ISSN: 2755-6468

Review Article

Open Access Journal of Clinical Pathology Research

Artificial Radionuclides and Evolutionary Mismatch: Vulnerability of the Colon, Pancreas, Diabetes, and Arteries

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Received: February 26, 2026; **Accepted:** March 05, 2026; **Published:** March 11, 2026

ABSTRACT
The global dissemination of artificial radionuclides since 1945 represents a novel environmental condition in evolutionary terms. Isotopes such as Cesium-137 (Cs-137) and Iodine-131 (I-131), generated through nuclear weapons testing, reactor accidents, and nuclear energy production, were not present during the evolutionary history of terrestrial life. The new radio-iodine decays into inert Xenon, while carbon-14 decays into nitrogen-14 via beta emission; this transformation occurs at the atomic level and does not directly disrupt established macromolecular carbon frameworks, although local molecular damage may occur during decay events. This paper suggests an evolutionary hypothesis" that chronic internal exposure to such radionuclides may constitute an underexplored factor in long-term metabolic, pancreatic, colon and arterial disorders. The framework integrates evolutionary biology, ion mimicry theory, oxidative stress mechanisms, and internal dosimetry. It further addresses an ongoing scientific disagreement regarding the adequacy of prevailing low-dose radiation risk models, particularly with respect to non-cancer metabolic endpoints. While no causal association between environmental Cs-137 exposure and arteries, diabetes, pancreatic or colorectal cancer is currently recognized by major international health authorities, this position paper of CCPDA-hypothesis, supports that targeted interdisciplinary investigation is warranted.

Keywords: Iodine, Caesium-137, Colon, Pancreas Cancer, Diabetes, Atherosclerosis, CCPDA-Hypothesis

Foreword

Fukushima radiation continues to seep into the Pacific Ocean

In 2015, the highest Cs-137 marine concentration off the coast of North America is 6 Bq/m3. Off the coast of Japan after the accident, is a high of 4,500 Bq/m3 [1]. (From WHOI, Woods Hole Oceanographic Institution - https://www.whoi.edu)

Cesium-137 in Oceans and in Our Planet

Sr-90 and Cs-137 radionuclides combined with the longer-lived 129-Iodine (T 1/2 = Million year) can trace hydrologic, atmospheric, oceanic, and geochemical processes. Buesseler (Woods Hole Oceanographic Institution) reported that, in the 1960s, immediately after testing on the Pacific atolls, the concentration of radioactive cesium in the Pacific off the coast of Japan was about 50 (Bq/m^3) and 10 (Bq/m^3) in California. Recently he recorded, aside from the extremely high levels detected at the point of release from the reactors, a maximum of 4,500 (Bq/m^3) off the coast of Japan, after the Fukushima accident [1,2]. Concentrations (2020) are still elevated compared to pre-accident levels reaching up to 198 (Bq·m–3) for 137-Cs, and (114) × 10–5 (Bq·m–3) for 129-Iodine, and today should be roughly 25–30% of the original 1960s peak.

Introduction

Oppenheimer and the "New Nuclear Era" as an Evolution Discontinuity

The Chernobyl nuclear accident, on 26 April 1986, marked a turning point in both the history of energy production and our understanding of the relationship between human technology and life. So, J. Robert. Oppenheimer, as he witnessed the first detonation of a nuclear weapon on July 16, 1945, evoked a piece of Hindu scripture, "Now I am become Death, the destroyer of worlds ", reflecting the profound realisation that nuclear fission represents a significant departure from the natural history of the world, as marking a new era [3]. In historical terms, this statement referred to geopolitical and ethical transformation. In biological terms, the nuclear age can also be interpreted as the beginning of persistent anthropogenic isotope circulation within Earth's ecosystems. The metaphor of a "new era of atoms" captures the idea that human technological capacity has modified the elemental landscape of life: artificial isotopes now

Citation: Venturi Sebastiano. Artificial Radionuclides and Evolutionary Mismatch: Vulnerability of the Colon, Pancreas, Diabetes, and Arteries. Open Access J Clin Path Res. 2026. 2(1): 1-8. DOI: doi.org/10.61440/OAJCPR.2026.v2.39

participate in global biogeochemical cycles. Life evolved within a stable isotopic environment shaped by cosmic nucleosynthesis and long-term biological selection. Iodine was progressively integrated into biochemical pathways, including antioxidant and regulatory mechanisms. However, nuclear fission has introduced an anthropogenic production of radioactive isotopes that were absent from evolution. This evolutionary integration of iodine and potassium into living systems has provided robust mechanisms for antioxidant defense, redox regulation and metabolic stability. Artificial radionuclides, such as iodine-131 and cesium-137, mimic these essential elements but differ in radiophysical properties, exploiting conserved transport pathways and creating cellular vulnerabilities [4,5]. The new radio-iodine decays into inert xenon, while carbon-14 decays into nitrogen-14, which is no longer able to form the vital organic carbon chains. Fallout dispersed isotopes across continents, oceans, and food chains. From a geological perspective, this event occurred abruptly.

