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PREFACE

This issue of the journal *Dose-Response* features 11 papers that were originally presented at the 15th Pacific Basin Nuclear Conference (PBNC), 15-20 October 2006, in Sydney, Australia. The PBNC is organized every two years by the Pacific Nuclear Council (PNC), which is composed of the nuclear societies around the Pacific Rim. Similar sessions related to the biological effects of low dose radiation were held for the first time at the 14th PBNC conference in Honolulu, USA in 2004 (*Dose Response* 4(4) 2006 and 5(1) 2007). Two of the objectives of the PNC are to “provide a strong voice as an internationally recognized regional, non-government organization at important and regional forums” and to “identify nuclear-related topics of interest warranting consideration by the PNC members”. This collected set of research and review papers from the 15th PBNC question the relevance of the LNT (linear no-threshold) model of risk-assessment, which is currently used worldwide as the basis for all ionizing radiation protection practices. The LNT model is based on the assumption that the risk of detrimental health effects is directly proportional to dose, such that that even the smallest dose of radiation increases health risks. The official acceptance of this LNT model has understandably generated a public fear of radiation doses, no matter how low the dose, thus fuelling inappropriate concern about low doses of radiation in occupational, diagnostic and environmental settings. Just as at the previous PBNC conference, the papers presented here by some of the world’s leading researchers in this area continue to contradict the LNT model for radiation risk assessment, and clearly demonstrate that at low doses, radiation dose does not predict risk. For low acute doses and for continuous, protracted or intermittent exposures, the harmful effects of high doses are reduced, cells respond to the exposure by inducing protective, adaptive responses and detrimental effects give way to beneficial effects. Evidence and physical reasons for these biologically positive hormetic effects of radiation are discussed in several of the papers.

The papers address low dose radiation effects by focussing on different areas where our knowledge of radiation has advanced, and all conclude that the LNT model is outdated. Brooks and Couch emphasise the fact that radiation is a very weak carcinogen and that very large doses of

radiation are required before it would be possible to detect an increase in cancer incidence from radiation exposure. Strzelczyk *et al.* emphasise the new molecular techniques that are now available to identify cellular responses to radiation and give a broad overview of biological mechanisms that can help to explain why the LNT model is outmoded. They also emphasise that scientists have a responsibility to educate the public about the science of low dose radiation. Mitchel approached the problems of the LNT model by demonstrating the inadequacies of the radiation protection assumptions which form the basis of present radiation risk assessment, namely tissue and radiation weighting factors, dose and dose-rate effectiveness factors and additivity of doses. Cuttler uses the lessons learnt from Chernobyl to emphasise that the LNT model is not an accurate predictor of radiation induced cancer and genetic abnormalities, and that low doses of radiation appear to be protective against cancer. Higson presents the position statement adopted by the Australasian Radiation Protection Society on risks from low levels of ionizing radiation, which divides radiation dose levels into different categories for risk assessment. Sykes and Day note the paucity of biological data for doses of radiation below 1 mGy, and discuss assay requirements, study design and the practical problems in measuring mutations required to detect hormetic responses after low dose radiation. This same group use such an assay to demonstrate adaptive responses for chromosomal inversions at doses that are relevant to population and occupational exposure. Cassidy *et al.* describe the lack of radiation-induced mutation data for non-human biota, and use the micronucleus assay to measure radiation effects in fish cells. They demonstrate differences in radiosensitivity between different species and the inability to detect mutations at low doses in such species. Marked differences are also observed in individual radiosensitivity in human lymphocytes in the paper by Schnarr *et al.*, highlighting that risk is unlikely to be the same in all individuals. The mechanisms underlying the adaptive responses to radiation open up the possibility of using low doses to protect individuals from cancer or to enhance antitumour therapy. Lui *et al.* describe the use of whole body low dose radiation to increase the efficacy of tumour killing by high dose radiotherapy and anti-tumour gene therapy in mice.

A joint summary statement was written by a number of the participants at the end of the 15th PBNC low dose radiation sessions. The joint statement constitutes the first of the papers of this set.

The papers presented in this issue of *Dose Response* place radiation dose in perspective with known biological outcomes, based on fact and not assumptions. Present expenditures on radiation protection for doses that are lower than background radiation in some regions of the world

P. Sykes, R. Mitchel, and D. Higson

are in the order of billions of dollars and appear to be unwarranted. The information provided in this issue has the potential to influence policy decisions about radiation protection standards and implementation of nuclear technologies.

Guest Editors

Pamela Sykes

Department of Haematology and Genetic Pathology, Flinders University and Medical Centre, Bedford Park, South Australia, 5042, Australia. pam.sykes@flinders.edu.au

Ron Mitchel

Radiation Biology and Health Physics branch, AECL, Chalk River Laboratories, Chalk River ON, Canada, KOJ 1PO, mitchelr@aecl.ca

Donald Higson

260 Glenmore Road, Paddington, New South Wales, 2001, Australia. higsond@bigpond.net.au