Interview with Barry J. Marshall. Winner of the Nobel Prize in Medicine for the Discovery of *Helicobacter pylori*

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Fabián Emura (FE): It is September 9th, 2016, and we are in Bogota D.C. It is a great honor to interview one of the most famous and recognized physicians in the world, the 2005 Nobel Laureate for the discovery of *Helicobacter pylori*, from Perth, Australia, Dr. Barry J. Marshall. Hello Barry, good morning.

Barry Marshall (BM): Hello Fabian, how are you doing? I'll introduce myself. I'm Barry Marshall and I won the Nobel Prize in 2005 for the discovery of the *Helicobacter pylori*, and at the moment I am the clinical professor of microbiology at the University of Western Australia in Perth, and I'm also a gastroenterologist, so that is why we are here today.

FE: That is great. I am very honored to be conducting this interview. I know that this is the first time you are visiting Colombia for the Pan-American Congress of gastroenterology, and I am thrilled to be here with you and with my very good friend and "adopted father", Dr. David Peura, who is right behind the camera.

BM: This is great. So far I'm enjoying it here in Bogota.

1. **FE:** In 1981, you were a fellow in gastroenterology at the Royal Perth Hospital in Australia, and later, you turned out to be the recipient of the Nobel Prize. What made you so different?

BM: So, one useful thing about being new in any kind of field is that you don't know all the teaching, you don't know the dogma, and so you are more receptive to new ideas. And… so as a young internal medicine fellow, I was just starting training in gastroenterology for 6 months, when my friend Dr. Warren showed me this bacteria. He said, "What do you think of those?" and I said, "Well, it looks like they are living in the stomach, let's do some research on this to find a new bacteria." We had no intention to

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discover the cause of ulcers, but after we had studied the bacteria for a few months, we were there thinking, do we really know the cause of ulcers? Everybody says we already know it. They are always caused by acid or stress or genetic factors. But I think we had an open mind on it because we had not been indoctrinated as much in the gastroenterology teaching. And that was the advantage. So it is good to be a little bit isolated, you can build your own hypothesis, test a few ideas, and also be more receptive to new discoveries and new technology.

FE: At that time, same year, your fourth child was born. You were facing a lot of difficulties at home, getting an extra loan for home renovations, and you were very busy at work. How did you manage all these situations at home and at the same time try to discover and cultivate the bacteria?

BM: Well, at this point, I should say my wife is a very strong person and very smart. And so she had me well organized, and of course, anybody with four children knows that is a full time job. Not many people have four children these days. So it meant that the normal medical work that I was doing was maybe from 8 am to 5 pm, but then I would have 2 hours before that, so I would probably be going to the hospital at 6:30 in the morning and see the patients before endoscopy, and at the end of the day, I would see some other patients at 6 pm. So I would be coming home quite late, 6:30 - 7 pm.

FE: That was very tough, wasn't it?

BM: It was also tough on my wife who didn't have as much help with the children, and managed most things herself. So I have to give her credit for letting me do my work and stay at the hospital and lab long hours. She has always been quite interested in my research, and actually helped me write some of my very early papers.

FE: Yes, I know, I read that.

BM: So in some ways it was very selfish, and I suppose most people who succeed in research have to be selfish to a certain extent. So I hope I can make it up to her because I owe her a lot.

2. FE: I would like to know your opinion about your first letter, the first report that you submitted with Dr. Warren to Lancet back in 1983. Tell me about that.

BM: Dr. Warren and I started out working in 1981, and we did the 100 patient study in 1982, and of course I made the discovery that year. We cultured the bacteria and noticed the association with peptic ulcer. So it took us a few months to write these letters. Then we were having an argument about it because Dr. Warren, said he should be first author and he thought I should be second author. I said, well, I did the microbiology and the endoscopy gastroenterology part of it. And so we had these arguments over the discovery because he had obviously seen the bacteria two years before that and in the end, we decided that he would write one letter as a single author and would write a second single author letter. As a result, the first letter in the Lancet was single authored by Robin Warren himself, describing the histology and the discovery of the bacteria. The second letter, which was immediately afterwards, was my observation that the bacteria could be cultured by microbiology and I was the one who noticed that they were associated with gastric lesions . Also, I was the one who did the computer work and the analysis and first noticed the association with peptic ulcer. After I made these observations I showed them to Dr. Warren, and we were both still a bit skeptical. We reached a compromise, had some more arguments, got some more data until we both believed what we saw. Then we submitted our papers and letters to the Lancet. And of course, everybody says letters to the Lancet!- you don't really have to believe them, they are just preliminary. But of course we had already been working on this bacteria for two years by then, and had lots of other data, even some treatment data. So when we published what looked like an hypothesis in those letters, have most of it had already been proven, but not published (Figure 1).

