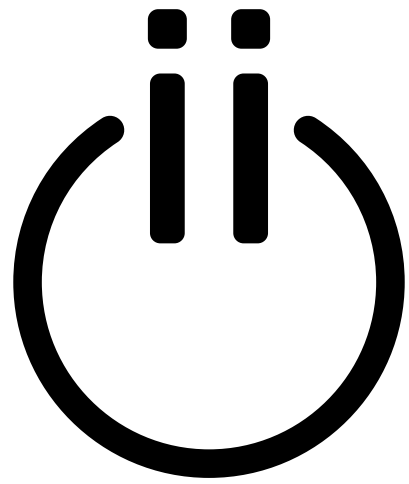

The Innovation Initiative

Human Life Extension

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We can change the way things are when we stop accepting the status quo. It's not science fiction for us to live longer, for each of us to be restored to the physical and mental performance of a young adult. Today's topic is human longevity, also known as life extension.

I'm Cat Volz for The Innovation Initiative- Entrepreneur TV. Next to me is Dr. Aubrey de Grey. Aubrey is one of our most intelligent people solving THE #1 problem facing humanity- we're all going to die, and for most of us, our declining health will be unpleasant. Over 6300 people die every hour. We are holding back progress if we do not care about aging related deaths. Dr. de Grey is a biomedical gerontologist, the chief science officer and co-founder of SENS Research foundation. He was the first person to get started on this problem, before there was commercial opportunity, while most of the world ignored it. Dr. de Grey is adding unlimited productivity, health and enjoyment to the lives of us people on the planet.

This is an expensive problem. It will take a concerted application of therapies to repair the accumulated damage that kills us. To put it into perspective, curing disease is low hanging fruit. It's attractive to investors to solve side issues around aging (Alzheimer's, Atherosclerosis, Cancer), the near-term successes to return on investment. But the SENS foundation is working from a complete solution backwards- a perfect maintenance plan so these problems don't even occur. Because there's already funding in place to focus on stem cell research, Dr. de Grey has elected to solve the harder long-term problems that are not yet being funded. Please visit SENS.org to learn about their research and to donate so these treatments will be here for us to have the option of living longer. The SENS link is below as well as a pdf to explain the types of aging damage.

Cat: Welcome Dr. Aubrey de Grey

Aubrey: Thank you very much

Cat: What's the problem with society's healthy aging stereotype?

Aubrey: I think the real problem is that there is no such thing as "healthy aging." I mean, of course the term aging is used in many ways, depending on context, but most of the time, what we mean by it is a decline in performance; a decline in physical performance and a decline, of course also in mental performance—one's cognitive abilities. And nobody wants either of those things. Both of them are identical with ill health. So the idea of healthy aging is a contradiction in terms.

Cat: Why should we treat the system instead of targeting the disease?

Aubrey: The reason why these diseases are diseases of old age is because they're actually diseases of being alive. They are side effects of simply being alive. And for many decades they are asymptomatic because their early stages are so minor at the molecular level and the cellular level that the body just works its way around them without significant performance impact. But eventually the accumulation of this damage that the body does to itself reaches a level that the body is not able to just work its way around. And that's when these diseases emerge and progress. So what that means is that if we're trying to actually eliminate and make sure that we don't get these diseases then we have to look at the originating cause which is the mechanisms that keep us alive in the first place. And clearly we can't eliminate the mechanisms that keep us alive because that would rather defeat the object, so the goal therefore is to identify where as to intervene, to let us be alive without the side effects accumulating to a point that is bad for us.

Cat: You say aging is a phenomenon of physics. What does that look like?

Aubrey: Well what I mean when I say that aging is a

phenomenon of physics and not of biology is that it is fundamentally the same process whether it happens in a living organism or in a simple man-made machine like a car or an airplane. And it is the same process. The fact is that any machine with moving parts simply does itself damage. A car will accumulate rust, it will accumulate grit in the engine or whatever. And different machines of different levels of sophistication have different ways of coping with this damage-- either they're designed simply to tolerate a large amount of it or they're designed to create the damage slowly as a side effect of operation. But one way or another, whether they are living or not living, these machines all at some point have enough of their, if you like, metabolism behind them but they've accumulated an intolerable amount of damage.

Cat: Can you give an example of a side effect of metabolism that's killing us?

Aubrey: Well one very obvious example is that we get cancer. Cancer, most cancers, are diseases of old age. And that's because they result from the accumulation of damage to our DNA. The mutation in certain genes that normally control when the cell divides, that happens when the cell divides uncontrollably and that's what cancer is. Now that damage comes from chemical reactions that cause mutations to occur. Those chemical reactions are initiated by a family of compounds called free radicals which come out of a place in the cell called the mitochondrion. And that happens, every side effect, every central component of being alive, mainly breathing. Mitochondrion is often called the power house of the cell. What that means is it's where the chemistry of breathing happens, where oxygen is combined chemically with nutrients to extract energy from those nutrients. The details of how that happens are very intricate. And sometimes they don't go quite right and these chemicals called free radicals are created. And occasionally those free radicals interfere with other molecules such as DNA.

Cat: So the Hayflick Limit is a proposed finite number that a cell can divide. Is this real and what is the work around?

Aubrey: The Hayflick limit is certainly a real phenomenon in certain circumstances. But its relevance to aging is actually rather complicated. The Hayflick Limit arises because when a cell divides the ends of the chromosome which are called telomeres get a little bit shorter. That's an inherent and absolute inescapable consequence of the molecular detail of how DNA is replicated during the cell division. Now some cells in the body need to divide rather a lot and for that reason they need to do something about it the shortening of the telomeres. And they do. They have a special enzyme called telomerase which adds additional DNA on to the end of the chromosome, on to the telomere. That DNA doesn't have any information content, all it's there for is to compensate for the shortening that happened during cell division. But even though this sounds a little bit bad news we have to look around a little more closely in order to determine whether it really is bad news. In most cells there is none of this enzyme telomerase that compensates for the shortening I'm talking about. But in most cells that's okay because the cells do not divide, or even if they do divide they only divide very, very rarely, for example cells that make up the heart, simply don't divide. And if they're not dividing then of course their DNA is not replicating so the telomeres don't get shorter in the first place so they don't need to have the telomeres extended. Now there are some cells that do divide but only on demand. For example, the cells in the lower layer of the skin called the dermis which divide like crazy when you have a wound, when you cut yourself. First the cut, then close the wound very quickly. But normally they're just hanging out and not dividing. So those cells don't express this enzyme telomerase. Because they divide so well when they are induced to do so, they turn out to be really easy to work with in the laboratory, in the petri dish, in cell culture. And that is the model in which this thing called the Hayflick Limit was discovered. But if we ask what its relevancy is to real life, we have to look at what's going