Evolutionary Biology and Elemental Selection

Life emerged in primordial oceans characterized by a specific ionic composition. Over approximately 3.5 billion years, organisms adapted to: stable ratios of sodium, potassium, calcium, magnesium, iodine and natural background radiation from primordial isotopes. Adaptive systems, including DNA repair pathways, antioxidant networks, and membrane transporters, evolved under these conditions. Therefore the persistent internal incorporation of anthropogenic isotopes may represent a selective pressure too recent for adaptive refinement [6,7].

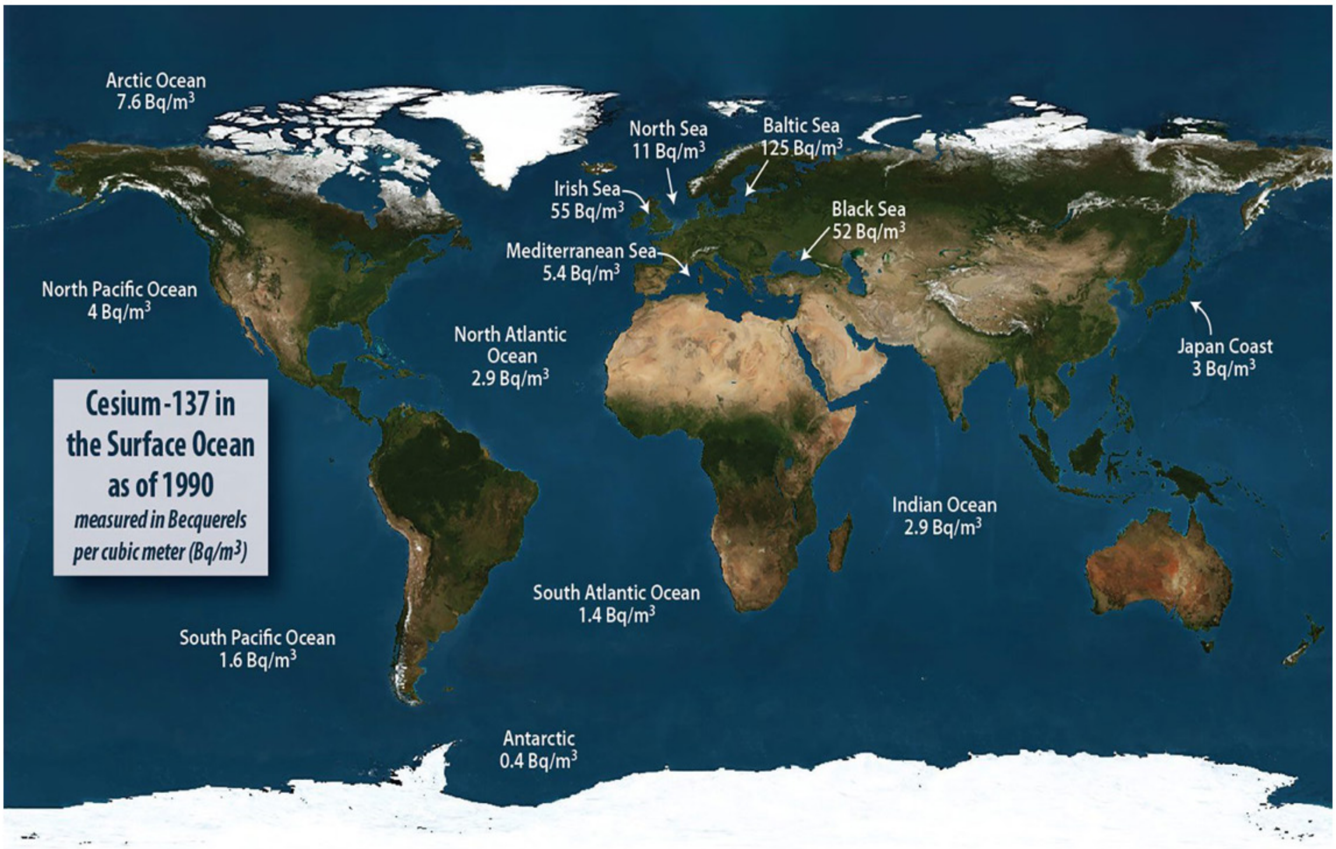

Figure 1: Cesium-137 in the surface oceans as of 1990 (pre-Fukushima)