3. FE: Just now I want to jump into my next question, because 1 year later, when all this literature appeared, there was not even 1 person who believed in this discovery. People really didn't think this was a real fact, and I believe you felt very frustrated and disappointed. So how did you remedy this situation?

BM: Well, I was a very immature researcher, and of course I expected everybody to believe me once I produced the data. But when you do publish your first data, your first letter if you like, with no track record of respected research, well then I think everyone is going to be skeptical. And the teaching that was in all the medical books said "bacteria cannot live in the stomach." The stomach is full of acid pH less than 2. We can show you that kills bacteria. There's no question. So how could bacteria possibly live in the stomach? It must be wrong. So I had a lot of skepticism from my colleagues, most gastroenterologists. They were skeptical because they had never seen it. And they had never actually looked for bacteria in the stomach. They knew there couldn't possibly be there, so why check, why look? So it was really ignorance and the illusion of knowledge. There is a quote that says "the illusion of knowledge is what

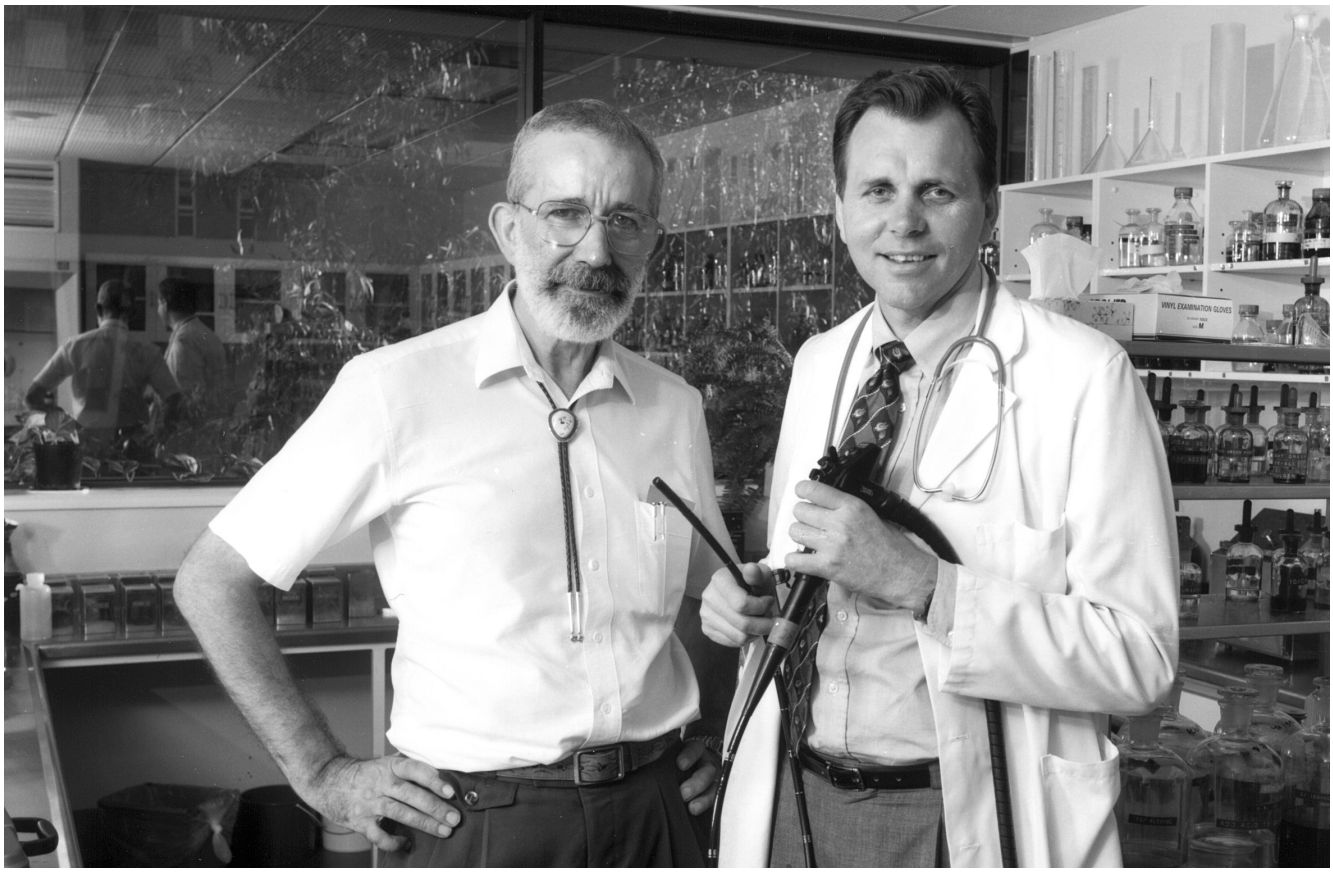

Figure 1. Doctors Robin Warren and Barry Marshall in the lab at Perth University, Australia. Unpublished photo.

holds up medical progress". And the prevailing knowledge was that bacteria could not live in the stomach and ulcers were caused by stress. So if you had that knowledge then you could not possibly believe the new observations. Thus it took a few years after that before we had converts. And of course, when people challenge me when I presented this paper in Australia on two occasions, it was challenged from the audience. I was extremely hostile. And so people ask, "Dr. Marshall, did you hold a grudge?" Well, probably I did for quite a few years.

FE: They thought you were a crazy researcher?

BM: I could have been. Is a difficult thing to remain objective about your own research. So one of the most important things in your training is the scientific process, be skeptical of your own data, and then try to be objective and repeat and perform other experiments, which would be some other way of checking your data, so that you could be confident. And that means it is going to be slower process to publish it, but you can be more solid and confident when you finally do publish it.

4. FE: Lets look back again to that special day -that long weekend in Australia, which by coincidence was Easter weekend, and nobody checked the culture, and of course nobody on Sunday, nobody even on Monday, and you came back to the lab on Tuesday after several days and the bacteria were there. Just tell me your first impression on Tuesday morning.