on in cell culture. Cell culture is functionally equivalent to putting these cells in an infinite sized wound that they can never close. And they divide and divide and divide far more than they would ever need to divide in the body and so it doesn't prove that there's actually any relevance. The cells that do divide a lot, stem cells for example in the gut lining or in the blood, those cells certainly do divide more often than they would be able to if they didn't do anything about telomere shortening. But they have a little bit of this enzyme telomerase, so again, they don't have a Hayflick Limit. In most cases there isn't a Hayflick problem. However there may be some cases where there is-- white blood cells which of course, the main constituents of the immune system sometimes divide like crazy in order to get rid of an infection. And sometimes they have to do the same, the same cells that divide like crazy for infection have to do it repeatedly because the infection comes back. Those cells have been implicated substantially in the declining function of the immune system during old age. And some people believe, and it's a reasonable hypothesis, that inability to divide because of telomere shortening is a large part of what goes wrong with these cells. So the short answer is, it's a bit of an open question. The Hayflick Limit is by no means as important as some people thought it was 50 years ago.

Cat: Okay.

Aubrey: But it may still have some relevance in normal aging.

Cat: Weren't you just doing a crowdfunding campaign around telomeres?

Aubrey: So the crowd funding campaign that we have just done actually kind of turned the Hayflick Limit upside down. The thing about our campaign is that it's designed to fund a project that we have that is targeting cancer. Now cancer of course is a disease of uncontrolled cell division. So if cancer cells could not extend their telomeres,

then we wouldn't have a problem with cancer. Cancers might initiate because they've figured out how to divide indefinitely and escape from the immune system and break down the extracellular matrix and all the other things that cancer cells have to do in order to divide, but then, if their telomeres were getting shorter and shorter, eventually the cells would kill themselves just by dividing too much. They would divide themselves into oblivion because when the telomeres get too short all hell breaks loose. And that's what we would like to have happen, we would like to have that happen way before the cancer got big enough for us to even notice it. But what actually happens in a real cancer that we do notice is that the cancer figures out how to introduce mutations that upregulate, is what it's called, activate this enzyme telomerase. So they kind of escape from the Hayflick limit that way. And for some time people have been interested in the possibility of reverting that, of suppressing the activity in telomerase in cancers so as to make them divide into oblivion.

Cat: Divide.

Aubrey: Now what we're doing here and what we're doing a crowdfunding campaign for, and I should emphasize it that it's still going. This is for a kind of complement to the anti-telomerase work that I just mentioned. And it's needed because it turns out that some cancers, about 15% of cancers in humans, actually, they do the same kind of thing, they figure out and they mutate a way into a state where they're extending their telomeres, but they don't use this normal enzyme, telomerase. They use a different system. (sneaky!) And that different system is not understood. Telomerase is well understood. The genes were cloned a long time ago and everyone understands what's going on. This other thing which is called ALTS, which stands for alternative lengthening of telomeres-- virtually nothing is known about it. We don't know the main fundamental genes that are responsible for making it go. And once we can find those, of course we can do corresponding thing to what's already being done with telomerase and knock it

back, and thereby knock all cancers back.

Cat: So you're working on that?

Aubrey: That's what it's all about.

Cat: Super cool. What are you thinking about introducing the new genes from bacteria? That was something that seemed novel that you're doing.

Aubrey: Sure. So the idea here is something I first published back in 2002. And the concept is very simple when you hear it but a little bit left field when you first hear it. The idea here is to identify bacteria in the environment which have the capacity to break down substances which accumulate in the body-- things like oxidized cholesterol or artificial or protein aggregates in the brain during Alzheimer's for example. These things accumulate very slowly over time. And the reason they do so is because the cell just doesn't have the genetic machinery to break them down. They're a problem when we get older, but they're not a problem when we're young. Evolution only cares about youngsters. Evolution is all about perpetuating genetic information, not about perpetuating individuals. Once you've had your offspring evolution no longer cares about you at all. So, the idea here is, we find these bacteria, now we do not do what you might think is the obvious thing-- inject these bacteria into the body so it can break down the substances that we cannot naturally break down. The reason we don't do that is because these bacteria do plenty of other things as well and there will be plenty of bad side effects. But what we can do instead is we can identify the specific gene or genes that the bacteria are using to allow them to break the stuff down. And then we can incorporate that gene, just that one gene, into the human genome of the cell so that human cell is then augmented. It has now this new ability to break down the stuff that was previously accumulating and poisoning it.

Cat: Can women remain fertile longer with these

treatments?

Aubrey: I firmly believe that female fertility is going to be actually one of the easier parts of aging to deal with. The easiest way to do that is to give women new ovaries. Now once they're new, that can be done either by tissue engineering, by creating new ovaries in the lab and then surgically implanting them or in principal it could be done by rejuvenating the pre-existing ovary in situ by introducing stem cells for example that would re-grow new follicles with new egg cells in them. Either way though what this means is that both the fertility aspect and the endocrine aspect, the hormonal aspect of menopause and the declining female fertility, ovary function, can be addressed by this way. Essentially this is because the ovary is just another organ, so the same kind of approach will work there just as with any other organ.

Cat: That's fascinating. What's the worst that could happen as far as adverse side effects from these treatments?

Aubrey: Well of course these treatments are in many cases only rather on the early stage of development. And it's normal for any medical idea when it's in the early stage of development to proceed very iteratively through mouse models and phase 1 trials and phase 2 trials and so on so as to look for side effects. And when that happens the side effects do not actually get to completely derail the idea. What happens is that people look for ways to get the best of both worlds—to get the effect without the side effects. Sometimes that means re-engineering the whole concept that was being pursued, but very often it doesn't mean that, very often it just means co-administering some kind of second treatment that direct, that will end that side effect. The whole spectrum of things exists-- emphasized really by your question is that this does not in any way distinguish our work or work on postponing aging from any of the rest of medicine.

Cat: It would happen with everyone. Of course. Yeah. If you

get enough funding, how long do you think it would be until these would be accessible to the general population?

Aubrey: I think that we will probably be able to get to the point where all of these technologies are simultaneously functioning in humans at the same time within the next couple of decades. I think that's only a 50-50 probability of course for anything even more than a couple of years away, there's plenty of things that could go wrong that would extend the timeline for any primary technology. So I certainly wouldn't bet on it being a hundred years but I think the 50-50 chance is about 20 years. But as you rightly say, it is dependent on funding. I think that if funding continues to be limited as it has been over the past 10 years that we could add at least another decade to that and let's remember that you gave your number of 6300 per hour, I think it was?

Cat: Yeah.

Aubrey: That adds up to well over a hundred thousand per day or you know, 40 million per year. So if we're talking about losing perhaps half a billion people if we actually delayed these therapies by a decade. That really puts into perspective just how important this problem is.

