## EMERGING RESEARCH FRONTS - 2009

**August 2009**

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**Marcus S. Cooke talks with *ScienceWatch.com* and answers a few questions about this month's Emerging Research Front Paper in the field of Molecular Biology & Genetics. The author has also sent along an image of his work.**

**Article: Oxidative DNA damage: mechanisms, mutation, and disease**
Authors: Cooke, MS;Evans, MD;Dizdaroglu, M;Lunec, J
Journal: FASEB J, 17 (10): 1195-1214 JUL 2003
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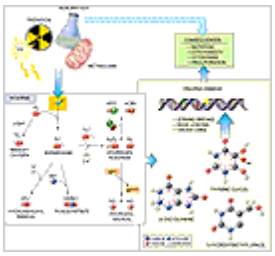
**SW: Why do you think your paper is highly cited?**

I think the paper is highly cited as it is a very comprehensive review that covers three important, interrelated areas concerning oxidatively damaged DNA. These are (i) mechanisms of formation; I was very happy that Miral Dizdaroglu agreed to become a co-author, and he provided the expertise for this section; (ii) effects of oxidatively damaged DNA at a cellular level; Mark Evans, here at Leicester, wrote that section; and (iii) role of oxidatively damaged DNA in disease; I wrote that section.

The breadth of the topic is a clear strength of the review, it stretches from the description of events at the level of individual nucleobases, via repair and mutation, to the possible role of such damage in disease—right from conception, this was my plan for the review. It is therefore very easy for authors to find information relevant to their studies, and hence able to cite the review. I was very pleased that the review was accepted in the *Journal of the Federation of American Societies for Experimental Biology (FASEB)*, which is a highly read and prestigious journal, and certainly maximized the coverage that the review received.

Figure 1 [+] details



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

It is very much a synthesis of knowledge, not just a summary of literature. Bringing the three areas (described above) together, I think, gives an interesting, and useful perspective upon the importance of oxidatively damaged DNA in health and disease. This is probably what made our review attractive to

*FASEB J.*

Too often I've heard reviews dismissed as being "something that anyone can do." Yes, perhaps anyone can summarize the literature, but that is not really a review. To my mind, a review should bring new ideas together, in some novel way, or give new perspectives on existing data; hence it should very much be a synthesis, not regurgitation.

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

In layman's terms, this review summarizes the effects of damage to DNA, the cell's blueprint, and how this damage might be involved in diseases, such as cancer. It describes how highly reactive chemicals known as free radicals, which can be generated by radiation, sunlight, and smoking, for example, are constantly damaging DNA. Antioxidants help to prevent this damage, and the DNA can also be repaired. However, if the antioxidants and repair processes are overwhelmed, levels of damage increase, and this leads to an increased risk of disease.

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

Whilst studying on the MSc in Molecular Pathology and Toxicology, here at Leicester, back in 1993, there was a lecture on free radicals. This grabbed my interest, and I've pursued this field ever since.

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

Pretty much like the review, my research examines the effects of oxidatively damaged DNA at a cellular level, but then translates those findings into human studies, and vice versa. I see this continuing to be my approach for the foreseeable future, but increasingly taking into account, and understanding the basis of, interindividual variability.

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

There are social and political implications for any kind of medical research. The fact that oxidatively damaged DNA is linked to so many diseases, as highlighted in the review, makes it of profound importance. Therefore society, via politics, needs to have the will to improve our understanding of how this damage is formed, so we can develop intervention strategies, and thereby reduce morbidity and mortality.

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**Figure 1:**

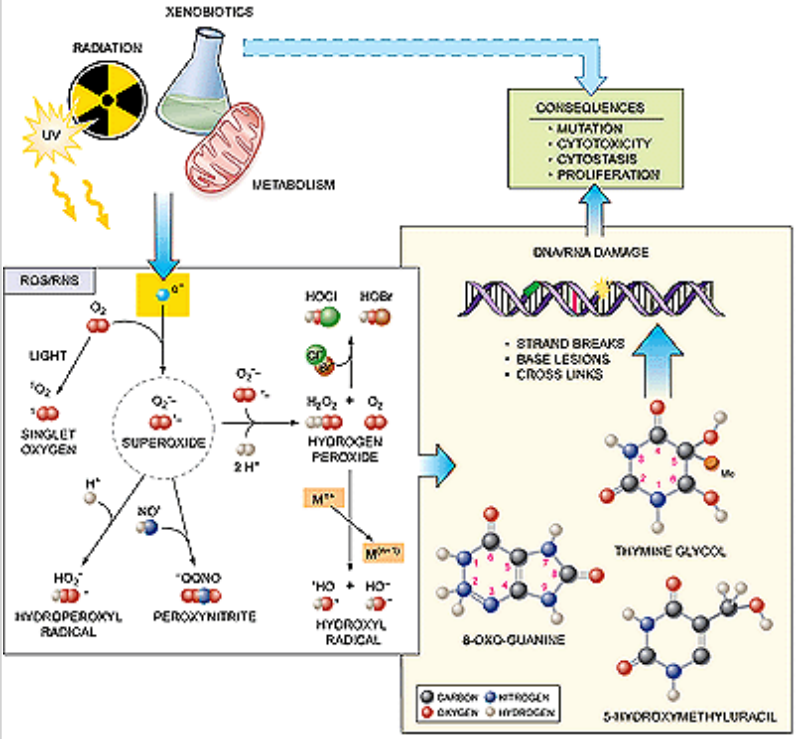


**Figure 1:**

Reactive oxygen species: causes and consequences (from Cooke, MS. and Evans, MD. [2005] *Science & Medicine*, 10, #2, 98-111). The production of reactive oxygen species (ROS) and reactive nitrogen species (RNS) may arise from common exogenous and endogenous processes. Free electrons produced by these processes are captured by molecular oxygen to yield superoxide (O2•-). Further interactions produce other reactive species, including the highly reactive hydroxyl radicals (•OH) and HO-. (Mn+/M(n +1) are reduced and oxidised metal ions, respectively.) Interaction of these reactive species with nucleic acids may lead to a wide variety of nucleobase products, deoxyribose products, strand breaks and DNA crosslinks. Many of the modifications are substrates for DNA repair. However, a consequence of unrepaired damage is, potentially, mutations which can lead to cancer. Click for a larger view.

KEYWORDS: BASE EXCISION-REPAIR; TRANSCRIPTION-COUPLED REPAIR; COLI ENDONUCLEASE-III; SYSTEMIC-LUPUS-ERYTHEMATOSUS; RADICAL-INDUCED FORMATION; CYTOSINE-DERIVED LESIONS; SINGLE-STRANDED-DNA; HUMAN CELL-EXTRACTS; HUMAN MUTY HOMOLOG; ESCHERICHIA-COLI.

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