1996

Characterization of uranium distribution in Baja California Sur, Mexico

Cecilia Serrano Hidalgo
San Jose State University

Recommended Citation

Hidalgo, Cecilia Serrano, "Characterization of uranium distribution in Baja California Sur, Mexico" (1996). *Master's Theses*. 1334.
DOI: https://doi.org/10.31979/etd.we22-p8t7
https://scholarworks.sjsu.edu/etd_theses/1334

INFORMATION TO USERS

This manuscript has been reproduced from the microfilm master. UMI films the text directly from the original or copy submitted. Thus, some thesis and dissertation copies are in typewriter face, while others may be from any type of computer printer.

The quality of this reproduction is dependent upon the quality of the copy submitted. Broken or indistinct print, colored or poor quality illustrations and photographs, print bleedthrough, substandard margins, and improper alignment can adversely affect reproduction.

In the unlikely event that the author did not send UMI a complete manuscript and there are missing pages, these will be noted. Also, if unauthorized copyright material had to be removed, a note will indicate the deletion.

Oversize materials (e.g., maps, drawings, charts) are reproduced by sectioning the original, beginning at the upper left-hand corner and continuing from left to right in equal sections with small overlaps. Each original is also photographed in one exposure and is included in reduced form at the back of the book.

Photographs included in the original manuscript have been reproduced xerographically in this copy. Higher quality 6" x 9" black and white photographic prints are available for any photographs or illustrations appearing in this copy for an additional charge. Contact UMI directly to order.

U·M·I

University Microfilms International
A Bell & Howell Information Company
300 North Zeeb Road. Ann Arbor. MI 48106-1346 USA
313/761-4700 800/521-0600

CHARACTERIZATION OF URANIUM DISTRIBUTION IN BAJA CALIFORNIA SUR, MEXICO

A Thesis

Presented to

The Faculty of the Department of Biological Sciences

San Jose State University

In Partial Fulfillment

of the Requirements for the Degree

Master of Science

By

Cecilia Serrano Hidalgo

August, 1996

UMI Number: 1381445

UMI
300 North Zeeb Road
Ann Arbor, MI 48103

APPROVED FOR THE DEPARTMENT OF BIOLOGICAL SCIENCES

Kenneth H. Coale

Dr.Kenneth H. Coale

Dr. Leon Dorosz

Adrian Rodriguez

Dr. Adrian Rodriguez

APPROVED FOR THE UNIVERSITY

M. Lou Lewandowski

San Jose State University
One Washington Square
San Jose CA 95192-0100

CHARACTERIZATION OF URANIUM DISTRIBUTION IN BAJA CALIFORNIA SUR, MEXICO

APPROVED FOR THE DEPARTMENT OF
BIOLOGICAL SCIENCES

Kenneth H. Coale

Kenneth H. Coale, Ph. D.
Adjunct Professor Marine Biogeochemistry
Moss Landing Marine Laboratories
P. O. Box 450
Moss Landing, CA 95039

Leon Dorosz, Ph. D.
Professor Chairman
Duncan Hall, Department of Biology
One Washington Square
San Jose, CA 95192-0100

Adrian Rodriguez

Adrian Rodriguez, Ph. D.
Associate Professor
Duncan Hall, Department of Biology
One Washington Square
San Jose, CA 95192-0100

APPROVED FOR THE UNIVERSITY

M. Lou Lewandowski

CHARACTERIZATION OF URANIUM DISTRIBUTION IN BAJA CALIFORNIA SUR, MEXICO

By

Cecilia Serrano Hidalgo

The manuscript's purpose is to increase the current knowledge of uranium levels in a sea shore environment near phosphate deposits in Baja California Sur for future identification of natural enrichment in coastal habitats. A study research plan was designed and presented in this thesis. It includes: 1) a uranium overview (radiation principles, uranium chemical- and radio-toxicity, and environmental distribution); 2) a preliminary study results, discussion, and conclusions; 3) a research proposal to quantify the environmental levels in La Paz Bay, Mexico; 4) suggestions of other potential methods to evaluate the association between the uranium leached from the phosphate deposits and cancer risk.

The proposal's approval and subsequent completion would reveal the most important pathways (air, marine and terrestrial sediments, natural waters) of accumulation with relevance for human populations.

ACKNOWLEDGMENTS

This manuscript would never have been completed without the help and encouragement of Drs. Kenneth Coale and M. Suzanne Connors.

First and foremost, I wish to express my sincere and unreserved appreciation to my advisor Dr. Kenneth H. Coale who provided a spirit of friendly cooperation, a wealth of ideas, constant support, encouragement, enthusiasm, and guidance throughout the project.

This work was also greatly enhanced by the patience and rigorous review of Drs. Connors, Dorosz, and Rodriguez. For this I am extremely grateful.

I wish to thank the personnel of Moss Landing Chemical Laboratory, especially Ms. Jocelyn Nowicki, for the radiochemical analysis of the samples.

I like to extend my genuine thanks to FUJITSU AMERICA, INC. for sponsoring the Ernesto Galarza Scholarship that I was awarded at San Jose State University.

I would also like to express my sincere gratitude to my family. My husband, Jose Francisco, provided moral and financial support as well as invaluable patience throughout my work. And my children, Oscar Antonio and Araceli Karina, despite their young age, were immensely understanding for the length of the project.

Finally, I wish to acknowledge Dr. William E. Bros for sharing his computer system to finalize the figures needed for this manuscript.

DEDICATION

This thesis is dedicated to the memories of my father, Moises Serrano, and to my friend, Annette Shappiro, who did not live to see me achieve my goal.

TABLE OF CONTENTS

PART I RADIATION PRINCIPLES AND URANIUM BACKGROUND

PART II PROPOSAL

Preliminary Study

Further Investigations

LIST OF FIGURES

LIST OF TABLES

PREFACE

This preliminary study and detailed proposal grew out of a desire to document the environmental uranium concentrations for the State of Baja California Sur, Mexico, to aid in future biological and environmental impact assessments. The proposal's objective is to present a research plan to measure the uranium content in environmental media for further evaluation of human and animal exposure to uranium in a coastal ecosystem.

This thesis is organized in two parts. Part one gives an introduction to the subject that provides an overview of natural radiation, uranium and its distribution and a summary of the basic radiation and carcinogenic principles.

Part two presents the proposed research plan to study the natural uranium radioactivity in Baja California Sur. The preliminary study section presents the region, the technique and results of a preliminary study. An examination and discussion of the preliminary results is also covered in this section. The further investigations section states the need, significance, hypothesis, objectives, and budget for the proposed study to further our understanding of uranium in this area. A review and selection of the biotic and abiotic media is done for sampling as well as the potential techniques for the uranium isotopic analysis. A rationale and choices of software models are presented to examine the trophic levels likely to be affected by the local uranium levels. It also outlines other potential methods to apply the findings based on the conventional belief that all doses of radiation no matter how small have some cumulative effect and that no susceptible tissue in an organism is exempt from malignant transformation.

This work is important because of the lack of baseline data on the magnitude and variability of uranium environmental concentrations, transport, trophic pathways, exposure, or inherent radiological effects in the study region.

INTRODUCTION

Little attention has been paid to the environmental radiotoxicity of uranium enrichment caused by mining and natural weathering of phosphate deposits. Still, phosphate deposits around the world contain levels of uranium and its decay products that are elevated relative to average background levels (Manyama and Holm, 1993; Barisic et al., 1992; Luther et al., 1992; Laiche et al., 1991; Berish, 1990; UNCEAR, 1988; Kim and Burnett, 1985; Guimond, 1976). Uranium in phosphate rock deposits throughout the world ranges from 3 to 400 mg/kg^{-1} (Guimond 1977, 1976).

In the course of natural weathering processes or anthropogenic activities, phosphate rocks release, transfer, and redistribute the natural uranium and its progeny to the surrounding environment. Uranium decays by alpha radiation and this type of radiation has been classified as more cytotoxic and oncogenic than other types.

Exposure to the naturally occurring mixture of uranium (99.275% ^{238}U, 0.720% ^{235}U, and 0.0055% ^{234}U) may be radiologically significant for populations who work in the phosphoric industry, people who live in regions of high natural radioactivity and humans who consume foods grown in areas with enhanced levels of uranium (Augustin and Zejda, 1991; EPA, 1985; NCRP, 1984; Rayno, 1983; Crounse et al, 1983; Doll and Peto, 1981; West et al., 1979; Miller, 1977; Yamamoto et al., 1971).

There is evidence that cancer predisposed individuals may be more susceptible to radiation induced oncogenesis (Cox 1994 a, b). Hence, naturally enhanced uranium levels may have great significance for cancers induced by ionizing radiation (Sankaranarayanan and Chakraborty, 1995). Consequently, it is important to obtain a better understanding of the natural levels of uranium in biotic and abiotic media in geographic areas characterized by phosphate rock deposits. Baja California Sur is such an area (Fig. 1). A preliminary radiochemical analysis of coastal waters there suggests anomalously high uranium levels.

In this thesis I propose a research plan to examine the following hypothesis: the natural uranium levels in La Paz Bay are enriched and deviate from average natural levels in ocean waters. The elevated uranium levels may be propagating through the food chain into human populations with deleterious effects.

This paper outlines the study design and presents preliminary findings for local uranium levels in La Paz Bay, Baja California Sur, Mexico.

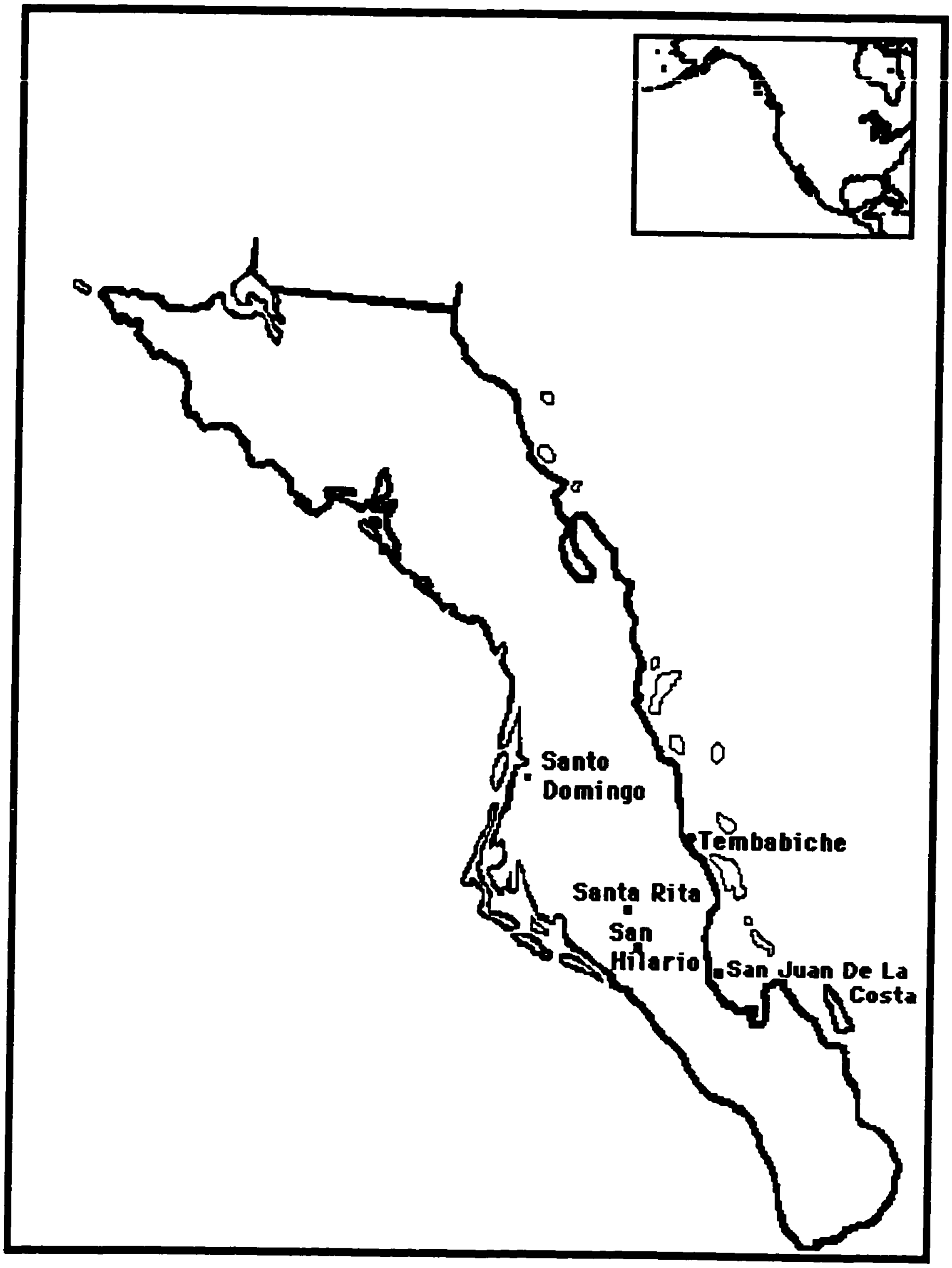


Figure 1. Baja California Sur, Mexico and phosphorite bed locations.

NATURAL RADIATION

Radiation occurs naturally throughout our environment. Yet, natural radioactivity was only discovered within the last 100 years. Natural radiation was first reported on February 24, 1896 by Henri Bequerel. Research and public attention has focused on anthropogenic radioactivity resulting from thermonuclear weapons detonation and the production of nuclear energy, although natural radiation is by far the largest (82%) source of radiation exposure (NRC, 1990).

Natural sources of radiation include: solar rays, cosmogenic particles (arriving at the earth's surface), internal radiation, and radiation that comes from primordial or terrestrial radionuclides at the earth's surface.

Naturally occurring radionuclides can be divided into three classes:

(a) primary radionuclides formed in the synthesis of the elements that have survived to the present;

(b) cosmogenic radionuclides formed by the interaction of cosmic rays with matter and;

(c) artificial radionuclides introduced into the environment by human activity over which the experimenter has no control.

Primary radionuclides account for most of the natural terrestrial radioactivity. They contribute to external radiation fields, as well as to the internal doses incurred by all living systems on the earth's crust. Virtually every organism in this world contains trace amounts of radioactivity of terrestrial origin and is constantly exposed to natural background radiation sources from the rocks, soil, and water in varying degrees.

Of the approximately 340 nuclides found in nature, about 70 are radioactive. Most of these naturally occurring radionuclides are heavy-elements (all isotopes of elements with $Z>82$ are radioactive). The most important way for radionuclides to enter the hydrosphere is by leaching from rocks and soils. Airborne dust that is leached in the ocean provides another important source of these radionuclides for a coastal habitat.

Continental weathering releases uranium into solution, where it is complexed with carbonate, sulphate or dissolved organics (Langmuir, 1978). This is especially true in regions with geological formations rich in radionuclides. Those factors make uranium isotopes a significant contributor to the "background" radiation to which all living things are constantly exposed.

Radiation falls broadly into two categories: (1) radiation in the form of waves, and (2) radiation in the form of rapidly moving electrically charged or neutral subatomic particles or charged atomic nuclei of a wide range of masses. To some extent, particularly when very high energies (velocities) are involved, the effects of waves and particles are virtually the same and it is convenient to think of any radiation as "particles" of energy that interact with matter. Indeed, under some conditions, electromagnetic waves show properties usually associated with particles; they are then described as photons (quanta of energy). Similarly, moving particles may show some characteristics of waves (Potten, 1985).

Radioactivity is a natural process that takes place when the protons and neutrons of certain nuclei cannot maintain a stable energy balance and spontaneously change (decay) to reach a more stable level. Radiation can take different forms including: a) α-particles; b) negative β-particles, or negatrons; c) positive β-particles, or positrons; d) X rays resulting from electron capture; e) γ-rays, either from isomeric transition or, more commonly, as excess energy following particle emission; f) internal conversion electrons, resulting from the electromagnetic interaction between the nucleus and the orbital electrons; g) protons; h) neutrons; and i) cosmic rays.

The physico-chemical changes caused by the ionization of the atoms of living matter occur in a fraction of a second, whereas the radiological processes that result from physicochemical changes eventually lead to such biological changes as genetic mutations, cell death and cancer may take months or even decades. The effects of radiation insult depend upon a large number of variables other than radiation dose, such as time of exposure (fractionated, acute or chronic), the character of the tissue irradiated, the time course of the radionuclide in the body (governed by both physical decay and biological elimination rates), and the specific properties of the radioactive emissions.

