The Linear No-Threshold (LNT) theory of radiation induced cancer risk is questionable and should be reevaluated for low dose exposures.

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NSE 319 Term Paper
March 12, 2018

I. Introduction

The linear no-threshold model of radiation induced cancer risk is the currently accepted model used to quantify the health risks related to radiation exposure. Based on a handful of epidemiology studies from the second world war, this model has been used to make major policy decisions for public health that have played an integral part in spreading fear of radiation and slowing the progress of nuclear technology. In this paper I argue that this model is scientifically questionable and needs to be reevaluated at low doses. Epidemiological studies of high radiation dose nuclear bomb survivors should not be extrapolated to describe the biology of low dose, variable rate exposures.

II. The No Threshold, Linear Model

The LNT model asserts that cancer risk is directly proportional to radiation exposure. This means that 1 mSv dose has one hundredth the risk from a similar exposure of 100 mSv [4]. The "no-threshold" part also means that this model suggests there is no minimum cancer inducing dose i.e. every exposure is dangerous regardless of magnitude.

Essentially this theory relies on two key hypotheses. The first is that the relationship between dose and DNA damage *in vivo* appears linear (evaluated by DNA strand breaks) and each DNA strand break has the same probability of inducing cell transformation regardless of how many strand breaks are occurring in a given space. The second hypothesis is that each transformed cell has the same chance of developing into cancer regardless of the applied dose [1]. These two assumptions make it surprising simple to calculate the number of expected cancer cases for an irradiated population. Simply take

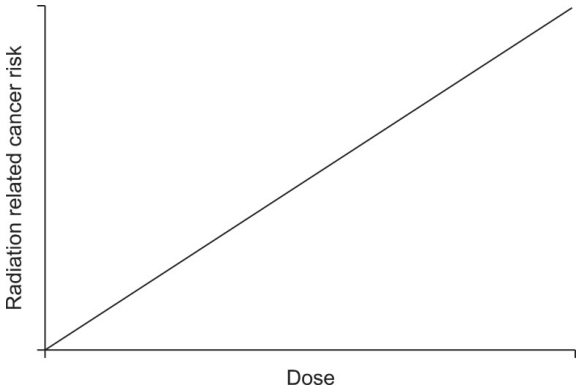

Fig. 1: The Linear no-threshold model

$$(\text{number exposed}) \times (\text{effective dose}) \times (\text{excess risk})$$

The main reason for accepting this model is that it fits observational data decently well and no other model has been conclusively shown to provide a *better* fit. The problem though is that there is very little data for low dose exposures meaning that in order to apply this model, data must be extrapolated. This has led some to argue that imaging techniques as in nuclear medicine are completely safe while others argue that these methods carry appreciable risks [4].

III. The Evidence

Imagine that for a brief instant a massive glowing fireball of plasmonic gases were to appear in the sky above you. Ignoring the dangers of the large temperature and possible shock waves from any

prior explosion, the light from such a source would be full of ionizing radiation that would surely pose dangerous health risks to you and those around you. Now what if I told you that I want to use this example to determine whether or not shining a UV light on your braces to set the glue is *safe*. Surely to reach any conclusion given this prior evidence would require a grocery list of assumptions. That is exactly what is done with the data used as a basis for the LNT model for low dose exposures.

Widely cited sources for radiation carcinogenesis in humans come from doses of the order 1 Sv or higher (one to two orders of magnitude higher than those in medical imaging). The most popular of these are epidemiological studies of atomic-bomb survivors from the second world war. Since 1947 a group of 120,000 survivors of the bombing of Hiroshima and Nagasaki as well as 77,000 of their children have been extensively studied [2]. Their exposed radiation levels can be fairly precisely determined (roughly 1 Sv scale) and so the increases in cancer in these populations as well as the effects on their offspring have been the single largest tool in studying the effects of radiation on human health.

While these studies have pretty accurately explored the health effects of large doses of radiation there are some important distinctions that make extrapolation of this data to low doses and variable dose rates suspect. Radiation was not the only carcinogen that survivors were exposed to. Atomic bombs release a host of other nonradioactive toxins into that sky that condensed in clouds and fell as black rain. The burns that most victims also suffered can lead to chronic inflammation that can also be carcinogenic. Once source even says, "It is difficult to separate the effect of radiation and, possibly, of contamination from the consequences of burns since most victims suffered both" [2]. The scale of these catastrophes is undeniable but trying to apply conclusions from this range of doses ignores fundamental biological processes our cells have evolved to perform. Even if we concede to the LNT mode it must also be noted that it *cannot* be applied to accurately individuals because it relies on large enough populations to be able to average out individual differences in radiation sensitivity.

