

From the Birth of Atoms to Life: Iodine and Caesium as Evolutionary Protectors and Anthropogenic Disruptors in Pancreatic Cancer and Diabetes

On The 40th Anniversary of the Chernobyl Nuclear Disaster and In Remembrance of the Heroic Sacrifice of the Russian Liquidators

Dedicated to the memory of Alexey V. Yablokov(1933-2017) and Valery A. Legasov (1936-1988), whose scientific and ethical contributions highlighted the long-term biological consequences of nuclear technologies

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ABSTRACT

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. J. Robert. Oppenheimer evoked the famous 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 matter. 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, cesium-137 and **strontium-90** mimic these essential elements but differ in radiophysical properties, exploiting conserved transport pathways, creating cellular vulnerabilities and **a radionuclide evolutionary mismatch**. But the total correct amount of iodine (I) in the human body is still controversial and only thyroidal iodine content has been estimated of 5-15 mg. The World Health Organization hypothesized the total content to be only 15-20 mg, with 70–80% in the radiation-insensible thyroid gland and a more little part 20–30% in extrathyroidal tissues. Recently (2020), Venturi tried to calculate the amount of extrathyroidal and pancreatic iodine uptake -(at 20 hours from i.v. injection), using sequential scintigraphies, with the help of AI and reported different values about 30% in the thyroid and 70% in extrathyroidal tissues with about 0.3 mg in whole pancreas. The damage caused by artificial iodine-131 and cesium-137, from now on, it will be present for a long time in the future. These isotopes are chemically mimicking biological elements but differing in their radiophysical properties, and by exploiting conserved cellular transport, create a mismatch between biochemical recognition and radiological behavior. According to agreement of 1959, WHO cannot publish on nuclear radiation without consulting the IAEA and loses scientific independence in radiation protection, so many WHO-assessments are conservative towards industry. “Food Standard Safety Limits” report that cesium-137, which persists for over 300 years in the environment, after disaster of Chernobyl and Fukushima, are revised downward, to apply different “Precautionary Principle”. Japan lowered the limits from 500 to 100 Bq/kg, while Europe maintain 600 and USA-FDA 1200 Bq/kg. These radionuclides interfere with cellular signaling and causing genetic and metabolic disorders as pancreatic cancer and diabetes.

Keywords: Iodine, 137-Caesium-137, Pancreas, Diabetes, Atherosclerosis, CCPDA-Hypotesis, ChatGPT A.I, Radionuclide Evolutionary Mismatch, Arteries, Heart

Foreword

The Chernobyl nuclear accident on 26 April 1986 marked a turning point not only in the history of energy production, but also in our understanding of the relationship between human technology and biological systems. As famously evoked by J. Robert Oppenheimer from the Hindu scripture, “Now I am become Death, the destroyer

of worlds”, nuclear fission represents a profound departure from the natural evolutionary history of matter. Life evolved within a stable isotopic environment shaped by cosmic nucleosynthesis and long-term biological selection, progressively integrating essential elements such as iodine into conserved biochemical, antioxidant and regulatory pathways [1]. The 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, strontium-90 and cesium-137, mimic these essential elements but

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differ in radiophysical properties, exploiting conserved transport pathways, creating cellular vulnerabilities and **a radionuclide evolutionary mismatch** [2]. A recent paper of Alwadi et al. In **Nature Communication, 2026**, repoerted that in **U.S. counties located closer to operational nuclear power plants have higher cancer mortality rates than those farther away**.

The World Health Organization (WHO) hypothesized the total content to be only 15-20 mg with 70–80% in the radiation-insensible thyroid, and 20–30% in extrathyroidal tissues [3]. But the correct amount of iodine (I) in the human body is still controversial and only thyroidal iodine content has been estimated of 5-15 mg (Hays, 2001) [4]. Recently in 2020, Venturi tried to calculate the amount of extrathyroidal and pancreatic iodine, using sequential scintigraphies, with the help of AI and reported different values of about 70% in extrathyroidal tissues, 30% in the thyroid, and 0.3 mg in whole pancreas [5].

Introduction

Radioiodine and Radiocesium vs Iodine and Potassium: Anthropogenic Disruptors and Evolutionary Protectors

Artificial radionuclides such as iodine-131 (^{131}I) and caesium-137 (^{137}Cs) have similar chemical properties to essential elements such as iodine (I) and potassium (K), but differ fundamentally in terms of their radiophysical properties. Iodine-131, which is produced almost exclusively via nuclear fission, has a short half-life of around eight days and emits beta and gamma radiation. Cesium-137 has a longer half-life of ~30 years, meaning it remains present in the environment for over 300 years. It decays by gamma and beta (electron) emissions, producing highly ionising radiation [2].

Internal beta emission is very dangerous because it deposits all its energy within a distance of just 3–4 mm (several hundred cells of pancreatic, salivary and internal organs). Ionising radiation can irreversibly break the DNA backbone or disrupt the hydrogen bonds of the complementary nucleic acids strands, thereby preventing natural repair processes such as those involving BRCA-1 and BRCA-2. Both these radionuclides exploit evolutionarily conserved transport

pathways: iodine via the sodium-iodide symporter (NIS), and cesium via potassium channels. This gives them access to tissues that evolved to utilise the stable isotopes. Pancreas, particularly the insulin-secreting beta cells, is metabolically active and sensitive to oxidative stress. Introducing radionuclides that follow the same chemical pathways as essential elements may compromise the cellular redox balance. This can lead to impaired insulin secretion and increased oxidative damage. It can also contribute to genetic and metabolic disorders, such as diabetes. Additionally, chronic oxidative stress is a recognised co-factor in carcinogenesis, suggesting a potential mechanistic link between exposure to these radionuclides in the environment and an increased risk of cancer in metabolically active tissues. This integrative perspective emphasises the dual nature of essential elements: while they provide evolutionary protection under stable isotopic conditions, they may also render tissues vulnerable when anthropogenic isotopes exploit their biochemical pathways. Understanding this duality is crucial for evaluating environmental health risks in populations affected by radionuclide contamination and for devising preventive measures, such as optimising dietary iodine and 'stable' potassium intake. This highlights the importance of integrating evolutionary biochemistry, environmental radiology and metabolic physiology in order to assess long-term health risks. Strategies such as optimising dietary iodine and potassium intake may mitigate radionuclide uptake and protect vulnerable tissues, particularly thyroid, salivary glands and pancreas [5].

