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## ABSTRACT

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**Radiation from Medical Procedures in the Pathogenesis of Cancer and  
Ischemic Heart Disease:  
Dose-Response Studies with Physicians per 100,000 Population.**

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### ORIENTATION:

For decades, xrays and other classes of ionizing radiation have been a proven cause, in vivo and/or in vitro, of virtually all types of mutation --- especially structural chromosomal mutations (such as deletions, translocations, and rings), for which the doubling-dose by xrays is extremely low. Additionally, xrays are an established cause of in vitro genomic instability.

This monograph looks at the impact of medical radiation --- primarily from xrays, including fluoroscopy and CT scans --- upon mortality-rates from both Cancer and Ischemic (Coronary) Heart Disease, from mid-century to 1990. The evidence in this book strongly indicates that medical radiation has become a necessary co-actor (but not the only necessary co-actor) in causing over 50% of the death-rates from Cancer and Ischemic Heart Disease (IHD) --- a finding which is consistent with participation of non-xray causes as necessary co-actors in the same cases (Introduction). In multi-cause diseases such as Cancer and IHD, more than one necessary co-actor per fatal case is very likely. Absence of any necessary co-actor, by definition, prevents such cases. The concept, of cases due to medical radiation, means cases which would be absent in the absence of medical radiation.

### PURPOSE:

Xrays have been a well-established cause of human Cancer for decades. This monograph was undertaken (a) to quantify what share of U.S. age-adjusted cancer mortality, for each gender, is caused by medical radiation, and (b) to check on the author's 1995 finding, based on completely different data, that exposure to medical radiation accounts for about 75% of Breast Cancer incidence in the USA. In the process of evaluating cancer mortality vs. noncancer mortality for this monograph, it became obvious that the impact of medical radiation upon death-rates specifically from Ischemic Heart Disease also demanded evaluation.

### MATERIALS AND METHODS:

This study is based on mortality rates among 130-250 million persons --- namely, the entire United States population, 1940-1990. Age-adjusted cancer mortality rates (MortRates) per 100,000 population are available by gender for each of the Nine Census Divisions (USA), for the 1940-1990 decades, from Vital Statistics. Such rates for noncancer mortality rates also are available. For Ischemic Heart Disease, such rates are available starting in 1950, which means that NonCancer NonIHD MortRates, by Census Divisions, are available starting in 1950.

For reasons presented in Chapter 2 (Parts 2+3), there are no reliable estimates of average per capita population dose, accumulated from medical radiation, currently or in the past. Also not available, for reasons presented in Chapter 2 (Part 7c), are reliable estimates of cancer-risk per unit of dose from medical xrays. This monograph avoids these two types of uncertainty by using the number of physicians per 100,000 population (PhysPop) as a reasonable approximation of the RELATIVE magnitude of exposure from medical radiation in the Nine Census Divisions. The ranking of averaged PhysPop values by Census Divisions, over the 1940-1990 period, is remarkably stable.

MortRates are regressed upon PhysPop values, by Census Divisions, to determine the presence and direction of any dose-response. When a significant positive dose-response exists, the line of best fit is extended to the y-axis, where the intercept's value indicates what the MortRate would have been

for that disease, if there had been NO physicians per 100,000 population in a Census Division. The national MortRate for the disease under study, minus the intercept's value, provides a reasonable estimate of the share of that national MortRate which is due to medical radiation (i.e., the share which would be absent in the absence of medical radiation). Confidence limits are provided in Chapter 22, Box 1.

### RESULTS:

Cancer and IHD MortRates each have very significant positive correlations with PhysPop, for males and females separately. By contrast, NonCancer NonIHD MortRates have a significant negative correlation with PhysPop. The following groups of Cancer were studied: All-Cancers-Combined, Breast Cancers, Digestive-System Cancers, Urinary-System Cancers, Genital Cancers, Buccal/Pharynx Cancers, Respiratory-System Cancers, Difference-Cancers (All-Except-Respiratory). Only female Genital Cancers failed to have a significant positive dose-response with PhysPop. The percentages, of the death-rates from Cancer and IHD caused by medical radiation (i.e., the shares which would be absent, in the absence of medical radiation), are shown in Box 1 of Chapter 1. For example:

	Year	Percent	Year	Percent
● All-Cancers-Combined, m	1940	90 %	1988	74 %
● All-Cancers-Combined, f	1940	58 %	1988	50 %
● All-Cancer-Except-Genital, f	1940	75 %	1980	66 %
● Breast Cancer, f	1940	~ 100 %	1990	83 %
● Ischemic Heart Disease, m	1950	79 %	1993	63 %
● Ischemic Heart Disease, f	1950	97 %	1993	78 %

The growing impact of cigarette-smoking (Chapters 48, 49) almost certainly explains why the shares from medical radiation in 1980-1993 are somewhat lower than in 1940-50.

### CONCLUSIONS:

Since its introduction in 1896, medical radiation has become a necessary co-actor in most fatal cases of Cancer and Ischemic Heart Disease (IHD).

It is proposed that, for radiation-induced IHD, the probable mechanism is radiation-induction of mutations in the coronary arteries, resulting in dysfunctional clones (mini-tumors) of smooth muscle cells. A Unified Model of Atherogenesis and Acute IHD Events is presented (Chapter 45), which is consistent with the findings in this book, is consistent with the findings (first by Earl Benditt in 1973) of monoclonal cells in atherosclerotic plaques, is consistent with well-established knowledge about atherogenic lipoproteins and other non-xray causes of fatal IHD, and is consistent with recent findings about the weaker connection than expected between degree of arterial stenosis and the fatal rupturing of specific atherosclerotic plaques.

The evidence in this monograph has major implications for prevention of Cancer and IHD. This monograph points to demonstrations, by others, of proven ways to reduce dose-levels of nontherapeutic medical radiation by 50% or considerably more, without eliminating a single diagnostic or interventional radiologic procedure and without degrading the information provided by medical radiation. Reduction of exposure to medical radiation can and will reduce mortality rates from both Cancer and Ischemic Heart Disease.

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