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From Africa to a Career in the Life Sciences

Hello. I'm going to actually share my journey out of Africa and how I got into a career in life sciences.



Fig. 1

I was born in a small town beside Lake Victoria. It's about 18 miles south of the equator. This is a picture of me with my father outside what we call our tin house (Fig. 1, right). This is a one-bedroom house in which I was born. The thing about growing up in this town called Kisumu was it's beside the second-largest lake, it's Lake Victoria (Fig. 1, left), and there are a number of interesting living creatures in this lake.



Fig. 2

There are hippos, and the thing I found exciting was these fish, called cichlids (Fig. 2, right).

They are very fast-evolving, and there are about 500 species of this fish in this lake. This is now one of the key models for understanding how evolution works, and there are some people in Cambridge who are studying them.

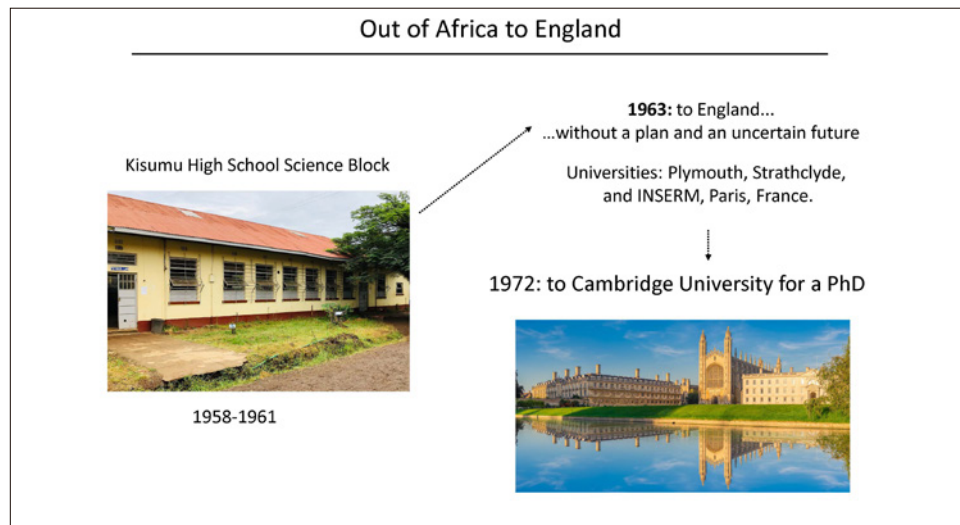


Fig. 3

I went to Kisumu High School (Fig. 3, left), and that's where I was lucky to have a biology teacher who inspired me to take an interest in living things. That was an important part of my growing up in this small town, but I didn't meet anybody who had been to a university. My parents hadn't been to university, so I didn't have any role models. I didn't understand how you could get into a career in life sciences.

At that time, in 1963, Kenya was approaching independence, so there were quite a lot of uncertainties about what you do. Politically, the situation was difficult, so my parents sent me to England on a one-way ticket, and my father told me to get some education because that's how I might survive.

So I went there without a plan and an uncertain future. I went to two universities, acquired an education in biochemistry and natural sciences. The situation was getting difficult in the UK because there were a lot of immigrants coming from former colonies that were becoming independent.

I decided to leave the UK. I went to Paris for a couple of years. While I was there, I met somebody who became very important to me in my career, and so I ended up in Cambridge in 1972, to do a PhD (Fig. 3, right).

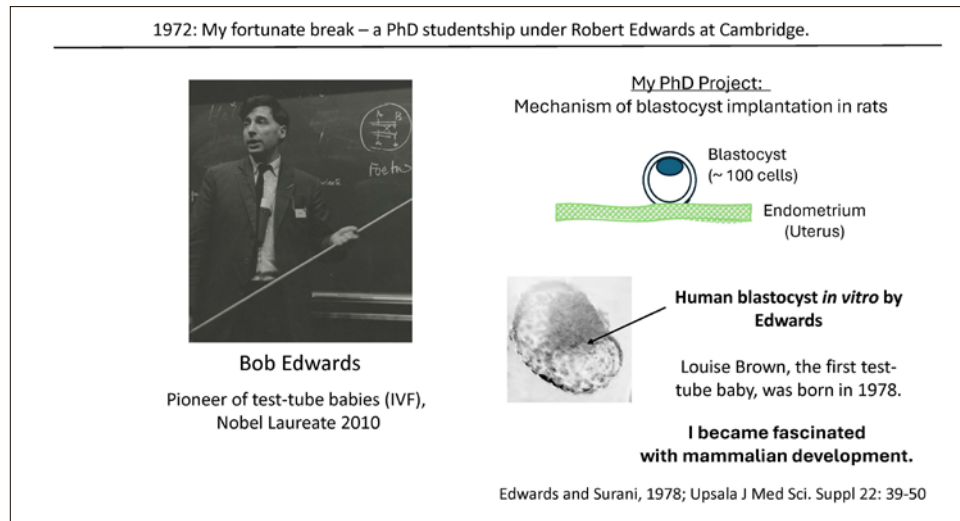


Fig. 4

Left image: Churchill Archives Centre, the Papers of Sir Robert Edwards, EDWS 18/4/4
Bottom-right image: Edwards and Surani, 1978; Upsala J Med Sci. Suppl 22: 39-50 Licensed under CC BY 4.0.