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How long these specific isotopes remain significant in the terrestrial environment:

Radionuclide	Physical Half-life (T1/2)	Total Environmental Presence (Approx. 10 Half-lives)	Biological Mimicry (Why it enters the body)
Cesium-137	~30 years	300 years	Mimics Potassium; spreads through muscle tissue.
Strontium-90	~29 years	290 years	Mimics Calcium; deposits in bones and teeth.
Plutonium-239	~24,100 years	241,000 years	Not essential; toxic heavy metal and alpha emitter.
Iodine-129	~15.7 million years	157 million years	Mimics Stable Iodine; settles in the thyroid.
Technetium-99	~211,000 years	2.1 million years	Used in medicine, but can accumulate in plants/thyroid.
Neptunium-237	~2.14 million years	21.4 million years	Long-term nuclear waste concern; highly mobile in soil.

Figure 2: Decay Timelines for these Artificial Radionuclides

Ion Mimicry: Cesium and Potassium

Cs-137 shares physicochemical similarities with potassium (K^+), an essential intracellular cation. Potassium plays central roles in: 1- membrane depolarization, 2- enzymatic activation, 3- mitochondrial function, 4- colon, pancreas and arterial wall cells.

Limited partial substitution of cesium within extracellular and intracellular compartments may occur, although homeostatic mechanisms generally restrict sustained accumulation. The hypothesis advanced here suggests that: Cs^+ incorporation may subtly alter intracellular ionic balance, persistent beta and gamma emissions from incorporated Cs-137 may create localized microdosimetric heterogeneity and combined ionic and radiative effects may contribute to cumulative oxidative stress [7,8].

Pancreas with Diabetes, Colon and Arterial Wall as Organs of Interest

These epidemiological disease trends are multifactorial and predominantly also attributed to lifestyle and demographic transitions. Most radiological risk models prioritize malignancy endpoints, particularly leukemia and solid tumors with well-established dose-response relationships. In medical literature, pancreas, colon and arteries are not historically been a primary focus in radiation epidemiology. Yet several features suggest pancreatic vulnerability: high vascular perfusion, elevated mitochondrial density, oxidative sensitivity of beta-cells and the dual endocrine and exocrine functions too. Colon, pancreatic and vascular cells exhibit relatively low intrinsic antioxidant enzyme expression compared to other tissues. This makes them particularly susceptible to oxidative stress. The hypothesis proposed is that chronic internal low-dose irradiation combined with ionic perturbation could act as a long-term co-factor in cell dysfunction, in pancreas, in colon and arterial microenvironmental instability. This does not assert causality. Rather, it proposes an expanded research focus beyond classical cancer endpoints [8,9].

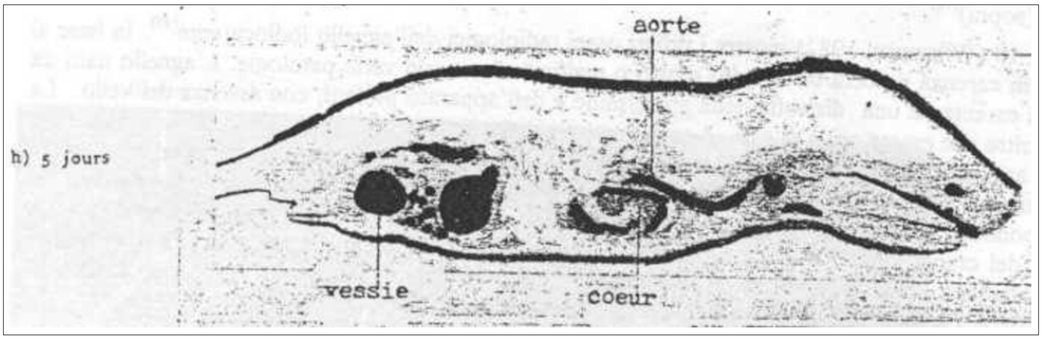

Figure 3: Distribution of iodine-131 (half-life: 8 days) in black in radioautographies of the body of a rat after a subcutaneous injection of radioiodine. High I-concentration is evident in gastric mucosa, intestine, colon and arterial wall of aorta , where it is detectable up to 5 days after the injection. (from Pellerin,1961; Courtesy of Path Biol) [10].In 1964, high iodine uptake in arterial walls was confirmed by Ullberg and Ewaldsson [27].