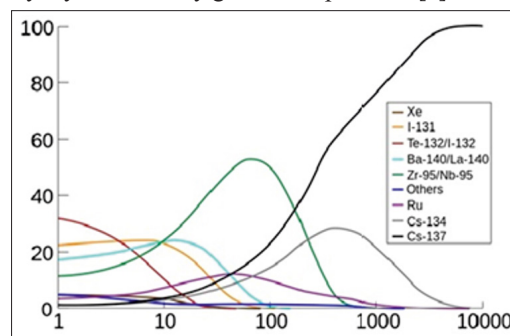


Figure 1: Relative Contributions of Major Nuclides to Radioactive Air Contamination after an Accident [2].

Iodine

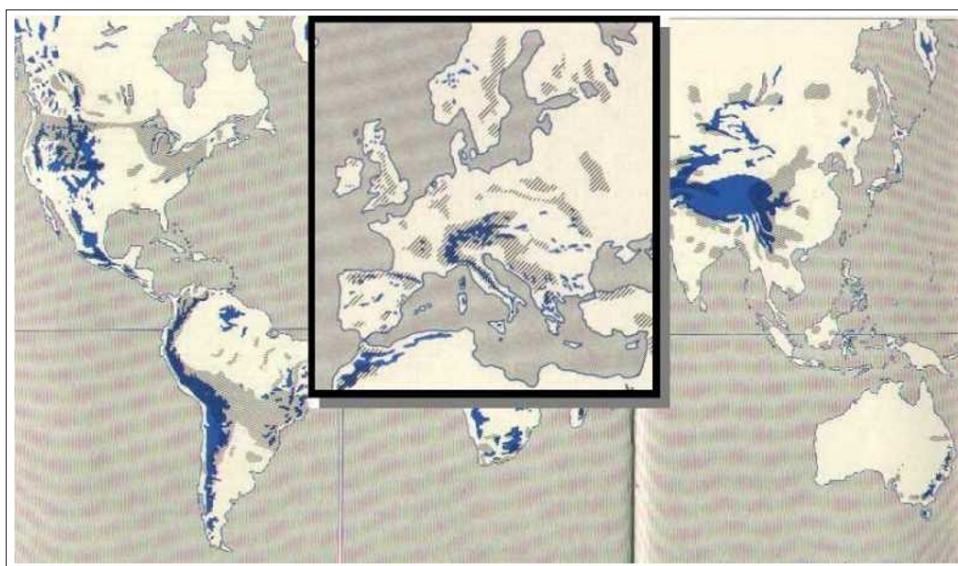


Figure 2: Map of the World's Iodine-Deficient areas (Shaded by Oblique Lines), with Populations Suffering from Iodine Malnutrition with its Specific Clinical Marker: The Goiter. The Planet's Mountainous areas (in dark) are the Most Iodine-Deficient, due to Millennia of Leaching, and where the use of Iodized Salt is Recommended as an Effective Prophylaxis. Some Coastal Regions of Japan and China have Iodine-Excess Endemic Goitre Caused by a Diet Richest in Marine Algae. (WHO, 2009).

Iodine is the only element, essential for life of humans and animals, that have a specific, visually diagnosable, specific clinical marker: goiter, which has been well mapped worldwide by the WHO, since the 1970s Figure. 2. The World Health Organization hypothesized the total content to be only 15-20 mg with 70–80% in the radiation-insensible thyroid, and 20–30% in extrathyroidal tissues. But the correct amount of iodine (I) in the human body is still controversial and only thyroidal iodine content has been estimated of 5-15 mg (Hays, 2001) [4]. Recently (2020), Venturi tried to calculate the amount of extrathyroidal and pancreatic iodine uptake (at 20 hours from i.v. injection), using sequential scintigraphies, with the help of ChatGPT AI and reported different values of about 70% in extrathyroidal tissues, 30% in the thyroid, with 0.3 mg in whole pancreas [5].

But WHO seems considers not important the extrathyroidal iodine compartments, as the mammary glands, stomach, salivary glands, pancreas, thymus, arterial wall, colon, pancreas, ovary, choroid plexus, articulations, liquor, eye, skin and others, that really share the same thyroidal gene expression of sodium / iodide symporter (NIS) and are capable of iodine uptake [6]. The author reports here a human scintigraphy where is well visible the great quantity of extrathyroidal radioiodine, in not breastfeeding thyroidectomized woman, so without the greater mammary quantity of radioiodine, necessary for fetal and neonatal development [7].

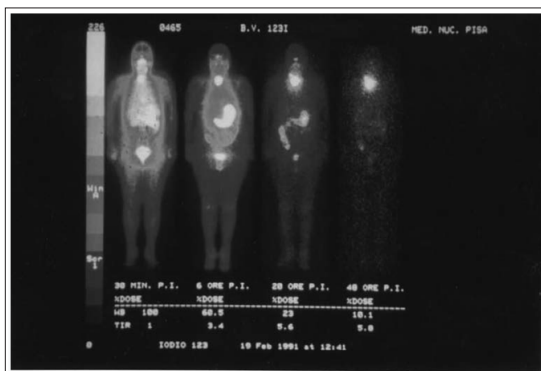


Figure 3: Sequence of 123-Iodide Total-Body Scintiscans of a Woman after Intravenous Injection of 123-Iodide; (from left) Respectively at 30 min, and at 6, 20, and 48 h. Here, Iodide-Concentration by the Mammary Gland is not Evident Because this Woman was not Lactating. A High Excretion of Radio-Iodide is Observed in the Urine. Venturi, with the Calculation of AI, Reported Different Values of Iodine uptake (at 20 hours from i.v. injection): about 70% in Extrathyroidal Tissues and 30% in the Thyroid Gland [5].

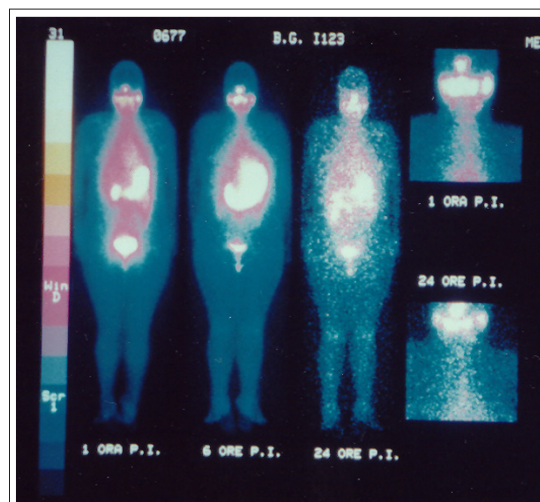


Figure 4: Sequence of 123-I Total-Body Scintiscans of a Thyroidectomized Woman, who is not Breastfeeding, after Intravenous Injection of 123-I; (from left) Respectively at 1, 6 and 24 Hours. Highest uptake of Radio-Iodide (in white) is Evident in Salivary Glands, Gastric and Oral Mucosa, Pancreas, Colon, Ovary ecc. (In stomach 131-iodide, would be Evident for more than 72 h.). In the Thyroid, I-concentration is more Progressive, as in a Reservoir (from 1% [after 30 min] to 5.8% [after 48 h] of the Total Injected Dose).