Cat: What is your response to the people that are concerned with overpopulation?

Aubrey: I have a variety of responses to people who raise concerns, whether it's overpopulation, whether it's inequality of access, whether it's, you know, dictators living forever, whether it's economic issues like the paying of pensions and so on, I have variety of answers. First of all for each of the specific concerns, I have particular ways of thinking about the problem that show that actually it's unlikely to occur or that we would actually be able, very straight-forwardly to avert it. So in the case of overpopulation for example I always point out that all the other technologies that are coming along, they're

improving, for example the use of fossil fuels and replacing them with renewable energy and artificial meat, they're reducing their agriculture and so on, these things are going to be reducing our carbon footprint and therefore the amount we pollute, and the same is happening for other pollutants. So of course what we already have done with CFC's that were affecting the ozone layer. So what this adds up to of course is that we're going quite soon to be in the position where we can have more people on the planet with less environmental impact. In fact it's precisely what overpopulation is all about, so I don't think it's going to happen. So that's kind of answer I often give but I want to complete that answer by giving you my two generic answers.

Cat: Okay.

Aubrey: These two answers apply across the board whether it's concerns over overpopulation or any of the others, they apply equally. The first one is sense of proportion. The same question is supposing, let's suppose we didn't invent renewable energy fast enough or whatever and we genuinely did have a situation maybe 50 years from now where we will be faced with a choice of having fewer kids than we would like because we need to make room for all these irritating old people who are not getting sick, or instead, letting them get sick. Now, seriously which are we gonna choose? We're going to choose to have fewer children and keep people healthy. So raising this as a reason not to develop the therapies is just not ethical. And the same applies to all of the other things. The other answer is the right to choose. I believe very strongly here that if we were to say, "Oh Dear, overpopulation, let's not go there" and we were not to work as hard as we can to develop these therapies, and if therefore we were to delay the time in which the therapies actually arrive then what we would be doing, we would functionally be condemning an entire cohort of humanity to an unnecessary early, unnecessary unpleasant death; the kind of death that we're familiar with today, whereas conversely if we had got on

with the job then humanity at least would have had the option with the information available to them that isn't available to us like whether or not they fix their renewable energy problem and so on. They would have the option to decide whether or when or how to use these therapies.

Cat: We should have the right to have an option.

Aubrey: They should have the right, the people in the future should have the right and therefore we have a duty to develop the therapies and give them that choice.

Cat: What is your inequality of access answer?

Aubrey: So of course the generic answer to anything regarding inequality of access to new technology which is in fact, it always trickles down, it always becomes cheaper when there's demand and it becomes available to everybody. But I think with this particular case we have a stronger option than that, which is that this stuff is going to be enormously cost saving. The fact is today aging is ridiculously expensive. I'm not talking here just about the direct cost, the cost of medicine to treat people who are sick because of the diseases of old age. That's of course an astronomical cost. But even that is dwarfed by the indirect costs-- the loss of productivity of the kids of the elderly because they're looking after their sick parents, the fact that the elderly are no longer contributing wealth to society because they aren't in an able bodied state to do so, all of these things, either way you look at it. What this means is that these therapies, even if they're expensive to deliver, they will pay for themselves in absolutely no time. And that means that from the point view of governments, from the national point of view, it would be economically suicidal to let people get sick when they get old. It would only make sense financial mercenary economic sense for the federal government to actually make sure that everybody who is old enough to need these therapies does have an access to them and will therefore continue to contribute wealth to society.

Cat: We're feeling healthy and we're working longer. But we have automation and artificial intelligence. So what types of jobs will be available?

Aubrey: I used to work in artificial intelligence (before biologist) and the reason I did so was very much the same reason I now work in aging. It was humanitarian. I felt it was extremely miserable that people had to spend so much of their time doing things they wouldn't do unless they were being paid for it. And the fix for that is of course automation. So you're absolutely right to suggest that the advance of automation that we're seeing right now is very likely within relatively short amount of time to result in the pretty virtual elimination of most of the jobs that we know as jobs today. And of course we've seen this before. We saw it with the industrial revelation which resulted in the decimation of the size of the workforce in manufacturing and agriculture, but then the idea of maintaining a full, 40 year career with a 40 hour working week was perpetuated because we invented new jobs. We invented this thing called the service sector which of course expanded as the manufacturing and agriculture sectors contracted. But I don't think that's gonna happen this time around because this time around as the service sector gets automated to oblivion, you know, what have we got left? What sort of sector is there, how many people do you need in the entertainment industry you know. So I think we really are going to bite the bullet that we kind of dodged when the industrial revelation happened. We're going to have to find a different way to distribute wealth and to equitably share out the remaining things that do actually have to be done.

Cat: Do your donors get first dibs to be study participants?

Aubrey: We are perfectly happy to try to help people who help us. But you have to remember that we are doing the biomedical research, the early stage of biomedical research to develop these therapies. And as the therapies move through the pre-clinical and clinical stages and more and more money is required, the chances are very good that

we will have to relinquish a lot of the control over this intellectual property, even indications where we have some of that control early on. So it's not really, we're not really in a position to commit to anything like that.

Cat: Okay.

Aubrey: But also I want to say if I were a wealthy individual and I were paying for this research it wouldn't be because I wanted to be first. It would be because I wanted it to happen soon enough. Nobody really wants to be first in line for any experimental treatment.

Cat: Right. Why do you think calorie restriction isn't effective for humans for life extension?

Aubrey: The ineffectiveness or the relative ineffectiveness of calorie restriction in humans or indeed in other long lived species like monkeys is actually not a surprise. It's something that is very straight forwardly predicted from evolution. Essentially it just come from the very simple and obvious fact that long famines in nature happen more rarely than short famines. Now that matters because the frequency with which a particular environmental situation occurs determines the extent of which evolution is motivated to develop genetic machinery to be optimized for that environmental situation. If something only happens once in a million years than evolution just dust doesn't care enough to develop machinery to deal with it.

Cat: That makes sense.

Aubrey: It's rather like the situation we have with Vitamin C. So Vitamin C is of course a vitamin, we have to actually consume it in our diet in order to get along. But most mammals don't have that problem. Most mammals make their own vitamin C internally in their own cell. And the reason we don't, and the reason also why two other families of mammals, namely guinea pigs and fruit bats...

Cat: They don't either?

Aubrey: That's right. The reason is believed is agreed to be simply that in our evolutionary ancestry there was a time when there was so much Vitamin C in our diet all the time reliably that there was no selection, no evolutionary pressure, to maintain the machinery to make it ourselves. So the machinery mutated into an inactive state and by the time the Vitamin C supply became less reliable again it was too late.