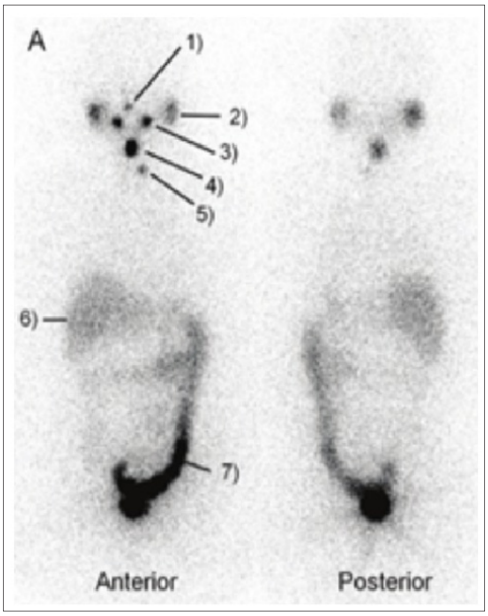

Figure 4: Physiologic uptakes of radioiodine in the whole body scintigraphy in nasal secretion (1), parotid gland (2), thyroid tissue (5), liver (6) and pancreas (not very visible here) and colon (7) are observed. (From Oh , 2012) [11].

Differences in Institutional Mandates between WHO and the International Atomic Energy Agency (IAEA) Have led to Distinct Emphases in Risk Communication and Radiological Protection Frameworks

WHO and IAEA have an historical tension regarding the perception of risks from low-dose radiation. IAEA tends to downplay the damage of low doses, focusing primarily on "deterministic" (acute) effects. WHO, ideally should follow the ‘precautionary principle’, but it is often aligned with UNSCEAR reports, which critics view as overly conservative. The author recently (2025) argued that the IAEA and WHO-limits are based on obsolete mathematical models that ignore selective bioaccumulation and uptake of cesium in specific organs, such as the pancreas, salivary glands , intestine and colon, causing inflammatory damage, pancreatitis and cancer. These authorities often only count immediate deaths or certain cancers (as thyroid cancer which has high morbidity but very low mortality), ignoring long-term genetic damage and the chronic illnesses (as diabetes, arterial hypertension associated with atherosclerosis in hyperlipidemic and hypothyroid people with hypercholesterolemia) reported in contaminated people) [11]. In 2025, the FDA has recalled from the U.S. market frozen shrimp, produced in Indonesia because they were contaminated with Cs-137, for reasons that are not yet known.

The Low-Dose Controversy

Regulatory bodies such as the FDA, IARC, and IAEA generally rely on the Linear No-Threshold (LNT) model for radioprotection. The LNT framework assumes that cancer risk increases proportionally with dose, without a safe threshold. However, this model primarily addresses stochastic carcinogenic outcomes. It does not comprehensively evaluate: chronic internal emitters versus external exposure, microdosimetric heterogeneity within specific organs and long-term metabolic or endocrine outcomes. The disagreement articulated in this position paper concerns two points: 1. whether low-dose chronic internal exposure may exert non-cancer biological effects not adequately characterized; 2. whether current regulatory emphasis may underestimate tissue-specific metabolic vulnerability. However, the international agencies do not recognize a causal relationship between environmental Cs-137 exposure and diabetes, atheroslerosis, pancreas and colon cancer and epidemiological findings remain heterogeneous. Nevertheless, scientific disagreement over model sufficiency does not equate to rejection of the principles of radioprotection. In 2020, Hauptmann and other 15 international researchers of 8 nations and from Institutes of Biostatistics, Registry Research, Centers of Cancer Epidemiology, Radiation Epidemiology, U.S. National Cancer Institute (NCI), International Agency for Research on Cancer (IARC), and Radiation Effects Research Foundation of Hiroshima, conducted a study. They investigated definitively through meta-analysis the damage resulting from the "low doses", that have affected the survivor populations of the atomic bomb explosions in Hiroshima and Nagasaki, and also in numerous accidents of nuclear plants that have occurred in the world. These scientists reported in JNCI Monographs: Epidemiological Studies of Low Dose Ionizing Radiation and Cancer Risk, that the new epidemiological studies directly support excess cancer risks from low-dose ionizing radiation [13].

Russian and Japanese Research

Following nuclear contamination events, some Russian and Japanese researchers reported associations between radionuclide exposure and metabolic or endocrine disorders, including increased diabetes prevalence too. Nowadays these findings have not achieved international consensus validation, but have not expressly denied. However, they contribute to a body of literature suggesting that metabolic endpoints merit systematic re-evaluation. [14,15].

Tissue Uptake and Tumor Trend Argument

An additional argument raised within this framework concerns tissue uptake patterns. Soft tissues and skeletal muscle account for a relatively small fraction of overall tumor incidence (commonly estimated below 1% for primary soft-tissue and musclular sarcomas). This raises a question: if distributed radionuclides primarily localized uniformly in muscle mass, would we not observe higher incidence patterns there? This reasoning suggests that organ-specific susceptibility, rather than simple mass distribution, may influence outcome patterns. However, even if tumor incidence reflects complex biological variables including cell turnover rates, DNA repair capacity, and microenvironmental factors, the significant increase of incidence of colon, pancreatic cancer, diabetes an atherosclerosis too, is also stimulating and supports further necessary inquiry.