Upper right: I-uptake in salivary glands and oral mucosa after 1 hour.

Bottom right: I-uptake in salivary glands and oral mucosa after 24 hours. Excretion of radioactivity is also seen by the kidneys with accumulation in the bladder (Venturi, 2009.)

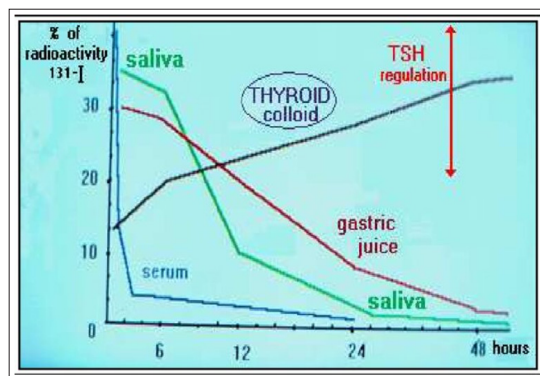


Figure 5: Percentage of Radioactivity in Serum, Gastric, Salivary Secretions and in Thyroid Colloid, after Intravenous Injection of i-131. Secretion of Iodine is Higher and Prolonged in Milk, During Breastfeeding, and in Pancreatic Juice it is Slightly Lower than the Salivary One [7].

Iodine in Evolution: From Marine Antioxidant to Endocrine and Metabolic Regulator

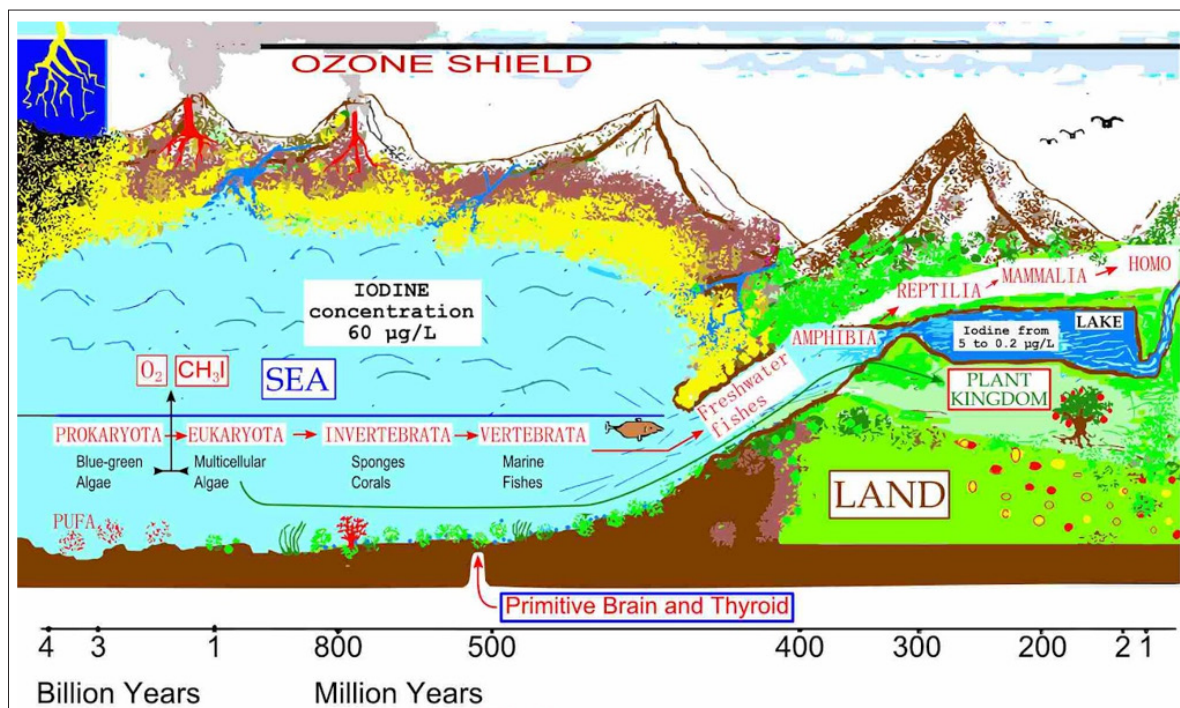


Figure 6: Iodine in Evolution. Over 3 Billion years ago (BYA), in the Primitive Sea, Blue-Green algae were the first Living Prokaryotes to Produce Oxygen and Halo-Carbons (such as CH_3I) in the Atmosphere, and also PUFAs in Lipid Membranes. About 500 Million years ago (MYA), when the Primitive Brain Evolved in Marine Animals, Thyroid Cells Originated from the Primitive Foregut in Vertebrates, Migrated and Specialized in uptake and Storage of Iodo-Compounds in a Novel Follicular “Thyroidal” Structure, as a Reservoir for Iodine. 350 MYA, Some Vertebrates Evolved into Amphibians and Reptiles when they Colonized the I-Deficient land in Vertebrates thyroid.

Over three billion years ago, blue-green algae (Cyanobacteria) were the first living prokaryotes to produce oxygen and halocarbons (such as CH_3I) in the atmosphere, as well as polyunsaturated fatty acids (PUFAs) in lipid membranes. 500 million years ago, when the primitive brain evolved in marine animals, thyroid cells originated in the primitive foregut of vertebrates. These cells then migrated and specialised in the uptake and storage of iodo-compounds in a novel follicular ‘thyroidal’ structure, acting as a reservoir for iodine. Around 350 million years ago, some vertebrates evolved into amphibians and reptiles when they colonised iodine-deficient land. In vertebrates, thyroid hormones became active in metamorphosis and about 200 Ma, in thermogenesis as an adaptation to the terrestrial climatic variations [8]. Hundreds of millions of years, iodide became integrated into biological systems, particularly in ancestral marine photosynthetic organisms (Cyanobacteria) and algae, where oxygen production and oxidative stress are high. One key adaptation is the formation of iodinated lipids, which protect polyunsaturated fatty acid-rich membranes against peroxidation [9].