IV. Biology of Radiation

Life evolved in a constant barrage of radiation and thus has developed systems to repair damaged DNA and remove damaged cells. DNA repair mechanisms have existed in the cells of mammals for nearly 800 million years [1]. The efficacy of these mechanisms has been shown to vary depending on the scale of applied radiation. Sensor molecules detect damage and can halt the cell cycle to allow for repair which involves either recombination or end joining. These methods are nearly zero error for low doses of x-rays but become worse as more DNA is damaged. DNA repair also varies by dose rate and type of damage– neither of which is accounted for by the LNT model.

One of the many misconceptions about Atomic Bomb survivors is that there is an increase in cell mutation. In truth, persistent DNA damage can lead to aberrations in chromosome expression which can result in genomic instability but doses of less than 250 mSv have been shown to display no instability . Furthermore, studies in mice have shown that there is no statistically significant correlation between radiation induced genomic instability and cancer [1].

Finally, the mechanism of cell death is effective at preventing cancer associated risks for low doses because the effect on tissue is minimal. This does not apply to high doses because the larger amount of cell impairment and death would be much more dangerous to tissue. Thus at high doses, manipulating the DNA is the only reliable mechanism which does involve a higher probability of forming defects.

So clearly there are demonstrable differences between the effects of high and low radiation doses on human biology. This suggests that it is unreasonable to 1) extrapolate high dose data to low doses and 2) assume that there is no minimum dose threshold. Now we will explore a select few counterproposals to the LNT model.

V. Alternatives

The next step up so-to-speak from the LNT model would be a linear threshold model. This can be seen in 2 as the straight line that has been

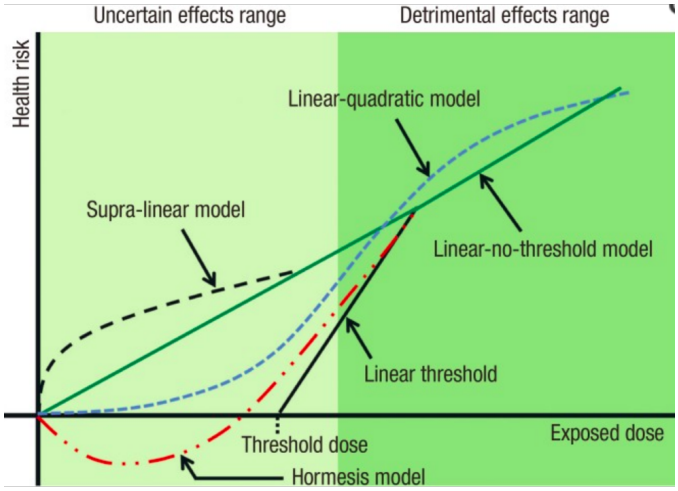

Fig. 2: Other models for radiation induced health risk

shifted to the right. This competing model supposes that there exists a minimum dose below which we do not experience any adverse effects. Qualitatively this is reasonable as we know that it is safe to got out in the Sun during the summer and get a little tan (from the UV radiation) so long as we don't overdo it. Because the confidence intervals are so wide for the low dose data, this curve and many others can be made to reasonably fit the low dose portion of the curve.

Another possible model considers the effect of a biological process called radiation Hormesis. For some biological systems, it has been found that low doses of radiation can actually be beneficial. One study even goes as far as to say that low dose radiation increased the average lifespan of laboratory animals [3].

Thus there are *many* different possible models that can fit the epidemiological data that also account for the biology of low dose radiation. Currently policy decisions that define allowed yearly dosages as well as how much radiation medical equipment can expose patients to is determined by these models so it is vitally important to try and find one that is true to human biology.

VI. Conclusions

First we examined the linear non-threshold model for radiation induced health risks. The data used to support this model comes largely from epidemiological studies of atomic bomb survivors in Japan. We explained how these studies are all of people exposed to radiation doses orders of magnitude higher than medical imagining uses however extrapolations from this data into low dose ranges are dangerous because they do not have good statistical confidence and fail to account for the biology of low radiation dose.

Then we considered some specific biological mechanisms such as DNA repair and cell death that provide defense against low dose radiation. In all of those cases, increasing the dose reduced the ability for the cell to protect it self.

This combined with the fact that there is not enough data for low dose associated cancer risk leads me to conclude that the LNT model must be reevaluated in order to see if some kind of threshold or hormesis model can be used in its place. Because cancer is such a widespread fear in modern society it is vital that we do not over react to radiation as this increases fear that can prevent the invention of new lifesaving technologies.

References

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