Life on Earth 101: The Genetics of Aging, Identity, and Everything In Between Transcript

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Podcast Intro and Regenerative Medicine Overview

Hey there, welcome to the Recharged Biomedical Podcast. I'm Dr. Edward Park, and if you're curious about regenerative medicine, you've come to right place.

We're diving into the latest breakthroughs in telomeres activation, stem cell exosomes, all the cutting edge science that's shaping the future of healing and longevity.

Let's get started. Hi everybody, this is Dr. Ed Park, the host of the Recharge Biomedical Podcast and I'm planning a vacation so I wanted to provide some interesting content.

Today's content is going to be about the genetics of life. And in 20 minutes of this improvised conversation, I am going attempt to explain to you what genetics is, why it matters, how it involves all living cells and how all behaviors such as aging relate to this.

So let's go. First of all, there are some things which are not alive. For example, prions are shaped mimicry proteins that cause things like Jakob-Kreuzfeld disease.

What Genetics Is and Why It Matters

These are NOT alive! Bacteria are alive, bacteria have Circular chromosomes generally, double stranded. Viruses can have single or double-stranded DNA or RNA encoding.

But for the most part, life on earth, if you're talking about plants and animals, involves chromosomes. Okay, so what's a chromosome? Chromosome, if it's not bacterial, is a linear set of base pairs.

A base pair is A plus T or G plus C. These are the four flavors of all known life in plants and animals. How many chromosomes do we have? 23 pairs. One from mom, one from dad.

Insects can have 10 to 20, plants varies a lot, but in general everyone has pairs of chromosomes. Another truism is that sexual dimorphism, is very common.

Flowering plants up to 80% of them can be both male and female So there are exceptions as with everything to every rule, but in general You have male or female plants and very much so male-and-female animals Now why is that?

I submit this is for genetic diversity think of your chromosomes as decks of cards now if you only had cards from one parent, eventually mistakes would enter.

You don't have the advantage of a variety of things that are working in terms of evolutionary theory. So, for example, you have, let's say blue is for boy and pink is girl.

We have 23 chromosomes from mom and 23 from dad, which are the blue deck. Now what makes this so advantageous is that when you make sperm and eggs or whatever it is you're making for the next generation, there's a step whereby your blue deck and your pink deck will get mixed.

This is called meiosis or the creation of sperm in eggs in our case. And because grandma on maternal and paternal, and grandpa on maternal and maternal are represented in your parents' chromosomes, you get effectively four decks of cards that are mixed into your gametes.

And this provides a great advantage in mixing of the decks. Okay, he got it? So all plants and animals, when their cells divide, they line up these 23 pairs of chromosomes and they get separated.

In point of fact, when you have 46 chromosomes and then you unzip them as when a cell divides, then, you'll get a perfect 46 copies of that. So, nothing changes.

Now, having said that, There is something that I need to point out. One of the doctors at this, he has a billion dollar company testing gut biome and I walked into his talk and he said, I only know two things and that is that, that your chromosomes or your genetics never change.

Chromosomes, Meiosis, and Genetic Diversity

Well, he actually was wrong on both counts. Well he, actually he was only right about not being a doctor because your chromosomes can vary. Every time you copy, it's incredibly rare to get a mistake even though there are about 6.4 billion base pairs, 6 4. 4 billion typographical errors possible, it's estimated that a typographic error only occurs one in three divisions.

But eventually over the course of a lifetime over an unknown number of reproductions and copyings, you do get errors. Think of it as photocopying a photocobe.

Every time you there's degradation. And in fact, if you put into AI image generation a request to copy an image by about eight to ten generations, the final copy looks nothing like the original.

So who cares? Well, that doctor or that businessman was wrong because you do experience genetic drift. Witness my podcast on the twins who look nothing alike.

There are other reasons for that, but indeed we have to presume that you yourself don't make perfect copies every time. And so, this may not matter, it may matter and we'll get into that right now.

So, forget about epigenetics, we're going to circle back to that in a second. Basically, you have what is a pretty decent set of chromosomes with mostly functioning genes.

