

- [ScienceWatch Home](#)
- [Inside This Month...](#)
- [Interviews](#)

- Featured Interviews
- Author Commentaries
- Institutional Interviews
- Journal Interviews
- Podcasts

Analyses

- Featured Analyses
- What's Hot In...
- Special Topics

Data & Rankings

- Sci-Bytes
- Fast Breaking Papers
- New Hot Papers
- Emerging Research Fronts
- Fast Moving Fronts
- Corporate Research Fronts
- Research Front Maps
- Current Classics
- Top Topics
- Rising Stars
- New Entrants
- Country Profiles

About Science Watch

- Methodology
- Archives
- Contact Us
- RSS Feeds



- Interviews
- Analyses
- Data & Rankings**

2009 : February 2009 - Fast Breaking Papers : Ros Eeles

FAST BREAKING PAPERS - 2009

February 2009



Ros Eeles talks with ScienceWatch.com and answers a few questions about this month's Fast Breaking Paper in the field of Molecular Biology & Genetics.



Article Title: Multiple newly identified loci associated with prostate cancer susceptibility

Authors: Eeles, RA, et al.

Journal: NAT GENET

Volume: 40

Issue: 3

Page: 316-321

Year: MAR 2008

* Inst Canc Res, 15 Cotswold Rd, Sutton SM2 5NG, Surrey, England.

* Inst Canc Res, Sutton SM2 5NG, Surrey, England.

* Royal Marsden NHS Fdn Trust, Sutton SM2 5PT, Surrey, England.

* Royal Marsden NHS Fdn Trust, London SW3 6JJ, England.

(addresses have been truncated)

SW: Why do you think your paper is highly cited?

Until recently, little has been understood about the heritability of prostate cancer. Historically, it has been regarded as a "Cinderella" cancer, lagging behind illnesses such as breast cancer and lung cancer in terms of both awareness and research. However, as prostate cancer is now the second biggest killer of men over the age of 60, more attention and resources have been paid to understanding the causes and development of this illness.

Our research has confirmed that there is indeed a heritable component to the disease causation, and these initial findings show the incredible potential scope for further study in this area. With better genetic knowledge about the causes of the disease, doctors and researchers will be equipped to target care more personally for those who show a genetic susceptibility.

As the findings in cancer genetics progress at an exponential rate, prostate cancer is one of the first disease areas where we can see a potential benefit for patients in terms of targeting screening for patients, and we hope, a more individualized approach to prevention and drug treatment.

SW: Does it describe a new discovery, methodology, or synthesis of knowledge?

The research uncovered that genetic alterations are very close to a gene called "microseminoprotein, beta-" (MSMB), which could possibly be used in screening for prostate cancer and disease monitoring. Another of the sites harbors a gene called "lemur tyrosine kinase 2" (LMTK2) which might be a target for new treatments. The

"These results were only possible due to the Human Genome Project and the developments in genetic technology which have enabled studies of thousands of genetic variants to be

data suggest these newly identified genetic alterations are present in over half of all prostate cancer cases. They each increase a person's risk of the disease by up to 60 %.

There are probably many different factors that influence the development of prostate cancer, but particular combinations of genes are thought to play a major part. These results represent the largest number of genetic risk factors found in one genome-wide cancer association study to date.

observed in thousands of samples at an affordable price."

SW: Would you summarize the significance of your paper in layman's terms?

The team studied the differences in the genetic makeup of over 10,000 men in total, making it the largest genetic prostate cancer study ever undertaken. They started by scanning the DNA of men who were thought to be at higher genetic risk of prostate cancer because they had been diagnosed with the disease at or younger than the age of 60 or had a family history of prostate cancer. They then compared these results with a control group of men who did not have the disease but lived in similar areas.

In the next stage, they looked to see if these genetic variants could be found more frequently in men with prostate cancer than in men without the disease. They studied 3,268 men with prostate cancer from the UK and Australia and 3,366 men who did not have the disease.

SW: How did you become involved in this research, and were there any problems along the way?

This work is the result of over 10 years of collaborative preparation when we set up the study blood sample collections and collaborations. These results were only possible due to the Human Genome Project and the developments in genetic technology which have enabled studies of thousands of genetic variants to be observed in thousands of samples at an affordable price.

SW: Where do you see your research leading in the future?

A more tailored approach towards screening, prevention, and treatment for prostate cancer. If we have a fuller understanding of man's genetic profile, we can provide more specific advice and recommend those treatments which have the greatest chance of overcoming cancer.

SW: Do you foresee any social or political implications for your research?

These results make the genetic profiling of populations, in order to be able to tailor healthcare, a reality. This will result in a mindshift in the way that medicine is practiced from a population-wide screening approach to a tailored one.

Medicine will become more proactive and preventative based on tailored risk assessment. The challenge will be interpretation of the genetic variants and assessment of their interaction with lifestyle in order to enable healthcare professionals to develop tailored prevention programs which are individualized.

Dr. Ros Eeles
Team Leader
Translational Cancer Genetics Team
Section of Cancer Genetics
The Institute of Cancer Research
& The Royal Marsden NHS Foundation Trust
Sutton, Surrey, UK

Web

KEYWORDS: GENOME-WIDE ASSOCIATION; AUSTRALIAN CASE-CONTROL; RADICAL PROSTATECTOMY; ANTIGEN LEVELS; RISK LOCUS; GENE; MEN; POLYMORPHISMS; VARIANTS; PROMOTER.



[back to top](#)

2009 : February 2009 - Fast Breaking Papers : Ros Eeles