The person I met was Bob Edwards (Fig. 4, left). He's the pioneer of IVF, in vitro fertilization, IVF babies, so at that time, when I went to Cambridge, he was working on IVF.

My project there was to study the mechanism of how the embryo implants—blastocyst implantation (Fig. 4, right). This was a big problem for IVF at the time. So I was working on this problem in rats to understand the mechanism of how this embryo can implant.

Actually, I didn't get so excited about IVF, but at that time, there was pioneering research that was ongoing to do with how mammals develop. This was a new field at that time. I became fascinated with mammalian development. In fact, Louise Brown, the first test-tube baby was born in 1978 anyway, so I thought the problem was, in principle, solved. I wanted to do something different.

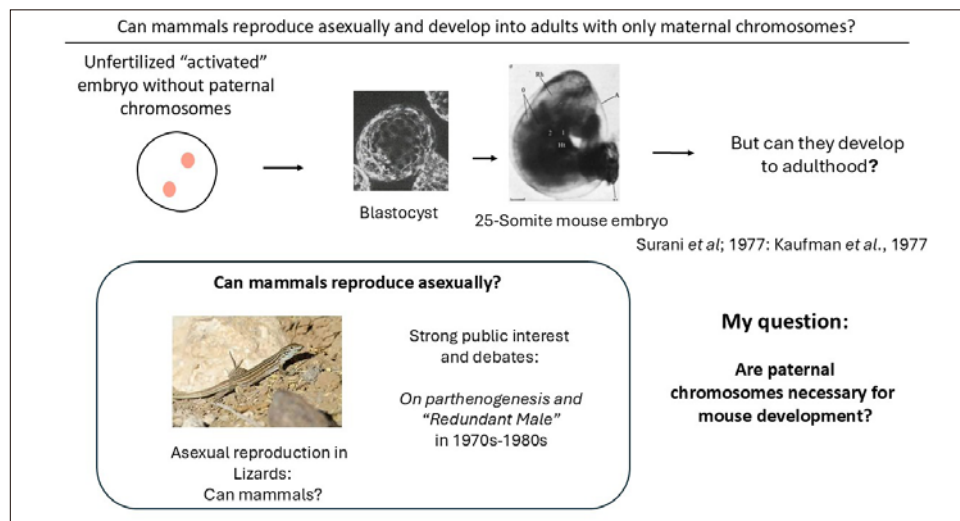


Fig. 5

Bottom-left image: Bill Gorum / Alamy

So I started working with my friend Matt Kaufman, and what he got me excited about was working on these embryos (Fig. 5). These parthenogenetic embryos are unfertilized, and so we kind of became interested in thinking about whether mammals can develop asexually, without fertilization of an egg because the idea was that there are many vertebrates, like lizards and frogs, that can develop without fertilization parthenogenetically, asexually.

At that time, there was also a strong debate about topics like the redundant male. There was a

strong feminist movement at that time in Cambridge. So there was this idea that the males were redundant, you know, you could develop human beings without fertilizing eggs. This was the background to all of this.

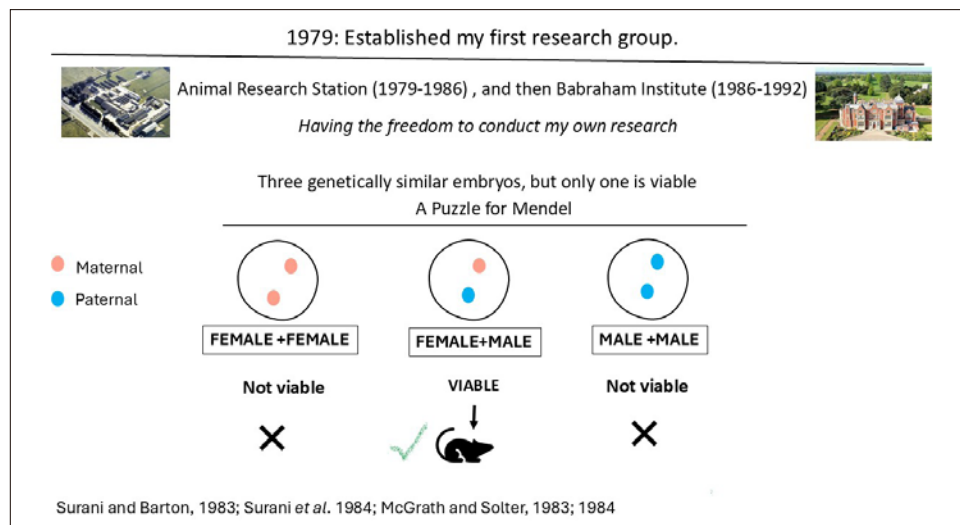


Fig. 6 Left image: *The work of the Animal Research Station, Cambridge* (38 (2)) by Chris Polge. Copyright 2007 by Elsevier Ltd. Reproduced with permission of Elsevier Ltd. in the format of post on a website via Copyright Clearance Center. Right image: © The Babraham Institute Bottom-center icon: Vecteezy.com

I formulated this question in my head: Could mammalian embryos develop without fertilization and whether a paternal genome was needed. It's a very esoteric idea but this was something I got absolutely obsessed with. Fortunately, I got my first lab which started in 1979, just outside Cambridge (Fig. 6, top), and I got this position, which was also extremely fortunate because when I got to this institute and started my first group, I could do whatever I liked. I was not restricted to what subject I could follow.