What are genes? Genes simply put are sequences of these base pairs that will encode mRNA. What is mRNA? sequence of I guess you could say anti-sense. And when that's translated by the ribosomes, it'll make a protein.

Why is this so important? Why isn't the essential dogma of biology? Well, because everything we know, all that emerges is because we make these genes, about 20,000 in our case.

How that makes life is quite the mystery. But it is true that our genetics are to some extent destiny. So let's talk a little bit about taboo. Why is it tabo to have incest?

Well, because there's lack of genetic diversity. Let's say you're in an isolated population. You keep on meeting with close relatives. Then let say a gene mutation has been acquired and gets reshuffled into that four-deck solution.

of both maternal and paternal grandparents, where are you going to get the good gene if your sperm and egg producing cells only have bad genes? So let's say you've got 20,000 good genes or the ones that nature has designated as wild type and you have all of a sudden one dad that has a deletion, let say that causes cystic fibrosis.

Well, if his cousin has the same deletion, we're getting to these Mendelian genetics problems. So let's get into that now. Gregor Mendell was a monk who mated peas and he was able to somehow brilliantly deduce that this kind of sexual dimorphism was occurring.

It's the basis of genetic theory that we have let's say a wild type or a quote-unquote good gene.

Mutations, Mendelian Inheritance, and Disease

Now a mutated gene, let say one single nucleotide is different or these SNPs, single-nucleotid polymorphism, it may provide an advantage like classically they call out malaria.

Some types of sickle cell might have protected from malaria in Africa. So the laboratory of nature is always tinkering. In general, you want the wild type variants and you'll want two copies, okay?

So, let's say a gene is like a kidney, and in the case of cystic fibrosis, if you have two kidneys from mom, two from dad, on your four chromosome copies you're good to go.

Once you start deleting those, diseases can emerge. So, in general, that's the point of sexual dimorphism. Well, dimorfism is really more the different traits, but true hermaphroditism, is practically non-existent in animals.

So who cares? So we were born one single fertilized egg, and from that, we differentiate into different types of stem cells. And we're hoping that the different kinds of stems cells have good copies, even if their generations of daughters and daughters start to acquire abnormalities.

So it's kind of an elegant system throughout our body. We hope that we have original as good as we can get copies. Certainly in the future and now, So, in the present, recent present that people have done gene editing on zygotes using things like CRISPR cas, that opens up the way to different kinds of solutions to genetic diseases.

But in general, we have a certain number of stem cells and the ways that stem cell interact is kind of mysterious. We think it has something to do with epigenetics.

So here at Minute Nine, let's talk about epagenetics, so in the 6.4 billion base pairs and your 20,000 genes, chromosomes are actually like strings, but they're not just strings.

They would be impossibly long. so they are wrapped up into spools. called histones they're wrapped around the histone's themselves are clustered so everything is very tightly packed in the chromosomal structure when you see a picture of a chromosome it's packed ready for division and then it'll be unpacked right so what you're doing when your dividing a cell is taking 46 single strands and splitting them into 46 you're taking 46 double and making them into 46 single.

So you get a copy of each of the four grandparents' contribution. What people don't often consider is something called epigenetics. So epigenex is which genes are gonna be turned on and off and therein lies the magic.

Let's say you have a thousand musical instruments in eight different genre orchestras and they can play all different kinds of notes. The way that those notes play in what genre is kind of how the species emerges.

Why do I say that? Well, we share about 98% of the genes with chimpanzees and yet chimps do a hard time and we have a harder time looking the same.

Epigenetics and Gene Regulation

Some of the actions, the core functions of metabolism, cell biology differentiation are the In contradistinction, humans, despite mixing with Neanderthals, homo sapiens, sapians in general, can mate with anyone and have viable products because we're close to 100% similar in our genetics and in the chromosome structures and everything else.

So the concept of race, which was invented in 1800s, is really not a very robust one. in so far as you can't really tell someone's race from their genetics.

It's much more of a cultural aesthetic, if you will, view. So, what does that mean? The histones can be methylated, which means that the genes are silenced.

