Testimony Before NRC Public Hearings Regarding the
Draft Programmatic Environmental Impact Statement
for Decontamination of TMI Nuclear Station, Unit 2

Both the NRC and Metropolitan Edison admit that they are unable to remove tritium from the hundreds of thousand of gallons of contaminated water resulting from the infamous accident that occurred at Three Mile Island, one and a half years ago. According to their draft EIS, this tritiated water will ultimately end up in the Susquehanna River and be carried downstream into Chesapeake Bay. The plan is to release about 3,500 Ci of tritium over a period of a few months. Now the average annual release of tritium from a nuclear power plant is only 400-500 Ci, which means that on a similar annual basis TMI will be releasing about twenty times more tritium than it would under normal operating conditions. We are told not to be concerned because the tritiated water will be sufficiently diluted with non-tritiated river water so that the actual concentration of tritium shall fall within the NRC safety standards. This assurance does not assuage my concern for at least two very good reasons; namely, it is the cumulative amount of tritium rather than its concentration that is the significant statistic in this case - never before have the people near a nuclear plant been subjected to three and a half thousand curies of tritium in their fishing and drinking water and, secondly, the NRC safety standards for tritium are based on outdated population dosage calculations that grossly underestimate the radiotoxicity of tritium to human life.

The remaining part of my testimony is meant to amplify the two reasons given above in a slightly more scientific vernacular that should be comprehensible to the NRC Commissioners and to the public in general. If the NRC is interested in a more detailed scientific presentation, including documentation of the appropriate research, that is now in the publication process and can be forwarded at some future date.

Inhomogenious Dispersion Versus Uniform Dilution

Conventional engineering wisdom asserts that dissolved tritium or tritiated water rapidly diffuses throughout any body of water, reaches its equilibrium concentration, and remains uniformly distributed in that body of water forever. This rather simplistic view does not take several additional factors into consideration such as convection currents, thermal differences and different rates and strength of physical adsorption. For example, if a nuclear power plant (e. g., Three-Mile Island) discharges its tritiated water into a naturally flowing river (e. g. Susquehanna River) then that tritium does not instantaneously diffuse throughout the total volume of river water to achieve maximum dilution; but rather, it may very well stay within certain currents or be adsorbed by the sediment of the river bed (or its aquatic contents) or even remain within the cooler regions of the river wherethermal diffusion is less vigorous, all of these additional factors would prevent a rapid equilibration of the discarded tritrium within the river thereby resulting in an uneven distribution of the tritium. In other words, parts of the river would have much higher concentrations of the tritium than other parts and thus any ingestion of this more highly tritiated water by fish, animals or even humans would result in greater irradiation of their tissues (by the beta particles) than one would anticipate by the engineering hypothesis of a totally uniform tritrium distribution.

Biological Accumulation or Concentration

The toxicity of any hazardous substance is typically, a function of the quantity of that substance to which a living organism is exposed. Radiation is no exception, the larger the concentration of the radioisotope the greater the risk of genetic and somatic damage resulting in birth defects, stillbirths and cancer. When it came to evaluating the effect of tritium (T), the International Commission of Radiological Protection (ICRP) calculated its population dose based on the tritium activity that could equilibrate with the body fluid (i.e., the inorganic compartment) and totally neglected the covalently bound tritium (i.e., the organic compartment). The implicit assumption of the ICRP dose estimate is that the tritiated body water exchanges its tritium for hydrogen only in a polar or ionic transfer with other molecules. Understandably, real life is not that simple and there is now considerable scientific evidence demonstrating that the tritium to hydrogen ratio (T/H) is much greater in the organic molecules or biopolymers (such as polysaccharides, lipids, proteins and nucleic acids) than in the inorganic tritium source (HTO). This results from at least three distinct biological or biochemical phenomena including (1) isotope effects in metabolic pathways (2) concentration of tritium within the organic compartment along a food chain and (3) radiation damage induction of unscheduled DNA synthesis. The metabolic route can, for example, produce covalent tritium-carbon bonds which are much stronger than the more polar hydrogen-oxygen bonds found in the inorganic compartment. Since many of these organic biopolymers are quite stable (i.e., long half-lives), the tritium tends to "hang around" for relatively long intervals. The data also suggests that tritiated organic precursers are more easily incorporated (than simple HTO) into organisms,

further along a food chain with several trophic levels. Thus the greater chemical stability of the tritiated organic molecules and their concentration along the food chain results in a much greater biological accumulation of tritium than one would anticipate from the oversimplified ICRP hypothesis. The incorporation of tritium into any biopolymer is clearly a function of the concentration of tritiated precursors, the rate of synthesis and the half-life of that macromolecule in vivo. In the specific case of DNA, the beta decay of tritium causes radiation damage to this biopolymer which increases its rate of synthesis, that is, the tritium has an autocatlytic effect on the synthesis of DNA. All three phenomena therefore come into play, producing a greatly increased steady-state concentration of tritiated DNA (T-DNA). In fact, several investigators have found that the incorporation of tritium into DNA was 3 or 4 times that found in the water (HTO), clearly demonstrating the importance of biological accumulation.

Microdistribution Affects Relative Biological Effectiveness

The radiotoxicity of tritium depends, in part, on its exact tissue, cellular and molecular localization. The marked differences in the radiosensitivity of various tissues has been well recognized, however, the affect of the microdistribution of the radioisotope within the cell has only recently been demonstrated. A measure of that cellular radiotoxicity is called the relative biological effectiveness (RBE) or quality factor (QF) and it may be assayed in various ways such as the inhibition of erythropoiesis, killing of oocytes or spermatogonia, frequency of dominant lethal mutations; tissue culture growth rate (e.g., HeLa Cells) inhibition, or the number of single strand breaks in DNA. It appears that the toxicity of tritium varies greatly with its molecular form, for example, the QF of tritiated DNA(T-DNA) is larger than tritiated water (HTO) or even other organic molecules (e.g. tritiated proteins or lipids). Recent studies indicate that the RBE for tritiated-DNA is closer to 4 rather than the 1.7 or 1 designated by the ICRP. The greater RBE for tritiated-DNA is consistent with the increased importance of DNA strand breaks and chromosomal structural aberrations as being primarily responsible for the mutagenic and carcinogenic effects of radiation. In addition to its well-known capacity for rupturing the DNA strand or macromolecule, there have been at least four other mechanisms identified that tend to augment its radiotoxic potential, namely, the (1) beta radiation from tritium retards the rate and efficacy of DNA repair (2) DNA may be altered so that point mutations are introduced by errors in the rapid mechanisms (3) induction of repair mechanisms (by radiation damage) may also facilitate viral transformations of the cells into abnormal or malignant forms and (4) synergistic

effects due to the presence of toxic chemicals may enhance the radiotoxic effect of the decaying tritium nuclei within the DNA. Thus any calculation or estimate of the population dose resulting from exposure to tritium or tritiated water must consider both the greater concentration of tritiated DNA than was previously suspected as well as its much larger QF. These two factors alone may represent a ten-fold increase in the radiotoxicity of tritium and must be properly reflected by new government standards for the "acceptable levels of tritium" to which the public may be subjected.

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