I went to the Animal Research Station with this question in mind about trying to explore this question of asexual reproduction in mammals. To cut a long story short, after a lot of experiments, most of them failed, we came to the conclusion that you cannot reproduce mammals asexually, but what we became aware of was that if you make these embryos which are genetically similar, with two maternal genomes or two paternal genomes, they will not develop; they stop developing at a certain time. So you needed a maternal and a paternal genome for normal development (Fig. 6, bottom).

According to Mendelian laws of genetics, all three of them should develop because, according to Mendel, the genomes are equivalent if they are genetically similar. However, we only found that the embryos that could develop would have a male and a female genome, which was the precursor to the discovery of genomic imprinting.

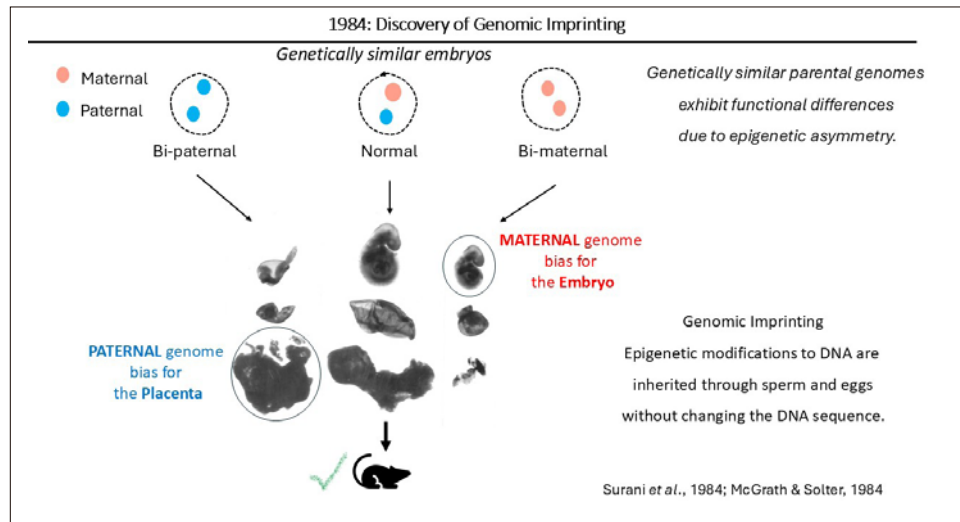


Fig. 7

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What became clear in around 1984 was that the parental genomes in mammals, although they are genetically similar, are functionally different; they function differently (Fig. 7). What we found was that the genome that comes from the father is much more important for the development of the placenta, and the genome which comes from the mother is much more important for the development of the embryo. So that's why you need both of them.

This was a really quite amazing kind of idea that this experiment taught us. We had no explanation for this because we couldn't understand why this should be, because this was going against the laws of genetics established by Mendel in the 19th century. Although we were excited to see this kind of result, and we thought this was something quite interesting and something new, we had no explanation for it, and when I first started giving talks, I had a hard time because people just didn't believe it.

So we needed to actually look and find an explanation for this observation—and are still trying to find the full explanation for this observation. There are still many things that are not known.

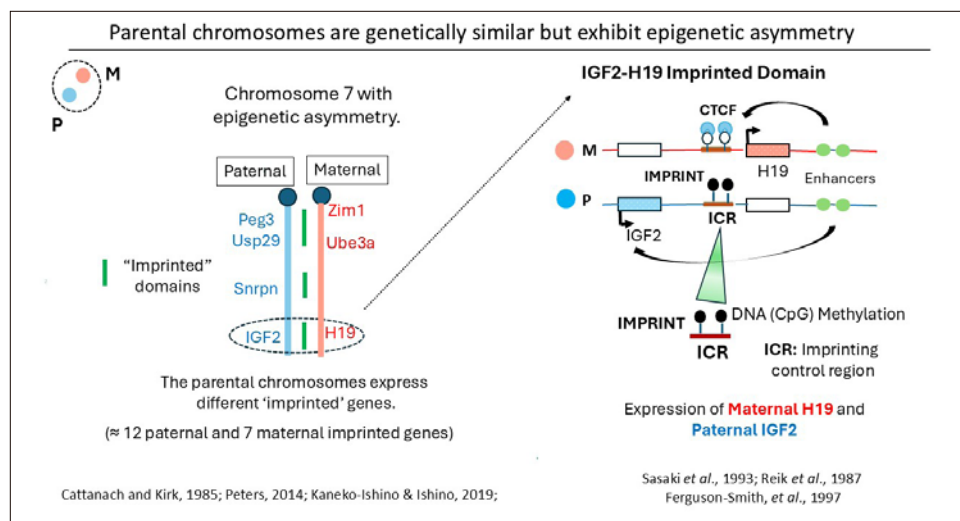


Fig. 8

Let me try and guide you through some critical issues. This is a slightly complicated slide (Fig. 8), but what we found was that, although the parental genomes are genetically similar, they are epigenetically not similar. They are asymmetric. So the maternal and the paternal genomes

have epigenetic asymmetry, and that's why you need both of them.