It means the spools are tightly wound and if they're tightly-wound they can't be opened up and they cannot be accessed for gene expression. So you don't need 20,000 genes to run a heart cell or a brain cell, or kidney cell.

You might only need a suite of them and that suite would include you know, basic metabolic things, proteins to express in any of these cell types, and it would vary.

There would be different nodes played, yeah? So the opposite of that is acetylation. See, methylation is adding a methyl group around the histones to open up the genetics in an interested or a gene of interest and acedylation is opening and methylation closing.

So that's what you have. So even though your computer program or your data is in every cell, which is hugely inefficient in a way, only certain genes are being expressed.

And so that's like the software. Okay. So good. We have the genetic codes, we understand chromosomes, so we understands epigenetics. Now, how does cell types, how do they emerge?

And this is a very interesting thing. Last year, or a year and a half ago, I went to Hong Kong and went a stem cell conference and the exhibitors at these booths, you know, they're not just giving out pens and beers.

They have recipe books where they figured out exactly how either with Gene expression, proteins, or even chemicals, they can make every step of the way to any cell type.

And, you know, with possible exception, their magic trick over the last eight years or so is called IPS, which is Induced Pluripotent Cell. So they can take any differentiated cell with any kind of epigenetic software, de-differentiate it like a slot machine, and send it to a zero early state.

And then they know the steps to create the different types of tissue, then the iterations, finally the deployments. So, they could make you a specific nerve type cell, To produce dopamine, they can make the cell to produce insulin in the pancreas, and they know each step, which is quite godlike in my estimation.

I even met and blogged about a Chinese doctor who had cured 7 out of 7 insulin-dependent diabetics by doing exactly that.

Stem Cells, Reprogramming, and Cell Differentiation

So great, we have different cell types. We know that cells can roll back. An interesting thing I learned only two weeks ago is that my hunch was validated in certain retinal cell type, which is my hunt was that regular differentiated cells could transiently go back to acting more primitive.

And so just because your destiny is to be a differentiated ho-hum kidney cell, you can go back to a more stem-like state either within that specific kidney or something even earlier.

So it's kind of like magic that can multi-directionally. So who cares about stem cells? The major reason we understand stem cell's role in aging, and we're talking about aging now at minute 14, is why do we get old?

Well, consider the humble lint trap, right? Every time you do a drying load, there's a little lye in there. So what is the implication of that? Implication is that You don't have as much clothes as when you put them in.

And if you keep on drying your clothes over many, many times, your close will disintegrate, right? So the same with the three base, I'm sorry, one error per three cell replications.

We're in a state of constant entropy. Now, are there hacks to regenerate muscle? Definitely. Are there hack to re-generate liver? Yes. Can we harness those hacks? Yes.

But what is the journey from one fertilized stem cell or egg or zygote to being dead? Well, you make a bunch of

stem cells and some of them gain genetic mutations, some gain epigenetic errors.

And it's really important to understand that the epigenetic switches on or off, right? That would be acetylation, methylation. They're kind of inherited cell to cell.

Now, that's not as 100% reliable as the genetic code, but it is pretty darn close. And over the last 12 years, I met the two inventors of this technology.

and they can attempt to read every single one of those gene locuses, or loci, and say how old you are. And this epigenetic age has a reliability of about 99% correlation with what's written on your driver's license, which is amazing.

Does that mean that they understand why the switches are on or off? No, clearly it's just data mining. Just like I went to 23andMe yesterday, because I had a question about my sister asking me, hey, did mom have a love child?

Aging, Stem Cell Decline, and Immune Exhaustion

And so I looked up the math and it turns out my sister Anne and I are 52% related, me and my two sons are 49% as per the 23andMe company. But what isn't there is my Japanese.

When I first looked at it eight years ago, I was 17% Japanese, the next year I 9% and now I'm 100% Korean. What does that mean? That means that these are just models generated from statistical sampling, self-identification through what's called haplotyping.