Cat: Some of your critics have, unsuccessfully, gone out of their way to try to discredit you. This was about 11 years ago. They even offered a prize of $20,000 if someone could prove you wrong. Why would they do this?

Aubrey: Well actually it's not quite that simple. The $20,000 that was put forward, was actually half of it from the magazine that orchestrated the prize, MIT Technology Review, the other half came from us, because we wanted to actually smoke out the opposition.

Cat: That's awesome.

Aubrey: It was to solve a rather unfortunate situation that had risen over the previous couple of years, namely that a lot of people like you, all the journalists were coming to the more established grandees of the field and they were asking these grandees about my work and asking whether my conclusions in terms of people's potential longevity had any basis in fact or in legitimate hypothesis. And they didn't want to know about it because they didn't know my work well enough to be able to make a cogent case one way or the other so they thought that the simplest way I can get the journalists off the phone was to engage in off the record ridicule. Because it was off the record of course I didn't have the opportunity to rebut any of it. So essentially what I did, both with this prize and also simultaneously in the academic literature around the same year, I was able to essentially pick a fight with my more vocal critics

and get them to set out in print the details, the scientific details of why they thought that this idea of SENS was unscientific so that I was able of course very easily to write very comprehensive rebuttals and the result was exactly as hoped. It took a while of course for the dust to settle but eventually people began increasingly to acknowledge that what I was saying did make a good deal of sense. And the result now is that people are even re-inventing these ideas and calling them their own, which is, you know..

Cat: It's so true.

Aubrey: Imitation is the sincerest form of flattering and all that.

Cat: They couldn't poke holes in your rebuttals and then your book, Ending Aging, came out and now all the things that you said then, they're trying to do now. So, that's pretty awesome.

Aubrey: It is.

Cat: Calico is the Google backed company that's tackling aging. And they claim to be funded for long term research. You're obviously extremely intelligent and passionate. Why do you think they're so secretive and what are your thoughts about why they haven't brought you onboard yet?

Aubrey: Well the answer to the question about why they're so secretive is not something that I'm really in a position to guess. What I can say is you know, they're a company and they're being run by a guy who made his name running another company, Genentech. You know they have a corporate attitude to information flow, I think it's pretty normal to be secretive when you run things this way and you want to make money. Now the curiosity of that of course is that as you say they've already got all the money they could possibly need from Google.

Cat: Right.

Aubrey: So it kind of makes no sense but on the other hand they have very clearly positioned themselves so that some of their effort, a lot of their effort in fact, is actually geared towards actually making money fairly soon; they've done deals with other biotech companies including some pretty large ones that are likely to be quite profitable within the next few years. Now everybody recognizes that those deals, those projects that are collaborations with the rest of big pharma and such like are not the ones that are going to defeat aging. And that the other part of their work which is really targeted to defeating aging is somehow being protected, being insulated from the pressure or whatever by this kind of approach. Fine so far. The difficulty, which comes to the second part of your question about why they aren't working with us is that the real science part, the part where they're really trying to tackle aging is being dominated as far as we can tell, and again because they're so secretive we can only infer this from basically who they hire, is they're dominated by basic scientists, by which I mean by people who are interested in understanding nature and are good at understanding nature but are not so good at manipulating nature and the consequence of that understanding. They're not technologists so much.

Cat: Okay.

Aubrey: This is a real problem because of course we believe that actually aging is sufficiently understood at this point, that it is possible to engage in the design and implementation of methods that will comprehensively postpone aging. And it's very frustrating that the people at Calico, especially the decision makers at Calico appear to have decided that no, that's not true, and that more exploration and hypothesis testing and so on needs to occur before any kind of actual technological exploitation can be contemplated. It's a great shame. I have of course spoken somewhat to Calico. I and a couple of other people here went there a couple of years ago shortly after they

got going and gave a long presentation of all of our work and they were terribly polite but they didn't call us back shall we say.

Cat: That's so frustrating. Were there any correlations in the habits of the super centenarian population?

Aubrey: Not really. Super centenarians are extremely fascinating, no question about that.Any exceptional group in any sense is what is studied. Super centenarians of course constitute a very, very tiny proportion of the population that have lived longer than anybody else-- the definition is an age of 110. There are only about a couple of thousand of such people on record which is not very many at all. But when you ask about the habits or anything else for that matter like lifestyle, diet, genetics, whatever, there really isn't very much in common. I mean it won't surprise you to learn that Japan is over represented in super centenarians simply because Japan is the country with the longest life expectancy. But only by a couple of years which leads to a relatively small factor, relative to other countries. So the question then is what can we learn from super centenarians? The difficulty is that first of all no, they don't seem to have very much in common except for one thing, which is they seem to be very good at, and this applies down the list a little bit, down to centenarians as well. They seem to be very good at handling stress. They haven't necessarily had a particularly stress free life. But the thing is nothing bothers them. When enduring kind of stressful situations they cope with them very well. And so that kind of makes sense, we know that stress is bad for you, it elevates the level of certain hormones, that accelerate the creation of damage. However the best way, the most informative way to study super centenarians is very probably to study not them themselves but rather their offspring who are of course genetically very closely related. The idea there is that then you can compare the offspring with other people of the same age. There's no point really in comparing the health or functionality of a super centenarian with someone who's 80 because the

super centenarian is very impaired in their function both mental and physical.

Cat: Sure.

Aubrey: They're about to die, after all. But the other thing that's special about them is that they were born a very long time ago. If you study their offspring and you compare those offspring with people of the same age then you're factoring out all of that problem.

Cat: So other people are now working on the problem of aging. And there's been recent patents around removing the intra and extracellular junk and the cross linking, will the patents interfere with your work at all?

Aubrey: Well first of all we ourselves are taking out IP, taking out patent protection on some of the work that we're funding. But also one thing that is quite useful is that we are seen as very much the nexus of this rejuvenation biotechnology, by near fellows and the people who know everybody. So we often have very good, a few different collaborations not just with other groups but even bringing other groups together with each other so as to minimize the difficulty of competition and trade secrets and such like. The patent situation in some of these areas is stronger than in others. So for example with extracellular waste products, such as amyloid in the brain of Alzheimer's patients, there's been quite a lot of work done, lots of money has gone into the developmental of antibodies for example, they're able to remove this stuff. And you know, we don't expect that there's any real need for us to get involved. But there are other types of garbage like another type of amyloid that accumulates in the heart in older people where actually the group that is doing the most promising work is the group that we funded and we do have an equity position in the company that spun out doing that work. It all depends on the specifics of the individual project but the short answer is no, we're not too worried.

Cat: What can we do if we want to live longer?