RADIOBIOLOGICAL CONCEPTS

Six major radiobiological concepts describe the effect of ionizing radiation on biological tissue.

1) The principal target for radiation-induced cell killing is DNA. Although it is not the exclusive target, it is generally the most consequential. While the evidence for this conclusion is mostly circumstantial, it is also compelling (Lea, 1956). The effects of the absorption of radiant energy arise from excitations and ionizations along the tracks of the charged particles that occur when radiant energy is absorbed. Biological damage may be a consequence of a direct interaction between the charged particles and the DNA molecule, or the biological effects may be mediated by the production of free radicals (Michaels and Hunt, 1978). In the latter case, which is the indirect action of radiation, the absorption of the radiation may occur in, for example, a water molecule, and the free radicals produced diffuse to the DNA and produce a biological lesion at the molecular level. In the case of sparsely ionizing radiation, such as x and gamma rays, about two-thirds of the biological effects are produced by this indirect action, and this component of the radiation damage is amenable to modification by a variety of physical, chemical, and biological factors. As the radiation quality (LET) changes from low to high linear energy transfer, the balance shifts from the indirect action to the direct action (NRC, 1990). LET is defined as the spatial energy distribution, stated in terms of the amount of energy deposited per unit length of particle track. X rays and gamma rays set in motion electrons with a relatively low spatial rate of energy loss and thus are considered *low LET* radiations. The photon and electron energy degradation processes result in a broad distribution of LET values occurring in irradiated tissue. A typical value of LET for electrons set in motion by ^{60}Co γ-rays (average energy 1.25MeV) would be about 0.25 keV/μm. This can be contrasted with a densely ionizing 2MeV alpha particle which produces about 1000 times more ionization per unit

distance, 250keV/μm. Such particles are characterized as *high LET* radiation. Knowledge of LET is important when considering the Relative Biologic Effectiveness (RBE -biological potency of one radiation compared to a reference electromagnetic radiation (^{60}Co γ-rays) for the same biological endpoint-); LET is commonly used as a measure of radiation quality.

2) In general, the dose-response relationship for carcinogenesis in laboratory animals appears to vary with the quality (LET) and dose rate of radiation, as well as sex, age at exposure and other variables (Brenner, 1988). The carcinogenic response of humans influenced by age and sex has been characterized to a limited degree, but changes in response due to dose rate and LET have not been quantified. The body of radiobiological data available indicate that, in principle, RBE increases with decreasing dose, with limiting higher values generally reached at low doses or at low dose rates. This relationship results from the fact that the dose-response for low-LET radiation is often a linear-quadratic function of dose, whereas for neutrons and charged particulate radiations it approximates a linear function of dose. In spite of this hazard, studies of populations chronically exposed to low level radiation, such as those residing in regions of elevated natural background radiation, have not unequivocally demonstrated evidence of an associated increase in the risk of cancer. Continued research is needed, therefore, to quantify the extent of exposure and its carcinogenic effects (NRC, 1990).

3) The biological endpoint of a given dose of radiation varies with the LET of radiation. Although the RBE varies with the LET of radiation, it also varies with the dose, dose rate, type of cell or tissue used to score the biological effect, and the endpoint in question (Barendsen, 1968; Broerse and Barendsen, 1973). The pattern of variation of the RBE with LET appears to be similar for mutagenesis as for cell killing, but it has not been established to be the same for carcinogenesis as an endpoint. In general the RBE for various end points increases with LET and peaks at a LET of about 100 keV/μm (Rodriguez et al., 1992).

4) The wide fluctuation in the radiosensitivity of cells depends on their stage in the intermitotic cell cycle. Cells that are in mitosis at the time of irradiation (time zero) and cells that are in G-2 and reentering mitosis are by far the most radiosensitive. The surviving fraction at these times is less than a quarter of that seen for the most resistant cells. The most resistant cell population is the population of cells that is nearly complete in its cycle of DNA replication, the late S phase. A wide variety of cell lines of the mouse, hamster, rat, and human have been tested for the changes in radioresistance with cell cycle age. Most, but not all, cell lines show an increased radioresistance near the end of the S phase of the cycle. There are some significant exceptions. A rat cell line developed from gliosarcoma, which has been designated the 9L cell line, shows almost no age sensitivity at all during the various phases of the cell cycle. Even for this line, however, there is a small but significant increase in radioresistance near the end of the S phase (Sinclair and Morton, 1966; Terasima and Tolmach, 1963). Those few studies that have attempted to evaluate the cycle-dependent radiosensitivity of cells growing in *in vivo* tissue systems have demonstrated a similar age response function.

5) The effect of a given dose may be influenced greatly by the dose rate. In the case of low LET radiations, the reduced effectiveness of a dose delivered at low dose rates is a consequence of the interaction of a number of factors, most notably the repair of sublethal damage, the redistribution of the cells within the mitotic cycle, and the compensatory cellular proliferation during protracted exposure. In the case of high LET radiations, the dose rate effect is much reduced, at least those components of it that are a consequence of repair and redistribution. These general considerations appear to be equally valid for mutagenesis (gene alteration from one form to another) and carcinogenesis (multistep process in which two or more intracellular events are required to transform a normal cell into a cancer cell) (Hall, 1972). In most cases, for high-LET radiations such as neutrons, the effect of a given dose is relatively unchanged when the dose rate is lowered or when fractionation is

used. In a few important instances, however, (including neoplastic transformation *in vitro*, carcinogenesis in experimental animals, and mutagenesis) protraction by use of low dose rate or by fractionation actually enhances the biological effectiveness of a given dose (Hei et al., 1988). The overall conclusion is that the RBE of high-LET radiations compared with that of low-LET radiations may be larger for a low dose rate than for a single acute exposure at a high dose rate (NRC, 1990).

6) A variety of chemicals can modify the cell killing effects of radiation. Oxygen and other agents that mimic oxygen by being electron affinic tend to sensitize cells to the effects of a given dose of radiation, while scavengers, such as sulfhydryl compounds (glutathione), tend to protect cells (Mottram, 1936; Palsic and Skarsgard 1984). In general, the redox status of the cell affects its response to radiation. There are modifiers which have little influence on cell killing but may greatly modify the multistep process of carcinogenesis and its *in vitro* counterpart, oncogenic cell transformation (Hall and Hei, 1987). These modifiers include (1) hormones (Guernsey and Borek, 1980), (2) tumor promoters, that is, agents that do not affect initiations but that dramatically affect the later stages of carcinogenesis *in vivo* or transformation in vitro (Kennedy et al., 1980), and (3) protease inhibitors, such as antipain (Borek et al., 1979; Kennedy and Little, 1981). These factors, with little influence on cell lethality, can exert a profound effect on the response to radiation when carcinogenesis, transformation or both, are the endpoints being studied (NRC, 1990).

URANIUM DISTRIBUTION IN THE ENVIRONMENT

Natural uranium's importance lies not only in its inherent chemical and radiological properties (potential health hazards) but also its particle-reactive progeny that produce alpha radiation. Because it is a parent of two decay chains, the study of uranium is crucial for our understanding of isotopic disequilibria in the environment (Moore, 1992; 1967).

Environmentally, uranium is the primordial radionuclide of most importance because of its ubiquity. Primordial radionuclides are principally, but not entirely, the isotopes of three naturally occurring decay series: 4n+0, 4n+2, and 4n+3 (Bernat and Church, 1989; Osburn, 1965). The decay scheme for the three natural radioactive series is shown in Table 1. Some radionuclides decay too rapidly to have remained since the time of the creation of matter, as is the case for ^{236}U in the 4n+0 decay series. Geochemical evidence has indicated clearly that ^{236}U was one of the radionuclides when the universe was very young (Riss, 1924; Meyer and Urlich, 1923). ^{236}U, due to its relatively short half-life (2.4×10^7y), has decayed away since its cosmogenesis and all that remains is its daughter, ^{232}Th (Bernat and Church, 1989).

The parent nuclides of the present natural decay series are ^{238}U which decays through some 14 intermediates to stable ^{206}Pb, ^{232}Th which decays through a series of ten intermediates to stable ^{208}Pb, and ^{235}U which decays through a series of 11 intermediates to stable ^{207}Pb. If these parent isotopes are not chemically or physically separated from their daughter products, the various members of the decay chain attain a state of radioactive equilibrium in which the apparent rate of decay of each nuclide is the same as that of the primordial parent.

There are 14 naturally occurring isotopes of uranium; however, there are three isotopes (^{238}U, ^{235}U, and ^{234}U) that are significantly present in the environment. Natural uranium contains about 99.283% of ^{238}U by weight, 0.711% ^{235}U, and 0.0054% ^{234}U.

Table 1. Scheme Of Naturally Occurring Decay Chains (Based on General Electric Co., Chart of Nuclides 1989).

Atomic No.	Thorium Decay Series (4n + 0)					Uranium Decay Series (4n + 2)							Actinium Decay Series (4n + 3)					
92	^{236}U 2.34X10^{7} a					^{238}U 4.5x10^{8} a		↗ ^{234}U 2.5x10^{5} a					^{235}U 7.1x10^{8} a					
91	↓					↓	^{234}Pa UX$_2$ 1.17 m ↗ UZ6.69 h	↓					↓	↗ ^{231}Pa 3.4x10^{4} a				
90	^{232}Th 1.4x10^{10}a		↗ ^{228}Th 1.9 a			^{234}Th 24.1 d		^{230}Th 7.52x10^{4} a					^{231}Th 25.6 d		↓	↗ ^{227}Th 18.7 d		
89	↓	↗ ^{228}Ac 6.15 h	↓					↓							^{227}Ac 22 a	↓		
88	^{228}Ra 5.75 a		^{224}Ra 3.64 d					^{226}Ra 1602 a							⇣	↗ ^{223}Ra 117 d		
87			↓					↓							^{223}Fr 21 m	↓		
86			^{220}Ra 55.6 s					^{222}Rn 3.83 d							⇣	^{219}Rn 3.9 s		
85			↓					↓							^{219}At 0.9 m	↓		
84			^{216}Po 0.145 s		↗ ^{212}Po 3x10^{-7} s			^{218}Pb 3.05 m		↗ ^{214}Po 163.7 ms		↗ ^{210}Po 138 d			⇣	↗ ^{215}Po 1.8x10^{3} s		↗ ^{211}Po 0.52 s
83			↓	↗ ^{212}Bi 60.5 d	↓			↓	↗ ^{214}Bi 19.7 m	↓	↗ ^{210}Bi 5.02d 8 m	↓			^{215}Bi 8 m	↓	↗ ^{211}Bi 2.16 m	↓
82			^{212}Pb 10.6 h		^{208}Pb stable			^{214}Pb 26.8 m		^{210}Pb 22.3 a		^{206}Pb stable				^{211}Pb 36.1 m		^{207}Pb stable

Uranium isotopes are found primarily in rocks and soils with a mean total uranium concentration in igneous rocks of ~4 ppm (UNCEAR, 1988). ^{238}U has a very long half-life of 4.5×10^9 yr so that this isotope, although accounting for the largest fraction, by weight, of natural uranium in the soil, accounts for only half of the radioactivity. The remainder is derived from ^{235}U and ^{234}U.

Minerals containing uranium are widely distributed in nature's crustal rocks. Some are of commercial value and contain various oxides of uranium, including lignite, uraninite, carnotite, brannerite, coffinite, pitchblende, phosphate rock, and monazite sands. Uranium forms an integral part of minerals and rocks and is a source of radioactivity when exposed at the surface (Cowart and Burnett, 1994). Uranium weathered from igneous, metamorphic, or sedimentary rocks generally has been dispersed in the form of the dissolved ion (UO_2^{2+}), or one of its complexes (Hostetler and Garrels, 1962). The reaccumulation of uranium generally has been facilitated by reduction reactions that result indirectly from biogenic processes (Lovley et al., 1991).

Granites are characterized by high uranium concentrations, 30 mg/kg and the abundance is associated with silica content (Rogers and Adams, 1969). Granites present a deep weathering profile and uranium in mineral phase is easily weathered and removed (Ball and Miles, 1993).

Average shale uranium concentrations are 2 mg/kg, but the Chattanooga shale of the eastern U.S.A. has an average uranium concentration of 79 mg/kg (Rogers and Adams, 1969). A modest but measurable amount of uranium is almost always found in the carbonate sedimentary rock groups including biogenic carbonates such as corals and inorganic carbonates such as cave deposits (Rogers and Adams, 1969).

[1]On average most crustal rocks contain about 2.8 mg/kg (UNCEAR 1977, 1982). There are a number of rocks and sediments (phosphate rock and monozite sands) where uranium has become enriched beyond its average natural crustal abundance. Since

uranium's geochemical behavior (weathering and removal from the region and soils) depends on its mineralogy (Tandy 1974, 1973) it can be implied that organisms from regions near those rocks and sediments are subject to high levels of radioactivity.

Uranium enters the fresh and ocean waters in detrital form and dissolved state and these waters serve as important reservoirs and redistribution systems for uranium isotopes and other radionuclides. Natural fresh waters typically contain on the order of 0.024 to 200 mg/L, with sea water showing a more or less uniform concentration of 2 to 3.3 ± 0.2 mg/L, with a salinity of 35 per mille (Gascoyne, 1992; Chen et al., 1986; Ku et al., 1977; Turekian and Chan, 1971; Wilson et al., 1960; Rona, 1956). This uniformity in the concentration of uranium is largely due to its soluble stable carbonate complex $[UO_2(CO_3)_3]^{4-}$ in sea water.

From the biological standpoint, uranium is ubiquitous. A typical human (a 70 kg person) will contain about 100 to 125 μg of uranium. The intake of uranium is largely from food and it is excreted in the urine and feces (ICRP, 1988, 1979; Durbin and Wrenn, 1975) depending on the release from the main soft tissue repository. Soft tissue concentrations are relatively low, on the order of a few hundred picograms per gram of wet tissue, while bone contains a few thousand picograms of uranium per gram of bone ash (Hamilton, 1972, 1971,1970).

---

1. Note that the concentrations of uranium are expressed in units of mass rather than activity. This is commonly done in the environmental literature, and in part is due to the difficulties encountered by early investigators in determining the specific activity of uranium, which is, of course, affected by the state of the equilibrium. Thus, freshly separated uranium will have lower specific activity than uranium that has been removed from the ground, or that has been allowed to sit for several years since it has been separated from the daughter products. One gram of freshly separated natural uranium contains 0.333μCi (1.2×10^4 Bq) of ^{238}U, 0.015 μCi (555 Bq) of ^{235}U, and a negligible amount about 12 pCi (1584 Bq) of ^{234}U. The specific activity of natural uranium is 3.5×10^{-7} Ci (1.3×10^4 Bq)/g; this value refers only to the uranium, and does not include any contribution from the daughters. Another source of confusion has been the definition of the curie as applied to uranium, which in the past has been taken as 3.7×10^{10} disintegrations per second from ^{238}U, plus an identical amount from ^{234}U, plus 9×10^8 from ^{235}U, rather than the standard definition of 3.7×10^{10} disintegrations per second (Kathren, 1984). This special definition has been the basis for internal dosimetry calculations in the past (NCRP, 1959).

After examining the wide distribution and behavior of natural uranium in the environment, it can be implied that the uranium content depends on a variety of factors such as the migration from geological deposits to marine and ground waters, interaction with marine and terrestrial sediments and removal by living organisms. Thus the amount of radioactive material is to a large extent a function of the nature of the element itself and also of its chemical compound form (chemical species) (Piispanen, 1991).

PHOSPHORITE DEPOSITS AND URANIUM INPUT TO AND REMOVAL FROM THE COASTAL SYSTEMS

Uranium in phosphate deposits throughout the world ranges from 3 to 400 ppm. In the USA, Menzel (1968) reported a variability of concentrations of natural uranium and thorium in the phosphate ores from 8 to 399 ppm (5.4 to 267 pCi per gram) and 2 to 19 ppm (0.4 to 4 pCi per gram), respectively. Guimond (1977), a few years later, reported ranges from 50 to 200 ppm. Phosphate rocks processed to extract fertilizer may contain significantly more than 120 ppm. In analysis of ocean floor phophorites the uranium content is generally about 100 ppm (Cowart and Burnett, 1994). O'Brien et al., (1987) concluded that virtually all the uranium is more widespread in the phosphatic layer or crust based on the uranium oxidation state and isotopic ages of 33 phosphorite nodules from the sea floor off the East Australian margin. These layers are thought to subsequently break into discrete nodules that are periodically reworked and exposed on sea floor causing progressive oxidation of U^{4+} to U^{6+}.