So take it with a tremendous grain of salt as the model will improve. In contra distinction, what doesn't vary is they can sequence your entire genome by unzipping the chromosomes and reading them AGCT yada yadda yata out to 3.2 base pairs per chromosome.

What doesn´t change is that we have 23 pairs of chromosomes. The last pair is the sex chromosomes, and what does not change even though there are intersex variants and the most dramatic of which being testicular feminization very famous models have been people with testicles and no secondary sex characteristics of men because they did have the sex determining region Y or SRY or the TDF testes determining factor so they made internal testacles But in testicular feminization, there's no receptor for androgens.

So they literally can't receive any androgenic signals. And they become beautiful women with all the hallmarks of dimorphism, such as lack of hair and whatever it is that makes true beauties.

look feminine. So all right, so let's go to the last piece I want to address before minute 20, which is aging. Why do we age? Certainly we aged because of genetic mutations.

We probably age because of these epigenetic switches because the high correlation, although we don't know the meaning of that. But first and foremost, the lint trap tells us all.

We age, because we're losing cells. Now, don' worry about losing differentiated cells, cause the local stem cells will regenerate them. Over the course of creating errors, genetic silencing, this can lead to bad behaviors, old behaviors and that's bad enough.

But when the actual stem cells become depleted, the best example of this is aging. So aging is related to a lack of immune function. And the best example of this was a Dutchly Van Andelslipper who only had two clonal variants.

A clonal variant is a undifferentiated T cell that can respond to new challenges. You and I should have 10,000 clono variants that are undifferenciated.

or that are varied, but she only had two. So eventually her immune system was depleted of this flexibility, this improvisational capacity, and that is what begets cancer succumbing to infection like Jack Delaney in his 90s.

This is a big problem. Can we regenerate the immune systems, the hematopoic system? Yes, probably we can. And so stem cell scientists will get there. I think that if you could just stay alive for a little longer, you'll be able to reap the benefits of this.

So, I just wanted to, in these 20 minutes, explain to you what genetics was. People don't really understand how, just because we have 100% of the same genes as each other, 98% as chimps, why a chimp is a chimp, These things are very much emergent properties from concepts that haven't even been invented, things like biomorphic energy fields.

Closing Thoughts and Podcast Promotions

So all this stuff is a dialogue between cells, their stemness, differentiation pathways, and it's not something that we perhaps will ever understand. But I just want you to have the basics.

So yeah, there are certain diseases which are single gene, certain traits which aren't. But in general, the rule of thumb is that most traits, even things like eye color, hair color.

Cognitive styles are very much multifactorial and not deterministic like that. So don't let people convince you that you got this from your mom or that from you dad or this is genetic.

You know, genetics is not destiny. So I hope that's interesting to you, perhaps not, but if you like content like this, please like and subscribe and hit that notification button and I'll see you in the next episode.

So that is Life on Earth 101. See you next time. We age because our telomeres shorten and our stem cells deplete. But what if we could support both? I've been taking TA65 for 17 years.

It's the only supplement I trust to support better mood, better sleep, and exercise recovery. And at age 57, I don't have any gray hair and I do not need reading glasses.

TA65 is available now. Go to rechargebiomedical.com slash TA-65 and enter promo code RECHARGE10 to save 10% off. Many clinicians are getting interested in exosome therapy and they hesitate for good reason.

Questions like does this work? What forms do I need? How much should I charge? how do i stay out of trouble? All these questions are addressed in my online course.

That's why I created it to help you get started. The online course is your permanent turnkey resource to get started either treating yourself, friends and family, or to expand your practice and help more people, as well as increase your revenue.

Thanks for tuning into the Recharge Biomedical Podcast. If today's episode got you thinking, you'll love my book, Exosomes, Songs of Healing. It's packed with cool analogies, full color illustrations, and all the science you need to understand how exosome are changing the game in aging and regenerative medicine.

You can grab it in paperback, ebook, or audiobook, whatever works for you. Now head over to www.rechargebiomechanical.com to check it out. And don't forget to like and subscribe so you never miss another episode.

See you next time.