Aubrey: I'm afraid that I only have one answer to that question and it's not a very pleasant answer. The answer is all you can do is give SENS research foundation large amounts of money or possibly persuade somebody else to do so. Because at the moment we simply don't have any medical interventions or lifestyle or diet or other interventions that are available that can substantially postpone the ill health of old age. Therefore for any particular individual the only thing that they can do is to change the other end of the equation. In other words bring forward the arrival of therapies that do much more than anything that exists today. And that of course does require financial resources. One of the most important things I always have to emphasize about this work is that out of the three problems that I had to solve when I first entered the field 20 years ago, two of them were solved pretty quickly. Number 1, after only about 5 years I figured out the SENS plan, the idea of damage repair being a more practical approach than inhibiting the creation of damage by the body. And then one being the personnel, getting world leading scientists in all the various relevant disciplines on the side, getting them enthusiastic about doing all of this work, that has meant that at least for the past decade, the only problem that remained is the lack of resources with which to let those scientists get on with the job.

Aubrey: I take it you want me to open this box.

Cat: Yes.

Aubrey: Hope I can keep the skull. Alright well this is one of the best beers known to man.

Cat: So this is Pliny the Elder, and it's kind of a famous cult following microbrew in Northern California and the "Elder" is kind of pun intended. But I wanted to ask is it true that you drink 3-4 pints of beer a day?

Aubrey: I suppose that's a pretty good average.

Cat: How many hours do you sleep?

Aubrey: I actually don't get enough sleep. This is the one big thing that I do that is probably bad for my health and my longevity. I figure it's kind of a good trade off because I'm bringing forward the defeat of aging by my work and maybe I'm shortening my life but not by such an amount as the amount that I'm hastening the defeat of aging so it's a net win if you like.

Cat: You've got a lot of confidence that you're going to pull this off.

Aubrey: It's all probabilities but yeah I mean I don't have enough sleep that's for sure.

Cat: Do you have hobbies?

Aubrey: I used to have hobbies. I've pretty much given hobbies up. Many years ago I used to play competitively a board game called Othello which is actually not very well known in the West. It's quite well known in Japan where it's quite popular. It's very, very simple rules and it's got a very simple strategy. There are world championships in it, has been for 40 years almost. But yeah I used to be the Chairman of the British Othello Federation. I used to be play it quite a bit. But I haven't been able to do so for at least a decade now.

Cat: That's entrepreneurship.

Cat: You married pretty young to an older woman who was already an established biologist and geneticist. Was love an impetus to get up to speed in your field?

Aubrey: A lot of people have suggested that maybe I got into this business because my wife is 18 years older than me and I wanted to save her life so to speak. And of

course I would not object to saving her life, that would be very fine. But, no, that is absolutely not what gets me out of bed in the morning or whatever has. In fact saving my own life has not been the thing to get me out of bed in the morning. What really energizes and drives me is purely the humanitarian aspect-- the fact that I'm gonna be saving so many lives every time I bring the defeat of aging even one day forward—that's 100,000 lives. It's pretty easy to get excited about that.

Cat: Yeah it seems your pursuit of something important makes you happy. Millennials can respect and relate to the fact that you're not motivated by money. You could have had a luxurious life with your mother's inheritance but you assigned $13.5 Million to your SENS Foundation. How did she amass her fortune?

Aubrey: So my mother was I guess both very wise and very lucky. She inherited from her parents a small amount of money about 30,000 pounds which was you know, a respectful amount back then in the 50's and 60's. But still not a ridiculous amount, but she invested almost all of it in two houses in Chelsea, in central London. So the amount of income that she had and that we lived on thereafter was pretty limited because very little was left after the investment in the houses. But the result was in the end extremely fortunate because that area of London continued to appreciate, the property prices right through the rest of her life. And the result was that when she died in 2011 and we sold the houses, one of them we had, which originally cost 6 and a half thousand sold for 3 million, and the other one which was originally 23,000 sold for nearly 8 million, and that's all in pounds. So yes, so the result was I inherited I think we came to a total of 17 million and as you say I was able to donate 13 and a half million of that to the foundation. It was complicated because my mother of course was British and the foundation is a US charity so it has to be done by an affiliate charity that we created in the UK but it all worked out.

Cat: That must have been a pain.

Aubrey: Well it was okay, it was worth it. Of course we knew in advance of my mother's death what was going to be needed so all of it was in place by the time that happened. And I hold on a little bit so I did, I was able to buy myself a nice property in the Santa Cruz mountains, which is as far as I'm concerned, all I really need.

Cat: You've made such visible progress in your field. Has your father ever tried to contact you?

Aubrey: No.

Cat: You signed up to do cryopreservation with Alcor. Can you tell us about that?

Aubrey: Sure. So I think that pretty much the only tragedy in the world that compares in severity with the hesitance of humanity to embrace and pursue the anti-aging mission more aggressively is the reluctance of humanity to embrace cryonics more aggressively. Because cryonics is a massively valuable component of future healthcare. And therefore of today's healthcare, if only it were understood what cryonics really is then I believe that first of all, the actual quality of cryopreservation that can be achieved will be far higher now than what actually exists because more investment would have gone into the research. And secondly, that vast numbers of people would be cryopreserved. What we have to remember about cryopreservation is that it is just healthcare. We take someone who has just become legally dead and we learn to lower the nitrogen temperatures so that they don't become any more dead than they were before. The idea of someone being partly dead of course is the problem here because most people, society in general doesn't like to think that way. They like to think of people being either alive or dead and nothing in between. But that is that is biologically nonsense. Death is absolutely not a process that happens instantaneously. It's a process of steady and

certainly accelerating decay in the body, accumulation of damage. That rate of accumulation accelerates very sharply at the point that we stop breathing and our heart stops beating. But it's still only finite. So if you take someone who has just undergone cardiac arrest and may even have not any brain function anymore but still they don't have very much more damage than they had previously when their heart was still beating. If you can stop any further damage from accumulating which is what freezing someone does than they're still in a state where there's a substantial probability that they can be revived from that state using the benefit of medicine in the future that is first of all better at keeping people healthy with minimal amount of damage and second of all capable of repairing the damage and getting them back to their truly healthy, youthful state. Now the catch in all of this that everyone is quite well aware of, of course, is that the process of cryopreservation itself creates damage. Take someone who has just become legally dead but they're warm and you cool them down, if you cool them below freezing, they're going to become, they're going to crystallize and that's gonna rip the tissue apart. And it's extraordinary to me that still hardly anyone realizes that this problem was completely solved, more than 20 years ago, with the development of a rather elaborate cocktail of cryoprotectants that essentially stop this from happening at all. The body doesn't form crystals it forms a glass, it can amorphousize and that's why it's called vitrification. There are further advances that are certainly needed in to eliminate even other types of damage from cryopreservation but even with the current situation people who are cryopreserved in the best possible state have, in my view, a substantial chance of being revived. And another thing about cryobionics is cost. People always think, oh my God, Alcor charged $200,000 to cryopreserve someone, we think oh well that means it's only for wealthy people. But that's nonsense too and the reason it's nonsense is because society doesn't understand death. Society doesn't understand death. So when someone is pronounced legally dead their life insurance can pay out, even if the cryonics company and

the patient before they were legally dead, don't think they're dead, and they just were in a coma effectively. So life insurance pays out which means that if you have a life insurance policy, whose payout is let's say $200,000, then if you start it when you're in your 20's or 30's or 40's the monthly premium is going to be perfectly manageable just like any other life insurance policy, the only difference from the regular insurance policy is who the beneficiary is, rather that the beneficiary being the next of kin, it's Alcor, or whichever.