Iodide as Primitive Evolutionary Antioxidant in Lipids of Cellular Membranes

The first lipids appeared on Earth approximately 4,000–3,500 million years ago. Initially, in an oxygen-free environment dominated by simple chemical reactions, lipids were predominantly saturated. Unsaturation is a later biological innovation, linked to the need to regulate membrane fluidity when environmental temperatures varied. Iodide became integrated into biological systems over hundreds of millions of years, particularly in marine ancestral photosynthetic organisms

(Cyanobacteria) algae where oxygen production and oxidative stress is high. One key adaptation is the formation of iodinated lipids, which are in polyunsaturated fatty acid-rich membranes against their peroxidation [FIG. 4] [Venturi, 2000, 2007]. These molecules stabilize membrane integrity, reduce reactive oxygen species (ROS), and support redox-sensitive signaling pathways crucial for intracellular and intercellular metabolism of well-known “lipid language” as Arachidonic Acid Pathway (Leukotriene, Prostaglandin, Thromboxane Synthesis) [10-15].

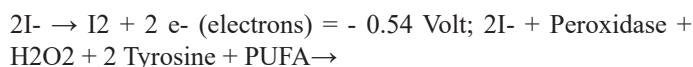


Table A: Antioxidant Biochemical Mechanism of Iodides (Venturi, 2011).



Table B: Antioxidant Biochemical Mechanism of Iodides, Probably the Most Ancient Mechanisms of Defence from Poisonous Reactive Oxygen Species (ROS). (Venturi, 2011).

In vertebrates, I-uptaking cells of gastric mucosa, salivary, pancreatic and mammary glands can produce iodothyronines, iodoproteins and iodolipids. In chemical terms, this is called ‘iodine number’ and these are often used to determine the amount of unsaturation in PUFAs. This unsaturation is in the form of double bonds which react with I-compounds. Some iodolipids [AA-iodinated called 6-iodolactone (6-IL)] have been shown to

regulate cellular metabolism and several authors have reported their effects on growth inhibition and apoptosis in different tumoral cells [14,15].

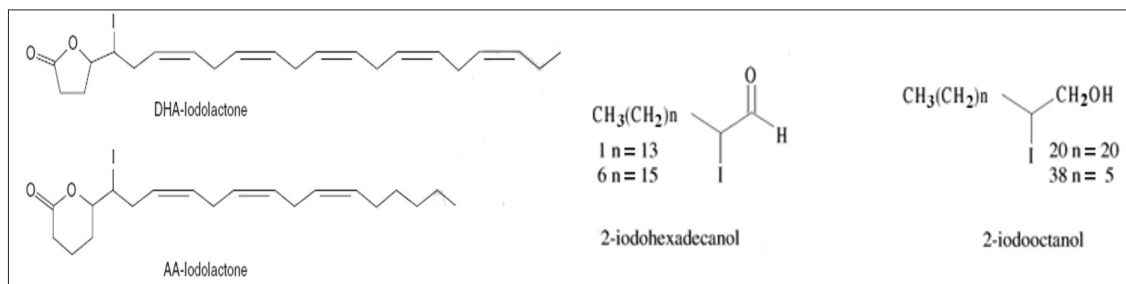


Figure 7: Formulae of Some Best known Iodolipid Molecules [14].

This article will not discuss the remarkable and well-known thyroid damage (thyroiditis and cancers) caused by radioiodine in population (especially iodine-131) that has been discussed by many other researchers. Such damage is very frequent and with high morbidity, but very low mortality. In particular, thyroid tumors are early diagnosed, well treatable and probably mitigated by the large presence in the thyroid of ‘stable’ non-radioactive and antioxidant iodide.

Iodine in Pancreas and Salivary Glands

In normal human pancreas, NIS immunoreactivity is located in ductal cells, exocrine parenchymal cells and Langerhans islet cells, demonstrating also the NIS protein in several exocrine glands and that iodide could have an important functions in their glandular secretions. Iodine acts as a powerful antioxidant, protecting pancreatic cells from ‘self-digestion’ or oxidative stress, which can lead to pancreatitis. Iodine also plays a role in protecting the islets of Langerhans against the risk of glucose intolerance. Acinar cells in the salivary glands and exocrine pancreas have similar histological and biochemical features and secrete the same amylase enzymes and iodides. Radioiodine and radiocesium of radioactive fallout could cause pancreatic and salivary gland cancers, which are both increasing in the world [5,6,16].

Potassium

The sodium-potassium pump is a vital protein found in the membrane of every animal cell. It acts like a cellular “rechargeable battery” by maintaining the electrical and chemical gradients necessary for life.

Radiocesium (Mimic of Natural Potassium)

Cesium-137 decays into Barium-137 and is one of the most concerning byproducts of nuclear fission (from reactors and weapons) due to its water solubility and chemical similarity to potassium, which allows it to easily enter the food chain. Its ecological half-life varies: while it can become trapped in clay soils, in forests and lakes, it continues to recycle indefinitely between soil, plants, and animals. USA, Europe, and Japan, following Chernobyl and Fukushima revised safety limits to apply the “Precautionary Principle”. Japan lowered the limits from 500 to 100 Bq/kg (Becquerels per kg), while Europe maintain 600 and USA (FDA) 1200 Bq/kg [2, 17-20].

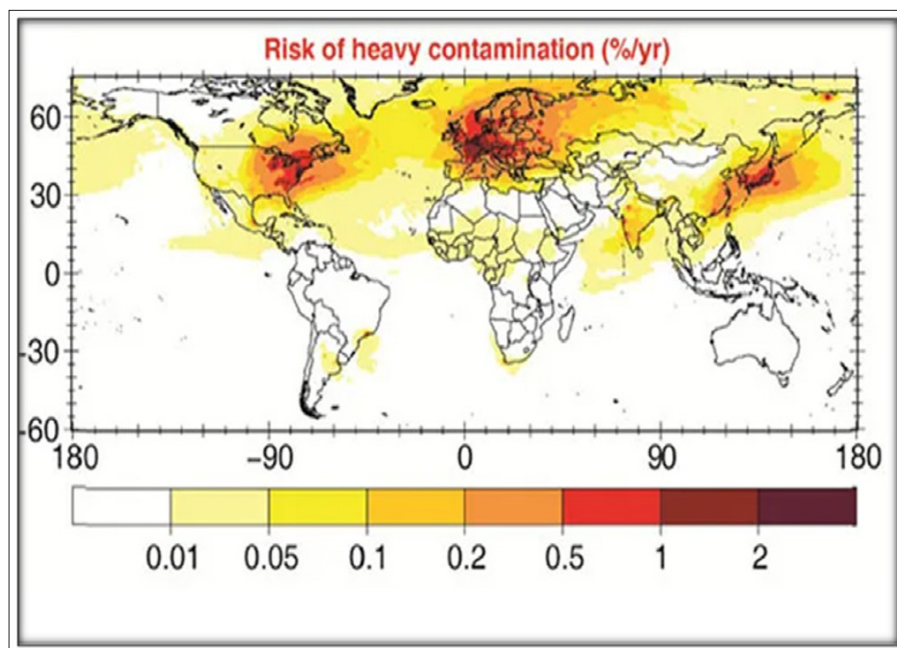


Figure 8: Spatial Distribution of Chernobyl Radionuclides in the Northern Hemisphere, 10 days after the Nuclear Incident. U.S. National Laboratory Modeling. 76% of Total Radiation was Released in the Northern Hemisphere and 24% in the Southern Hemisphere (Yablokov et al, 2009)