Natural uranium isotopes are dissolved during chemical weathering when sedimentary marine deposits are exposed to subaerial conditions. Weathering disrupts the radioactive equilibrium within these deposits and under certain conditions. The oxidized uranyl ion (U^{6+}) complexes readily with carbonate [forming stable uranyl carbonate species $UO_2(C0_3)_3{}^{4-}$] in oxidizing aqueous environments at a pH of approximately 6 or greater (Langmuir, 1978)], phosphate, or sulfate ions and is easily transported in the hydrologic cycle. Also in reducing waters, uranium is in the U^{4+} state and has an extremely strong tendency to precipitate and remain immobile (Gascoyne, 1992).

In suboxic to anoxic environments in near-shore coastal waters, particularly in marine sediments, hexavalent uranium can be reduced to tetravalent uranium and removed from solution. The occurrence of hexavalent uranium in phosphorite has been explained as either a result of chemisorption of uranyl ions (UO_2^{2+}) on apatite crystallites, or as a result of postdepositional oxidation of primary U^{4+} (Burnett and Veeh, 1977). Subsequent oxidation of the uranium originally incorporated as U^{4+} would depend on the degree of exposure to oxidizing environments and the amount of auto-oxidation (Burnett and Gomberg, 1977; Ames, 1960; Altschuler et al., 1958).

Based on the characteristics of phosphorite deposits mentioned above, it can be implied that uranium is easily transported, fairly reactive and readily available in the nearshore and coastal oxic waters. Additionally, the uranium availability for an environment like Baja California Sur has biological and radiological significance because the region is semidesert and uranium is chemically stable in hot dry air and hot temperatures. It slowly oxidizes in cold moist air and forms numerous intermetallic compounds as well as cationic and anionic salts. The release of radiogenic species to the environment implies potential deleterious effects for the organisms in the region.

In the marine environment, the various nuclides of the uranium- and thorium-decay series from phosphorite deposits are classified into two groups: those which remain stable and dissolved in sea water (conservative) and those which associate with particles and are removed from sea water by adsorption, coprecipitation, or by biological processes (particle reactive and scavenged). Six main pathways provide for addition of these nuclides to the ocean waters:

1) Uranium and its daughter products are carried in the particulate load of rivers. Reactions occurring in river and estuarine environments may modify the fluxes of nuclides to the coastal and ocean waters through either uptake or release;

2) Depending on the extent of interaction of the longer-lived parents with the sediment particles, appreciable quantities of radioactive decay products may diffuse out of the bottom sediments into the overlying water;

3) Decay of the relatively soluble uranium isotopes, radium and radon, in coastal and open-ocean waters provides a mechanism for *in situ* production of daughter nuclides in the oceans. Introduction of ^{234}Th, ^{230}Th, ^{228}Th, ^{231}Pa, ^{210}Pb, and ^{210}Po occurs in this way;

4) Wind-blown particles and their associated radionuclides can enter the ocean through the atmosphere (Cochran, 1992);

5) Uranium is incorporated into marine sediments both by uptake in plankton and organic matter formed at the ocean surface and deposited at the sea floor, and by diffusion into sediments followed by reduction and removal from solution;

6) Aside from the natural chemical and physical weathering incorporation of uranium nuclides into the study area, there is potential anthropogenic input from a phosphorite plant in the region. Despite the beneficiation process and the variability of the uranium content, some nuclides are retained in the phosphate granules and the remainder is transferred to the by-products during fertilizer manufacture. Thus, the uranium from the phosphate rock and leach zone will be lost in the slime fraction (discharge) and enter the environment as a potential pollutant.

The processes described above account for a large fraction if not all of the input of uranium by continental runoff. Within the uncertainties of input and output, it is still possible to say that the oceans are in balance with respect to uranium.

Removal of uranium- and thorium-series nuclides from the oceans is accomplished by several mechanisms:

1) Radioactive decay is perhaps the simplest of these mechanisms but does not remove significant quantities of the longer-lived radionuclides;

2) Particle settling through the water column can remove dissolved nuclides through adsorption;

3) Precipitation of an oxide phase during oxidation-reduction reactions involving elements such as iron and manganese can also efficiently scavenge dissolved radionuclides;

4) Nuclide removal may also be biologically mediated through uptake on organic or in the siliceous or calcareous hard parts of marine organisms (Cochran, 1992). Anderson (1982) showed that dissolved uranium is fixed to organic matter in surface sea water but this particulate authigenic uranium is generally remineralized within the water column during descent except in areas of high organic carbon flux (for example the Panama Basin). Brewer et al., (1980) also observed that uranium is removed from the ocean waters by organisms (foraminifera, coccoliths, pteropods, radiolaria, diatoms, corals and molluscs).

Uranium in the coastal zone shows both conservative and non-conservative behavior. Conservative behavior is reflected by average isotopic activity ratios similar to the natural occurrance and non-conservative behavior deviates from this ratio. Thurber (1962) and a number of subsequent authors have reported an activity ratio for $^{234}U/^{238}U$ of 1.15. Conservative mixing has been demonstrated for estuaries in India (Borole et al., 1982, 1977), the UK (Toole et al., 1987), and France (Martin et al., 1978). Examples of non-conservative behavior of uranium include the Ogiichee and Savannah Rivers, Georgia, USA (Maeda and Windsdom, 1982), the Forth estuary (Toole et al., 1987), and the Amazon (McKee et al., 1987; Spalding and Sackett, 1972). The explanation for the diversity of trends is related to the redox chemistry of uranium in estuarine sediments. Reduction and removal of uranium from marine sediment pore waters produces an uptake of uranium in estuarine sediments (Toole et al., 1988; Barnes and Cochran 1987; Cochran et al., 1986). Preferential dissolution of ^{234}U can occur as a result of recoil from the decay of ^{238}U which mobilizes ^{234}U into the surrounding acqueous environment. Such a process can be seen as non-conservative behavior of uranium in the estuary if the rate of mixing is slow relative to uptake in the sediments.

In conclusion, the specific activity of natural uranium is increased by these enrichment processes [i.e. by increasing the proportion, of ^{235}U and ^{234}U (Hursh and Spoor, 1973)]. One can then hypothesize that organisms, human communities and structures built beside phosphorite deposits or mines are subject to high uranium levels and elevated indoor radon daughter concentrations.

The important environmental question then (posed by Cowart and Burnett, 1994) is how and under what conditions does uranium and its progeny become mobilized in the environment so as to present a risk to human health? To evaluate this aspect, the second part of this manuscript details a study to characterize the natural isotopic abundance of uranium. The results of the study can be used as a baseline or reference for further studies focusing either on the uranium radiotoxic effects or on the radiological cancer risk in the coastal biosphere of the region.

URANIUM EXPOSURE

Human exposure to primordial radionuclides is, and has been, an unavoidable consequence of their ubiquitous distribution in the earth's crust, surface soils, air, food, and water supplies. Radiation exposure results from the absorption by the body of penetrating gamma ray photons emitted externally, i.e., originating from the heavy elements (descendents of the uranium and thorium precursors) in the earth's soils and rocks, and from the incorporation of radionuclides emitting α and β particles within internal organs and tissues (Spencer et al., 1990). Irradiation of the whole body occurs as a result of external and internal exposure to radionuclides (Linsata, 1994; Linsata et al., 1991, Linsata, 1989; Linsata et al., 1989a, b; Linsata, 1986).

Radionuclides belonging to the four main decay series shown in table 1 are present in the atmosphere, in food and in potable water. These can be inhaled or ingested and so constitute internal irradiation. Natural uranium enters the body through inhalation, ingestion, or dermal pathways.

Although the effects are similar in many respects, the source and exposure pathways have some relevance to the tolerance dose. With internally deposited radionuclides the radiation dose to various organs and tissues of the body continues to accumulate until the radionuclide is removed by physical or biological processes. Thus, the radiation is delivered to various organs gradually, at changing dose rates, over what may be an extended lifetime. Therefore an internally incorporated radionuclide deposited in or near organs and tissues frequently produces nonuniform irradiation, depending on its radioactive emissions and metabolic characteristics. Differences in both dosimetry and biological response have a direct impact on the characteristics of the resulting dose-response relationships. Accordingly, any quantification of human health risks from exposure to ionizing radiation must consider, first, risk factors for exposure situations in which adequate data on dose and response are available, and second, the relative importance of various dosimetric or response factors that can alter the resulting risk estimates. This applies to both internal and external irradiation conditions (NRC, 1990).

The relative contribution of ingestion and inhalation to the human burden of uranium depends on several variables. These variables include atmospheric concentrations, particle size distribution, solubility (in lung fluids) and clearance rates of the aerosol fraction retained in the lungs, dietary intakes, GI absorption fractions, organ uptake fractions, and removal rates. Once absorbed systemically, the uranium distribution within the various tissues and their associated retention time functions are assumed to be equivalent for either initially inhaled or ingested concentrations (Linsata, 1994). Irradiation of specific organs and tissues is a function of the initial distribution within these tissues, the time course of the radionuclide in the body (governed by both physical decay and biological elimination rates), and the specific properties of the radioactive emissions. After systemic absorption, translocation by the blood (e.g., by circulating plasma proteins such as tranferrin) to all tissues and organs occurs. Organ uptake fractions, retention kinetics, and elimination routes (urine and feces) of those quantities absorbed systemically are element and species specific.

After inhalation, the deposition of uranium dust particles in the lungs depends on the particle size, and the absorption depends on the solubility of the compound. Particles larger than 1-5 microns are likely to be either exhaled or transported out of the tracheobronchial region by mucociliary action and swallowed. The more soluble compounds, such as uranium hexafluoride, uranyl fluoride, and uranyl nitrate hexahydrate, are more readily absorbed into the blood at the alveolar level within days. The less soluble compounds (uranium trioxide, uranium tetrafluoride, uranium tetrachloride) are more likely to remain in the lung tissues and associated lymph glands for weeks, or for years (uranium dioxide and triuranium octaoxide). When uranium insoluble salts are inhaled from dust, most leave the lungs by coughing or exhaling; the residual may have a prolonged retention time in the lungs and some may enter the blood system (Kathren and Moore, 1986).

From the various reviews (Leggett and Harrison, 1995; Cowart and Burnett, 1994; Mortvedt, 1994) and original works (LLobet et al., 1991) it appears that uranium intake (humans and animals) occurs largely through the ingestion of grains, green vegetables, and fruits. Animal products, sea food, and water become the primary sources in areas with high environmental levels . Bivalves (clams, scallops and oysters), which form a large portion of the diet among coastal populations living in areas rich in uranium, may contribute to higher dietary intake. In general, biota can be exposed to environmental uranium through various media and exposure routes and these can be measured by analyzing air, water, soil, food, or the organisms themselves (Sutter, 1993; EPA, 1993).

External exposure of human beings from natural radionuclides is from the radionuclides and progeny of the uranium-thorium series (Table 1). The local concentration of these radionuclides and their decay products varies widely depending on the geologic characteristics and history of the region. There will be a minor contribution of beta rays to the doses received by the skin. Any emissions of beta or alpha particles from the decay of natural radionuclides will not contribute significantly to the dose from externally located nuclides because of their short ranges (α particles of uranium have a range in tissue of approximately 0.05 mm). Thus, dermal absorption is thought to be negligible. One would not expect skin injury from them because of the two protective (dermis and epidermis) skin layers. But, when the energy of particles is greater, penetration in tissue becomes sufficiently great to produce skin injury (Parker and Cantril, 1986).

External sources of radiation are of relevance, principally, in regions where monozite sands are present. Major anomalies in the concentration of radioactive minerals in soil and sand are found in a number of countries such as China, the United States, Brazil, and India (Narayana et al., 1995). Beach sands of different regions, world wide, contain significant radioactivity due to the presence of thorium and uranium. It is also known that they offer radiation hazards especially to fisherman who reside close to the coast (Kalyani et al., 1990). The external dose rates associated with monozite sand regions are many fold higher than normal. Measurements in these sands reveal levels up to several mrad/h (several tens of µGy/h), in sharp contrast to the approximately ten microrad per hour usually associated with natural terrestrial background radioactivity (Katheren, 1984). Thus, external radiation may become a significant radiation source for beach dwellers or people using the beaches with high uranium and thorium content for recreational or occupational purposes.

URANIUM METABOLISM

Uranium is a heavy metal element that, after being absorbed, can be released into the body fluids through processes of anionic and cationic substitution. In body fluids, tetravalent uranium is likely to oxidize to the hexavalent form.

Uranyl salts hydrolyze in water at pH 7.4 and the products are insoluble. However, the salts form soluble $UO_2HCO_3^+$ ions in the blood due to high concentration of HCO_3^- ions. About 25% of injected UO_2^{2+} accumulates in bone due to the affinity of crystal surfaces for UO_2^{2+}, and the formation of complexes with phosphate and mucosal chondroitin sulfates. UO_2^{2+} in bone is in reversible equilibrium with that in blood. Among the soft tissues, kidney accumulates about 25% and the rest of the injected dose is excreted both in feces and urine depending on the release from the main repository in soft tissue (Durbin and Wrenn, 1975).

Soluble metal salts are highly toxic and have a strong capacity to complex with citrates, bicarbonates, glutamates, and proteins in the plasma such as albumins (Cooper et al., 1982; Stevens et al., 1980; Dounce and Flagg 1949). Uranium soluble salts form colloidal hydroxides and oxides. The stability of the bicarbonate complex depends on the pH of the solution, which will differ in different parts of the body (NRC, 1988). The low molecular weight bicarbonate complex can be filtered at the renal glomerulus, and be excreted in urine at levels dependent on the pH of the urine. The uranium bound to the protein (primarily transferrin) is less easily filtered and is more likely to remain in blood.

UO_2HCO_3 concentration, low in the blood, increases rapidly in the nephrons of the kidney due to water and salt reabsorption and acidification, and then binds to cellular components. The LD50 is 1 mg/kg for rats and 0.1 mg/kg for rabbits; it causes complete renal failure. UO_2^{2+} binding in kidney tissue causes loss of tubular reabsorptive function and cellular break-down. A single dose of 0.02 mg/kg in a rabbit will cause increased excretion of amino acids and kidney enzymes (Luckey et al., 1975).

Certain hexavalent uranium compounds, such as UF_6 and UO_2F_2, are quite soluble in the lung and are, therefore, rapidly absorbed into the blood following inhalation. This material is mostly excreted in urine. There is some generalized systemic deposition in soft tissues, primarily the liver, as well as in the skeleton (Kathren et al., 1989; Fisenne and Welford 1986; Fisenne et al., 1980). Hexavalent uranium oxide UO_3 and the tetravalent uranium compounds UF_4 and UCl_4 are more slowly absorbed. Only a fraction of the soluble U^{6+} entering the gastrointestinal tract, either from ingestion or by mucociliary clearance of inhaled particles, is absorbed into the blood. This fraction has been estimated to be 1-2% by Durbin (1984) and Wrenn et al., (1985), although the IRCP published an $f1$ value (transfer fraction from gut to blood) of 0.05 (5%) for hexavalent and tetravalent uranium compounds (ICRP, 1988, 1979). The remainder is excreted in the feces.

Uranium oxyfluoride (UO_2F_2) dissociates and is rapidly absorbed from the lung into the blood stream as the UO_2^{2+} ion, which behaves much like calcium and complexes with serum proteins and bicarbonate. The uranyl ion forms bicarbonate, $UO_2(CO_3)_3^-$, and citrate complexes in blood plasma (Durbin 1984). The UO_2^{2+} also binds to red blood cells. As it is filtered from blood and passes through the kidneys, the UO_2^{2+} ion dissociates within the tubular filtrate and recombines with cell surface ligands (Morrow, 1984). Removal of the uranyl ion from the kidneys has been described by a two-compartment exponential model indicating that 92-95% is excreted in the urine with a half-time of 2-6 d and the remainder with a half-time of 30-340 d (Durbin, 1984). Wrenn et al., (1989) showed from a variety of mammalian laboratory experiments that the short-term retention of uranium in the kidney should range from 2-6 d. Their earlier model for man used a kidney clearance half-time of 15 d. Fisher et al., (1991) provided a modified five-component exponential, systemic model based on uranium analysis data, on 31 workers acutely exposed to soluble UF_6 and its hydrolysis products at a 1986 accident in Oklahoma.