Cat: That's fascinating.

Aubrey: So it's really very affordable and it's a scandal that even though people like myself and the people who run Alcor and so on have been telling journalists this since forever, nevertheless it still isn't getting through to people's consciousness.

Cat: Do you believe we have eternal non-physical souls?
Aubrey: I have no idea whether we have any kind of non-physical attribute to the body. However what I will say is that if we do then very clearly it is intimately associated with the physical body for as long as the physical body keeps functioning, and therefore it doesn't really matter. We want the physical body to keep functioning and if there's any soul around that's inside it or tied to it in some way, then so what, really.

Cat: Until recently your beard had a Facebook fan page. If the reserve was set really, really high, would you consider auctioning your beard to raise money for SENS?

Aubrey: I have indeed been on the record for some time as saying that a million dollars will buy you my beard.

Cat: What about Peter Thiel?

Aubrey: Peter Thiel is awesome. He is one of very, very few wealthy individuals who are not only visionary but are also

rational enough to understand that not everything can be driven by capitalism. So he definitely likes making money and he's done very well investing in innovative technology but he's also donated very substantially to our research and to other research that was at a pre-competitive stage.

Cat: He is awesome. What about Ray Kurzweil?

Aubrey: Ray Kurzweil is pretty awesome too. He's definitely been the kind of thinker who has been able to see well outside of the box and he's invented a number of technologies that have been very successful. So he's definitely very much earned his national medal of engineering or whatever it was that he won a number of years ago. His work in longevity is also perfectly reasonable by in large. I believe that he is somewhat over optimistic when it comes to what we can do today using just vitamins and supplements and such like to postpone aging.

Cat: Yeah. He takes a lot.

Aubrey: But his attitude to future technology is pretty much spot on. He believes just like me that the next couple of decades will see the development of really powerful regenerative medicine, rejuvenation biotechnology. And his longer term vision of the use of nanotechnology, in particular a variety of different non-biological solutions to the medical problems, all of that is also very reasonable. It's not an area I'm an expert on so I don't want to say that whether it's right or wrong but it's not crazy.

Cat: What about Dr. Craig Venter?

Aubrey: So Craig is another guy who's achieved a great deal by not being scared to be different. He has of course been extremely prominent in biomedical technology for quite a long time. He was responsible for sequencing the human genome and more recently he has taken that technology, high-throughput sequencing, into a variety of other areas including environmental protection and

such like. Of course at the moment, his main claim to fame that's relevant to our work is that he along with, Peter Diamandis, a great friend of ours, started a company called Human Longevity Incorporated which is, well as the name suggests, interested in doing something about aging.Now the general value proposition of that company as it currently seems is somewhat short-termists-- they're more focused really on using genomic information to optimize personalized medicine, but as time goes on I'm quite sure that Craig will, because he is ultimately a really heroic pioneer, will be at the forefront of developing other technologies that will help make a big difference.

Cat: What do you think about Modern Meadow?

Aubrey: Modern Meadow is a great company trying to develop artificial meat that is good enough to eat and therefore can significantly reduce expenditure on agriculture and of course the pollution that comes with that. Modern Meadow is actually a spin out from a company named Organovo which is interested in the rather more difficult problem of developing 3D printed organs. The reason it's a more difficult problem is because with meat you don't need the circulation to work, the vasculature, which is the main physics problem, the main real material science problem with creating organs. However, I feel that both of these companies are very palatable, in fact we, in our previous forum at the Methuselah Foundation were the first investor in Organovo.

Cat: What do you think about eliminating sugar from the diet?

Aubrey: I really don't think that there's enough evidence to suggest that completely eliminating sugar is a particularly good idea. I'm not saying it's a bad idea. I just think that either way the difference that you're going to get is relatively mild. Of course we are not comparing normal sugar use to high sugar use, excessive sugar use. Excess of anything is definitely bad for you. But going from normal

amount of sugar to zero sugar, I think it's rather like any other area of dietary manipulation. We see sometimes we see mild beneficial effects in some people. You don't know whether you're gonna respond because every person is different. So you know, for me it's not really, it's not the real McCoy. It doesn't really rise to the level that we are interested in.

Cat: What about vegan, raw food or ketogentic diets?

Aubrey: Really I have exactly the same thing to say about all of these other dietary approaches that I do to, with regard to sugar. I think that any diet that is reasonably balanced in terms of its nutrient intake, both micronutrients and macronutrients is gonna be okay. You can always have too much of a good thing, so one mustn't get overweight or anything, but the difference between one reasonable diet and another reasonable diet is very unlikely to be enough to get us interested.

Cat: Okay.

Aubrey: We at SENS Research Foundation are all about doing things that will postpone the ill health of old age by a far greater amount than anything we can do today.

Cat: What about drinking deuterated water?

Aubrey: Deuterated water is very unclear as to whether it's got any interest. But since you mentioned deuterated water what I would like to mention is an alternative use of deuterium which seems to be very beneficial and indeed the benefits that it seems to confer make a lot of biological sense. So I believe this both theoretically and practically. The idea that is being pursued by a company actually local to here called Retrotope is to create versions of fats that have deuterium replacing the hydrogen atoms in certain judiciously chosen places in the molecule. And the purpose of this is to slow down the rate at which these fats actually react with free radicals, as I mentioned

earlier. Do that, then because of some of the details of how free radicals react with fats and how fats then react with each other, because of the details, it turns out that even a relatively small amount of deuterium incorporated in these judicious locations in molecules is enough to make a big impact on the oxidative stress of the organism on how much free radical damage occurs. And eventually that includes damage to DNA, which I mentioned earlier. So this is a radical and very innovative approach to dietary manipulation to postpone aging. It involves a lot of chemical sophistication as I've already indicated. But I believe that it definitely has a lot of merit.

Cat: What about Omega 6, Vitamin E, and other anti-antioxidants?

