# VITALITY *LIVE TO 100* **CanWe Live Forever?**

### FUTURIST AUBREY DE GREY SAYS SO BY VÉRONIQUE MORIN

IN THE LAST CENTURY, we have increased our life expectancy by around 30 years. But that is nothing compared to what we may soon be able to achieve, according to one of the most controversial scientists in the field of research into aging. Aubrey de Grey, 48, is the author of Ending *Aging: The Rejuvenation Breakthroughs That Could Reverse* Human Aging in Our Lifetime and a computer scientistturned-biogerontologist, with a Ph. D. from Cambridge. He has undertaken a quest to "cure" aging altogether, which potentially means attaining nothing less than immortality.

It may seem borderline crazy, but de Grey has been seriously pursuing his scientific dream for the past 15 years, and he gets annoyed when anyone calls it a theory. "It is not a theory," he says. "It is a very real biological probability."

When we speak, it's through cyber-space but face-to-face via Skype. De Grey is at his research centre, SENS Foundation, in Mountain View, Calif. He appears on the computer screen sporting his trademark beard – often likened to Methuselah's. "I have been growing it for 16 years. My wife would kill me if I was to cut it, which would defeat the purpose of my research," de Grey says with a gentle smile.

## *"immortality"?*

AUBREY DE GREY: I don't really like this work to be described as immortality. It is a word that has too many connotations. We are not really curing death. We are only curing one cause of it, the most important one, granted. But people are still going to be able to die from being hit by a truck. So what gave me the idea of defeating aging? I thought that people were not really working hard on it. I grew up presuming that, for biologists, aging was the No. 1 problem facing humanity. It seemed obvious to me. It was only when I was 30 and a computer scientist that I began to find out that that was not the case. Especially after I married a biologist and discovered that aging was regarded as rather boring and not very important. I was horrified. Eventually, I just switched fields. I always knew that aging was bad and was potentially fixable by medicine. For me, the main motivation is humanitarian.

### **VM:** We have been dying forever. Explain why and how aging can be stopped.

**AG:** It's not exactly that it can be stopped. Aging is a side effect of being alive in the first place. It is an accumulation of various molecular and cellular damages that are

side effects of normal metabolic processes. We don't want to mess around with metabolism and stop the side effects from happening because that would be too hard. What we can do instead is repair those various types of damage by cleaning up after them, so that they do not accumulate to the level of abundance that causes the problems of disability and disease associated with old age.

### **VM:** *How do you intend to do that?*

AG: Some of the most important diseases of old age are caused by the accumulation of molecules that are created as by-products of normal metabolism but that the body does not have the machinery to break down. Those molecules accumulate in the cell and, eventually, they cause problems just because they get in the way. This is why we get cardiovascular disease and macular degeneration, the main cause of blindness in the elderly. I realized that we may be able to combat this problem by identifying bacteria that can break these substances down, and then identify the genes that they use for that purpose and then introduce those genes in our own cells. One of our research initiatives involves the identification of non-human genes **VÉRONIQUE MORIN:** What gave you the idea of pursuing – usually bacterial – that make enzymes that break down these substances in the body.

The other project that we are doing here involves mitochondria, part of the cell and an important part of metabolism. Mitochondria has its own DNA, and that DNA goes through mutations and affects functions of the mitochondria. Those mutations accumulate throughout life, and a lot of people think that this is an important part of disability and disease in old age. We are interested in not actually repairing the mutations, which is the most obvious thing – but we do not know how to do that – but making the mitochondrial DNA unnecessary. Basically, to make those mutations a harmless part of the nuclear genome in the nucleus of the cell. We need to make modifications to those genes to make them work within the nucleus, and this is one of the projects we are working on in our lab.

**VM:** Scientists have been trying to find the key to cancers for decades. How can you be so certain that they will find it soon?

AG: You are right. Cancer is a hard nut to crack. In fact, I would say that cancer is by far the hardest aspect of aging to fix. But I think we can be very confident that eventually we will develop technologies that  $\rightarrow$  *Continued on page 128* 