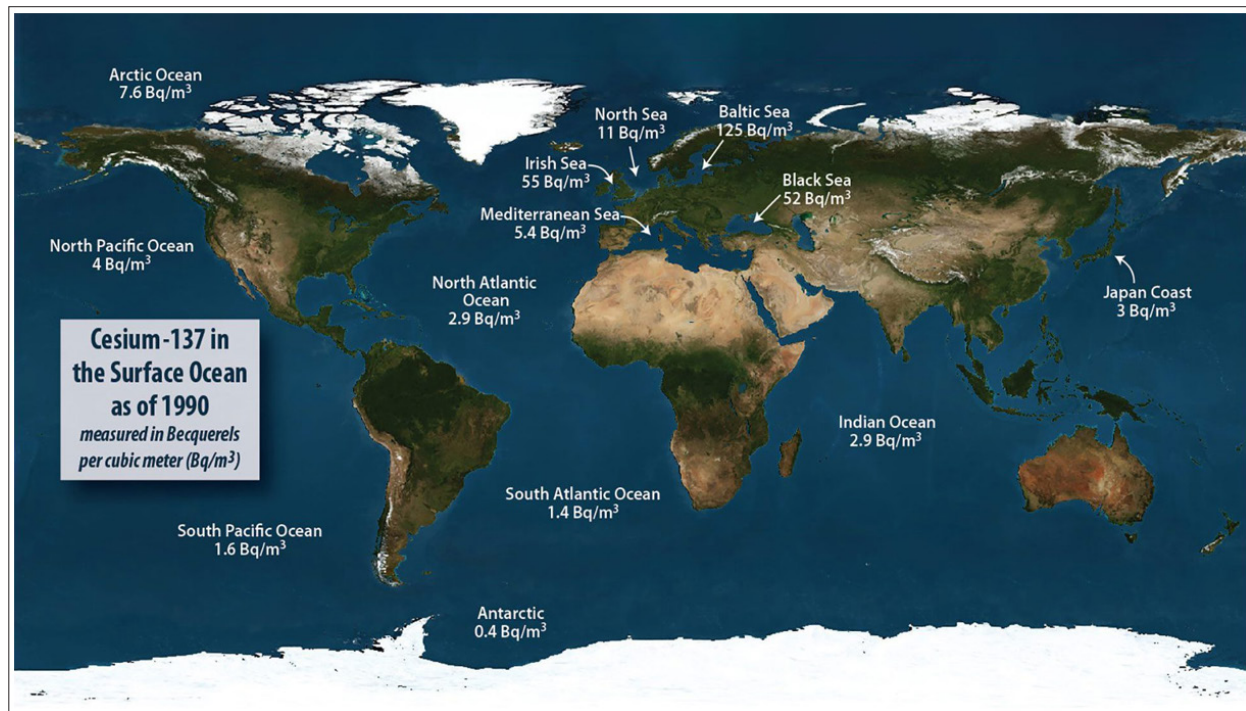


Figure 9: Cesium-137 in the Surface Oceans (pre-Fukushima) [21]. (From WHOI, Woods Hole Oceanographic Institution, 2016) <https://www.whoi.edu>

In the 1960s, immediately after testing on the Pacific atolls ended, the concentration of radioactive cesium in the Pacific off the coast of Japan was about 50 Bq/m³ and 10 Bq/m³ off the coast of California. Off the coast of Japan, aside from the extremely high levels detected at the point of release from the reactors, we recorded a maximum of 4,500 Bq/m³ after the accident.

Continuous low-level industrial discharges into the North Atlantic. The “expected” global inventory today should be roughly 25–30% of the original 1960s peak.

We can attempt to estimate 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. [21].

Cesium-137 in Pancreas

The pancreas is a metabolically very active organ. Despite weighing only 80–100 g in humans, it produces 500–2,000 ml of pancreatic juice daily, has high blood perfusion from the pancreatic arteries and can accumulate a considerable amount of radioactive cesium and iodine inside its cells. The pancreas expresses strong NIS protein (Na^+/I^- symporter) expression in islet cells, despite being weakly expressed in parenchymal cells and ductal cells. Salivary system and pancreas, both rich in iodine, have similar histological and biochemical feature and secrete same amylase enzymes, cesium and iodide also iodide which protects Islets of Langerhans and acinar and duct cell from oxidative damage. Their mortality cancer are worldwide both increasing [22,23].

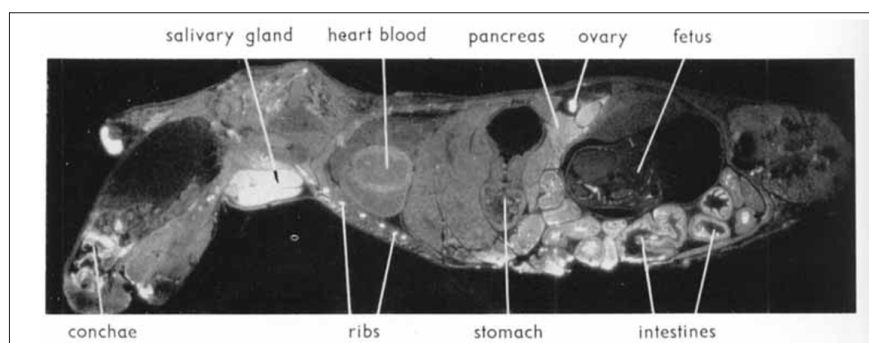


Figure 10: Autoradiogram Showing the Distribution of ^{137}Cs in a Pregnant Mouse 6 min after Intravenous Injection. White areas Correspond to High Radioactivity. High uptake is Present Initially, above all, in in the Salivary Gland, Stomach, Intestine, Colon and Ovary. The Pancreas Shows the Same High Level of Activity as the Intestinal Mucosa and Colon. (Reproduced from Nelson with permission of Acta Radiologica, 1961).

Nelson reported in old (1961) autoradiographies of a pregnant mouse high Cs-137 uptake in salivary gland, intestine, colon and particularly in pancreas, where the islets of Langerhans appear to have a slightly lower activity than the acinar tissue [24].

