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Sent: Thursday, November 19, 2015 10:18 AM
To: Rulemaking1CEm Resource
Subject: FW: Docket ID NRC-2015-0057 Arkansas Department of Health comments
Attachments: Arkansas Comment on NRC-2015-0057 Hormesis.pdf

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TITLE: Linear No-Threshold Model and Standards for Protection Against Radiation
COMMENT#: 533

From: David Stephens [mailto:David.Stephens@arkansas.gov]
Sent: Wednesday, November 18, 2015 4:18 PM
To: RulemakingComments Resource <RulemakingComments.Resource@nrc.gov>; Sherrie.Flaherty@state.mn.us; Bernard Bevill <Bernard.Bevill@arkansas.gov>; Jared Thompson (Pulaski-ADH) <Jared.Thompson@arkansas.gov>
Subject: [External_Sender] Docket ID NRC-2015-0057 Arkansas Department of Health comments

Greetings:

Please find attached comments from the Radioactive Control Section of the Arkansas Department of Health on Docket ID NRC-2015-0057, the hormesis issue. Hard copy to follow by FedEx.

Thank you,

David B. Stephens, Ph.D., AAHP Associate
Health Physicist, Radioactive Materials Program
Radiation Control
Arkansas Department of Health

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November 18, 2015

Annette L. Vietti-Cook, Secretary
United States Nuclear Regulatory Commission
Washington, DC 20555-0001

Dear Ms. Vietti-Cook,

The Arkansas Department of Health Radioactive Materials Program (Department) has reviewed the Petition for Rulemaking (Docket ID NRC-2015-0057), Linear No-Threshold Model and Standards for Protection Against Radiation. After careful review of the issues involved, the Department has several comments to make:

The Linear No-Threshold (LNT) model of biological radiation effect has been the standard in radiation safety regulations for over 50 years. On purely biochemical grounds, it bears a compelling logic. Ionizing radiation produces reactive oxygen species (ROS) in water, which are capable of damaging biological molecules, including DNA. Such damage to DNA is potentially carcinogenic. Thus, any exposure to ionizing radiation increases the chances of cancer.

There are several lines of evidence which have been brought against this model. There are studies which seem to indicate low-dose radiation causes no harm and perhaps even benefits the recipient. Critics point to the fact that ROS occur as a result of respiration and other mechanisms in normal cells. Recent research has indicated that a steady-state of approximately 40,000 DNA lesions per cell exist as a result of normal metabolism. Against this high level of background damage, the argument could be made that the much lower level of damage due to low-dose radiation exposure would be indistinguishable. Mechanisms to control ROS and repair DNA damage also exist and have been shown to be up-regulated in cases of radiation exposure.

In respect to this last point, supporters of the hormesis model of biological radiation effect propose a beneficial effect to low-dose radiation; some have stated that they support intentionally dosing the populace at low levels to improve health. This model proposes that low dose radiation stimulates the aforementioned control and repair mechanisms of the body, which then not only repair the radiation-induced damage but repair other preexistent damage as well, improving the net health status of the individual. To draw an analogy, it is as if the owner of a car with numerous preexistent small damages decides to intentionally have another accident, in order to trick the insurance company into fixing all the damage, including that damage that was pre-existent.

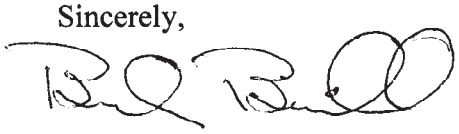
There is an important point to be made at this juncture, however. Radiation damage is different than the other forms of oxidative damage typically seen – it is *clustered.* Endogenous ROS tend to occur in association with the mitochondria and other areas of the cytoplasm and are dispersed in the media. Since radiation occurs along a track, the ROS produced cluster around that track; if the track transects a nucleus, clustered multiple DNA damage points have been shown to occur in high frequency. Further, these clustered DNA damage points have been shown to be quite refractory to the DNA repair systems, persisting for longer periods of time and potentially disrupting replication and other cellular functions. When an effort to repair this damage does occur, it frequently results in non-homologous recombination, an error-prone "last-ditch" repair attempt which further degrades the information carried by the DNA. To carry the previous analogy further, the hapless car-owner discovers that he bent the frame of the car during his accident, a type of damage his repairmen can't fix and permanently damages his car.

Another point can be made. Individuals exist with DNA-repair-deficient radiation-hypersensitivity conditions – Xeroderma pigmentosum is one example – which, although quite rare and an extreme example, indicate a more general issue. The human species is genetically quite heterogeneous, with many alleles for most genes. These alleles produce proteins of varying efficiency. As regulators, at what point of DNA-repair-system efficiency would we draw our threshold? Which part of the population with alleles of lower DNA repair efficiency would we consider of no consequence?

In light of these considerations, the Department finds that it does **NOT** support abandonment of the LNT theory in favor of hormesis as the standard model for radiation protection. Although evidence exists that other mechanisms may be at play, those considerations require quite a bit more study before becoming the basis of policy.

The Department appreciates this opportunity to comment on this issue. If a committee is formed to further consider this issue, the Department would like to recommend Dr. David B. Stephens, Ph.D., AAHP Associate, to this committee. For further questions please contact Bernard Bevill.

Sincerely,



Bernard Bevill, Section Chief
Radiation Control Section
Arkansas Department of Health

cc: Sherrie Flaherty, Chair
Organization of Agreement States