BM: My microbiology colleague showed me the cultured plates and said, "Barry, I think we have grown this bacteria and it is something new." I said show me the Gram stains. So I looked at it under the microscope, and I said, well, you might be right, but this bacteria doesn't look exactly like this bacteria which I see in the biopsy samples. As you know, the *Helicobacter* is like a *Campylobacter*, is like an "S" shaped. That is it's classical appearance. But when you culture it in a petri dish, it is not the same as the stomach. In culture it is slightly abnormal being much longer and much straighter, and so it look more like an E. Coli. So I said well, it could be anything. Lets wait and see some more cultures. But of course I was a little bit angry because I found out that Dr. Warren and I had been using the correct method for 6 months and sen-

ding all these biopsies to the lab to be cultured, and in the lab they had been looking at them after 2 days, they didn't see anything interesting, and they just threw them away. And so, we had probably cultured quite a few helicobacter already, but the colonies were slow growing and they couldn't see them, so they just threw them away. So at least we discovered the trick, and the trick was a special atmosphere, microaerophilic, they don't like too much oxygen. And the second part was really, they take 5 days, so you have to be a bit more patient with the *helicobacter*.

FE: Then, the resurrection day holiday really helped?

BM: Yes. Sometimes if you keep trying, some kind of accident, a little lucky accident will happen in the lab and you will succeed. And then you are going to figure out how it suddenly works. Then you can explore all the different variables and you might find something different on that particular day. So with us it was quite a simple thing. Just 5 days culture time.

5. FE: Now I would like to talk about what is probably the fact that made you the most important person in this field, which is the day that you decide to swallow the bacteria, because nobody was believing in your research. What day was that, where was it, and did your wife Adrienne know what you were trying to do?

BM: In the subsequent year, I decided to do a thesis an MD thesis, which is like a PhD. I submitted this plan to develop animal models. Of course to prove that the bacteria is a pathogen you have to create the disease in an animal, so I decided that I was going to feed it to piglets. So for nearly a year, I was doing this experiment where once a week I would make the pigs drink the *Helicobacte*r and then I would pass the endoscope to take the biopsies, but we could not infect the pigs. And of course, they started off as little piglets, but after 3 months or 4 months, they were extremely large pigs, and so this became a very difficult and expensive research project, and it failed. And so I always had this criticism, "Dr. Marshall, what's the issue? Is the *Helicobacte*r the chicken or the egg? Does the *Helicobacter* cause the ulcer or do the people with the ulcer just catch the *Helicobacter* because is so common?" And so it was necessary then to do a human experiment, and I said to Dr. Warren, "I think you should do it. I'll give you some *Helicobacter."* And he said, "oh no no!" He already had *Helicobacter* once. And I had given him some treatment by then, so he said, "you should do it." And so I said, ok. So I drank the *Helicobacter*.