If you look at this chromosome 7, taking this as one example, there are certain genes on chromosome 7 which only show expression of genes that come from the father. Here are the examples. For this gene IGF2, only the copy that comes from the father is expressed and the maternal copy is silent. If you look at the maternal chromosome 7, there are different genes that are expressed from this chromosome (Fig. 8, left).

This is where you get the asymmetry. You have some genes that are only expressed when they come from the mother and others that are expressed only when they come from the father. This is called monoallelic expression.

If you look at this region here, you get an idea about the molecular mechanism of how imprinting might work, and I'll just take you through this. This is an example, and there are variations on this scheme.

So here are these two genes, IGF2 and H19, on chromosome 7 (Fig. 8, right). There is a region called the imprinting control region on chromosome 7. If you look at this region here, there are the marks. These turned out to be DNA methylation marks of CpG dinucleotides. So, this is the imprint signature.

In this case, it's on the chromosome that comes from the father. This is the imprint that comes in from the germline, and it's inherited; it's heritable. It is present in the fertilized egg, and it's heritable throughout life. If you take your skin cells, you will see this imprint that has come from your parents' germline.

What this imprint here on chromosome 7 does is that it allows these regulatory elements called enhancers to activate this gene H19 on the maternal chromosome. The same enhancers will activate IGF2 on the paternal chromosome. So you can see that on chromosome 7, you have two genes: One is expressed when it comes from the father, and the other is expressed on the chromosome that comes from the mother.

This principle applies to other regions, other chromosomal regions, which work in similar ways, but there are variations on this. This is to give you an idea of how this imprinting works.

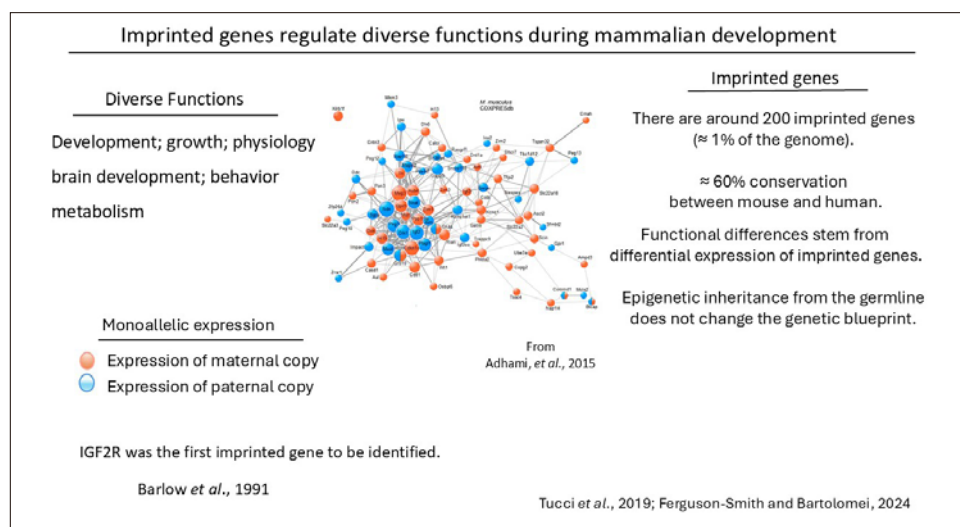


Fig. 9

If you now go forward, what we now know is that there are approximately 200 imprinted genes in mammals (Fig. 9), that is 200 out of 20,000 genes, which is around 1 per cent of the genome, so it's a small part, a fraction of the genome, which is imprinted in this way.

They have a variety of functions, from regulating development, growth, physiology, brain development, behavior and so on. So they don't have a specific function; they have a variety of

functions.

This is how imprinting works. There are imprinted genes that come from the mother and father. They have different functions. They are important in terms of balancing growth and other aspects of mammalian physiology and development.

I'll give you just two examples of what these functions are.

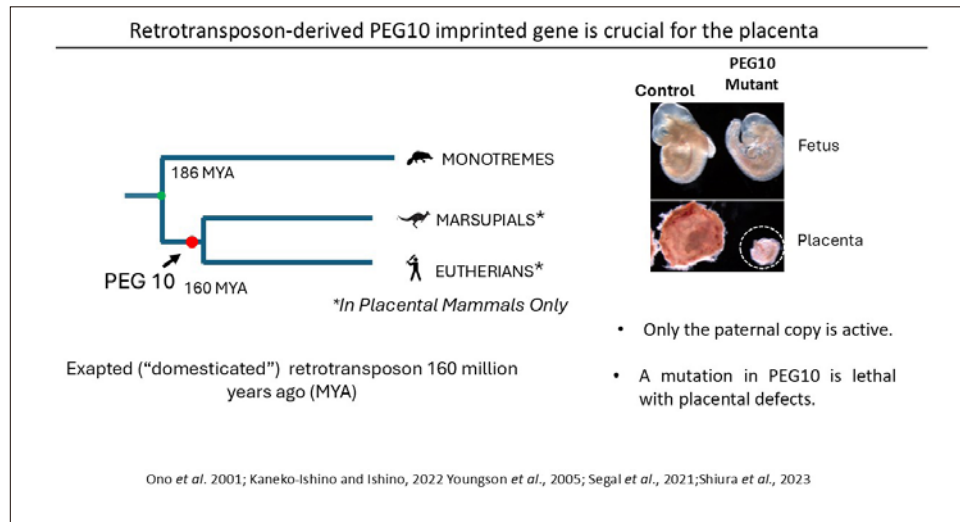


Fig. 10

This is one of the examples (Fig. 10). The gene called PEG10 is active when it comes from the father. The maternal copy is silent. So if you mutate this gene, what you see is that the placenta doesn't form very well and these embryos don't develop fully. They stop developing because when you mutate this gene, the placenta doesn't form.