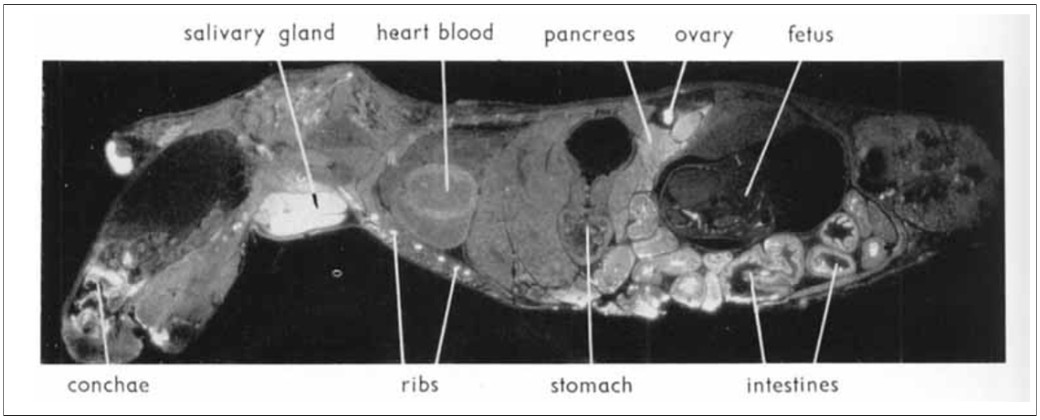

Figure 5: Autoradiogram showing the distribution of Cs-137 in a pregnant mouse 6 min after intravenous injection. White areas correspond to high radioactivity. High uptake is present initially, above all, in in the salivary gland, intestine, colon and ovary. The pancreas shows the same high level of activity as the intestinal mucosa and colon. (from Nelson with permission of Acta Radiologica, 1961) [12]

^{137}Cs level in the organs of children exposed to Chernobyl fallouts (adapted from [2]).

	1	2	3	4	5	6
Cause of death	**(Sepsis)**	**(Premature malform.)**	**(Sepsis bleeding)**	**(Cerebral malform.)**	**(Cardiac)**	**(Sepsis)**
Organ:						
Heart	**5333***	4250	625	**4166**	1071	1491
Liver	250	277	525	851	882	1000
Lung	1125	2666	400	1195	1500	2610
Kidneys	1500	1687	259	2250	812	583
Brain	3000	1363	305	90	1693	714
Thyroid gland	4333	6250	250	1900	n.d.	1583
Thymus	3000	3833	1142	**3833**	714	833
Small intestine	2500	1375	571	3529	2200	590
Large intestine	3250	3125	261	3040	**4000**	2125
Stomach	3750	1250	**1500**	n.d.	n.d.	n.d.
Spleen	3500	1500	428	1036	2000	2125
Adrenals	1750	2500	n.d.	2500	4750	2619

Pancreas	**11000**	**12500**	1312	n.d.**	n.d.	**2941**

Notes: *in bold are the highest levels found in other organs but the pancreas, and Underlined are the cases when the pancreas features a still higher level; ** not determined.

Figure 6: Cesium-137 level in the organs of children exposed to Chernobyl fallouts. Note the very high uptake activity of the pancreas, greater than that of other organs and up to 40-45 times greater in the liver (from Bandazhevsky, 2003) [16].

Diabetes and other Non-Communicable Diseases in Populations Following the Fukushima Disaster

After the Fukushima disaster in Japan , from 2009 to 2020, in affected populations, Murakami reported changes in the prevalence of major non-communicable diseases (NCDs), including hypertension, hyperlipidemia, diabetes, and also mental disorders [15]. Many authors stated that this increase was ongoing and had not yet peaked. Additionally, diabetes mellitus (“Type 3c” of pancreatic origin) and atherosclerosis which increased in the contaminated population of Chernobyl and Fukushima are a multifactorial diseases driven by genetic susceptibility, lifestyle and inflammatory stressors and β-cell failure is strongly linked to oxidative and mitochondrial stress and are particularly vulnerable. Low-doses ionizing radiation exposure is an emerging causal risk factor for cardiovasculardisease and radiation risk of cardiovascular diseases are reported in the cohort of russian emergency workers of the Chernobyl accident [16,17].