FE: Were you alone or were you with someone?

BM: So the story was I was in a different hospital in a different lab, and I had a technician called Neil. Well, 2 months before I had my own endoscopy and had biopsies taken to make sure that my stomach was clear of *Helicobacter,* and that it was 100 percent normal. And then we cultured that bacteria and we had it growing in the lab, and I said to Neil, "right now, I should take the bacteria and see if I get a peptic ulcer like the patient. So he cultured the bacteria over the weekend, and on a Monday or the Tuesday… Monday we were too busy, so maybe Tuesday morning we did it. He took 2 cultured plates of the *H. pylori* and mixed it up with a beef broth, just like beef soup, clear soup. Is a very good dark menu for culturing bacteria. Just put it in there and mix it up. I said, well, because I might have too much acid in my stomach, I'm not sure, I'll take some Tagamet. So I took 400 milligrams of Tagamet early that morning, so that I wouldn't have quite so much acid. We put the *Helicobacter* in this beef broth, about 20 milligrams of broth, and then I had to drink it down like a tequila shot.

FE: Why like that way?

BM: Quickly like a shot! I didn't know what it was going to taste like, but it just tasted like soup. And we now know the *Helicobacter* doesn't have any flavour, so it doesn't taste bad. I used to think it did, but it was just my imagination. So I drank that, and then nothing happened.

FE: And, nothing happened?

BM: Yeah. But of course I was a bit embarrassed because is very illegal to consume food in a microbiology laboratory. People should not be eating or drinking in the laboratory, so I felt a bit uncomfortable about this. And then, I just waited and see what would happen. At first, nothing much happened, I made it to 2 or 3 days, and after that 3 days, I started feeling a little bit of dyspepsia when I had the evening meal. Eating the meal, when I'm half way finished the meal I suddenly feel quite full already. And then I had sips of water to help the meal go down. I said, I'm probably imagining it… it is probably nothing. And so then 2 days later on the weekend I was visiting my mother and she said to me, "Barry, you have got very bad breath today." And I say, oh my mother, she always finds something wrong you know. And my wife said, I noticed it also. So then, 2 more days and then I started waking up early every morning, I woke up vomiting. And before I got out of bed, before dawn, suddenly at 6 am I get up and start vomiting in the toilet, and it was very strange because there was no acid in the vomit. It was just like water coming out. Clear water. So when you think about it, I had digested my meal, and

then my stomach had been secreting some kind of liquid all night without any acid in it. And so that happened for 2 more days, and on day 8 I had another endoscopy.

FE: And any biopsy?

BM: Yeah, and biopsies taken, and the biopsy showed extremely heavy colonization of *Helicobacter* and damage to the epithelial cells. They looked more primitive, they didn't have any healthy mucous inside the cell. The mucous had all disappeared from the epithelium. And the cells were just like primitive cells really, and lots of neutrophils polymorphs. So I had developed acute gastritis, active gastritis. And I didn't have any acid, so I didn't have any acid symptoms. Just a little bit of nausea, appetite not so good, and I was not sleeping very well. I just felt a bit sweaty or something. And so that was the acute illness.

6. FE: I'm impressed. Barry, you could be the only person in this world capable of doing that, and for what you did, millions of people in the world will benefit and have benefited from that. So, on behalf of humankind, I want to say thanks.

BM: Well, I appreciate it. However, I was very excited when I saw the histology and showed that I had been infected. So that means the chicken or egg still requires some discussion, but at least I can show a healthy person can be infected and gastritis develops. We know people with ulcers have gastritis and people with cancer have gastritis. So it was a logical hypothesis if you like that could happen. But of course I was quite excited and I had not told my wife about this, so this caused a problem. I came home that night and my poor wife, you see, she had four children, so not only that, but she had also been on a car accident and she had a broken rib and a whiplash, and she had a neck collar. She had been working very hard, I was not very well, I was not helping very much that week, and so I said, "Oh, great news!" And she said, "what is the great news?" and I said, "I drank the bacteria and I got the infection." And she said, "Oh my Goodness! What have you done?" She was very worried about me.