So you can see that this gives you an idea of how this differential expression of imprinted genes can impact on development.

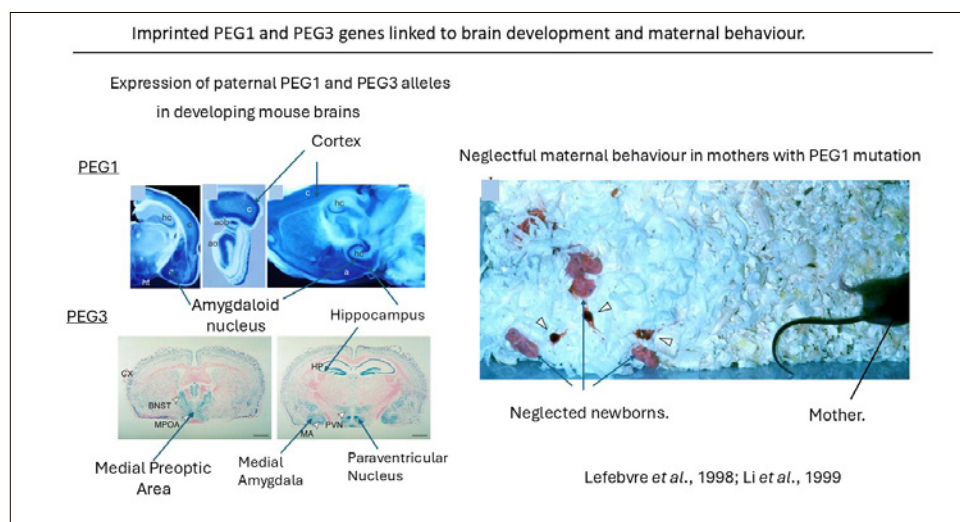


Fig. 11

The second example is these two genes: PEG1 and PEG3, where the paternal copy is active, and this shows the expression of these genes in the developing brain (Fig. 11, left). These experiments were done in mice. So in the mouse brain, you can see expression of these two genes.

What was found is that if you mutate this gene, PEG1, it affects behavior—maternal behavior.

What happens is that when the pups are born, the mother doesn't show normal maternal response. She doesn't build a nest, so these pups are left, and they will just die if you don't rescue them (Fig. 11, right). So this gives you an idea that another gene has a completely different function, and it's affecting brain development and behavior.

These are just the two examples of the 200 different genes having different functions, which gives you a broad view of how imprinting works. I should stress that one thing to remember is that these imprints do not alter the genetic sequence. They are marks placed on the DNA without altering the genetic blueprint of the genome, which remains intact.

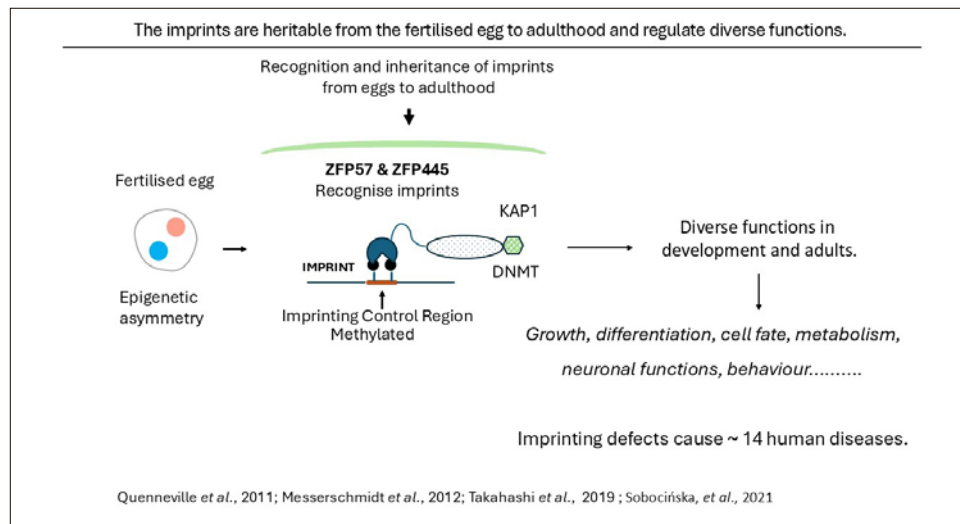


Fig. 12

These are imprints or imprinted genes (Fig. 12). This imprinting signature is heritable from eggs to adulthood. They persist, and there are specific mechanisms that recognize these imprints. These genes called ZFP57 and ZFP445 recognize these imprints, and the information is then transmitted from the egg to adulthood, and they will perform a variety of functions at different stages of development.

So we are coming from the start of very embryological experiments and observations, and we now have some understanding of the molecular basis of how this imprinting works.