Bandazhevsky performing autopsies of six children who died from different causes in the polluted area near Chernobyl, showed an high 137-Cs-radioactivity of in pancreatic and endocrine glands and also in thyroid and adrenal glands. He measured a 137-Cs level in the pancreas 40-45 times higher than in the liver [25,26].

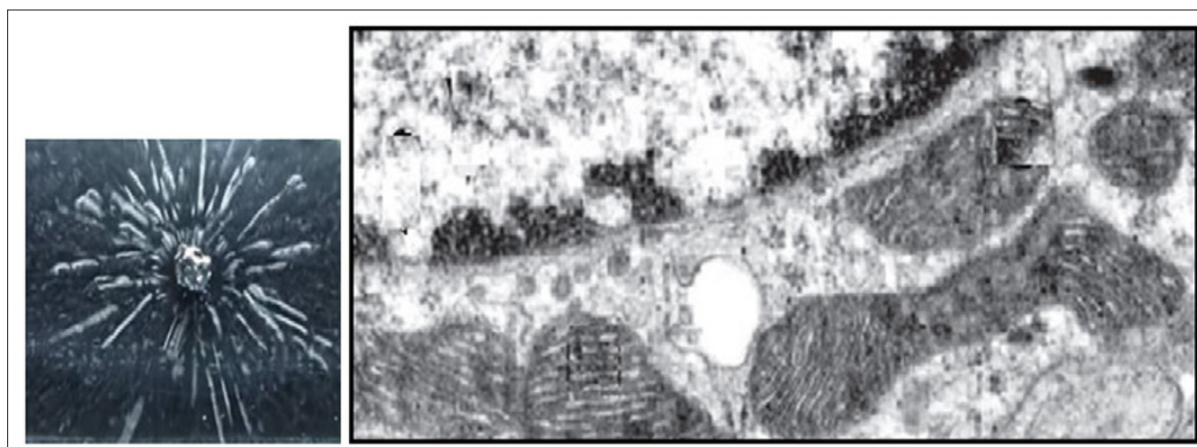


Figure 11: Sx: Radioactive Cloud Chamber of Uranium. DX: Ionizing Radiations of 137-Cs Cause Cytoplasmic Vacuolization, Dilatation of the Endoplasmic Reticulum and Destruction of Mitochondria of Various Sizes and Morphology, and Dense areas of Chromatin (DNA) was observed at the Periphery of the Nucleus. (From Venturi, 2025, and Boraks, 2008)

Pancreatitis

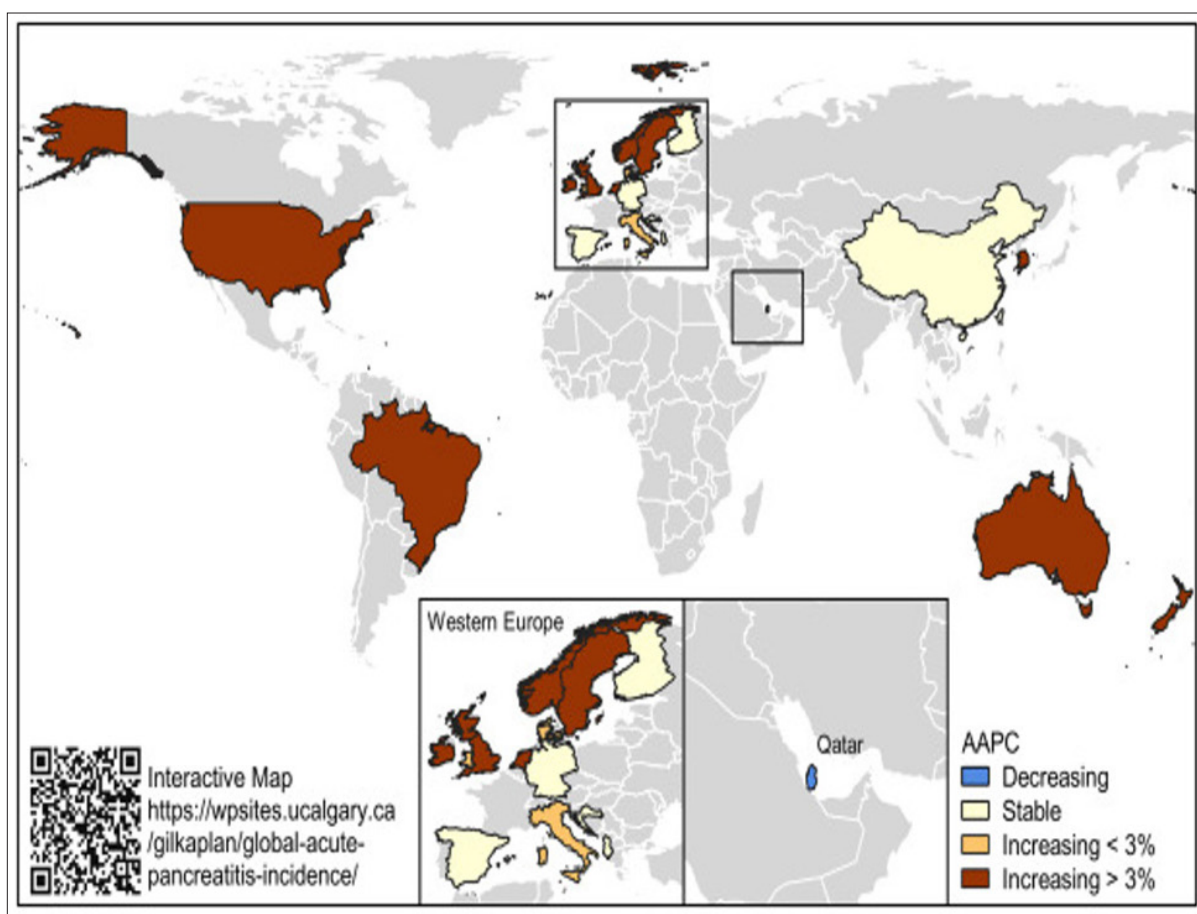


Figure 12: Shows the Global Incidence of Acute Pancreatitis, with the Direction of Change based on the Statistical Significance of a Country's Average Annual Percentage Change. Countries with a Statistically Significant Increase in AAPC (average annual percent change) were further Categorised as either above or below a 3% Increase Per Year. (Iannuzzi, 2022).

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

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 [27]. Martinucci et al. [28]. observed an increased risk of type 1 diabetes in Gomel after the accident compared with before it. 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, confirmed that the incidence of diabetes increased six years after the Fukushima-Daiichi disaster. These authors stated that this increase was ongoing and had not yet peaked. Additionally, diabetes mellitus (of pancreatic origin) increased in the contaminated population of Fukushima [29,30].

