now been hundreds of studies. And the effect is what is called moderate showing that oxytocin improves emotion recognition in students. So then the next question was, well, do we have something which might be useful for something like autism. We don't a lot of studies on lots of other populations, but we'll just talk about autism tonight. And in 2010 what we did is we got youth with autism, so young adults aged between 12 and 19, and we gave them a dose of oxytocin. And then we also -- or placebo. And then we asked them to complete emotion recognition tests. And what we found was that oxytocin to a moderate degree, it wasn't a huge jump, but it improved their ability to understand emotion in faces. Again, it was the first time that we'd shown any benefit of anything related to a drug to improve emotion recognition in humans and particularly in people with autism, sorry. And since then there's been a lot of studies showing that oxytocin seems to improve brain connectivity. It has impacts on the amygdala and [inaudible], the parts of the brain that are important for social function. And it improves eye gaze in single doses in adults with autism. So the next question was, well, how about it as a treatment? Is it a stand alone treatment? We can just give it to lots of people who have got social difficulties and get a great outcome. Well, of course, it never works out that simply. That's never going to be the answer, but we thought we'd try it anyway. So in 2012 we published a study, or maybe it was 2013, anyway we published a study looking at youth with autism. And we gave the oxytocin for 12 weeks. And the findings were really surprising. We got enormous change in the study. It was great. But it didn't matter if you got placebo or oxytocin, you showed lots of change, lots of movement. And we had this problem with lots of people were expecting benefit in the trial. And so they reported lots of benefit in the trial, and we didn't show any benefits in this particular study. At the same time there were other studies from Japan and Germany showing that there were brain changes that were important in treating autism but then predicted the benefits in social reciprocity. So we thought maybe actually this is something that's still working, it's worth further considering. The next step was to go, well, you know, the literature suggests that we need to really target that part where we're developing social skill. That's the most important part where oxytocin is important. So what we did in a study that's published in 2015 is we gave oxytocin for five weeks to young children with autism age three to eight. It turned out we recruited about 39 and 31 finished the study. And they took oxytocin for five weeks, and the same children took a placebo for five weeks, and we compared both the outcomes. And what we found was that oxytocin in this condition improved social responses. It improved the way that children were able to reciprocate, understand emotion and interact with their families, but only in 30 percent of children, only in 30 percent. And you might say, well, that's not great. But I tell you just about every medical is thought to work in psychiatric problems it's always about 30 percent, it's always about 30 percent. We thought that was pretty good, a clinically significant change. We don't know why. It was only in 31 kids, but it was the first evidence ever that a medical treatment could improve social responsiveness in child autism. It was really -- I mean for us it was -- I wouldn't say to parents go and start using it, but it's a first step in the right direction. But this is where it gets really exciting because I'm really pumped about what the world is doing today. The world has changed. We no longer think that autism and early development is set in stone, that the brain is hardwired. We no longer think there's nothing that we can do. There's lots we can do. We know there is lots we can do. I'm really proud to say that we've recruited and tested our 50th child with autism. In our next trial we're going to have 160 kids come through the trial. And we're doing one of the most sophisticated trials in terms of doing imaging and blood and eye gaze and social interaction assessments in all these children. We really thank the families. It's a really big protocol. But we're now starting to do research right where we can have at the end the answers about whether something works and why with all the right biological measures. Really excited by that. We'll have the results half way through next year, actually towards the end of next year. We're looking at different compounds. So we're starting to say, well, are there other things we can use than just the spray? And we're starting to look at other ones from the states and from other parts of the world. But most importantly, and where I think it's useful is we're starting to look at social development. So we're looking at social training and how that influences a child's development and whether there are benefits with oxytocin or not. And the point is I'm not trying to argue that we should just be drugging up children with oxytocin. That's not the point. We're starting to understand the neurobiology of social development, and oxytocin is a vehicle for doing that. But it might be that we can really engage the social systems of the neurobiological systems with greater interventions that lead to the right [inaudible] body release of the chemicals that are needed, and that's where we want to go. We're also looking at where oxytocin is going in the brain. So we've got this great collaboration with American universities, UQ and the Australian Nuclear Science Technology. But we're putting a stamp on the spray. We're spraying it up the nose, and then we're following that [inaudible] where it goes exactly in the brain. We want to know which part of the brain it directly hits. And that study hopefully we'll have some results in a few years. It's a really difficult study, but it will be groundbreaking if we get there. And finally we're a bit sick of families going to hospitals, going to hospitals, waiting for six months, doing assessments and then being referred back out to a variety of paediatricians in the community and health workers in the community and there being no linking between that process. So the most important thing we're doing now due to the energy and hope of what we might be able to achieve is we're getting all of the Sydney children's hospitals and the networks to work together so that every family that goes to the hospital the clinical information is put into a research database. That based on that database we can then start to personalise the intervention. We can say, well, based on this profile we're suggesting this treatment. So we'll have over 1,000 kids coming through each year into the hospital system. And we'll be able to provide suggestions about which treatment. The most important part is we want to develop the evidence so after a single year we can say 300 kids have done this treatment, and there was evidence for it or there wasn't. And the point of that is because it's been far too long. Autism is a diagnosis since 1918. It's been far too long that families go to the GP and ask about the evidence and there's nothing. We want the GP to be able [inaudible] basically these studies have been done and it's useful for this type and this problem. So while I might not have given you the chance and the clues to fall in love yourself and to convince that special person to fall in love with you, I hope I have given you some serious learning. I want to thank most importantly the families. I mean we have such desperate, beautiful families in our research. They give up their time. I can't tell you how much time they give up in our current trial. And we wouldn't have any research without them. And I wish that what we did helped everyone but it doesn't, and I thank them for their tie. And also my team, my young team -- sorry, I shouldn't say that, all of my wonderful team. And I hope the future science and the opportunities that exist in terms of really making a difference for young people excite me as much as it excites me. Thank you.

[ Applause ]

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