FE: Yes, you didn't know about the consequences, I mean the real consequences…

BM: So she was worried I would develop an ulcer. She was worried that I would give it to her, that I would give it to the children. It was a difficult time for her. And she said, "you have to take antibiotics. I can't have people with *Helicobacter* in the family even if some people think is not very bad." I think by then she believed that it was a pathogen.

7. FE: I know that you got cured from Hp before you had your antibiotic treatment, probably by an immune response, what we call a temporary infection of Hp. Why don't we jump into that temporary infection in patients?

BM: Yes, so a lot of people think, "oh yeah, Barry Marshall drank the bacteria and developed an ulcer," but that is not quite true. So I developed gastritis, and maybe 5 days later I had another endoscopy, and the idea was that I was going be taken a lot of biopsy samples, and after that I was going to take some antibiotics, because I expected that I would still be infected. But when we tested the biopsies on day 14, the bacteria by then had disappeared. Of course it takes a few days to see the biopsies, so in the mean time I already started treatment with antibiotics. But in retrospect, probably I have had a spontaneous cure. And this is the kind of immunity called innate immunity. Every animal has this with an acute infection, otherwise you would be dead. You must have an instant way of reacting against bacteria. So my histology showed very heavy polymorph infiltration. And you can see that if the acids secretion disappears, that is also a side effect of the inflammation, so no acid, lots of polymorphs, it is possible for the innate immunity to fight the bacteria and presumably, kill the bacteria with lysozymes, superoxides, all those natural things that you have when you fight bacteria. So by day 14, the bacteria were gone and when they tested me, there were no antibodies. I never had IgG, IgM, nothing like that. This is an interesting lesson. It means that not everybody gets infected with *Helicobacter* permanently on the first occasion.

So we can talk about Colombia a little bit. In Colombia, 70% of people have *Helicobacter*. That means they must have had several exposures to get that because on the first one, probably not more than 50% develop a permanent infection. The second exposure more people, will become infected. Maybe after 2 or 3 exposures you could get the population of up to 75% infected. At the moment this is an area for research. People don't understand really what is the necessary dose for infection or the relationship between the host and the pathogen. And countries like Colombia could be the place we could really do some interesting research on the epidemiology of the acute *H. Pylori* infection.

8. FE: I bet young researchers in Colombia and Latin America, after watching this interview, will be very interested in jumping into that arena of researching *Helicobacter.* I would like to talk about Latin American countries and Colombia. As you know, we have a lot of Hp infection and a lot of gastric cancer. But the question is, if somebody is infected should he or she get treatment if that person is asymptomatic?

BM: So that's a good question. Of course the question before that is, should we test people for *H. Pylor*i when they are asymptomatic? So this is where the controversy is. Because when you test the person, you are obliged to tell the person the result. And in Australia we have the same question, and the patient would say, "I've tested positive for *H. Pylori*, Dr. Marshall, what does that mean? Could I develop an ulcer?" Oh yes! Maybe you could, 10%. "Could I develop stomach cancer?" Yeah, possibly, maybe you could, maybe 2% or 3%, something like that. But usually after the age of 50. "Could I spread the infection to my children?" I say yes. Same way you catch it from your mother. "Could I spread it to my partner?" Yes! Probably, you can. So when you answer these questions for the patient, they will say please treat me. So the decision to treat is always, yes. Take the treatment. And so this becomes a little bit of a problem for the health authorities, because, ok, if we say that *Helicobacter* is a pathogen, we should treat it, but should we screen the whole population and treat 75% of people, 40 million people with antibiotics. So this becomes a big decision. It could be expensive, and the problem is that many people who feel fine with no symptoms are not very compliant to the antibiotics.

FE: And this will produce some treatment failures and antibiotic resistance?

