Free Rad. Res., Vol. 29, p. 461
Reprints available directly from the publisher
Photocopying permitted by license only

Introduction

In May 1998, the majority of the world's experts on oxidative DNA damage met at the Panum Institute in Copenhagen to discuss the "state of the art". In particular, there is growing evidence that oxidative DNA damage contributes to the age-related development of cancer and several other chronic human diseases. There is also growing evidence that steady-state levels of oxidative DNA damage in cells are modulated by diet. But which dietary constituents are the most effective and how much should we consume? A particular focus of the meeting was on methodology – how accurately can we measure DNA damage in the human body and what methods should we use? Only by answering such questions can we confidently use levels of oxidative DNA damage as a "biomarker" to evaluate the relation between diet, other aspects of lifestyle and cancer risk. Already controversies have crept in to the literature: have we been wrong in assuming that the pro-oxidant effects of ascorbate do not occur *in vivo*, for example? Or are the data recently published in *Nature* by Podmore *et al* just a methodological artifact?

In the Introductory paper to this issue, AT Diplock explains why the existence of accurate, validated biomarkers of oxidative damage is so important in antioxidant research. In a personal view, I then summarize where we are in relation to understanding how to document oxidative DNA damage and what it means, raising some of the issues that are discussed in detail in the subsequent papers. As you will see, there is not always agreement.

Barry Halliwell
Department of Biochemistry
National University of Singapore