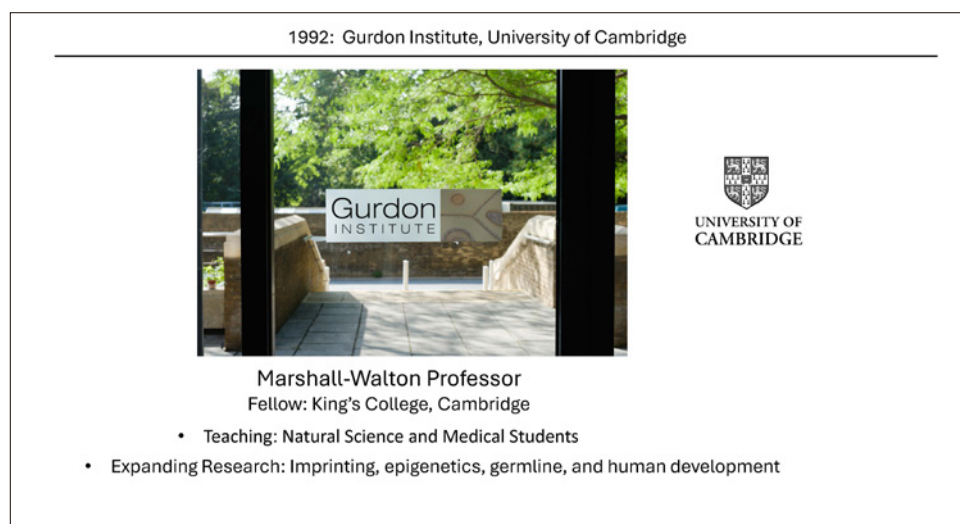


Fig. 13

Left image: Photo by Simon London

In 1992, I moved back to the university, where I was appointed as the Marshall-Walton Professor, and so I started doing some teaching (Fig. 13). I was teaching natural science and medical students, and again, I was fortunate to have my own lab. I started to expand my research on imprinting and epigenetics, which was the new field now that we were developing, emerging out of imprinting work, and also working on the germline and early human development. So we are expanding our research and going into different areas.

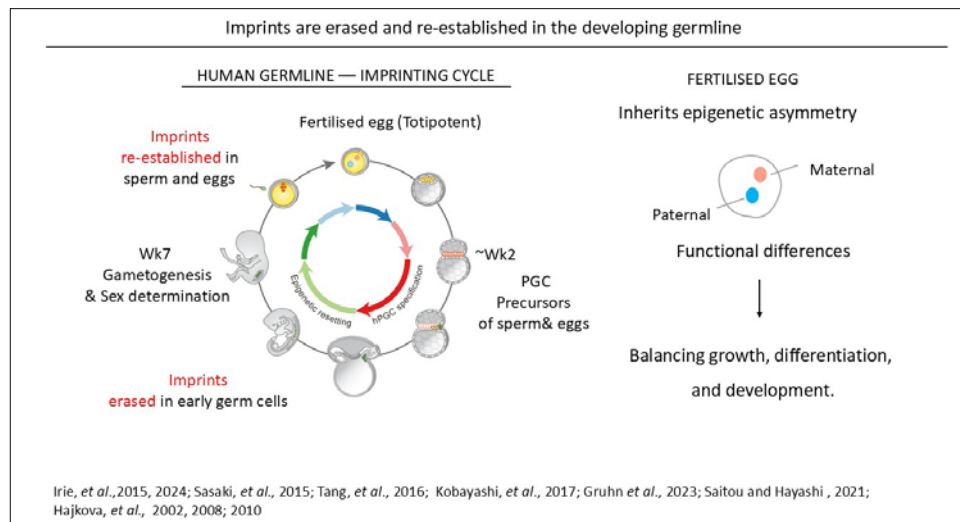


Fig. 14

One of the areas that we started to look at was the germline, which is the lineage which gives rise to sperm and eggs. This is the germ cell lineage (Fig. 14). As I told you, when the egg is fertilized, we have maternal and paternal genomes, which is the same in humans, and also have functional differences and inheritance of epigenetic asymmetry.

Now the epigenetic asymmetry is programmed during the time when the germ cells are developing, which is the same in mice and humans, but this is an example in humans. The primordial germ cells, the precursors of sperm and eggs, in humans form during the second week of development, the second week of gestation. So it's very early that the first cells appear, which are going to give rise to the next generation and the generations after that. These are very important cells, set aside very early in development.

As these cells start to migrate towards the developing gonads, the first thing that happens is all the imprints are erased, so that everything is wiped out by the time they get into the developing gonads. When you start to get the formation of sperm and eggs, new imprints are put on, depending on whether the embryo is a male forming sperm or the egg in females. When fertilization occurs, you recreate this asymmetry. So this is something that is repeated for each generation. It's not something that's heritable. It is wiped out and reintroduced.