BM: Yeah, so they won't be compliant, they won't take the treatment if they have any side effects, and they will create resistant bacteria. And not only to *H. Pylori*, but respiratory bacteria, urinary tract infections, and things like that. And so it creates too much difficulty. So in most countries now we say, ok, if we find *H. Pylori*, we treat it, no problem. But we are not going to look for it in every single person. Not just yet anyway. In a few more years, we might have a very easy treatment, and it might only cost just 5 dollars 1 tablet or something like that. So that is what we hope for, and then it would be much easier to treat everybody. But at the moment, we are going to test and treat patients with dyspepsia, with peptic ulcer, with a past history of peptic ulcer, with a family history of stomach cancer. Ok, so those ones no problem. It may be, when people have a health checkup at age 50, and in Colombia cancer is quite common, so maybe have a health check up at age 40, look for *H. Pylori*, if they have it, take a treatment. There would be plenty of work for gastroenterologists in Colombia for the next 10 years or 20 years.

FE: There is going to be a lot of work.

BM: Yeah, that is right. And so in many other countries as well. So not only Colombia in South America, Brazil is the same, and then if you go to Africa it is the same, perhaps more. But also Asia, China. Almost half the stomach cancer in the whole world is in China. And now they are getting this idea of probably treating the whole population over the next 20 or 30 years, which is a lot of people.

9. FE: Talking about Africa, you mentioned Africa before. As far as we know, in Africa and Latin America, Hp infection is very high. The prevalence is very high. But we have a lot of gastric cancer in Latin America, but no gastric cancer in Africa. At least not very much. How can we explain this phenomenon? What are the differences among people and among Hp infected individuals?

BM: So I think the most important difference is going to be cultural differences. Especially diet. So I don't understand what everybody in different countries eat. But the Western US diet, you know, lots of calories, lots of meat, high protein, that is the kind of diet which predisposes more towards duodenal ulcer and the acid levels are quite high. And that would give you ulcer, but it gives you some protection against stomach cancer. In developing countries, Latin America, used to be Japan, Korea, China maybe the diet is different in some way. So that people end up with low acid levels, and so those people over after many years will be susceptible to stomach cancer, rather than ulcer. So when we talk about Africa, I can say, also Africa, maybe the *Helicobacter* does not have so much toxic. So *Helicobacter*, makes a toxin called CagA toxin. The CagA toxin in Japan and Korea is the most dangerous. In China is a little bit less. As you travel west to Europe it is only about 50% as bad, if you go to Africa is less again. And so maybe the *Helicobacter* in Africa is not quite so bad, and the diet is more like a high acid level diet. So in Colombia, I believe that most of the strains of *Helicobacter* have the CagA toxin.

FE: Oh yes. 100% actually.

BM: Ok, so that's a bad place to start. That means is going to create more trouble, more inflammation. But again, obviously there is the host and the pathogen, and then on top of that the diet. Those 3 things can interact in different ways. But at the moment is a bit hard to be certain or dogmatic about that.

10. FE: Not only about Colombia, but thinking as a whole continent, I mean particularly Latin America. What do you think are the biggest challenges that we are facing in Latin America in relation to Hp?

BM: The biggest challenges are to educate the population and let them understand about hygiene, such as hand was-

hing, keep the toilets clean, make sure that the drinking water is clean. These days it is not so difficult, because we do have the internet. People are starting to get connected and they will understand why things happen. So 50 years ago, everything was like religion, witchcraft, and different theories, what your mother told you, everybody had different ideas about all kinds of diseases. But now, what we like to see is evidence based. But whenever your doctor or someone tells you the cause of some illness, your children will immediately say what is the evidence? They will pull out the iPad and check in the internet, look on wikipedia, and say that's not true, it is something else. So I think we are like that with *Helicobacter*. So you cannot be born with *Helicobacter*. When you are born you are completely sterile. But after about 1 year when your mother's antibodies go down, you know, your maternal antibodies to give you some protection. But if your mother has *Helicobacter*, she is kissing you, she is feeding you, so pretty soon you will catch the same *Helicobacter* that your mother has, so we know that. If you are the eldest, and you have the *Helicobacter* from your mother, well then your little brothers and sisters, they can catch it from your mother but also from the other children, the elder children. So younger children, the second born, the third born, they are more likely to get the *Helicobacter* even more so. So I think there are logical reasons for it. So what can we do in South America, well, we just want people to understand the good news, that ulcer is caused by *Helicobacter*, that you treat it with antibiotics, the reinfection is not very high, so usually you don't catch it back. And in an area where so many people have *Helicobacter*, ok, lets look at the hygiene, look at the drinking water, chlorinate the water or give people clean drinking water in a proper tank, and I see that in many countries, they get some clean drinking water delivery. In China for many years they always boil the water, so young people in China don't have *Helicobacter* so much now. So those things are going to make a big difference. So the knowledge is the most important thing to eradicate any infectious disease.