Aubrey: Vitamin E is a bit of a mixed bag and this has been understood for a long time. The thing about Vitamin E is that it transfers electrons from one place to another. In particular it's what's called a lipid soluble antioxidant which means that it can protect fats from arterialization I was describing a moment ago. But a given bit of a Vitamin E molecule can only do that once and then it has to be recycled by reacting with Vitamin C. Now Vitamin C is, it's not a lipid soluble antioxidant, it's a water soluble one which means that the rates for these reactions between Vitamin C and Vitamin E and between Vitamin E and the fats are dependent on details of where the molecules are. For example if the Vitamin E is in a membrane which is a very flat thin surface then it may be able to get greater rather rapid access to Vitamin C than if the Vitamin E molecule is in a kind of a spherical globule of fat, like a lipoprotein in the bloodstream. And this means that sometimes Vitamin E can actually not be an antioxidant at all but a pro-oxidant. Now you mentioned earlier, 6 and Omega 3 fatty acids, this is again part of what I was saying a moment ago about deuterium in fat. It turns out that different fatty acids have very different roles to play and very different susceptibilities to oxidation and therefore to contributing to oxidative damage throughout the body.

Cat: What about liposuction of abdominal fat? They say that's the most problematic fat.

Aubrey: So abdominal fat is indeed often mentioned as a big problem because a lot of it gets into a state where it secretes toxic molecules that will accelerate aging in other places. And there was some very exciting work done in New York probably as long as 15 years ago now showing that removal, surgical removal of that kind of fat from rats perhaps were diabetic could almost instantly relieve the diabetes. So that was pretty exciting and that kind of work has proceeded. People of course have looked at ways in which the fat can be removed non-surgically. But there's still plenty of interest in this area. It's got promise. And there's another thing about removal of abdominal fat that I should probably highlight as well which is that the abdominal fat you remove doesn't only contain fat cells, it also contains fat stem cells. Those stem cells can in today's world what we understand about stem cells, stem cells can be manipulated and turned into other types of cells and reincorporated into the body in beneficial ways.

Cat: Yeah they're starting to do that a lot. What do you think about exercise?

Aubrey: Exercise is a bit like diet really. You better have enough of it but having more than enough probably isn't gonna help you very much.

Cat: Hyperbaric oxygen therapy.

Aubrey: So people have been playing around with the oxygen pressure and indeed oxygen concentration in what you breathe for quite some time and there is some tantalizing data out there showing that it may have beneficial effects of a variety of types. I think tantalizing because I don't think the date are conclusive yet. There's plenty more to do but luckily enough people are tantalized that that data continues to accumulate. And so I think we will progressively learn more about this, again though,

I would bet good money that it is not going to be the fountain of youth.

Cat: What about Metformin increasing lifespan?

Aubrey: The Metformin is originally an anti-diabetes drug and of course diabetes is a prevalent disease of old age- - type 2 diabetes. So we would expect that there would be some benefit but the reason you bring it up of course is that there have been a number of experiments in the laboratory that have indicated that Metformin has a more widespread effect on processing the damage accumulation in aging than nearly the anti-diabetic effect. And the result is at this point that a number of researchers including incidentally the guy who did the work I mentioned on getting rid of fat from rats earlier. I have got together and have actually successfully petitioned the FDA to allow for a clinical trial of Metformin against aging. Now this is a real breakthrough, the first of its kind, because until now the FDA were unwilling to recognize aging as a condition for which there could be clinical trials and approval of a drug or anything like that. And in this case, this is really just a proof of concept firstly because Metformin is of course a very old molecule and no one's gonna make money out of finding that Metformin works against aging. And secondly because, to be honest, I don't think Metformin is going to have much effect in terms of overall impact on...

Cat: It's negligible.

Aubrey: That's right. I could be wrong about that so glad the trials are being done but the big thing here is that the negotiations between the researchers and the FDA have resulted in a definition of aging that everybody can work with. Essentially it's all about multi-morbidity, all about having more than one defined thing wrong with you at the same time. And the design of the clinical trial therefore is absolutely certain to be copied again and again and again for other drugs now that the precedent has already been set and now that drug companies can make money out of

a drug that actually achieves these ends.

Cat: Liz Parrish.

Aubrey: I know Liz very well and I wouldn't do myself what she's doing but that doesn't mean I think that what she's doing is wrong. She has taken good scientific advice from a number of people including myself and she has decided to volunteer as patient 0 to receive gene therapy for a couple of genes that may postpone aspects of old age. Now the results of that are of course difficult to evaluate for a variety of reasons. Number one it's only one person. Number two she's actually in the prime of health. She's in her forties and most people would not guess that she was as old as that, so it's unclear whether the can be any real performance indicators. However that still allows for the possibility of tentative preliminary indications at the level of blood work for example in terms of what effects these genes might have. And importantly the effect is supposed to be rejuvenative. It's not just supposed to be slowing aging down. So the effects may therefore show up quite quickly if they exist at all. So along further is you know, I mean she's not trying to make money out of this. She's just experimenting on herself and self- experimentation has a long and distinguished history in medicine. So you know, it's really her choice and I am very pleased that she's not being given too hard a time for it. Certainly her choice to do this and the publicity of it has attracted a lot of controversy but it hasn't been particularly vicious.

Cat: What about Gensight?

Aubrey: Gensight are a very interesting French company who are doing a few things but the main thing that's relevant to our work is that they are trying to do something called allotopic expression which is the copying of or the insertion of back-up copies of the mitochondrial DNA into the nuclear DNA. The idea here is that 13 proteins which are occurring in the mitrochondrial DNA are essential components of the mitochondria but there are more than

a thousand proteins that are also essential components of the mitochondria and they are encoded by nuclear genes naturally. So if we can kind of co-op the same machinery that transports the nuclear coded proteins back into the mitochondrion then what we would have is a system where it wouldn't matter if you got mutations in the mitochondrion. And that's really useful because mitochondrial DNA turns out to be vastly more prone to mutation than nuclear DNA. Now Gensight are pursuing one particular application of this, which is a type of blindness called Leber's Hereditary Optic Neuropathy and the reason they're doing this is because unlike the mitochondrial damage that you see in normal aging the damage that is the cause of this particular neuropathy is just a super mutation in one of us those 13 genes. So they're trying to put one of these backup copies in to correct that and they have pretty good initial data well good enough to get investment anyway.

Cat: Yeah.

Aubrey: But the reason we're interested in this of course is because we're trying to do exactly the same thing, though we're trying to do it for all 13 of these proteins because that's what you need to do in order to have a good effect in normal aging as opposed to this one type of blindness.

Cat: I have some questions about Dr. Aubrey de Grey as an entrepreneur. You started off in computer science. If somebody would have told you: You're going to be the first to work on the problem of aging, you'll be a famous author and pioneer and you'll be solving the hardest and underfunded problems, you're going to map a concerted set of therapies... did you feel back then like you know, I'm that guy, I could be that guy when you started?"