Animal studies, in general, have shown that inhalation, oral, or dermal exposure to uranium results in kidney damage. The mechanism for kidney damage is known, and it is likely that humans would develop kidney lesions by the same mechanism. In animals, two particular areas of the kidney are affected: the glomerulus and the convoluted tubules. Glomerular defects include a decrease in filtration rate, as assessed by creatine or inulin clearance and by proteinuria (Leach et al., 1973; Morrow et al., 1982b).

URANIUM CHEMICOTOXICOLOGICAL PROPERTIES

The toxicity of uranium is related more to its chemical properties than to its radioactivity. Chemically, the soluble uranium compounds (uranyl ion) and those which solubilize in the body by the formation of a bicarbonate complex, produce systemic toxicity in the form of acute renal damage. Laconte (1854) described the kidney lesions produced by uranium. Since then, the chemical toxicity of uranium has been observed in humans, quantified in animals and also has been the subject of recent studies (LLobet et al., 1991) and reviews (Leggett and Harrison, 1995; Cowart and Burnett, 1994; Mortvedt, 1994; Wrenn et al., 1985).

Uranium exhibits variable valance from +3 to +6 in the crystalline state; in aqueous media, U^{4+} and U^{6+} are the stable forms, U^{6+} existing as the uranyl $(UO_2)^{2+}$ ion. Uranyl ions readly form stable complexes with carbonate and phosphate ligands in biological fluids and tissues; carboxyl and hydroxyl groups are involved to a lesser degree. The water soluble uranium bicarbonate complex $[(UO_2HCO_3)^+]$ is very stable and will not dissociate at physiological pH (7). At lower pH the complex will dissociate. Low pH environments can be found in bone, leading to uranium accumulation or surface deposition in this tissue. The phosphoryl group of DNA and serum albumin can bind uranyl ions. Serum albumin binds 18 $(UO_2)^{2+}$ ions per molecule. At alkaline pH, a dinuclear complex involving uranyl nitrate with AMP, ADP and ATP are formed in which uranium is bonded to the phosphate and to the ribose hydroxy oxygen atom. The U-ATP system at pH 6.8 is a 2:2 sandwich chelate, where uranium is bonded to the β and γ phosphate groups and ribose hydroxy oxygen atom (Agarwal and Feldman, 1968). In the nucleic acid molecule the uranium would likely be bound to 3' phosphate, to 2' OH and to N-3 nitrogen. These bindings sites are thought to be involved in metal toxicity and carcinogenic action (Luckey et al., 1975). Uranium chelation by these bioactive compounds has the effect of interfering with their activity, and retarding the rates with which they interact with other metabolic compounds.

Renal (damage to the proximal convoluted tubules of the kidneys) and hepatic injury are the major symptoms of both acute and chronic uranium toxicity. Several potentially important, interrelated physiological events have been identified, at least for the case of acute exposure to high doses of uranium (Wrenn et al., 1985, 1984; Morrow, 1984; Morrow et al., 1982a, b; Moss and McCurdy, 1982; Durbin and Wrenn, 1975; Hodge, 1973; Hursh and Spoor 1973; Yuile, 1973; Nomiyama and Foulkes, 1968; Bencosme et al., 1960; Tanenbaum et al., 1951; Voegtlin and Hodge, 1953, 1949). These physiological events include binding of the brush-border membrane in the distal portion of the proximal tubules, which may result in reduced reabsorption of Na. Consequently, reduced reabsorption of glucose, amino acids, water, and other substances will occur, even before there is significant damage to the cell. Later, structural damage to the plasma membrane may lead to more extensive changes in membrane transport and permeability (Leggett, 1989).

Uranium salts can be absorbed through the skin (Luckey et al., 1975). X ray microanalytical methods have shown that uranyl nitrate can penetrate rat skin within 15 minutes and accumulate in the intracellular space between the granular and horny layers of the skin. After 48 hours, uranium is no longer found in the skin, and the rats showed severe toxic signs including weight loss and death, indicating that the uranium had been absorbed into the blood. Death due to kidney failure occurred when uranyl nitrate, amonium uranyl tricarbonate, or uranyl acetate was brushed onto the skin of rats once daily for five days. No deaths were reported from uranium dioxide or ammonium diuranate; exposures were approximately 7 g U/kg/day. Application of uranyl nitrate to the skin of rats resulted in disrupted membranes in the cell, mitochondria, and cell nucleus, as revealed by transmission electron microscopy. Light microscopy revealed swollen and vacuolated epidermal cells, and damage to hair follicules and sebaceous glands (De Rey et al., 1984, 1983).

Apparently, other uranium compounds (uranium tetrafluoride, uranium tetrachloride, uranium trioxide) can be absorbed through the skin, since they have toxic effects when applied to the skin of mice, rats, rabbits, and guinea pigs. Absorption was also shown to occur through the conjunctival sac of the eye (Orcutt, 1949). The mechanisms for the above mentioned effects are not fully understood.

URANIUM RADIOTOXICOLOGICAL PROPERTIES

^{238}U, a radioactive alpha emitter of considerable prominence, is highly toxic from a purely chemical standpoint, in addition to its radiation hazard. It has a specific activity of 2.52 x 10^4Bq/g (Bosshard et al., 1992), and undergoes changes 14 times before it becomes non-radioactive lead. Alpha particles are emitted from radionuclides with considerable kinetic energy and pose a threat to human health when inhaled or ingested. The range of energy from natural α-emitting sources lies between about 4 and 8 MeV. The most significant feature of α-particle energy is that it is discrete. All the α-particles emitted by a specific nuclide will emerge at one well-defined energie. As an example, ^{238}U emits α-particles with energies of 4.20, 4.15, and 4.04 MeV. Such discrete α-particle energies serve as a means of identifying specific nuclides (Wang et al., 1975).

The alpha particle is composed of two neutrons and two protons and is relatively massive. On passing through matter such a positively charged particle exerts a strong attractive force on the negatively charged orbital electrons of the atoms in its track. This attaction may pull off one or more of these electrons, and the energy required for such an interaction causes the dissipation of some of the energy of the alpha particle. The electron thus removed and the positive ion left behind constitute an "ion pair" and the process is called "ionization". The interaction between the alpha particle and the atoms of the media through which it is passing may not always be strong enough to cause ionizations, but may cause "excitations". Excitation differs from ionization in that it does not involve the removal of electrons from the atoms of the media, but raises some of the inner orbital electrons to higher energy levels within the atom. The number of ion pairs formed per unit length of the alpha particle track is called its "linear ion density" or "specific ionization", and depends on the energy and charge of the particle and on the density (and atomic number, Z, of the elements) of the material through which it is travelling.

The ionization damage of radioactive decay is dependent upon three factors: First, the number of ion pairs produced is directly proportional to the initial particle energy. Second, the rate at which the energy is lost along the track of a particle is proportional to the square of the particle charge. An alpha particle (2 positive charges) will lose energy four times as fast as a proton (1 positive charge), provided that both have the same velocity. Third, the velocity of a particle also governs the rate at which a particle is able to exert its electric field on the atoms of the media.

A faster moving particle is less likely to ionize a substance through which it is passing than a slower one. As a result of their mass, alpha particles move relatively slowly and so have ample time to ionize the atoms of the media through which they pass. Because of their charge (2+) and their speed, they give up all their energy in short, dense, straight tracks which have a specific ionization pattern. As the alpha particle penetrates deeper into the media, more and more interactions (ionizations and excitations) occur thus reducing its speed, which in turn increases the chances of further interactions. Eventually, ionizations decline to zero, when all the alpha particle's energy has been dissipated. The exhausted particle then attracts two electrons to itself and becomes a neutral helium atom.

The range of a 1 MeV alpha particle is a few centimeters in air at atmospheric pressure. In living tissue, because of its greater density, a 1 MeV alpha particle is only able to travel a few micrometers. The high ionization density in the track of alpha particles (high LET) and the general nature of the alpha emitters, make them very radiotoxic if absorbed (Alpen, 1990).

Alpha radiation is the most harmful to living cells because it delivers more radioactive energy per unit path length than other types of radiation. When α-emitters enter the body by ingestion or inhalation, the particles dissipate their energy in such an exceedingly small volume of tissue that very great local damage can occur. Moreover many natural α-emitters have long half-lives, and some such as radium, plutonium and uranium are incorporated into metabollically less active tissue, thus increasing the internal hazard.

The maximum permissible concentrations of most α-emitters in the body is quite low (Wang et al., 1975).

Comparisons of various high- and low-LET ionizing radiations for their ability to induce oncogenic transformation in several cell systems have been reported. In general, high-LET radiations are far more cytotoxic and oncogenic than low-LET radiations such as x rays or gamma rays. Furthermore, the RBE for oncogenic transformation and cytotoxicity increases with increasing LET of the radiation. Hence if the transformation frequencies for each type of high-LET particle are plotted against the corresponding survival values, the curves obtained cannot be superimposed. This suggests that there is a real difference in the RBE between cell killing and transformation (Hei et al., 1988a; Yang et al., 1985) and also indicates that there is a significant frequency of transformation at doses of high-LET radiations that have very little effect on cell survival (NRC, 1990). Previous studies by Lloyd et al., 1979 showed that at a dose corresponding to a surviving fraction of 37%, about 14 particles traversed the nucleus for each cell killed. The fact that on the average 13 particles may traverse a cell nucleus without killing the cell may explain the high efficiency with which high-LET particles induce transformed loci (NRC, 1990).

The transforming ability of alpha particles also has been studied extensively with *in vitro* transformation sytems. Robertson et al., (1983) showed that the RBE for transformation by ^{238}Pu alpha particles in Balb/3T3 cells was substantially higher than that for cell lethality. It was also demonstrated that potentially lethal damage was repaired in x-irradiated 3T3 cellls and was not repaired in alpha-particle irradiated cells, resulting in a high RBE value for oncogenic transformation in alpha-irradiated plateu-phase cultures. At equivalent doses, alpha particles were substantially more cytotoxic than gamma rays and were more efficient in inducing oncogenic transformation. The calculated RBE for alpha particles ranged from 2.3 to 9 over the range of doses studied, with the highest RBE value at the lowest dose. Recent results have suggested the absence of a dose-rate effect with alpha particles (Hieber et al., 1987).

The radiological toxicity in human skeletal tissues is rather hypothetical (Wrenn et al., 1985). However, from the biological stand point, bone is considered to be the critical organ for long-term radiation exposure to soluble uranium compounds (Bosshard et al., 1992). The deposition of uranium in skeletal tissues was originally considered not to be important, because of the low specific radioactivity of natural uranium. But, uranium behaves chemically like calcium, and the skeletal system might be the target organ in the case of enriched uranium. The use of autoradiography with isotopes with high specific activities has provided much experimental information on the mechanism of deposition of uranium in bone. It was shown that uranium shares characteristics with radium, which is distributed throughout bone (a volume seeker), and with plutonium, which remains on the surface of the bone (Priest et al., 1982; Rowland and Farnham, 1969). The initial deposition is nonuniform and occurs at the sites of active calcification (Tannebaum, 1951; Neuman et al., 1949). Subsequently, there is a slow relocation throughout the volume of the bone. The mechanisms or relationship between any ionizing or activation event and its radiobiological effect are extremely complicated, and at present, not completely understood, because it involves a complex sequence of events initiated by the absorption of energy followed by biophysical, biochemical, and biological changes. Although the mechanisms are not completely understood, all forms of ionizing radiation have been shown to be harmful. For instance, when DNA was identified as the universal genetic material, it soon became obvious, and was confirmed by overwhelming genetic evidence, that, in principle, the genetic material of all living beings is susceptible to radiation-induced damage (Vogel, 1992).

URANIUM CARCINOGENESIS

For decades, scientists have suspected a link between natural radiation exposure and cancer. Yet, the effect has only been demonstrated for a few types of radiation. For instance, ordinary sunlight with its ultraviolet component has been recognized as a cause of skin cancer. Nevertheless, the evidence on the effects of ionizing radiation has been indirect. It consists primarily of epidemiological studies of human cohorts living in highly radioactive areas and toxicological studies on animals.

At this time the etiology of radiation-induced cancer is complex and incompletely understood because the susceptibility to radiation as a carcinogenic physical agent can be affected by a number of factors. These factors include: the cancer type and tissue at risk; the genetic constitution; the age and sex of the person exposed; the magnitude of the dose to a particular organ; the quality of radiation; the nature of the exposure, whether brief or chronic. The presence of factors such as exposure to other carcinogens and promoters may modify the radiation induced carcinogenesis. Physiological state and individual characteristics that as yet cannot be specified may help to explain why some persons do and others do not develop cancer when similarly exposed.

Cancer induction by uranium is presumably the radiobiological effect of chronic exposure to environmental levels. Nonetheless, there is conciderable debate about the extent of the cancer risk when people are chronically exposed to very low levels of radiation. A few authors have suggested association between areas with enhanced uranium levels and cancer incidence (Crounse et al., 1983; Doll and Peto, 1981).

Experimentally, metallic uranium powder dispersed with lanolin was implanted in rats, either once into the marrow cavity of the femur (50 mgU) or in six monthly injections into the pleural cavity (50 mgU each). Acute mortality and renal damage indicated that a small fraction of the uranium dissolved. Tumors of parietal serous membranes and connective tissue developed at or adjacent to the uranium injection sites. However, the authors were unable to decide whether the local tumors induced by insoluble uranium were caused by its chemical or physical properties (Hueper et al., 1952).

Human uranium occupational exposure through inhalation has been linked to lung cancer deaths (Saccomanno et al., 1986; Samet et al., 1984; Gottlieb and Husen, 1982; Archer et al., 1973a; Lundin et al., 1969). People occupationally exposed have also developed lymphatic malignancies. Waxweiler et al., (1983) in a study of 2002 uranium millers reported 6 deaths from lymphatic malignancies with a latency period of 20 years noting that only 2.6 were expected. Archer et al., (1973b) also found a slight excess in deaths from tumors of the lymphatic and hematopoietic tissue.

It is difficult to determine whether inhalation exposure to natural uranium will cause cancer in humans, although an inhalation study of rats, dogs, and monkeys showed that exposure to airborne concentrations of 5 mg/m^3 of uranium dioxide (UO_2) dust for up to 5 years produced accumulation in the lungs. Tracheobronchial lymph nodes which accounted for 90% of the body burden of uranium. Lung cancer was produced in rats and dogs but not in monkeys after continuous inhalation for 2-5 years (Leach et al., 1973; 1970). Though the studies are not extensive or conclusive, the possibility of cancer induction can not be ruled out due to the fact that uranium and its isotopes are α-emitting radionuclides, especially enriched uranium. Still, other α-emitting radionuclides, such as radon, radium, and plutonium, are known to cause cancer from radiation.

For the long-lived members of the decay series (Table 1) and some of their shorter-lived progeny, the radiation doses to osteoprogenitor cells (stem cells) lining bone surfaces and the bone marrow are usually considered to be of greater significance than retention of these nuclides in the body and because these tissues are thought to be critical sites in the production of bone sarcomas and leukemias (Linsata, 1994).

Bone sarcomas have been induced in mice with high-specific-activity ^{232}U and ^{233}U. A retrospective dosimetric analysis (Durbin and Wrenn 1975) showed that, with respect to a linear-dose relationship, the effectiveness of both uranium isotopes was equal to and not distinguishable from that of ^{226}Ra in the same strain of mouse. Investigators from the USSR have shown that highly enriched uranium can induce bone sarcomas in rats. Accordingly, it is reasonable to believe that high-specifc-activity uranium can produce bone sarcomas in humans. If there is a linear dose-response relationship (Mays et al., 1985) one could speculate that uranium or slightly enriched uranium could also induce bone sarcomas in humans, but the likelihood would be very small because of the low specific activity of uranium. Nonetheless, Mays et al., 1985, have estimated, using toxicity ratios, the risk of bone sarcoma induction from chronic ingestion of natural uranium, on the basis of an assumed linear dose-response function for ^{226}Ra. The proportionality of response to average energy deposition in the skeleton by alpha particles is a reasonable assumption, because there is metabolic evidence that uranium is a skeletal volume seeker similar to, although not identical with, radium.