11. FE: I have 2 more questions, one of those two is about the vaccine. Tell us your opinion about this probable new vaccine, because we have been fighting against Hp for many years and there is no vaccine yet. What do you think about that?

BM: Well, there has been a vaccine developed in China and they could show that when they gave it to small children, school children, they cut down the *H. Pylori* infection by about 50%. So there is hope. That's not perfect, is not a perfect vaccine and is not a very good commercial prospect for vaccine company, because you just take it once, and then you only need 1 dose. And it could be quite expensive for developing countries. So most people wouldn't want to have it. However, if you gave it to all the small children, then the infection rate in children would be much lower. And you know, you could see after 20 years the *H. Pylori* would be disappearing like hepatitis vaccine or something. So there are possibilities there. The other use of the vaccine would be if I came to work as an Australian, probably never got exposed to *Helicobacter*, like ever. So if I bring my family to Colombia, I say, "you kids watch out. You could catch *H. Pylori*." So maybe I would buy the vaccine and give it to the children to give them some protection. Otherwise the *H. Pylori* from Colombia would come into my family, so maybe it could cause some problem. So that is how we would do it. So is not a perfect vaccine, and there is still going to be some research over the next 10 years or 20 years. Is quite difficult to make it, but there is some hope.

12. FE: I see, and the last question is about treatment. Many people just abandon the treatment because is quite tough. The triple therapy. Do you have any news about possible new therapy, or what about the role of probiotics and something that is sensitivity based personalized treatment for the Hp?

BM: Ok. So that is a good question. The question of antibiotic resistance in *H. Pylori*, and the difficulty in some patients. How do you treat it? Recently I have been visiting in China where they have the same problem, and what we have done is start up a few centers called "Marshall Centers" and we give personalized treatment. And personalized or precision medicine is when you come to me and you say I had three treatments and I still have *H. Pylori*, and I need to get rid of it. Well I can say, let's do some testing on you so that when we give you the next antibiotic treatment it is exactly the one you need. It is not just an average for everybody. So this is the precision medicine. This is where we are going to go in the future. At the moment people say it is quite difficult to culture *H. Pylori* to get this information, because it needs a culture. In a few years, 2 or 3 years probably, we will be more able to maybe just do the DNA on a biopsy and then after 24 hours or so we will say you should use this antibiotics, you don't have to culture the bacteria. Just put it in some kind of machine. So this is going to be the future for *H. Pylori*. But good news for you, there is still going to be lots of gastroenterology work to get those biopsies. And will be learning how to do this kind of precision medicine. So is important to have sense of excellence for *Helicobacter*, because what we found in China, a lot of doctors say, well, I don't want to treat *Helicobacter*, because if the treatment fails, I have got anything else to offer the

patients, it is too difficult. And the patients will be worried about it. So I don't even like to do follow up. But if I say, ok, you can go to the Emura Foundation, or a specialist gastroenterologist center, in China, a Marshall Center, and the difficult cases can all go there and 99% cure. So don't worry, take this treatment after I do follow up, if is cured good, if is not cured, don't worry, we are going to send you to the next level, and those guys will cure you almost certainly. So everybody feels, you know, relaxed. All of a sudden is easier. And then, just with that type of process after another 10 years you can see that the *Helicobacter* will be declining, and there will be a lot of *Helicobacter* experts.

FE: I'm impressed. Thank you very much Barry. This interview will be uploaded to our webpage at the Colombian Society of Gastroenterology, and it will be edited as a paper in our journal. It will be of tremendous benefit for, not only Colombian gastroenterologists, but for all Latin American gastroenterologists and endoscopists. Thank you so much, and welcome to Colombia again, and I hope you enjoy Cartagena a lot and the Pan-American Congress of Gastroenterology and Endoscopy (Figures 2, 3 and QR code).

BM: I'm looking forward to it, thanks Fabian.

FE: Thank you so much.

Acknowledgments

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Figure 2. Doctors Barry Marshall and Fabián Emura by the end of this interview in Bogota. September 9, 2016.

Figure 3. Doctors Barry Marshall, Fabián Emura and David Peura during a research meeting related to the future of *Helicobacter*. Bogotá, September 9, 2016.