Aubrey: Well only in part. I mean when I was in my twenties, my early twenties or my teens and I was making career decisions I had given up biology already. I only went back into biology after I met my wife. So I definitely never

thought that I would go into biology per se but I certainly did think that I would wanted to spend my life trying to change the world, where could I make a big difference? And I went into artificial intelligence research simply because I found, at the age of maybe fifteen, sixteen, that I was a pretty good programmer. And so I thought well this is an area where my talents can be applied for good humanitarian benefit. Then the progress I made when I was doing that was really very specialized—I was working in a particular area called software verification. However, you know, it was an important area and I certainly had a strong belief that my work there could translate and generalize into full blown artificial intelligence of one kind or another in the long run. But more to the point it also taught me that I was not just good at programming, I was also good at working on really hard problems, breaking them down and figuring out how to solve them. So when I started to talk to my wife and other biologists about aging at the age of about twenty-seven, twenty-eight, twenty-nine, it wasn't too much of a struggle for me to start to realize that maybe this was an even more important target than the problem of tedium and having to work on all these things that we don't really want to do and the fact that I knew I was good at working on hard problems was a big start. So by the time I switched, most of what you just said really was true. I felt that I have a respectable chance of making a big contribution if I switched field.

Cat: Wow. You founded your first company within a year after undergrad. Is that true?

Aubrey: Kinda yeah. So Man-made Minions, which was the company you're talking about that was the company within which I pursued this software verification work. And but it's not really fair to say that I founded my own company. First of all I co-founded it with another guy from the same company where I was working in that intervening a year. And that other guy with whom I worked was 2 years older than me, 2 years more experienced, generally he was the person who really founded the company. And I was really

the back room guy doing research. Second thing I want to mention is that this company wasn't selling anything. It was a company that was really a company only in name with my colleague going out and doing contract programming work that paid the bills and me just doing full-time research. So I think it would be misleading to say that I founded my own company.

Cat: That was very honest answer. How did you get a publisher in 2007 for Ending Aging?

Aubrey: It wasn't too hard actually to get Ending Aging published. The reason it wasn't too hard was because I went to TED. TED was not quite so glitzy as it is now back then. They didn't even have TED Talks let alone TED X. But it was still pretty glitzy, and I was invited as speaker in 2006 and of course the book was in gestation by then, it was largely written and one of the people I met there was a very famous literary agent named John Brockman who has handled books for a general audience for pretty much every scientist you can think of who has written a popular book. And he, having seen my talk, and having talked to a few other people, he didn't have too much difficulty persuading himself that this book was going to be worth taking on. So once Brockman's on board, the rest is easy. It's all downhill from there. So Brockman found us St. Martin's Press, which took on the actual publication.

Cat: You have a 25 member advisory board and a ton of very talented people working for you. How do you onboard reputable people?

Aubrey: The main way in which one gets scientists to associate themselves with a particular project, whether it's senior scientists who are working with advisors or professors or whether it's community people who are doing the actual grunt work at the bench, the main way you get good people is by doing good work. And that means, that comes down to two different things. First of all it's what you've already done, the track record of grants

and publications you put out and so on. And second it's just how it feels, how legitimate, how exciting it feels. You can look at, someone would look at our work for example and will say oh a lot of this seems very speculative, but the more I get into it the more they see that any initial skepticism they might have about the feasibility of this or that, actually we've already thought of their objection and we've got a good answer, and the more times you get around that cycle the more your confidence increases that actually the objections are invalid and the excitement is what's valid.

Cat: In light of what happened with Theranos, are the SENS studies published and peer reviewed?

Aubrey: We do publish our work in the peer reviewed literature and we think that's very important. We think that credibility of our work needs to extend across all audiences and there are many scientists who simply aren't going to pay really good attention to any work unless it's been published in peer reviewed academic literature, which is fine. What we don't do is submit to what's often called the publish or perish paradigm. In other words essentially the philosophy of publishing as much as we possibly can and indeed choosing what work we do in order to be able to publish as much as possible. This is very, very prevalent in academia, in fact I would say that it's absolutely ubiquitous in academia, simply because it's the only way for people to distinguish themselves in competitions for grants and promotions and tenure and so on. But it's immensely damaging to the long term progress of science because it means that people aren't actually working on the most important problems, they're only working on the easiest problems so they can get publications quickly. So we do publish but we don't publish nearly as much as a grip of our size of our total budget would publish if we were doing things the normal academic way.

Cat: What's the best and worst part of your job?

Aubrey: The best part of my job is the achievements. Every time that we make a breakthrough, we get something working, I know that we are one step closer to ending the biggest problem of humanity. And also even when people come to me and they say that I've inspired them to move into the field for example or it works out financially or anything like that, again it's a sense of achievement. The fact that I really am being given external validation, that I really am making a difference that I wanted to make. The worst thing about the job-- hard to say. I mean I feel very fulfilled by the job. The fact that I've made such a difference already and the fact that I continue to make a difference of course is huge. I guess the worst part of the job really is the frustration that it's not going faster. The frustration that despite my best efforts, and spending an awful lot of time on camera and on stage trying to get the word out, trying to explain to people that this is both feasible and desirable, nevertheless it remains a really hard sell.

Cat: You have an operating budget of about $4 million a year. How does the research break down- what costs the most?

Aubrey: At the moment our budget which, as you say is about 4 million a year, is mostly on research itself. We also have a minority that is spent on outreach, on you know, conferences for example and then a minority that is spent on education, on funding internships in this facility and also in various extramural labs that we support. The research breaks down into the obvious things really, personnel, salaries and reagents and consumables and equipment and that varies enormously from one project to another, there's no single path.

Cat: Do you have any regrets?

Aubrey: Honestly I don't have any significant regrets with regard to how this whole process has gone. I don't even regret the fact that I started out in one field and then didn't

discover the utility that I might have in the anti-aging effort until relatively late in my life. I don't regret that because I think that my work in artificial intelligence per se equipped me very well to make more of a difference than I could have otherwise have done after I switched fields. With regard to how I've actually conducted this whole thing going about learning and thinking and coming up with ideas and getting the ideas out there and so on. Again I don't really look back and say to myself that I really made a bad mistake here or there. I think by in large any mistakes I've made have been pretty minor.

Cat: What's the most important thing a human can do in this lifetime?

Aubrey: So that is probably the most unanswerable question it's possible to ask because for sure in certain what's important is a value judgement and what any given human can do varies very much from one human to the next. So it seems to me that the main thing, the main way to think about that question is to think about how many things we can do in a lifetime which of course is somewhat limited by how long our lifetime is, and that's what we're trying to fix.