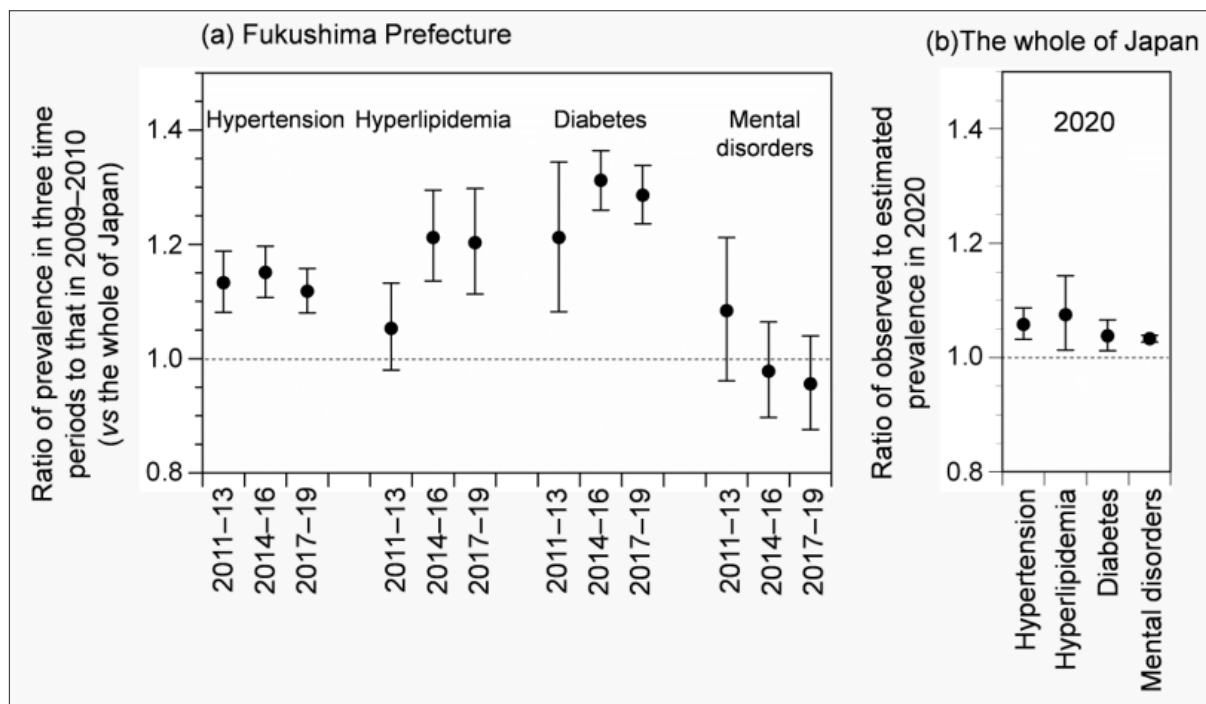


Figure 13: The “Annual prevalence of non-communicable diseases and identification of vulnerable populations following the Fukushima disaster,” in the ‘Intern.J. of Disaster Risk Reduction’ reported a significant and progressive increase of annual prevalence of non-communicable diseases as diabetes, hypertension and hyperlipidemia in Fukushima Prefecture. and also in the whole of Japan, after the Fukushima disaster, compared precedent years of 2009-2010.

Diabetes mellitus is a multifactorial disease driven by genetic susceptibility, lifestyle factors, inflammation, and environmental stressors. Increasing evidence indicates that β -cell failure is strongly linked to oxidative and mitochondrial stress, processes to which pancreatic islets are particularly vulnerable. This article explores the hypothesis that chronic low-dose internal exposure to ^{137}Cs provides a biologically plausible framework to explore the pancreatic β -cell dysfunction and diabetes, a possibility largely absent from pancreatic and endocrine literature. Biological Behavior of Cesium-137 Relevant to the Pancreas Internal irradiation following ingestion, ^{137}Cs distributes systemically and emits: β -particles (dominant) and γ -radiation. When localized intracellularly, β -emissions result in continuous low-intensity irradiation of subcellular structures, particularly mitochondria. Unlike external radiation, internal β -emitters generate heterogeneous microdosimetric patterns, with localized energy deposition near DNA and mitochondria [32].

After the Fukushima disaster in Japan, from 2009 to 2020, in affected populations Murakami et al. reported changes in the prevalence of major non-communicable diseases (NCDs), including hypertension, hyperlipidemia, diabetes, mental disorders and atherosclerosis (FIG.14) and colon diseases too (FIG.15) [35,36].

Trends of Deaths by the Most Frequent Cancers

Pancreatic cancer is currently following a concerning upward trend that sets it apart from many other major cancers. While mortality rates for lung, breast, prostate and partially colon cancer have been declining due to better screening and treatments, pancreatic cancer is moving in the opposite direction. As of early 2026, the data confirms your observation: pancreatic cancer is on track to become the second leading cause of cancer-related deaths by 2030. (WHO, 2025)

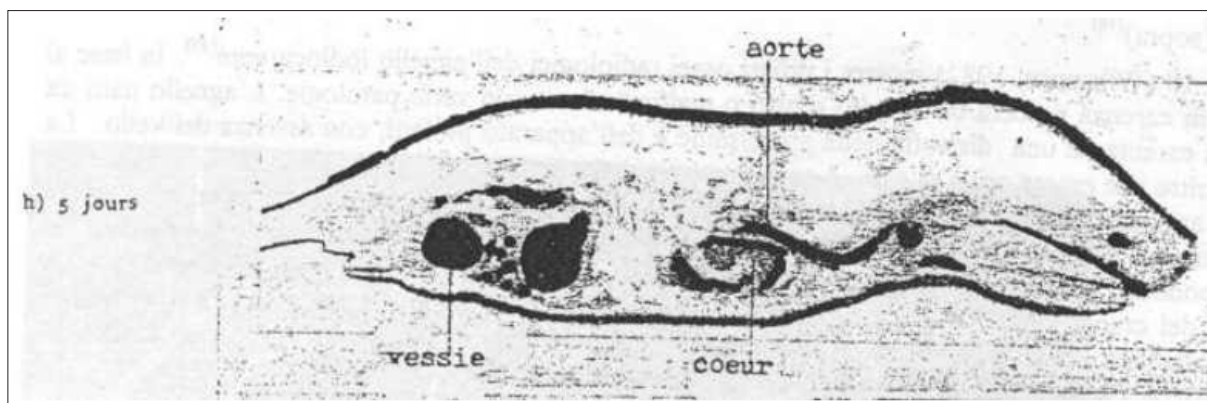


Figure 13: Temporal Trends of Deaths Caused by the Most Frequent Cancers, Diabetes and their Forecast Worldwide from 2010 to 2030.