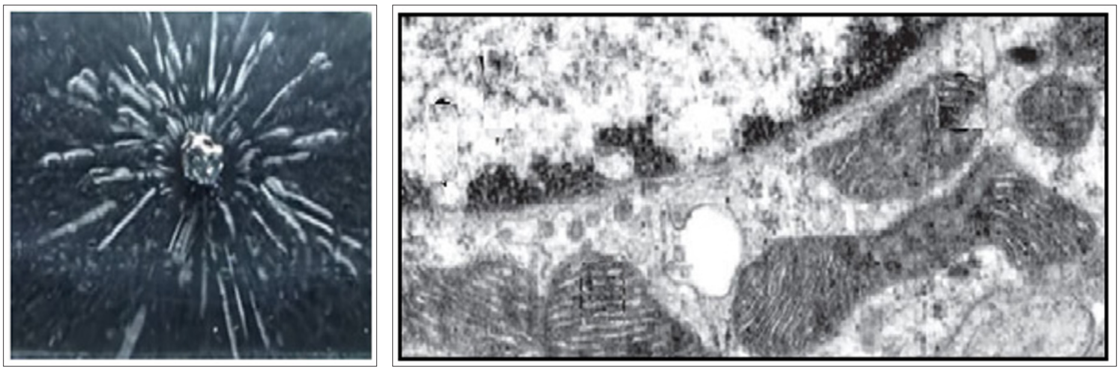

Figure 7: Sx: Radioactive cloud chamber of Uranium. DX: ionizing radiations of Cs-137 cause cytoplasmic vacuolization, dilatation of the endoplasmic reticulum and destruction of mitochondria of various sizes and morphology, and dense areas of chromatin (DNA) was observed at the periphery of the nucleus. (From Venturi, 2025, and Boraks, 2008)

The article explores the CCPDA-hypothesis that chronic low-dose internal exposure to Cs-137 may contribute to pancreatic β-cell dysfunction, diabetes and atherosclerosis too. Following ingestion, internalized Cs-137 emits beta particles and gamma radiation, resulting in continuous low-intensity internal irradiation. β-emissions result in continuous low-intensity irradiation of subcellular structures, particularly DNA and mitochondria. Following the Chernobyl disaster, the incidence of type 1 diabetes mellitus in children and adolescents increased by 25%. Ito revealed a 2.1-fold increase in male prevalence and a 2.0-fold increase in female prevalence in Japan in mass diabetes screening of adult survivors of the Hiroshima bomb between 1971 and 1992 [18]. Iannuzzi (2022) reported an high acute pancreatitis global incidence in Nations, were we showed high diabetes and environmental Cs. Pellerin reported a high quantity of I-131 in radioautography of arterial in wall of aorta, where it is detectable up to 5 days after the injection, after a subcutaneous injection of radioiodine. (Figure 3). [10].

Martinucci et al. observed an increased risk of type 1 diabetes in Gomel after the accident compared with before it [21]. Subsequently, a more detailed study by Zalutskaya et al. also found a significant increase in diabetes in children and adolescents in a more contaminated area compared with a less contaminated area [22]. Chung confirmed that the incidence of diabetes increased six years after the Fukushima-Daiichi disaster [23].

Iodine as an Evolutionary Counterpoint

Iodine occupies a unique evolutionary position. Beyond thyroid hormone synthesis, it has documented antioxidant and halogen-mediated biochemical roles. The conceptual contrast proposed here frames: natural halogens (e.g. iodine) as evolutionarily integrated elements and artificial radionuclides as evolutionarily novel intrusions. This metaphorical distinction, sometimes described as “protective vs disruptive elements”, is heuristic rather than doctrinal. It underscores the broader evolutionary mismatch thesis [4-6].

We tried to calculate, using publicly available historical datasets and radiological decay models, with the help of ChatGPT, the continuous and progressive quantity of Cs-137 produced from 1945 to the present day, taking into account of documented atmospheric testing, industrial accidents and clandestine activities. From the above concentration data of radioactive cesium in the seas, and from the secret nuclear tests, submarine accidents, illegal dumping, lost sources, we must consider the sediment sink and deep water sequestration more than 1100 PBq.

