

# EPPENDORF AWARD FOR YOUNG EUROPEAN INVESTIGATORS

Presented in partnership  
with **nature**



Kerri Smith and 2009 winner Óscar Fernández-Capetillo

## NEW DEADLINE FOR ENTRIES!

We invite biological and biomedical researchers not older than 35 years, working in Europe, to apply for the 2011 Eppendorf Award. There is a new deadline of 15th January 2011. The prize ceremony is also moving to the new EMBL Advanced Training Centre (ATC) in Heidelberg, Germany. To find out more visit [eppendorf.com/award](http://eppendorf.com/award).

The Eppendorf Award for Young European Investigators was established in 1995 to recognise outstanding work in biomedical science. It also provides the opportunity for young European researchers to showcase their work and communicate their research to the scientific audience. *Nature* is pleased to partner with Eppendorf to promote the award and celebrate the winner's work in print and online. Below, Podcast Editor Kerri Smith talks to the 2009 winner Óscar Fernández-Capetillo about his work, and how it felt to win the award. To listen to the full interview, visit [nature.com/nature/awards/eppendorf](http://nature.com/nature/awards/eppendorf)

***"...the DNA damage to which you are exposed in the womb can have a massive impact on your ageing rates later in life."***

**Kerri Smith:** How did it feel to find out that you'd won?

**Óscar Fernández-Capetillo:** It was impressive - you don't get to win that many awards, at least I don't! When I got the call, I didn't think it was related to the award, so my first intention was to hang up. But luckily I didn't, and then I was super happy - in the next five minutes I made about ten phone calls to friends and family to tell them about it.

***"... I believe we have now built up an independent original niche..."***

**KS:** Give me a sense of the work your lab does.

**OFC:** We work on how cells detect and signal DNA damage. When cells detect DNA damage, they stop growing, and they also start the repair machinery. We use the mouse as a model system to understand how cells detect damage, and the consequences of having an inefficient damage response. If you don't repair damage very well, that leads to mutations, which lead to cancer. There's also this concept that DNA damage is the cause of ageing. We play with different proteins involved in the damage response to find out how we age and whether we can design strategies that will delay ageing, and/or protect us from cancer.



Óscar Fernández-Capetillo received the Award from Kai Simons

## EPENDORF AND NATURE

Óscar Fernández-Capetillo is the fifteenth recipient of the Eppendorf Award for Young European Investigators, which recognizes talented young individuals working in the field of biomedical research in Europe. The Eppendorf Award is presented in partnership with *Nature*. The winner is selected by an independent jury of scientists under the chairmanship of Kai Simons (Max Planck Institute for Molecular Cell Biology and Genetics, Dresden, Germany). *Nature* and Eppendorf do not influence the selection. For more information see [eppendorf.com/award](http://eppendorf.com/award).

**"I've had quite a diverse career... I think that's one of the things the jury appreciated"**

**KS:** Worthy aims! Can you apply any of your findings to ageing in everyday life?

**OFC:** One of the things we've learnt about ageing is that the DNA damage to which you are exposed in the womb can have a massive impact on your ageing rates later in life. This is the concept of intra-uterine programming. The other thing we're doing is dedicating part of our research to drug development directed towards cancer treatment.

**KS:** What studies are you doing at the moment?

**OFC:** As I said, DNA damage leads to cancer and ageing. But what is not clear is the ultimate cause of this damage. Is it your telomeres shortening? Is it oxidative stress? One trend we are exploring is the role of replicative DNA damage. When a cell replicates, there's a lot of gymnastics in the DNA and it tends to break. We're studying the role

of a protein called ATR, which protects cells from DNA damage. We know that limiting ATR activity is bad for cancer cells because they replicate their DNA very fast and generate lots of replicative DNA damage. So maybe if we can eliminate a protein that is important to protect cells from replicative DNA damage, it'll be more toxic for cancer cells than for normal cells.

**KS:** So you can selectively kill cancer cells without harming normal cells?

**OFC:** Well - that's the idea.

**KS:** What do you think the jury particularly liked about your work?

**OFC:** I've had quite a diverse career. I started off doing immunology, then informatics, and then I jumped again to study DNA damage at the National Institutes of Health... then I jumped to my lab here and I almost completely changed focus...I believe we have now built up an independent original niche, developing these ideas on ATR and cancer and ageing, and I think that's one of the things they appreciated.



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