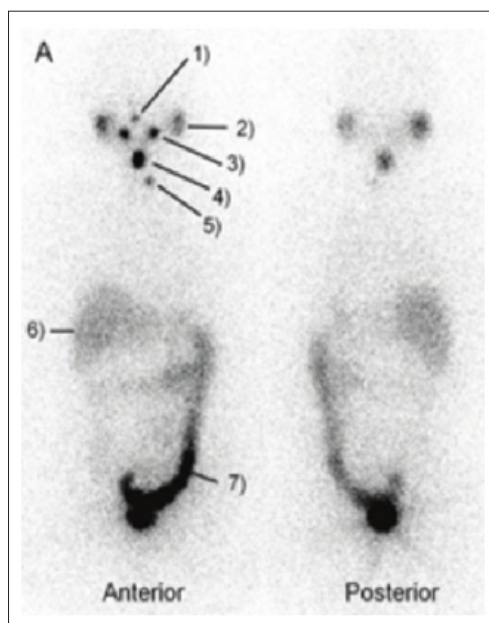


Figure 14: Distribution of Iodine-131 (half-life: 8 days) in Black in Radioautographies of the Body of a Rat after a Subcutaneous Injection of Radioiodine. High I-Concentration is Evident in **Stomach**, Intestine, **Colon** and Arterial Walls of Aorta, where it is Detectable up to 5 days after the Injection. (from Pellerin, 1961; Courtesy of Path Biol).

Physiologic uptakes of radioiodine in the whole body scintigraphy in nasal secretion (1), parotid gland (2), thyroid tissue (5), liver (6), **stomach** and pancreas (not very visible here) and **colon** (large intestine di Figure 6), (7) are observed. (From Oh , 2012).

Conflict between WHO and the International Atomic Energy Agency (IAEA)

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 current limits are based on obsolete mathematical models that ignore selective bioaccumulation highlighting how cesium concentrates in specific organs, such as the pancreas, colon and salivary glands potentially causing

pancreatitis and cancer, even at doses considered “safe” by current regulations. Venturi points out that 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 chronic illnesses (such as diabetes reported in Chernobyl and Fukushima contaminated people) [21,22,23].

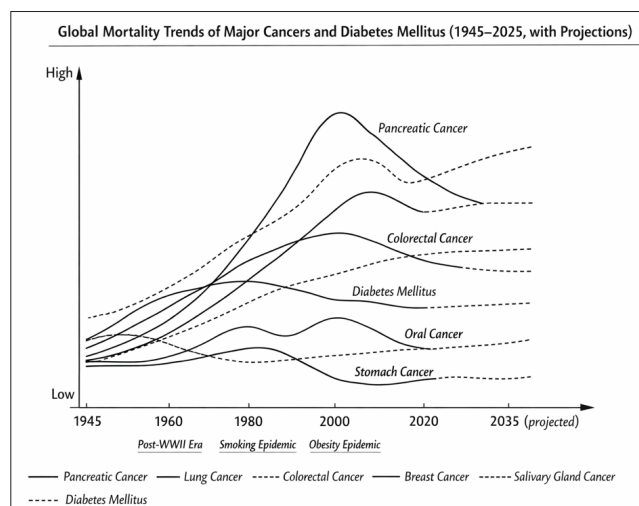


Figure 15: Temporal Trends of Deaths Caused by the Most Frequent Cancers and Diabetes Mellitus (M+F) and their Forecast Worldwide from 1945 to 2035 (From ChatGPT- AI).

Discussion

Although according to the 'Agreement of 1959': WHO cannot publish on nuclear radiation without consulting the IAEA, in this field the IAEA has the preeminent role and actually commands. WHO loses scientific autonomy. This explains why: WHO has never produced its own independent radiation protection. So, many WHO assessments are conservative towards industry. This work proposes an integrative, hypothesis-driven framework to interpret the biological effects of artificial radionuclides through the lens of evolutionary biology, redox biochemistry, and metabolic physiology. Iodine and potassium became embedded in living systems over geological timescales, shaping antioxidant defenses, membrane stability, and electrical signaling. The pancreas represents a convergence point of vulnerability. Despite its small mass, it exhibits high metabolic activity, substantial blood perfusion, and limited antioxidant capacity in endocrine β -cells. Experimental, autoradiographic,

and autopsy studies have reported organ-specific accumulation of radiocesium and radioiodine in metabolically active tissues, including salivary glands and pancreas. Epidemiological observations from Chernobyl, Fukushima, and atomic-bomb survivor cohorts consistently report increases in diabetes incidence and pancreatic-related disorders years after exposure. These findings are multifactorial and influenced by genetic, nutritional, psychosocial, and environmental factors.

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 [37].

Conclusion

In conclusion, iodine and potassium illustrate the dual nature of essential elements: evolutionary protectors in their stable forms and potential disruptors when replaced by anthropogenically altered radionuclides. Recognizing artificial radionuclides as

evolutionarily novel agents interacting with ancient biochemical pathways provides a biologically grounded framework for reassessing long-term health risks associated with nuclear technologies (as a radionuclide evolutionary mismatch). Ensuring adequate dietary iodine intake, improving environmental monitoring, and prioritizing research on organ-specific uptake and internal microdosimetry could contribute to more effective public health strategies. By integrating evolutionary biology, radiobiology, and metabolic science, this work aims to stimulate a more nuanced and precaution-oriented discussion of chronic low-dose internal radiation exposure and its possible role in pancreatic disease. A recent paper of Alwadi et al. In Nature Communication, 2026, reported that in U.S. counties located closer to operational nuclear power plants have higher cancer mortality rates than those farther away [38].

3 -Tables A, B, C

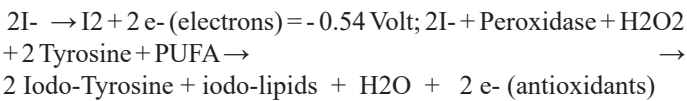


Table A: Antioxidant Biochemical Mechanism of Iodides (Venturi, 2003).

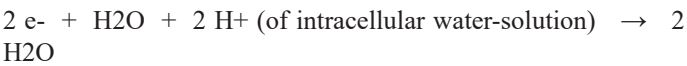


Table B: Antioxidant Biochemical Mechanism of Iodides, Probably the most Ancient Mechanisms of Defence from Poisonous Reactive Oxygen Species (Venturi, 2006).

Table C: 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).

	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

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Interest conflictS absent