Sarcomatous tumors have also been induced in humans by large, long standing deposits of extravasated (blood), insoluble, finely divided $^{232}ThO_2$. The primary α-emissions of this thorium isotope are comparable in energy to ^{232}U (Mays et al., 1985; Mays and Speiss, 1984). These studies demonstrate a high likelihood for natural uranium exposure to increase the risk of cancer in mammals. In addition, there is evidence that individuals with specific inherited mutations such as recessively inherited conditions (retinoblastoma), or genetic polymorphism, may be more sensitive to radiation-induced tissue transformations.

Patients with retinoblastoma have a high risk of developing osteosarcoma of the orbit following radiation therapy. They also have a lesser predisposition to osteosarcoma in the absence of irradiation. In either case, the genetic change in the tumor cells is the loss of the two normal alleles of the retinoblastoma gene; thus, this gene is a tumor suppressor gene for osteosarcoma (Hall and Hei, 1985) as well as for retinoblastoma. The probability of mutation or loss of the normal gene in persons born with one mutant gene in the germ line is apparently increased by radiation, as would be expected (NRC, 1990).

Recessive inherited conditions like xeroderma pigmentosum, ataxia telangiectasia, Fanconi's anemia, and Bloom's syndrome, that predispose the chromosomes of an individual to breakage and/or defective repair of DNA damage (Hanawalt and Sarasin, 1986) do not involve cancer genes of the types discussed above but can be viewed as conditions that increase the probability of a cancer-producing mutation. Thus, in xeroderma pigmentosum, a defect in excision repair permits an increased rate of mutations at all genetic loci in cells exposed to sunlight. Ataxia telagiectasia predisposes the chromosome to breakage, especially in lymphocytes; the underlying molecular defect is not known, but it is thought to involve a defect in DNA repair. Patients with this syndrome are especially predisposed to lymphoid neoplasia, and their cells are highly sensitive to ionizing radiation. Chromosome breakage and rearrangement are regular features of Fanconi's anemia, which predisposes an individual to acute myelomonocytic leukemia; the underlying molecular defect for this is not known. Finally, Bloom's syndrome is associated with high rates of mutations (sister chromatid and even homologous chromosome exchanges). The molecular defect apparently involves a ligase that is important in the repair of DNA damage (Chan et al., 1987; Willis and Lindahal, 1987). The syndrome predisposes an individual to several kinds of neoplasia, perhaps by facilitating mutation, somatic recombination, and the expression of recessive oncogenes (NRC, 1990). In contrast to the aforementioned DNA repair disorders, in which the response to an environmental agent is altered, there are cases in which the response may be normal but the amount of radiant energy imparted is increased. By way of illustration, albinos are sensitive to ultraviolet light because they absorb more of it, not because they have a defective DNA repair mechanism. Traits affecting the metabolism of a chemical may alter susceptibility to radiation carcinogenesis (NRC, 1990). Another kind of inherited mutation that may predispose an individual to cancer are hereditary fragile genetic sites (chromosomes). Although several sites have been found to be situated at or near break points that are known to be involved in various cancer-associated translocations (LeBeau and Rowley, 1984), cancer does not appear to be common in families with such abnormalities. The importance of these mutations in carcinogenesis thus remains to be determined.

Animal feeding studies have shown that natural uranium causes nonneoplastic kidney damage, but the studies have not shown any tumors in any organs after natural uranium oral intake. However, theoretically, it is possible that exposure to any radioactive substance will cause cancer.

Studies in the current literature linking natural uranium to dermal exposure and cancer are lacking. Nonetheless, enriched uranium administered by other routes (intravenous or intratracheal) in experimental animals has been shown to cause tumors. Intravenous injections of ^{233}U have been shown to cause bone tumors in mice (Finkel, 1953). Intratracheal administration of ^{235}U and ^{234}U have been shown to cause bone, lung, and kidney tumors to 90% of rats (Filippova et al., 1978).

A 1990 NRC report, concludes that: cancer rates are highly age dependent and, in general, increase rapidly in old age. The expression of radiogenic cancer varies with age so that the age dependent increase in the excess risk of radiogenic cancer is conveniently expressed in terms of relative risk; that is, the increased risk tends to be proportional to the baseline risk in the same age interval. In some cases, however, such as breast cancer, the change in baseline cancer rate with age is more complicated and possibly related to variations in hormonal status with age. Susceptibility to radiation-induced breast cancer may be similarly complicated, and there is some indication that protective factors for breast cancer in nonirradiated women, such as early age at the birth of the first child, may also be relevant for radiation-induced breast cancer. For lung cancer and most other non-sex-specific solid cancers, it is unclear how a person's sex affects the risk of radiogenic cancer. In general, baseline rates for such cancer in males exceed those in females, possibly because of increased exposure to carcinogens and promoters in occupational activities and life-style factors, such as increased smoking and the use of alcohol.

For ingestion in water or food at a constant daily rate of 5 pCi/day and nonoccupational intake, Mays et al., (1985) estimated that the risk of bone-sarcoma induction over a lifetime is 1.5 bone sarcomas/million persons for ^{233}U, ^{234}U, ^{235}U, ^{236}U, or ^{238}U, if the dose response is linear. If the dose-response relationship is quadratic, then virtually no effect would be expected as a result of exposure to natural uranium. In a million people in the United States, the naturally occurring bone sarcomas would number about 750 (Mays et al., 1985).

PRELIMINARY STUDY

REGIONAL BACKGROUND

The State of Baja California Sur, Mexico (Fig. 1), is located on the meridional portion of the peninsula of Baja California, to the NW of the Mexican Republic, over the Tropic of Cancer, between parallels 22°30′ and 28° North, and between the meridians 109° and 115° W of Greenwich. The Northern limits of the State of Baja California Norte are, along parallel 28° which serves as a divisory line; to the West the Pacific Ocean; to the East the Gulf of California; and to the South the sea where the waters of the Pacific and the Sea of Cortez meet. Its territorial surface is 73,475 km^2 with a length of 750 kms, a median width of 100 kms, and median altitude of 600 m above sea level.

The waters surrounding the Baja California peninsula are rich in piscivorous resources. In the inlets, coves, and coastal lagoons there are lobster, shrimp, clams, tuna, sardine, garropa, cabrilla, mero, turtle and abalone. In addition, there is an abundance of sport fishing species: marlin, jurel, bonito, pez vela, pez gallo, dorado, sierra, totoaba, and others. Most of the fisheries production is consumed in Baja California Sur but some is exported to Ensenada, Baja California Norte. In recent years, the rest of the supply goes to packing industry and fish flour factories.

In Baja California Sur fresh water sources are mainly subterranean, since there are no proper rivers, although there are temporary brooks and streamlets during the seasonal rains or storms. The coldest months are December, January, and February; the warmest are July, August, and September. Rains are scarce, although most frequent in August. From November to May the NW offshore winds blow from around 9 AM - 4 PM, and at dusk the winds start blowing from the south onshore lasting all night; during the rest of the year the dominant winds are SE and SW.

La Paz Bay according to Murillo (1987) is the largest on the east coast of Baja California (Fig. 2). It is located on the southeast coast of the Gulf of California, Mexico between the parallels 24.1° - 24.8° North latitude and longitude 110.2° -110.8° West.

The Bay is a shallow marine depression, with increasing depth from south to north (~1% slope), until it reaches the 500 m isobath in its northern limit (mouth of the bay). The eastern limit is provided by the Espiritu Santo and La Partida islands. Between the islands and the mainland there is a shallow channel called El Canal de San Lorenzo (aproximately 50 m in depth) through which an exchange of waters from the gulf and the bay takes place. To the south, the bay is limited by a sandy barrier called 'El Mogote' consisting of alluvial deposits. The western limits present a steep escarpment from a regional fault that forms part of La Paz Valley. On the escarpment, tertiary sedimentary rock outcrops consist of sandstone and tufa quarry and are crowned by splay lava and volcano sediments (Hausback, 1984).

The bay receives natural minerals from the weathering and leaching-out process of three geologic formations (two sedimentary and one volcanic) with eight well defined phosphate layers of 0.15 to 2.0 m thickness (ROFOMEX). The bay waters also receive the tailings of the anthropogenic extraction of the phosphorite from those layers. The precise current movement (circulation) of the bay's waters is not known. However, the superficial circulation is related to the seasonal wind patterns and the relatively stable coastal currents generated by the tides. Based on the geomorphologic coastal features of the region and the above processes, two currents have been inferred. The currents are established from NE to SW, depending on the tidal currents. During October, and February such currents reach considerable velocities, approximately 3 knots (Obeso Nieblas, 1986).

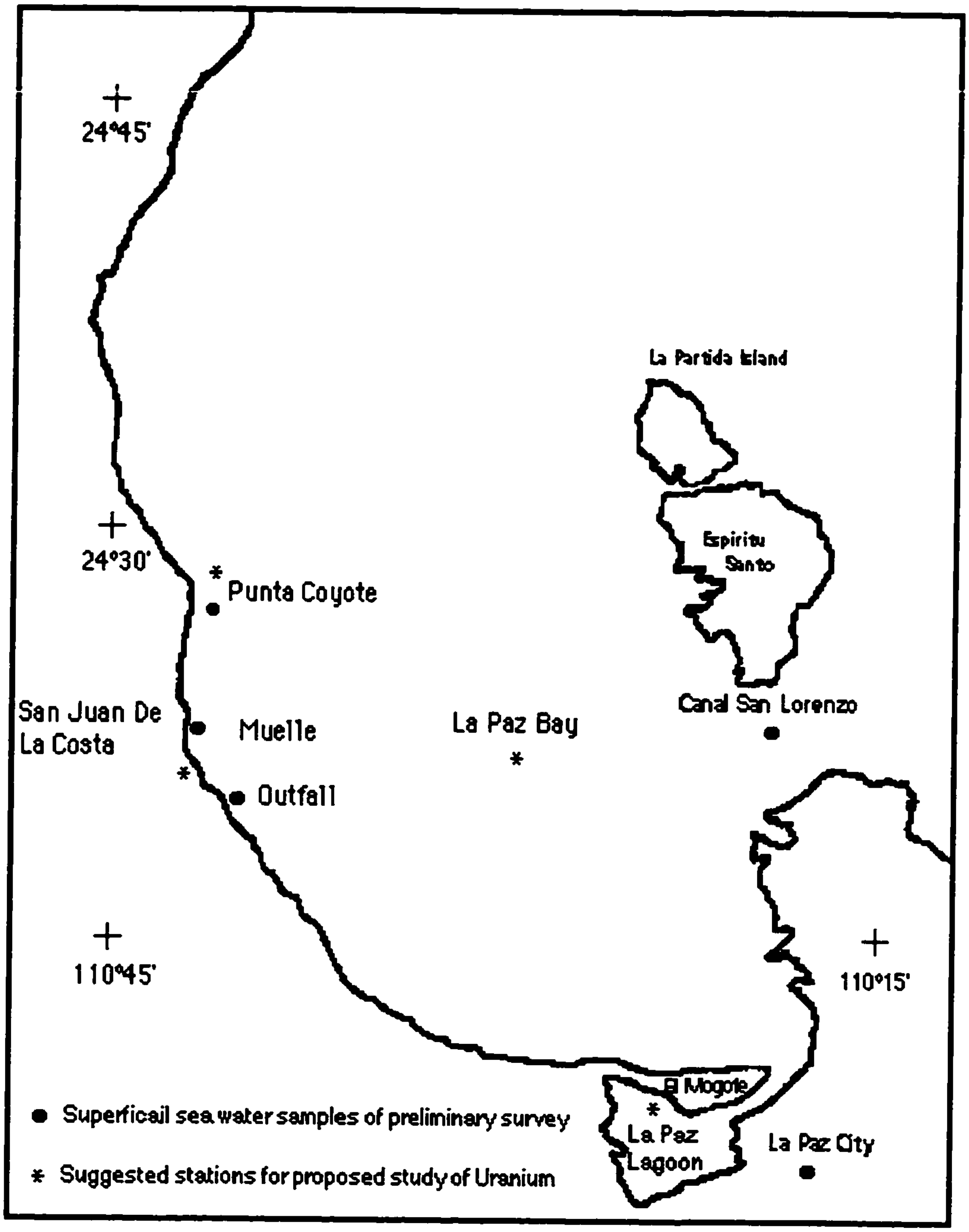


Figure 2. Bahia De La Paz, Mexico.

Bahia de La Paz is the natural receiving system for the fluvial runoff and mineral leaching of the neighboring phosphorite ore in San Juan De La Costa (Fig. 2). Phosphate ores are characterized by moderate to high uranium concentrations. Uranium in mineral phases is easily weathered and mobilized (Ball and Miles, 1993). Evidence of the extensive mobilization of uranium from the rocks was provided by Tandy (1974, 1973). Naturally occurring uranium concentrations in the media rock-water-air-biota are a function of the regional geomineralogy, climate, and their many interactions all acting through time. Therefore, it appears that La Paz Bay is the natural reservoir for the erosion, leaching and weathering of the minerals from the regional phosphate ores with the subsequent increase of uranium levels in the nearshore waters of Bahia de La Paz.

The city of La Paz (Fig. 2) is located in the south part of the bay , at an altitude of 10 m above sea level. La Paz city is the capital of the State of Baja California Sur, Mexico, with 250,000 inhabitants (Campillo, 1991). Around the bay there are a number of dispersed fishing communities and small farms in areas adjacent to the phosphorite rock region. Located in this region is San Juan De La Costa, a town of 383 people, situated between the phosphorite plant and the Bay. Formerly a fishing village, it has become, in recent years, an increasingly popular camping spot during the summer season. The people from this semi-developed and tourist region are potentially chronically exposed to enhanced uranium levels that are leached from the immediate geologic environment.

The rationale for investigating the natural isotopic abundance of uranium and the potential exposure of a human population is based on the following:

1) a well-characterized exposure area;

2) potential exposure of the population to chronic doses;

3) no substantial exposure to uranium from other sources;

4) a large enough population to have sufficient statistical power to distinguish differences in cancer rates with exposure levels of the population at risk.

The author believes that La Paz municipality has the characteristics listed above. Furthermore, this region permits the use of Tembabiche, in the municipality of Comondu, as a control population. La Paz municipality has two inland phosphorous deposits (San Hilario, Santa Rita) and one on the Sea of Cortez (San Juan de la Costa) currently being exploited commercially (Fig. 1). Because the area is geographically isolated, the uranium input since the Miocene comes only from the natural weathering of the phosphorite deposits (ROFOMEX). This translates into a uniform exposure.

In any case, an assessment is required whether the uranium introduced to the Bay's waters originates from natural weathering processes or is indirectly related to anthropogenic actions. The assessment is needed to increase our understanding of the radiochemistry for this particular geographic area to answer specific questions such as: What are the local isotopic abundances of uranium?; What is the uranium content at different levels in the biologic food chain?; How do we evaluate the radiobiologic effects of the uranium concentrations on the local populations?

A preliminary study was conducted to address the first question. The preliminary assay included sampling of superficial nearshore sea water and subsequent isotopic dilution analysis.

SAMPLE RECOVERY AND PREPARATION

Preliminary sea water samples were obtained from 4 stations around La Paz Bay from an out-board motor boat on Jan. 6 1993. These were (Fig. 2): A station to the North at "Punta el coyote", to the South at San Juan de La Costa ("Outfall" and "Muelle"), and to the west at "Canal San Lorenzo". The samples were collected from the surface waters (at 0 meters) according to the standard depths procedure (Grasshoff, 1976). The samples were neither acidified nor filtered because stored uranium samples in 20 liter polyethylene cubitainers are not altered within approximately one year. Moreover, most of the uranium in sea water is present in the dissolved state (Ku, 1977).