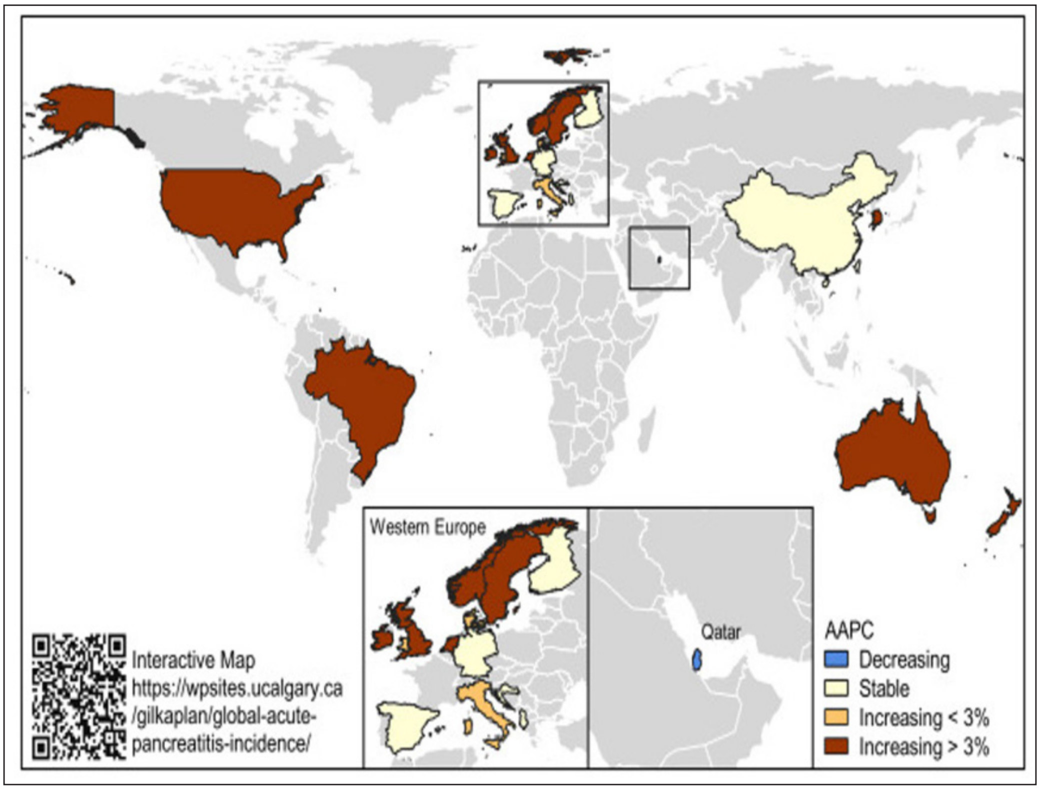

Figure 8: Acute pancreatitis global incidence map: direction of change based on statistical significance of a country's average annual percentage change [19]. Countries with a statistically significant increase in AAPC (average annual percent change) were further stratified by whether the increase was above or below 3% per year. (Iannuzzi, 2022) [20]

