EPPENDORF AWARD FOR YOUNG EUROPEAN INVESTIGATORS

Presented in partnership with nature

Kerri Smith and 2008 winner Simon Boulton

Now in its fifteenth year, the Eppendorf Award for Young European Investigators is well established as giving prestigious recognition to a young scientist's early career. It also provides the opportunity for young European researchers to showcase their work by communicating to the *Nature* audience. *Nature* is pleased, in partnership with Eppendorf, to provide visibility to the winner in print and online. Below, podcast editor Kerri Smith talks to the 2008 winner Simon Boulton about his research and the effect that the award has had on his career. To listen to the full interview, visit nature.com/nature/awards/eppendorf.

"Maybe it's a little bold to say this, but I think our work provided a very important new piece in our understanding of how recombination is controlled."

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

Simon Boulton: I was shocked! I thought it was a joke initially. I expect most people feel the same way. As it turned out, I knew quite a lot about the award from my past supervisor, Steve Jackson, who was the first ever recipient of the award. So I knew the implications of this for my career.

KS: *I understand it gave Steve's career a real boost?*

SB: Yes, speaking to Steve, which I did after finding out that I was the recipient of this year's award, he told me how important it was to him in terms of making his name and his lab known throughout Europe — it really opened doors and provided many opportunities for him. It was, for him, a defining point in his career.

KS: *Give us a bit of background to your work.*

SB: So, we have an interest in DNA repair, primarily double-strand breaks where the DNA duplex is broken completely. This is a catastrophic event for cells — you need to be able to repair double-strand breaks correctly

EPPENDORF AND NATURE

Simon Boulton is the fourteenth 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.

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Simon Boulton and colleagues at Cancer Research UK

CALLING FOR ENTRIES!

If you are a biological or biomedical researcher under the age of 35 you could be the next Eppendorf Award winner. The winner receives prize money of €15,000 plus other benefits. To find out more, visit eppendorf.com/award. The deadline for complete applications is 30 June 2009.

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otherwise you lose genetic information. The work that led to this award looked at an aspect of DNA repair that was very poorly defined, called homologous recombination. Homologous recombination uses an intact chromatid as a template to repair the damaged chromosome. You might think recombination is always a useful thing, but it can be invoked at the wrong place or the wrong time, so you need events that can restrain it.

KS: *What specific findings won you the prize?*

SB: There's quite a lot of work in yeast that's identified a protein called suppressor of Rad six (Srs2), which is an antagonist of recombination. However, there was no obvious orthologue outside yeast. So we used a genetic screen in *Caenorhabditis elegans* worms, and in mammalian cells and mouse models, and discovered this new protein, which to a large extent acts like Srs2.

KS: *So it was originally discovered in yeast, and then you found an analogue in C. elegans and mice — what about a human version?*

SB: Yes, indeed, it turned out that a number of people had observed that this protein was amplified in gastric cancers, for example, and nobody knew why. What we've discovered subsequently* is that when you overexpress this helicase you also cause problems. We're now using mouse models to look at the consequence of loss or overexpression of the protein. We're also working with others to see if it's deregulated in other cancers.

KS: *What do you think it was about your work that the jury liked so much?*

SB: It's difficult to say exactly. I think it's fair to say that our work solved a conundrum in the field. Srs2 was identified nearly 30 years ago, but nobody had discovered anything equivalent in more complex organisms. Maybe it's a little bold to say this, but I think our work provided a very important new piece in our understanding of how recombination is controlled.

**Unpublished work*

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