ANALYTICAL METHOD

The surface samples were analyzed for uranium by an isotope dilution technique with a ^{232}U tracer of known activity. The activity ratios between ^{232}U and the other uranium isotopes were determined by an alpha-particle spectrometric analysis (Ku, 1965). This method has been verified as an accurate technique by several authors (Williams,1988; Ku 1977; Veeh, 1968; Minoru and Goldberg, 1965; Ku, 1965; Blanchard, 1965; Mitsunobu and Goldberg, 1959; Wilson et al., 1960; Rona, 1956).

One of the advantages over other methods is that we can tolerate losses in the chemical separation without adversely affecting the results (Rona et al., 1956). However, the procedure to measure the Activity Ratio (AR) must satisfy three requirements: first, complete isotopic equilibrium between natural isotopes and added ^{232}U tracer; second, the counting sample must be extremely thin to eliminate energy degradation of the emitted particles (for alpha spectroscopy), and, third uranium must be completely separated from radium and thorium (because of their similarities in alpha-particle energies). The details of the analytical technique are described in Williams (1988).

URANIUM SEPARATION AND COUNTING METHODS

Samples were returned to the laboratory where the salinity was determined using a refractometer prior to acidification with 50 mL concentrated HCl. Samples were stored-acidified for one week. The yield tracer, ^{232}U, together with 50 mg Fe (as $FeCl_3$) were added to the samples. The tracer and iron were mixed by bubbling with nitrogen (3 hours) and allowed to sit overnight. The following day, samples were shaken vigorously to insure isotopic equilibration prior to the addition of 50 mL 12 M NaOH. The iron hydroxide precipitate which formed was allowed to settle and was collected on glass fiber filters. The precipitate was dissolved in 12 N HCl and loaded onto anion exchange columns (Biorad, AG-1 x 8 anion exchange resin, 20 mL col. vol., charged with 8 N HCl). Uranium and iron both adsorb to the column under these conditions. After the sample was completely loaded, the column was rinsed with about 3 column volumes of 8 N HCl. Nitric acid (7.5 N) was then added to the column. The first HNO_3 fractions containing iron were discarded until the column effluent cleared. The uranium fraction that followed was collected in about 100 mLs and taken to dryness in a 150 mL glass beaker. Further uranium purification was accomplished using anion exchange on a 7 mL column charged with 7.5 N HNO_3. A 5 mL sample was loaded, washed with 4 column volumes and eluted with water and weak HCl. The purified residue containing the uranium isotopes was oxidized with a few drops of concentrated HNO_3 and brought to dryness. The residue was then taken up in 2 mL 2 M NH_4Cl adjusted to pH 2 with HCl and transferred to an electrodeposition cell containing a 1" stainless steel planchet. The sample beaker was rinsed with two additional 2 mL rinses of the NH_4Cl solution and these rinses were also transferred to the electrodeposition cell. A platinum anode was inserted into the cell and electrodeposition proceeded at 7 volts, 2 amps for about 15 min. after which a 1 mL H_2O rinse was added to the plating cell. Electrodeposition continued for another 15 min. before termination by the addition of 1 mL NH_4OH. The planchet was rinsed with H_2O and air dried prior to alpha counting.

Uranium samples plated on platinum planchets were inserted into Tennelec TC-256 alpha spectrometers fitted with Ortec silicone surface barrier detectors. Signals were processed through a DMR 108A digital multiplexer router to a PCA-II Universal ADC interface. The alpha energy spectra were then collected using the Nucleus DMR-II multichannel analyzer on an IBM PS-I 386 SX computer. Alpha spectra of the uranium isotopes were quantified by integration of the regions of interest for each isotope and corrected for detector backgrounds (negligible in these regions).

Uranium activities were determined by ratio of the ^{232}U spike integral to the ^{238}U integral from the offshore sample. We assume the ^{238}U activity of the San Lorenzo sample is consistent with the relationship determined by Ku (1977) and the activity of this sample is 2.3 dpm/L. By calibrating the internal tracer ^{232}U activity in this way, all other isotope activities were determined.

RESULTS

The results of the preliminary analysis of the four coastal surface samples are presented in the spectra in Fig. 3. Sample salinities, counting times, activity ratios and concentrations are tabulated in Table 2.

The findings in Table 2 suggest a radioactive disequilibrium between ^{234}U and its parent ^{238}U at the San Juan de La Costa station (outfall and muelle) compared to normal ratios at San Lorenzo and Coyote. This last two samples are in excellent agreement with the averaged expected equilibrium values (Coale and Bruland, 1987). The results were unexpected for two reasons:

1) The two isotopes are separated in the decay chain by two short lived nuclides and therefore should have an isotopic ratio close to 1.0.

2) The two uranium isotopes are expected to behave the same chemically and therefore the reaction should not favor one isotope over the other.

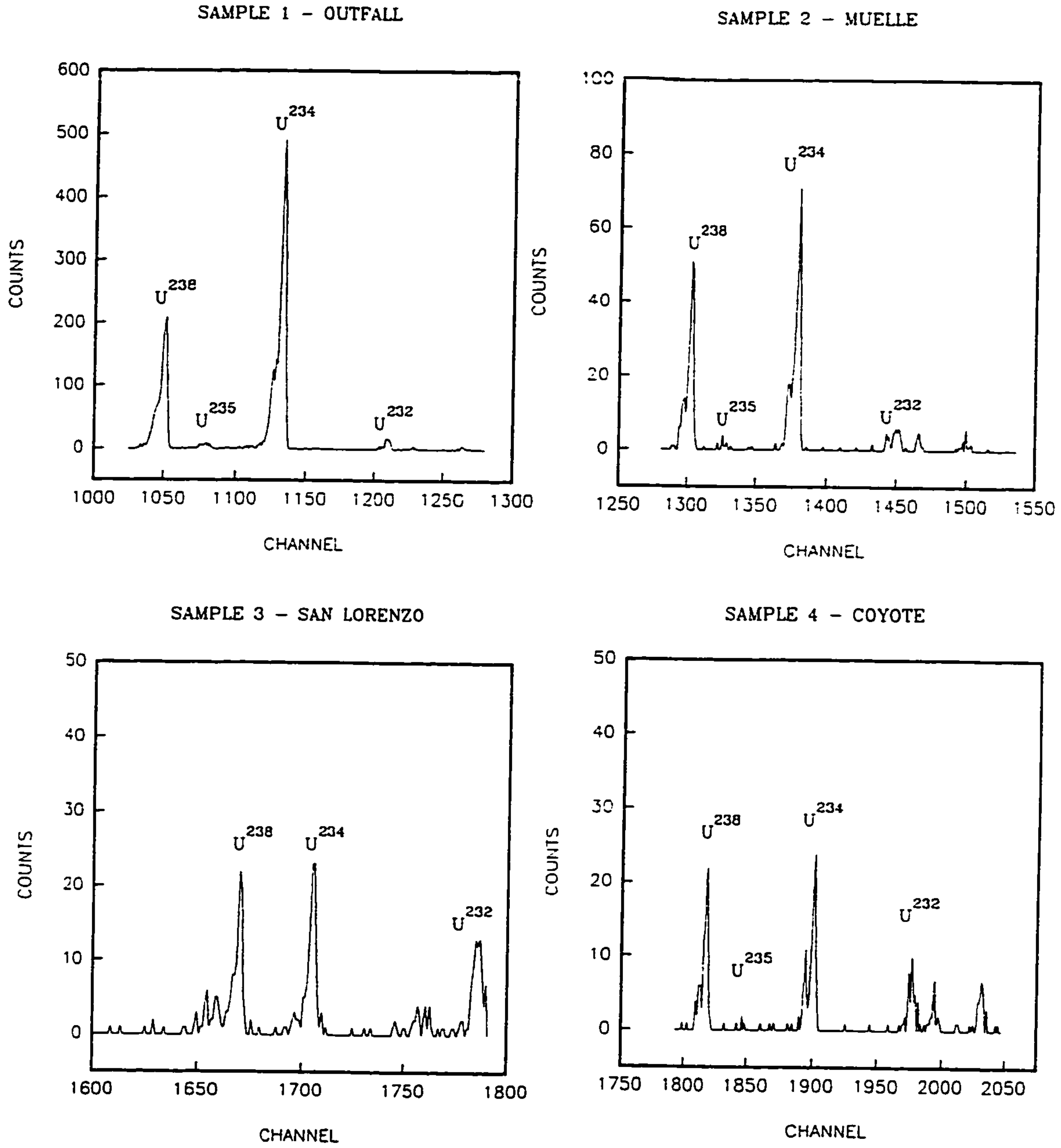
The results of the preliminary uranium isotopic analysis point to the following main findings:

1) Concentrations and isotopic composition of uranium from offshore stations (San Lorenzo and Coyote) appear to reflect that commonly found in seawater.

2) Nearshore stations deviate in concentration and composition with A ^{234}U > A ^{238}U characteristic of a distinctive non-equilibrium source.

3) The point source (effluent from the phosphorite plant) sample has an extremely anomalous ^{234}U : ^{238}U ratio, probably reflecting the preferential mobilization of ^{234}U due to alpha-recoil and ore processing.

4) The uranium activity in the effluent samples (outfall and muelle) is extremely high (~10 times the natural concentration).



SAMPLE 1 - OUTFALL

SAMPLE 2 - MUELLE

SAMPLE 3 - SAN LORENZO

SAMPLE 4 - COYOTE

Figure 3. Alpha Spectra of surface coastal samples from La Paz Bay, using ^{232}U as a yield tracer. Natural ^{238}U and its isotopes can easily be identified by the well defined [total counts/energy (Mev-channel)] ratio of peaks.

Table 2. Concentrations Of Uranium Isotopes From La Paz Bay By Isotope Dilution Alpha Spectrometry And ^{232}U As A Tracer.

Sample	Salinity per mille	Counting time (seconds)	^{238}U	^{235}U	^{234}U	^{232}U	$^{234}U/^{238}U$ Isotopic Activity Ratios	Concentration Disintegrations per minute (dpm)
Outfall	50	604600	1365	56	2815	90	2.0622	322
Muelle	40	604600	256	11	324	49	1.2656	110
San Lorenzo	40	258887	122	0	110	75	0.9016	21
Coyote	40	604600	113	4	123	25	1.0884	95

DISCUSSION

The findings are significant in three important ways: First, they identify phosphate ore processing facilities as a large but localized source of uranium into the marine environment. The local effects appear to dissipate from the source within the natural boundaries of the Bay and therefore, are likely only to impact the local environment. Second, the marine discharge from this source has a distinctive isotopic composition. The isotopic composition would be helpful in the identification of uranium incorporated into the local marine biota. Third, since the isotopic composition of the phosphate deposits are assumed to be in secular equilibrium, it may be possible to distinguish terrestrial from marine sources of uranium in the human population. The resultant mineral phase (particulates, sediments, dust,) should have $^{234}U : ^{238}U$ activity ratios which are depleted in the same mass/ratio to the extent that the dissolved effluent is enriched. Therefore those solid phase products should have a $^{234}U : ^{238}U$ ratio < 1 and should be a trace of terrestrial/particulate exposure.

CONCLUSION

From the average activity ratios (A.R.) of the preliminary uranium isotope measurements (table 2) and the location of the sample stations (Fig. 2) it can be concluded that the disequilibrium is local and temporal rather than general. Further sampling and analyses are needed. It would be helpful to have data to determine the mobilization of the activity ratios found in the preliminary study as well as to evaluate the possible local effects of uranium for further association of health risks for those living near the enriched area.

FURTHER INVESTIGATIONS

RESEARCH PLAN

The natural isotopic abundance of uranium in a coastal setting and the potential radiologic hazard may be evaluated in various ways. Detailed studies using radioanalytical methods to monitor environmental uranium variations and epidemiologic analysis in relation to carcinogenesis should be done in regions with enhanced levels of uranium. Geographic variation of uranium suggests, geochemical influence in the prevalence of different kinds of cancers (Crounse et al., 1983; Doll and Peto, 1981). Links between local uranium contents and cancer incidence are plausible. Only recently has attention been paid to the possibility that local geochemical anomalies might be a cancer factor.

General knowledge allows us to infer that the uranium distribution is influenced by chemical, biological, physical, and atmospheric processes. Consequently, a seasonal sampling carried out every four months is suggested for this study in order to investigate the uranium isotopic content in a coastal region close to phosphate sediments.

In the present research plan we propose a three stage study design. For stage one we will conduct concurrent sampling of environmental media using radiochemical analysis to determine uranium isotopic abundances and to identify trends in the food chain and localized enhancements. This study will determine the frequency of occurrence of high uranium activities according to specified attributes, such as geographic and seasonal patterns. The data may also help to determine the magnitude of the exposure (for organisms and the human population at risk).

For stage two we will use software modeling programs to analyze the area-specific data obtained from stage one. Such an approach will provide a unique analysis to determine local uranium sources and levels, pathways of radionuclide transport and the radiotoxic risk in the coastal environment of La Paz Bay.

For stage three we will complete an analysis of data from stage one and two to obtain estimates of radiation exposure from localized uranium levels.

Such an integrated approach would allow for further qualitative and quantitative studies of habitat conditions, ecosystem composition or other confounding factors that may be important for the evaluation of potential human health and ecologic risk.

PROBLEM STATEMENT

There are five phosphorite bed formations in the state of Baja California Sur, Mexico (Fig. 1): Santo Domingo, San Hilario, and Santa Rita on the Pacific Coast. Two lay on the Mar Bermejo Coast at Tembabiche and San Juan de la Costa (ROFOMEX). The marine phosphorite along the Baja California Margin are associated with areas of strong coastal upwelling and influence biological productivity (Burnett and Veeh, 1992)

Natural weathering processes play an important role in the localized enrichment, transport and behavior of uranium (Barisic, et al. 1992) in these ecosystems. In addition, mining and processing of the local ores redistributes much of the uranium and its daughters. The annual mining and milling of phosphate rock extracts several thousand curies of uranium and its decay products. The industry's waste products, including slimes, tailings, slag, slurries, and gypsum contain the bulk of the radioactivity extracted from the ground and are discharged into the environment. Consequently, any use of these materials may result in increased population exposure to radiation. Sources of contamination include waste water leaching into the aquifers, surface water systems and sea water. The airborne suspension of radionuclide bearing particulate matter by vehicular traffic on loose-dry roads and wind erosion is another route of contamination.

It is likely that wind suspended particulate emissions are of great significance because of the extreme dry conditions of the area. It also has been shown that certain structures built on reclaimed phosphate land contain high indoor radon concentrations. Bottrell (1993) describes how uranium can be concentrated by physical processes during weathering and resedimentation, and redistributed in a form more amenable to radon release. This emphasizes the potential for population exposure due to mismanagement of the wastes. If exploitation of the phosphate is done by open-cast mining, workers at the mine are likely to be affected by direct external radiation from the phosphate rock and through inhalation of dust from the mine. When beneficiation of the phosphate rock is done by the dry method, the dust arising during this process could also affect workers and immediate communities through inhalation (Makweba and Holm, 1993). Thus, the radioactive constituents from phosphate mining, manufacturing products and wastes have the potential of becoming environmental pollutants.

Although uranium is present in most phosphate deposits, the higher concentrations are associated with the marine phosphate deposits (Kim and Burnett, 1985). For example, the typical concentration of ^{238}U in sedimentary phosphate deposits is about 1500 Bq kg^{-1} (UNSCEAR, 1988). This is probably due to the dissolution of the uranium by the marine environment and later redeposition (Osburn, 1965).

Data on the enrichment of uranium levels as a result of natural weathering or mining of phosphorus rock in Baja California is lacking. Presently, limited data exist on the relationship between naturally enhanced uranium levels and cancer as an endpoint of environmental high-LET radiation. How such an increase will affect the health status of human populations is part of the service that scientists are expected to offer. It is incumbent upon us to understand the consequences of our actions both as they affect humans and other organisms with which we share this environment.

The lack of analytical data has prevented detailed knowledge of isotopic regional variation of uranium in the natural environment of Bahia de La Paz. A preliminary study was conducted in order to assess the uranium isotopic composition and concentration of uranium in the nearshore environment. The preliminary study was designed to test the hypothesis that both isotopic composition and concentration would be altered close to shore as a result of local weathering and mine activity. The resultant alpha spectra provided evidence to this effect (Fig. 3). These spectra demonstrate that some points in the Bay present anomalous $^{234}U : {}^{238}U$ ratios (Table 2). The ratios are enhanced in uranium activity by an order of magnitude and they are likely to impact the local environment.