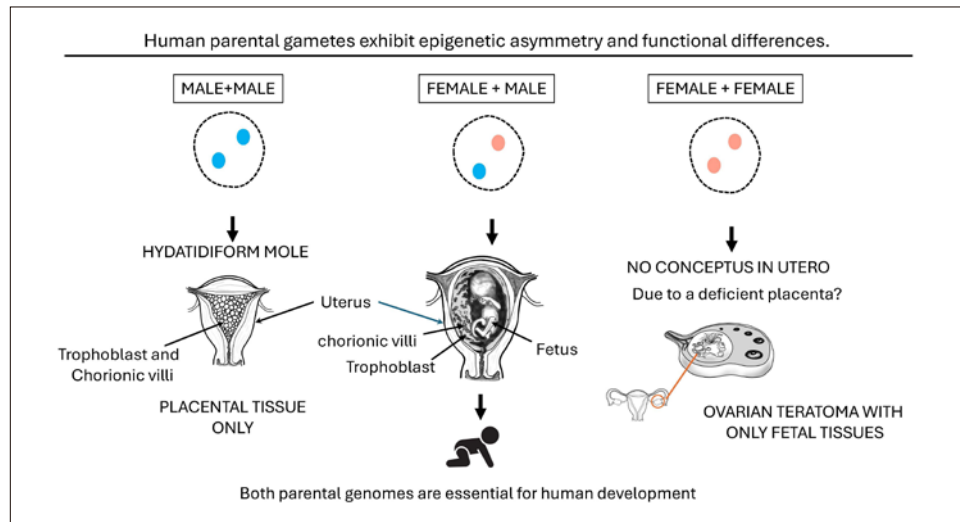


Fig. 15

The same thing that I showed you in mice works in humans. So in humans as well, you have the two parental genomes which balance the development of the embryo and the placenta, and you need both of them for normal development of humans (Fig. 15). If by chance you have fertilization of an egg which lacks the maternal genome, then you have two paternal genomes. What you see is excessive proliferation of the placental tissue, and you don't get any fetal tissue. Conversely, if you have two maternal genomes, you also don't get development because there's no placenta, so they can't implant. Sometimes these embryos stay in the ovary. They form what are called ovarian teratomas, which contain only fetal tissue.

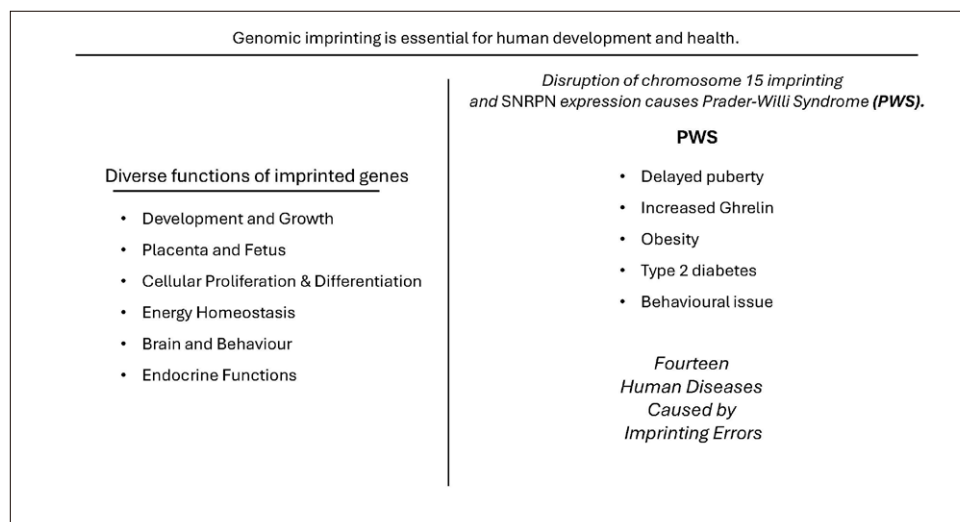


Fig. 16

What I've told you applies to mice and humans and all the mammals that have been looked at so far. So this is something which is universal to all mammalian development systems. As in mice, this imprinting has diverse functions in humans as well (Fig. 16, left). For example, it's involved in regulating development and growth, the regulation of the placenta and fetus, and the proliferation and differentiation of cells as they develop. It is also involved in energy homeostasis, brain and behavior and endocrine functions. There's a very diverse range of functions that is performed by these 200-odd genes. So they are very important.

Now, if imprinting breaks down for some reason and you get a disruption of imprinting, you

have human diseases. Here's one example of what happens if there are imprinting errors on chromosome 15 of humans involving this gene called SNRPN, and what happens is you get this syndrome called Prader-Willi Syndrome, where you have delayed puberty, obesity, type 2 diabetes, and behavioral issues (Fig. 16, right). So you can see that imprinting is important for development, but if something goes wrong, there's a cost which results in human diseases. It's estimated that there are about 14 diseases that are caused when you have errors in imprinting.

So, one of the questions which is really quite fascinating, and we need to find answers to, is on the evolution of imprinting. When did it evolve, and what does it tell us about mammalian life and mammalian development?