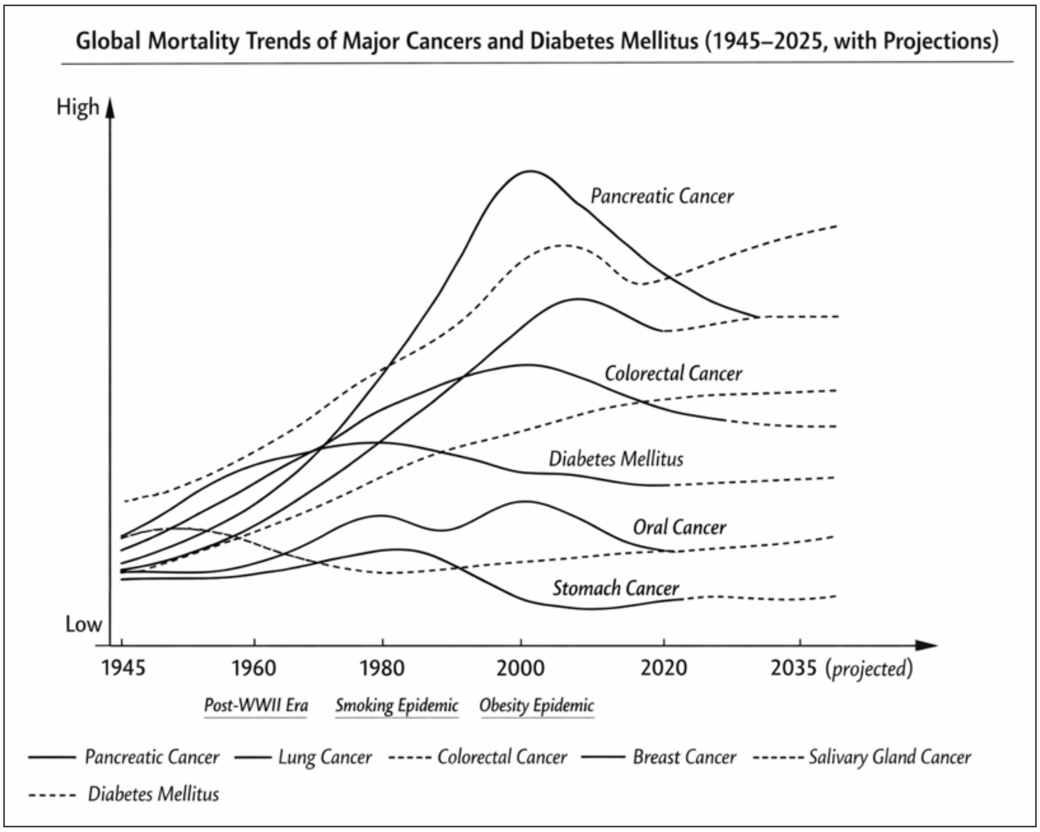

Figure 9: Temporal trends of deaths caused by the most frequent cancers and diabetes mellitus (M+F) and their forecast worldwide from 1945 to 2035 (From ChatGPT- 2026) [8,9]

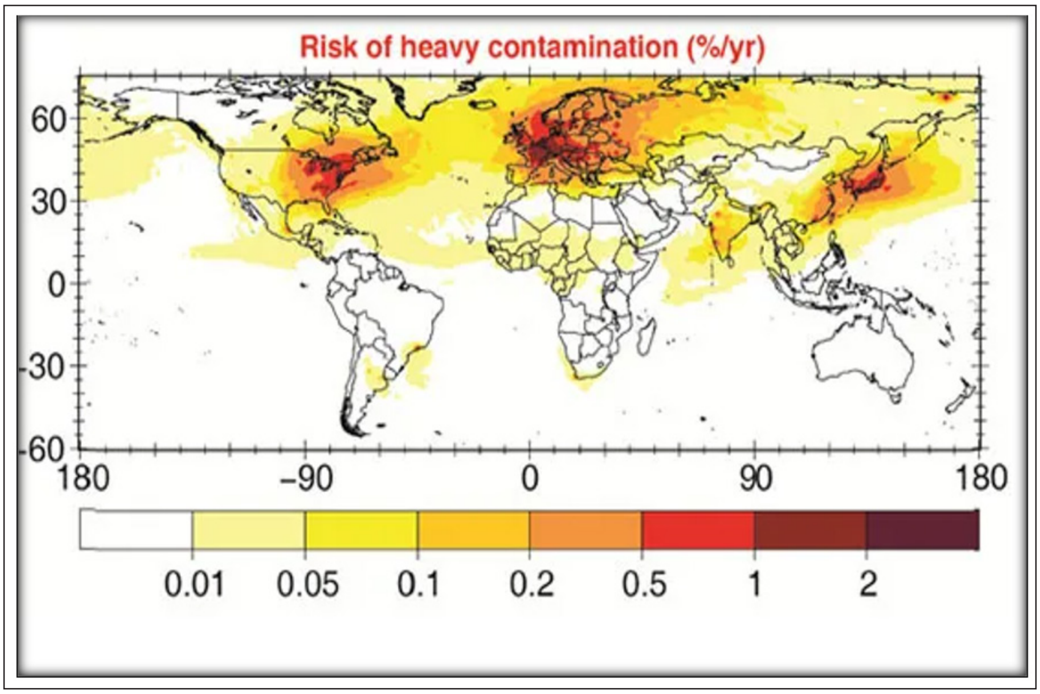

Figure 10: Spatial distribution of Chernobyl radionuclides in the Northern Hemisphere 10 days after the explosion. U.S. Livermore National Laboratory modeling (Yablokov et al, 2009) [14]

Conclusion

Artificial radionuclides represent a historically recent addition to Earth's elemental environment. From an evolutionary perspective, their persistence raises legitimate scientific questions regarding long-term biological integration and tissue-specific vulnerability. The colon, pancreatic and arterial walls vulnerability hypothesis does not claim established causality. Rather, it calls for expanded radiobiological inquiry beyond traditional pathogenetic endpoints, integrating evolutionary biology, metabolic pathology, and internal dosimetry. The nuclear age introduced new isotopes into the biosphere. Whether these isotopes exert subtle long-term metabolic consequences remains an open scientific question, one that warrants rigorous, methodologically sound investigation [24].

Resilience and the Perspective on Life Return

While it may feel "unscientific" to talk about hope in a radioactive land, we hope that the life can treat radiation as a resource rather than a death sentence observing the Resilience of Life at Chernobyl as a Biological Adaptation in the discovery of radiotrophic fungi (like Cryptococcus neoformans). These organisms don't just survive radiation, they "eat" it, using melanin to convert gamma rays into metabolic energy; and moreover, the thriving populations of wolves, lynx, and wild horses prove a bittersweet scientific truth: human presence is often more destructive to biodiversity than chronic radiation [25,26,27].

Funding

The author declares that he not received funding and he has no conflicts of interest for this study.

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