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SPECIAL ISSUE INTRODUCTION

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SPECIAL ISSUE INTRODUCTION

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Ionizing radiation protection regulations for humans are based on the presumption that any amount or radiation, no matter how small, will cause cancer in a very large population. The regulations impact on the use of ionizing radiation in routine diagnostic procedures (e.g., computed tomography), on allowable radiation levels in the environment (e.g., residential radon levels), and on allowable radiation exposures in the workplace (e.g., nuclear facilities). Cancer risks evaluations are based on the linear-no-threshold (LNT) paradigm for which risk is assumed to increase linearly as the radiation dose increases (Puskin 2009). Organizations such as the U.S. Environmental Protection Agency, the National Council on Radiation Protection and Measurements, and the International Commission on Radiological Protection rely on the LNT risk model for establishing limits for human exposure to radiation. *Indeed, dose units such as millisievert (mSv), which apply to all forms of ionizing radiation and their combinations, depend on the LNT hypothesis for their justifiable use.*

Justification for the continued use of the LNT risk model is based largely on results from epidemiological studies that employ inappropriate methods (Scott *et al.* 2008) such as the following: (1) giving considerable weight to high-dose, high-dose-rate data where excess cancers clearly occur while ignoring the absence of excess cancers after low doses and dose rates; (2) dose lagging (i.e., discarding some of the dose thereby shifting the dose-response curve to the left, making smaller doses appear more harmful than they actually are); (3) attributing observed low-dose-associated reductions in the cancer incidence among radiation workers to a presumed healthy worker effect; (4) not allowing for the possibility that a threshold or a reduction in risk at low doses may occur because of radiation-stimulated adaptive protection; and (5) averaging over wide dose intervals so that nonlinearity is removed.

This issue provides additional support for the rapidly growing view that the LNT risk model should not be used for low-dose cancer risk assessment. The paper by Fornalski and Dobrzyński (2010) discusses the inappropriate use of the healthy worker effect assumption in attempts to

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explain away actual cancer suppressive effects of low-dose radiation. Jaworowski (2010) in his paper makes a clear presentation of how LNT-related radiation phobia after the Chernobyl accident has caused significant human suffering and unnecessary very large costs to Eastern European populations impacted by the accident. He points out that unlike the thousands of hypothetical late cancer deaths projected by some (which did not occur) based on the LNT model, a 15% to 30% deficit (relative to the general population) of solid cancer mortality was found among the Russian emergency workers at Chernobyl and a 5% deficit of solid cancer incidence among the population of the most contaminated areas. The paper by Vaiserman (2010) reviews some of the abundant epidemiological and other data on radiation hormesis as it relates to cancer suppression.

Mitchel (2010) points out in his paper that radiation adaptive responses occur in all organisms and describes a dose window (which corresponds to the hormetic zone) over which cancer suppression via stimulated adaptive protection occurs. The hormetic zone is thought to depend on the type of radiation (Scott 2005; Elmore *et al.* 2009) and how the radiation is delivered (e.g., brief high rate vs. protracted low rate exposure [Elmore *et al.* 2006; Feinendegen *et al.* 2010]). The hormetic zone is more pronounced for sparsely ionizing forms of radiation (e.g., x-rays and gamma rays) than for highly ionizing forms (Scott 2005). The sparsely ionizing forms are sometimes also in combination with highly ionizing alpha radiation (e.g., as for residential radon) in which case a hormetic zone may also be evident at low doses and dose rates if data are properly analyzed (Thompson *et al.* 2008).

The paper by Nowosielska *et al.* (2010) presents evidence from animal studies of low-dose radiation stimulating antineoplastic immune surveillance. The stimulated immunity can protect against cancer and metastases and raises the possibility for use of low doses of radiation in preventing cancers in high risk populations (e.g., long-time heavy smokers) and curing early stage cancers (Scott and Di Palma 2006).

Feinendegen *et al.* (2010) review the current state of knowledge about radiation-stimulated adaptive protection against cancer and present a Dual-Probability Model of cancer induction that allows for multiple levels of protection (including metabolic-dynamic defenses) and for a hormetic zone. The model integrates biological responses to irradiation over multiples scales (molecular, cellular, and tissue levels). Based on analyses conducted, the authors conclude that adaptive protection (radiation hormesis) preventing only 2 – 3 % of endogenous life-time cancer risk would fully balance a calculated (based on LNT) induced cancer risk at about 100 mSv, in agreement with epidemiological data.

Collectively, the papers in this issue indicate that low-dose radiation-stimulated adaptive protection can no longer be justifiably ignored when

evaluating risks from exposure to low radiation doses. This includes risks from diagnostic procedures such as computed tomography that employ radiation.

I was asked by Edward (Ed) Calabrese (2009 Marie Curie Prize recipient) to serve as Guest Editor for this issue of the journal and was happy to do so. It was a pleasure working with the contributing authors. All of the invited authors readily volunteered to contribute papers. On behalf of all the authors I would like to express our gratitude to Denise Leonard (Managing Editor) for her help in preparing this special issue and to Ed for supporting its publication. It is hoped that the issue will stimulate interesting discussions world-wide as well as help foster new research related to the use of low doses of sparsely ionizing radiation to prevent and cure cancer and thereby lengthen life.

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