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Special issue on DNA oxidation: Mechanisms, measurement and consequences

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EDITORIAL

Special issue on DNA oxidation: Mechanisms, measurement and consequences

In recent years, the study of oxidatively-generated damage to DNA has moved towards the forefront of the field of free radical and oxidative stress research. From its radiation chemistry beginnings, the field has now broadened, encompassing both *in vitro* and *in vivo* studies, related to health and disease. The topics covered in this special edition give some indication of exactly how broad this field has become.

The edition begins with a commentary by Cadet et al. [1], which provides a well-argued rationale for the establishment of IUPAC-consistent nomenclature for various free radical intermediates and their DNA reaction products. A victim of its own success, the ever-increasing numbers of publications involving oxidatively damaged DNA means that there is often uncertainty concerning what is appropriate and accurate terminology. This report should support those embarking on studies involving oxidatively generated damage to DNA nucleobases, and produce greater uniformity across the field.

There then follows a series of reviews associated with the formation, prevention and repair of oxidatively damaged DNA, starting with a description of the 'Mechanisms of free radical-induced damage to DNA' by Dizdaroglu and Jaruga [2]. This provides detailed description of the damage mechanisms on a lesion-by-lesion basis. Delany et al. [3] put this work in a more biological context by considering the mutagenic and genotoxic properties of oxidatively damaged DNA, together with possible roles in disease. This subject is extended further by Winczura et al. [4] to encompass lipid peroxidation-derived DNA adducts and their repair, together with the modification of proteins and potential roles in disease. Unlike damage to lipids and proteins, damaged DNA molecules cannot be removed and replaced, hence there is intense interest in the repair of oxidatively damaged DNA, not just single- and double-stranded breaks, but also modified nucleobases. The subsequent review of Harini et al. [5] focuses upon base excision repair, being one of the major routes for the prevention of damage persistence, and the implications for the organism if these pathways are deficient.

The next section relates to the measurement of oxidatively damaged DNA. Ravanat provides a review of 'Chromatographic methods for the analysis of oxidatively damaged DNA' [6], which includes comparisons between the various chromatographic approaches and certain non-chromatographic methods. There is also attention paid to the methods for extracting DNA, and hydrolysing or digesting DNA to its constituent nucleobases or 2′-deoxyribonucleosides – issues which remain central to avoiding artefactual generation of damage. Immunochemical approaches to the detection of oxidatively damaged DNA and 8-oxo-7,8-dihydro-2′-deoxyguanosine (8-oxodG) in particular, are a popular alternative to chromatographic methods, not least due the availability of commercial kits for this purpose. Rossner and Sram provide an extensive review of this literature, discussing the benefits, as well as the potential short-comings of immunochemical techniques, along with their widespread application to the study of 8-oxodG in disease, and after environmental and lifestyle exposures [7]. However, such damage is not uniformly distributed throughout the genome, and to evaluate this, new approaches are needed. Akatsuka and Toyokuni describe how 'omics technology' can be applied to the genome-wide, sequence-specific study of oxidatively damaged DNA [8]. Urine represents a non-invasive alternative to cellular DNA, in which nucleic acid-derived oxidative stress biomarkers can be measured, and Weimann et al. [9] summarise and compare the technologies used for this. As the provenance of these lesions is not entirely clear, they give considerable attention to the interpretation of these measurements. This section ends with a review by Moller et al. of the three major international strategies for harmonising assessment of DNA biomarkers of oxidative stress: European Standards Committee of Oxidative DNA Damage (ESCODD), European Comet Assay Validation Group (ECVAG) and European Standards Committee on Urinary (DNA) Lesion Assessment (ESCULA) [10]. As the names suggest, the groups evaluated commonly used methods to quantify damage to nuclear DNA, comet assay (single cell gel electrophoresis) and

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commonly used methods for the evaluation of urinary 8-oxodG, respectively.

The final section of this special edition provides two examples of diseases where oxidatively damaged DNA is proposed to play a pivotal role. Malik and Herbert highlight the emerging evidence that DNA damage is more than a bystander in the process of cardiovascular disease [11]. Santos et al. provide an overview of oxidative stress-related processes underlying the pathogenesis of Alzheimer's disease, specifically increased ROS production, damage to nuclear and mitochondrial DNA, combined with disrupted base excision repair [12].

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