In summary, the La Paz Bay system, surrounded by the phosphorite ore, presents an anomalous uranium content, as a result of the uranium released in the course of natural weathering and mining processes. Thus, this area is subject to high natural radioactivity. Regions of this kind offer valuable opportunities to investigate the long term consequences of chronic exposure of living systems to natural uranium.

HYPOTHESIS

The proposed uranium assessment is designed to test the following hypothesis: the environmental exposure to uranium and its daughter products is enhanced where weathering is accelerated through natural or anthropogenic activities.

OBJECTIVES

The overall goal is to identify the naturally occurring isotopes of uranium and their concentration in environmental samples and human tissues at selected sites in the State of Baja California Sur, Mexico. The research plan will enhance the scientific knowledge of the regional content of natural uranium, at shorelines immediate to phosphate beds.

Specific aims for the proposed study include:

1) Determine the distribution and isotopic ratio of ^{234}U:^{238}Uin abiotic media (water, soil, dust) in the area of La Paz Bay.

2) Determine the distribution and isotopic ratio of uranium in organisms at critical levels of the food chain for humans.

3) Employ a computer software application (predictive model "Environmental IMPACT") to assess the data and model the distribution and transport of uranium in the environment and ecosystem in the surrounding region of La Paz Bay.

4) Estimate the uranium toxicological and radiation health risk to the human population of La Paz Bay.

SIGNIFICANCE

It is necessary to distinguish, for the context of this proposal, that uranium in its natural, depleted or slightly enriched state has the potential to exert deleterious effects. The proposed will add to the existing pool of uranium data for shoreline ecosystems when phosphorus beds are present in the region.

The data that will be obtained will be valuable in predicting the uranium transport through the ecosystem. Furthermore, baseline data on a local and regional scale would be useful to determine the modes of uranium exposure. The data would complement the existing information and provide a basis for future epidemiologic studies to elucidate the relationship between chronic uranium exposure and oncogenesis. The findings that emerge will have substantial human health and regulatory significance for these communities and others with similar radiogeology.

In conclusion, the undertaking of the present research plan will increase knowledge and understanding of natural uranium radiation exposure in a coastal environment. It will enable local estimates to be made. It will indicate those critical living systems where remedial or preventive action might be required.

REVIEW AND SELECTION OF POTENTIAL MEDIA FOR URANIUM ASSAY

It is an almost impossible task to examine all the biotic and abiotic media because of the vast array of biotic media, sample availability, time (collection and laboratory) and expense. There is no evidence in the literature to support the view that the distribution of uranium in animals and in human volunteers, following intake of simple inorganic salts of uranium, will resemble that following intake from natural sources in which uranium will be present in a variety of inorganic and organic chemical forms. In the present research plan we suggest the following:

a) Surface sea water sampling will be done at five stations, representative of the bay system according to geographical location. Sampling will include potential point sources such as the phosphorite plant outfalls to rule out anthropogenic sources.

b) Fresh water will be sampled from wells feeding La Paz City and San Juan De La Costa, since the human communities in the region receive water from different aquifers. Maps for the well locations can be obtained from the Water Department ("Secretaria de Recursos Hidraulicos").

c) Settled dust (at the N. S. E. W of both La Paz city and San Juan de La Costa Town) will also be sampled.

d) Sea cucumber samples will be used as indicators for uranium content in marine biotic media because they may reflect the uranium levels of the coastal sediments and the persistence of radioisotopes in soft tissue in the absence of bone.

e) Human teeth extracted during routine dental procedures will be used to determine uranium incorporation in human tissue. Teeth are sampled because of the potential health hazards posed by other human tissues or fluids. Teeth samples will be obtained from persons who have not been directly exposed to uranium except from the natural environment and also from workers directly exposed from the phosphorus mine.

Suggested sampling sources to survey uranium levels in La Paz Bay have been summarized in Table 3. Still, there is an immense array of materials to sample and process for isotopic determination. The advantages and disadvantages of some of the suggested and other potential media are discussed in the following section.

Table 3. Suggested Media For A Four Season Set Sampling (1997) For The U Survey Of La Paz Bay.

Station/Locality	Media	Total Samples per Media
San Juan de La Costa	Surface Sea Water 1-20L	3
	1 settled dust sample from North, South,East and West	4
	1-20L Ground water sample/well	4
	100g collective teeth sample	1
	1-100g Sea cucumber sample	4
	1-marine sediment core sample	1
	1-sand core sample	1
La Paz City	1-20L Ground water sample/wells	10
	1 settled dust sample from North, SouthEast and West	4
	100g collective teeth	1
	1-marine sediment core sample	1
	1-sand core sample	1
La Paz Lagoon	Surface Sea Water 1-20L	2
	1-100g Sea cucuber sample	4
	1-marine sediment core sample	1
	1-sand core sample	1
La Paz Bay	Surface Sea Water 1-20L	2
	1-100g Sea cucuber sample	4
	1-marine sediment core sample	1
	1-sand core sample	1
Punta Coyote	Surface Sea Water 1-20L	2
	1-100g Sea cucuber sample	4
	1-marine sediment core sample	1
	1-sand core sample	1
Total Samples For Terrestrial And Marine Media/Season		**59**
TOTAL NUMBER OF SAMPLES FOR 1 YEAR SAMPLING PERIOD		**236**

Abiotic Media

Sampling Settled Dust

Settled dust samples will be taken because uranium and the decay products that are electrically charged when they are formed readily attach to dust particles in the atmosphere. In general, the dustier the atmosphere, the greater the concentration of radionuclides. The dust particles with attached radionuclides settle to earth, a process that is expedited by rain that serves to scrub or wash the particles out of the air. Furthermore, marked differences in dose rates occur at the surface and in the lower levels of the atmosphere over different rock types. To derive the uranium action level in dust we will use a particle size measuring method (inertial separation using a specific cascade impactor) and determination of chemical solubilities (Reif et al., 1992).

Air samples are an alternative medium because the concentration of radionuclides in air are highly variable, and determined by the following conditions: concentration of precursors in soil, altitude, soil porosity and grain size, temperature, pressure, soil moisture, rainfall which affects the build up and retention in air, and season. In the atmosphere the nuclides undergo nuclear transformation and most of the radioactivity in the atmosphere in a coastal system diffuses from the earth into the atmosphere. Consequently, the uranium isotopic levels in the regional atmosphere may be high. Uranium intake by breathing in humans will be estimated by measuring uranium concentrations in air following the methodology of Yamamoto et al., 1974. Samples for airborne uranium concentrations could be taken from La Paz and San Juan de la Costa communities for alpha counting and analysis by gamma spectrometry. The annual average airborne concentrations will be evaluated by means of a dispersion model (Lopez and Martinez, 1982; Guedalia et al., 1980). The dispersion model offers a choice of compartments to consider a number of directions of the wind, and the surface of the region (Alvarez and Garzon 1989).

Beach Sand

Sand from the San Juan de la Costa beaches will also be sampled for uranium isotopic abundance. Sand radiation poses a hazard to fishermen who reside close to the coast and to sunbathers who frequent those beaches. Local sands and marine sediment core samples will be surveyed and are expected to reflect the uranium distribution, transportation, and isotopic composition in the marine system. We chose chemical separation as the most effective approach to study the elemental partitioning in this media because the process by which most radionuclides are incorporated into various components are themselves largely chemical in nature. Sampling methods, sample preparation, and radiochemical procedures for uranium analysis in settled dust, marine fractionated sediments and beach sands are widely explained by Narayana et al., 1995; Martinez-Aguirre, 1994 ; and Beckett, 1981. Briefly, samples would be subjected to weak acid leaching followed by chemical purification of the leachate prior to alpha-spectrometry.

Freshwater

A very effective sink for materials from weathered and eroded rocks during fluvial dispersal are fresh water and sea water. Thus, sampling of natural ground waters is directly useful in relation to water supplies because the uranium content varies markedly depending on the bedrock type and on the proximity to uranium or phosphorite deposits (Barisic et al., 1992; Bowie and Plant, 1983; Howarth and Thorton, 1983). Fresh water samples will be collected from different wells in both La Paz and San Juan de la Costa. For sample recovery, preparation, analytical and counting methods we will use the technique stated in the preliminary study section of this manuscript.

Seawater

Seawater samples will be taken from Punta Coyote, La Paz Bay, San Juan de la Costa, La Paz Lagoon, Espiritu Santo, and Canal San Lorenzo (Fig. 2). The uranium concentrations will be determined by using radiochemical separation procedures to isolate uranium from the matrix elements followed by alpha spectrometric measurement of the uranium fraction (Dang and Pullat, 1993; Pimpl and Yordanova, 1992; Miller, 1991). At present, many different analytical methods are available to identify isotopes such as uranium and its daughter products, and it can be detected in low concentrations in nearly all materials from the environment.

Biotic Media

Although many chemicals and toxins are biodegraded, uranium accumulates in human and animal tissues over a lifetime. Serial uranium bioaccumulation has a larger radiotoxic impact for organisms at the top of the food chain - including humans - than for animals lower in the trophic chain. In numerous instances, livestock, fish, shellfish, and other wildlife have been found to have accumulated environmental radionuclides to the point where their tissues may be hazardous to predators of these species and to humans who consume them. Additionally, transport of uranium within the biological chain is of interest because of the differential uptake by species. Therefore, uranium tissue-content analyses yield invaluable data to identify exposure to natural uranium.

Marine Organisms

Sea cucumbers which burrow in and ingest bottom mud are good indicators for benthic sediment contamination because in the absence of bone for the deposit of alkaline earths, the persistence of radioisotopes depends on the makeup of other tissues, especially on the binding of metallic cations by tissue proteins (Grosch and Hopwood, 1979).

Alternative biotic media, in the marine environment, include algae and other marine microorganisms that display remarkable affinity for heavy metals. Algae analysis can be particularly useful because it reflects accumulations that occur during a limited period of growth (Hanmin et al., 1991); they are potential indicators of localized short-term effects. Both the benthic and intertidal macroalgae and plankton may be sampled and analyzed to assess the uranium burden at this first step in the marine web. Analytical results would not only elucidate the uranium content at this level in the food web but would increase the knowledge on the use of algae and other forms of potential bioremediation agents. Macaskie et al., (1992) reported that bacterium *Citrobacter sp* tolerated spectacular loads of up to 9 g of accumulated uranium per gram of biomass (900% of the cellular dry weight) over several weeks without apparent ill effect. Other authors (Liu and Wu, 1993; Sakaguchi et al., 1978) have also reported algal uptake and recovery.

Crustaceans are also an alternative medium. Crustacea such as brine shrimp, true shrimp, and crabs, or filter feeders such as mussels, clams, scallops and oysters accumulate many chemicals and alkaline earth metals in their shells to concentrations much higher than those in the ambient water. Planktonic euphasids and salps are another alternative medium because they filter large volumes of water while feeding and because of their size, they reach equilibrium quickly and can serve as biological monitors of environmental radiation as well.

Among larger animals an alternative media are fish and amphibians. Fish are high on the food chain and are often exposed to chemical levels that are much more concentrated in the food they consume than in the sea water habitat. The study of cancer in fish and amphibians not only yields new insight into the origins of human cancers, but also provides numerous other benefits, because these animals may serve as sentinels of environmental contaminants and as models for studying neoplasia and basic mechanisms in oncology (NRC, 1990). However, the uranium concentration in muscle may be the aspect of most concern to humans, since this is the tissue used for food.

Seafood consumption in a coastal setting may be an additional contributor of uranium in the trophic chain. In some isolated farms the inhabitants subsist primarily on the food they grow and harvest from the sea waters. Human foodstuffs may be a major contributor to the annual uranium intake from natural sources. Indeed, some toxicologists may not have been aware that radionuclides can occur in some foods naturally (Rubery, 1989).

Other alternatives to use as marine indicators of uranium accumulation and bioavailability include gulls and seals but the latter are not linked to humans through the regional food web.

Human Tissues

People are at the top of the marine and terrestrial food webs, therefore, human tissues could be used to monitor the uranium intake from natural sources, e.g., diet, water, and air. The determination of internal exposure to uranium via inhalation or ingestion is extremely important because α-particles dissipate their energy in such an exceedingly small volume of tissue in which great local damage can occur, in addition to the increased internal hazard due to metabolic incorporation into mineralized tissues. Three different approaches may be taken: 1. quantification in mineralized tissues (bone and teeth); 2. human urinalyses; and 3. blood tests.

We have chosen human teeth because uranium is incorporated into mineralized tissues like bones and teeth. Hamilton, (1971) reported 0.7×10^{-8}gU/g as the concentration in unashed teeth with a close association with surfaces near the blood supply. There appears to be no significant difference within a factor ~2, in the concentration of uranium in the various bones of the human skeleton or for teeth when allowance is made for converting wet weight to ash weight. Thus the content of both tissues may serve as an index of population exposure. But, taking into account the difficulty of obtaining bone tissue, the collection of teeth eases the sampling problem because temporary and molar teeth extracted during routine dental procedures may be used. Masuda (1971a, b, c, d) provided evidence to support the empirical relationship between environmental concentrations and intake into the human body (Wrenn et al., 1985). Moreover, uranium concentrations on bone surfaces are higher shortly after intake, but are gradually and uniformly distributed throughout the volume overtime.

Human urinalysis is an alternative screening method for environmental and occupational chronic inhalation and ingestion. There are good experimental reasons to believe that urinary excretion of uranium is the only important route for loss of systemic uranium and that, therefore, the daily urinary output must be proportional to the uranium absorbed from the daily diet (Hursh and Spoor, 1973). Several authors have reported measurable quantities of uranium in urine excreted by humans without any history of industrial exposure and these values range from 0.012 to 26 mg of uranium per liter of urine (Welford et al., 1976; Welford and Baird 1967; Welford et al., 1960). The results of the urinalysis would be used to calculate the annual effective dose equivalent (AEDE) using REMedy software. Another potential, sensitive and rapid method to analyze uranium in urine samples has been used by Miller, (1991).

Invaluable information on the radiobiological effects of internal exposure is obtained by leukocyte (Moorhead et al., 1960), lymphocyte (Barcinski et al., 1975), or hepatocyte (Williams, 1977) short term tests. If the aim is to obtain information for species comparisons, the chosen method must be applicable to the same cell type for each of the species studied. Obviously the chosen organisms should accumulate uranium in tissues, their territory range must overlap the area to be studied, their population size and density must be sufficient to allow sampling, and have enough life span to allow the opportunity to couple chronic exposure with a variety of measurable responses.

Terrestrial Organisms

Terrestrial plant sampling is also an alternative source to detect biologic uptake of uranium. Differential uptake of radionuclides by terrestrial plant species was reviewed by Simon and Ibrehim (1988, 1987). In general, leafy vegetables contain higher concentrations than fruit and grain crops. Higher levels of radionuclides in plants generally are found in soils that have higher than background levels, namely near ore deposits or on contaminated soils and mine tailings (Morvedt, 1994).

Other alternative terrestrial media include cattle, swine and poultry among other farm animals, that apparently ingest soil both intentionally and incidentally. Therefore, tissue radioanalysis (Linsata et al., 1991, 1989, 1986) of these animals may also be used to study uranium transport from soil to terrestrial species.

The natural uranium in all samples will be separated, purified, and electrodeposited by standard ion exchange procedures. Concentrations will be resolved by the isotope-dilution method with a solid-state detector by alpha-counting spectrometry using ^{232}U as a tracer of known activity. The activity ratios will be determined as per the preliminary results section or by slight modifications of the radioanalytical method.

RATIONALE FOR ANALYSIS OF URANIUM PATHWAYS AND CANCER LIKELIHOOD THROUGH SOFTWARE MODELS FOR LA PAZ BAY

The problem of surveying the natural transport throughout the La Paz Bay ecological system presents a major challenge. The sources, toxicity, and exposure concentrations have not been identified. Moreover, many complex environmental pathways have not been addressed.

Still, with the data from the first stage of the proposed study and a model developed to predict the extent of physical and biotic transport processes that disperse uranium, it would be possible to identify unusual levels in the biologic chain.

Software applications that model hazardous materials will be used to determine the radiation risk. The models have the capability to simulate the pertinent chemical and physical processes associated with the release of the material and its mixing with the atmosphere. There are several choices available. We have selected the Environmental IMPACT model tailored specifically for natural uranium, a Model for Hazardous Materials presented by Rodean, (1991); and the RADFOOD model (Koch and Tadmor, 1986).