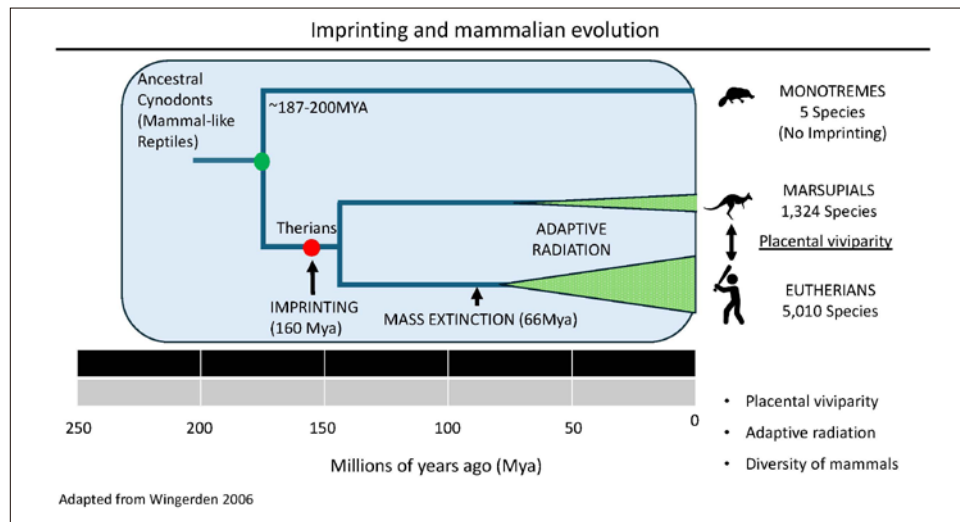


Fig. 17

It is supposed to have started around 160 million years ago (Fig. 17). This is before the split between marsupials and what are called eutherian mammals, which are the rest of the mammals. This is 160 million years ago in the Therian lineage when imprinting seems to have been initiated.

Then they split into two, marsupials and eutherian mammals, and so what this tells us is that imprinting somehow is associated with what is called placental viviparity, which means that the development of embryos occurs in utero resulting in live birth. So it's thought that imprinting through its influence on placental development was important for viviparity and development in utero.

66 million years ago, there was a mass extinction, and the dinosaurs were wiped out, and this gave an opportunity for mammals to proliferate and diversify. This is called adaptive radiation. After this time, over 66 million years, we have a huge diversity of mammalian life forms, which you see now in the world. To some extent it also happened with the marsupials—not to the same extent.

Now there is another group of mammals, which are called monotremes, like the echidnas. These are egg-laying mammals, and they don't have imprinting as they split off earlier. So what we can do now is, with the information that we are starting to collect on different aspects of imprinting, we can start to sort of piece together how this imprinting evolved and maybe understand better its significance and importance. This is something that I think might be interesting to focus on for the future.

I think for the discovery of imprinting, we started with a very esoteric idea, and a very curiosity-driven research, and I believe it has had quite a big impact, and I just want to illustrate different aspects that have been influenced by this discovery.

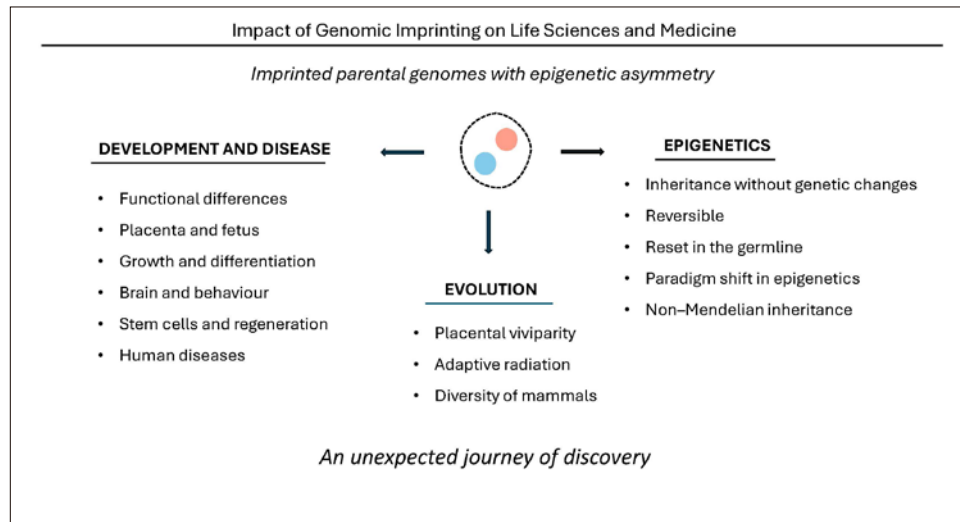


Fig. 18

As I told you at the start, this phenomenon, these functional differences between parental genomes, is due not to genetic changes but to epigenetic changes (Fig. 18). So these are modifications of DNA without alteration of the genetic code.

The discovery of genomic imprinting is very important for this field of epigenetics, which is now an extremely important area of research, both in terms of understanding development and disease. This is inheritance without genetic changes. It's reversible. Unlike genetic changes, it is reset each time in the germline. I think it presented a paradigm shift in the field of epigenetics because this is non-Mendelian inheritance, which is important in mammals, so you don't only have genetic inheritance, but you also have epigenetic inheritance.

It has an impact on development and disease, which I also told you about. These parental genomes are functionally different. The paternal genome affects the development of the placenta and the maternal for the development of the fetus. They regulate growth and differentiation, brain and behavior. Also, they are important for stem cells and regeneration. If things go wrong, they contribute to human diseases.

The important thing in the future is to find out more about the evolution of imprinting. I told you that it is important for the development of placental viviparity, the formation of the placenta and development in utero, and this might have contributed to adaptive radiation, to give rise to the diversity of mammals that we see now in the world.

I think this is a kind of a story of how you can start with a very simple question, and the answer you get can have implications beyond what you might imagine at the start.

Thank you very much.

The interview held after the commemorative lecture is available on the Kyoto Prize YouTube channel.

(<https://www.youtube.com/watch?v=2p9ocKdHV2M>)