The model for hazardous materials by Rodean is based on the thermodynamic equilibrium that incorporates the ideal gas law, temperature-dependent atmospheric vapor pressure equations, and temperature-dependent dissociation reactions. The model's greatest advantage is its versatility to interconnect hydrologic, atmospheric, marine,and terrestrial uranium sources, and determine the fate in biota and humans embedded in the environment.

The dynamic model, RADFOOD, was developed and based on different existing models. It simulates transport of fallout radionuclides through agricultural food chains to humans and evaluates the radiation doses resulting from consumption of contaminated food. Transport is modeled through compartments representing various environmental elements or food products. Internal radiation doses (whole-body weighted doses) following ingestion of contaminated foodstuffs can be estimated. Specific types of crops, soils and diets of humans and livestock can also be considered. Calculations can be made for individual or collective radiation doses, as well as associated health effects.

An analysis of the data through the use of predictive software will demonstrate periodic as well as widespread transfer of uranium throughout La Paz Bay system. Such an approach will also yield estimates of local enrichment if the research plan measures concentrations at the appropriate trophic levels.

BREAKDOWN OF OTHER POTENTIAL METHODS TO APPLY FINDINGS

The present research plan does not intend to examine toxicologically or epidemiologically the data acquired. Nevertheless, this section provides an analysis of potential methods to approach future studies.

Unmistakably, far less effort has gone into the oncogenic study of natural localized uranium exposure levels as etiologic agents. Clearly such studies must be considered for further resolution.

Potential epidemiological approaches are Toxicological studies and Epidemiological studies on cancer incidence involving a retrospective study of cancer in the municipality, a multi-city prevalence study, and a prospective cohort study.

BUDGET FOR ENTIRE PROPOSED PROJECT PERIOD

BUDGET DETAIL

January 1, 1997 - December 31, 1999

	Year 1 Jan. 97 Dec. 97	**Year 2** Jan. 98 Dec. 98	**Year 3** Jan. 99 Dec. 99	**Total** Jan. 97 Dec. 98
PERSONNEL				
Principal Investigator 100% effort for 3 years	54,646	56,285	57,974	168,905
2 Senior Project Associates 2 mos/yr @ 100% effort	18,000	18,900	19,845	56,745
Biophysics Research Tech., 1 Full Time Technician for 3 yrs	50,400	52,920	55,566	158,886
1 50% Time Technician counting @ 100% effort	25,200	26,460	27,783	79,443
Graduate Student Research Assistant 2 @ 50% for 9 months@$10/hr. and	15,480	15,480	15,480	46,440
100% for 3 months.	10,320	10,320	10,320	30,960
Total Salaries & Wages	174,046	180,365	186,968	541,379
FRINGE BENEFITS				
Partial Hourly @ 3% Yr 1, 4% Yr 2, 5% Yr 3	774	1,032	1,290	3,096
Full Benefits @ 27% Yr 1, 28% Yr 2, 29% Yr 3	40,026	43,278	46,739	130,043
Subtotal Fringe Benefits	40,800	44,310	48,029	133,139
Total Personnel	214,846	224,675	234,997	674,518
TRAVEL				
Foreign				
4 Air round trips investigator @	2,000	2,000	2,000	6,000
4 ground trips @ 1,000 ea.	4,000	4,000	4,000	12,000
Per Diem	13,000	13,000	13,000	39,000
Domestic				
1 National meeting	1,000	1,000	1,000	3,000
Total Travel	20,000	20,000	20,000	60,000

EQUIPMENT				
Gas bottles	1,000	0	0	1,000
4 Rn emanation ? @ 10K	40,000	0	0	40,000
Alpha spectrometer w/detector	30,000	0	0	30,000
Centrifuge	2,000	0	0	2,000
2 Computers for data analysis	6,000	0	0	6,000
Total Equipment	79,000	0	0	79,000
Materials, Supplies, Other				
Supplies				
Sampling containers	4,000	4,000	4,000	12,000
Lab ware/yr:	10,000	10,000	10,000	30,000
Separatory funnels, stainless steel planchets, columns, electrodeposition cells, glass fiber filters, filters, counting sample holders, Ion exchange resins, Misc. Lab supplies				
Radiochemicals	10,000	10,000	10,000	30,000
acids, standards, and spike of analytical grade				
Other				
Boat rent/driver, 12 samplings @ $150	1,800	1,800	1,800	5,400
Photocopy	300	300	300	900
Publication costs	0	0	500	500
Total Materials, Supplies, Other	26,100	26,100	26,600	78,800
TOTAL DIRECT COSTS	**339,946**	**270,775**	**281,597**	**892,318**
INDIRECT COSTS @ 49% MTDC	**127,864**	**132,680**	**137,983**	**398,527**
TOTAL PROJECT COSTS	**467,810**	**403,455**	**419,580**	**1,290,845**

TOTAL PROJECT COSTS 1,290,845

BUDGET JUSTIFICATION

The study comprises a major field and analytical effort. The majority of the budget is in salaries reflecting the amount of work to be done. The number of samples generated will completely saturate the 8 channel alpha spectrometer for three years plus Rn system for 2 years. This counting time is not available on shared use equipment at MLML or SJSU. The proposed study will contribute to the master's degrees of two students.

Sufficient funds are requested in the supplies category to conduct the uranium isotopic analysis required by this project for the environmental samples from La Paz Bay. In addition, we have budgeted sufficient funds for ground transport of the samples. Funds are also included for equipment.

Additionally, not only is this an analytically intensive proposal but its successful completion relies on bilingual researchers who can interview local doctors/dentists and health administrators in addition to carrying out the necessary analytical tasks.

TIMETABLE

The three year timetable (from January 1997 to December 1999) proposed study of environmental uranium in Baja California Sur includes a four set sampling to account for seasonal variation, which is intended to take place during the first year.
For the second year it is intended to continue with the analytical process and initiate the modeling of the results for analysis of the critical pathways. For the third year, the intention is to submit the anlaysis of acquired data and present final conclusions.

FACILITIES AVAILABLE

A broad suite of facilities are available to perform this work particularly the Moss Landing Marine Laboratories. In the past, MLML researchers have focused their work in developing techniques and investigating the marine environment with special interest in ichthyology and radiochemistry. Scientists from MLML have successfully published their results. All of the sampling and analytical instrumentation are at our disposal. All other facilities would be made available for this study. The MLML analytical facilities for metals include a Perkin Elmer Zeeman 5000 graphite furnace atomic absorption spectrometer, and four seagoing systems for metal analysis by flow injection analysis with either chemiluminescence or fluorescence detection.

We anticipate that the bulk of the group sampling needs would be met using the Moss Landing instrumentation. But, the existing 8 channel alpha spectrometer is dedicated to the analysis of ichthyological samples; consequently, the purchase of additional instrumentation is required. In addition to this equipment, we have access to several filter fluorometers for chorophyll analysis, spectrometers, a Dohrman CD190 DOC analyzer, microscopes (epifluorescence & SEM), a Control Equipment Corp. 440 Elemental Analyzer and sufficient lab space.

MLML maintains a modest library for access to the major oceanographic journals (75 current subscriptions and has rapid interlibrary loans available.

GLOSSARY

Absorbed dose Often referred to simply as dose. However, when the quality of radiation changes, absorbed dose alone no longer specifies biological effect. In other words, a given absorbed dose of x rays, does not necessarily result in the same biological effect as the identical dose of neutrons or alpha particles (such difference is characterized by the concept of RBE).

Acute Toxicity Refers to the ability of a chemical to do systematic damage as a result of a one time exposure to relatively large amounts of the chemical.

Authigenic Formed were found: said of the ingredients of crystalline rocks, or of crystalline ingredients of rocks.

Linear Energy Transfer (LET) Average amount of energy lost per unit track length. In other words, radiation quality, is a reasonable qualitative index for ranking radiations on an ordinal scale for biological effect. Low-LET, Standard radiation designated as x rays, gamma rays, electrons, or positrons of any specific ionization. High-LET, Radiation characteristic of heavy charged particles such as protons and alpha particles where the distance between ionizing events is small on the scale of a cellular nucleus.

Recoil To have a retroactive effect in consequence of elasticity or because of contact with an opposing force; a rebound; return.

Relative Biological Effectiveness (RBE) Biological potency of one radiation compared with another to produce the same biological endpoint. When the dose-response relationships for the two types of radiation differ in shape, RBE is necessarily dependent on the level of the effect that is considered and should be specified as such. It is numerically equal to the inverse of the ratio of absorbed doses of the two radiations required to produce equal biological effect.

Toxicity Refers to the ability of a chemical to damage an organ system, such as the blood forming mechanism, or disrupt an enzyme system at some site in the body.

Hazard The hazard presented by a chemical has two components 1) the ability of a chemical to do harm by virtue of its properties; 2) the ease with which contact can be established between the chemical and the system of concern.

Chronic toxicity Refers to the ability of a chemical to do systematic damage as a result of repeated exposures, during a prolonged period of time, to relatively low levels of the chemical.

Route of exposure Pathway by which a chemical enters the body: dermal, inhalation, oral.

Radiation Form of energy; either particles emitted by radioactive substances, such as electrons (beta or positrons), waves (x-, γ-rays), or α–particles (charged nuclei or helium).

Secular Existing, continuing or relating to a long term of indefinite duration.

Beneficiation To treat (a raw material) so as to improve properties.

REFERENCES

Agarwal, R. P. and Feldman, I. 1968. Chelation Of Uranyl Ions By Adenine Nucleotides Ii. Magnetic Resonance Investigation Of Uranyl Nitrate Adenosines Phosphate In D2o At Alkaline pH. J. Amer. Chem. Soc. 90:6635-6639.

Alpen, E. L. 1990. Radiation Biopysics. Prentice Hall Biophysics and Bioengineering Series ed. Abraham Noordergraaf.

Altschuler, Z. S., R. S. Clarke, and E. J. Young. 1958. Geochemistry Of Uranium In Apatite And Phosphorites. Geol. Surv. Prof. Pap. (U.S.) 314-D.

Alvarez, M. C., and L. Garzon. 1989. Assessment Of Radiological Emissions From Spanish Coal Power Plants: Radioactive Releases And Associated Risks. Health Physics. 57(5):765-769.

Ames, L. L. 1960. Some Cation Substitutions During The Formation Of Phosphorite From Calcite. Econ. Geol., 55:354-362.

Anderson, R.F. 1982. Concentration, Vertical Flux And Remineralization Of Particulate Uranium In Seawater. Geochim. Cosmochim. Acta, 46:1293-1299.

Archer, V. E., J. K. Wagoner, and F. E. Lundin. 1973a. Lung Cancer Among Uranium Miners in the United States. Health Phys. 25:352-371.

Archer, V. E., J. K. Wagoner, and F. E. Lundin. 1973b. Cancer Mortality Among Uranium Mill Workers. J. Occup. Med. (United States) 15:11-14.

Augustin, J., and R. Zejda. 1991. Cancer Incidence And Geochemical Factors In The Environment. The Science Of The Total Environment. 106:155-163.

Ball, T. K., and J. C. H Miles. 1993. Geological Factors Affecting The Radon Concentration In Homes In Cornwall And Devon, UK. Environmental Geochemistry and Health 15(1):27-36.

Barisic, D. S. Lulic, and P. Miletic. 1992. Radium And Uranium In Phosphate Fertilizers And Their Impact On The Radioactivity Of Waters. Water Research. 26(5):607-611.

Barcinski, M. A., M do C. A. Abreu, J. C. de Almeida, J. M. Naya, L.G. Fonseca, and L. E. Castro. 1975. Cytogenetic Investigation In A Brazialian Population Living In An Area Of High Natural Radioactivity.

Barendson, G. W. 1968. Response Of Cultured Cells, Tumors, And Normal Tissues To Radiation Of Different Linear Energy Transfer. Curr. Top. Radiat. Res. Q. 4:293-356.

Barnes, C., and J. K. Cochran. 1987. Uranium Geochemistry In Long Island Sound Sediments. EOS, Trans. Amer. Geophys. Union. 68:693.

Beckett, P. 1981. Agric. Adm. 8:177-208.

Bencosme, S. S., R. S. Stone, H. Latta, S. C. Madden. 1960. Acute Tubular And Glomerular Lesions In Rat Kidneys Aftewr Uranium Injury. Arch. Path. 69:470-476.

Berish, C. W. 1990. Potential Environmental Hazards Of Phosphogypsum Storage In Central Florida. In: Chang W. F. (ed), Proceedings of the 3rd Int. Symp. on Phosphogypsum, Florida Inst. Phosphate Res., Miami, Fl., pp 1-29.

Bernat, M., and T. M. Church. 1989. Uranium And Thorium Decay Series In The Modern Marine Environment. In: Handbook of Environmental Isotope Geochemistry, Vol. 3, The Marine Environment (ed. P Fritz and J. Ch. Fontez), pp 357-383. Elsevier Science, Amsterdam.

Blanchard L. R. 1965. ^{234}U/^{238}U Ratios In Coastal Marine Waters And Calcium Carbonates. Journal of Geophysical Research. 70(16):4055-4061

Borek, C., R. Miller, C. Pain, and W. Trom. 1979. Conditions For Inhibiting And Enhancing Effects Of The Protease Inhibitor Antipain On X-Ray Induced Neoplastic Transformation In Hamster And Mouse Cells. proc. Natl. Acad. Sci. USA. 76:1800-1803.

Borole, D. V., S. Krishnaswami, and B. L. K. Somayajulu. 1982. Uranium Isotopes In Rivers, Estuaries And Adjacent Coastal Sediments Of Western India: Their Weathering, Transport And Oceanic Budget. Geochim. Cosmochim. Acta. 46:125-137.

Borole, D. V., S. Krishnaswami, and B.L.K. Somayajulu. 1977. Investigations On Dissolved Uranium, Silicon And Particulate Trace Elements In Estuaries. Est. Coastal Mar. Sci. 5:743-754.

Bosshard, E., B. Zimmerli, and Ch. Schalatter. 1992. Uranium In The Diet: Risk Assessment Of Its Nepho- And Radiotoxicity. Chemosphere. 24(3):309-321.

Bottrell, S. H. 1993. Redistribution Of Uranium By Physical Processes During Weathering And Implications For Radon Production. Environmental Geochemistry and Health. 15(1):21-25.

Bowie, S. H. U., and J. A. Plant. 1983. Natural Radioactivity In The Environment. In: Applied Environmental Geochemistry. ed. Iain Thornton.

Brenner, D. J. 1988. Concerning The Nature Of The Initial Damage Required For The Production Of Radiation Infuced Exchange Aberrations. Int. J. Radiat. Biol. 52: 805-809.

Brewer, P. G., Y. Nozaki, D. W. Spencer, and , A. P. Fleer. 1980. Sediment Trap Experiments In The Deep North Atlantic: Isotopic And Elemental Fluxes. J. Mar. Res. 38:703-728.

Broerse, J. J., and G.W. Barendsen. 1973. Relative Biological Effectiveness Of Fast Neutrons For Effects On Normal Tissue. Curr. Top. Radiat. Res. Q. 8:305-350.

Burnett, W. C. and H. H. Veeh. 1992. Uranium-Series Studies Of Marine Phosphates And Carbonates. In: M. Ivanovich and R.S. Harmon (ed.) Uranium-Series Disiquilibrium: Aplications to earth, marine, and environmental sciences. 2nd ed. Clarendon Press, Oxford.

Burnett, W. C. and D. N. Gomberg. 1977. Uranium Oxidation And Probable Subaerial Weathering Of Phosphatized Limestone From The Pourtales Terrace. Sedimentology 24:291-302

Burnett, W. C. and H. H. Veeh. 1977. Uranium-Series disiquilibrium studies in Phosphorite Nodules From The West Coast From South America. Geochim. Cosmochim. Acta. 41:755-764.

Campillo C. H. 1991. Diccionario Ilustrado Y Enciclopedia Regional Del Estado De Baja California Sur; Fernandez eds, Mexico.

Chan, J. Y. H., F. F. Becker, J. German, and J. H. Ray. 1987. Altered DNA Ligase I Activity In Bloom's Syndrome Cells. Nature. 325:357-359.

Chen, J. H., R. L. Edwards, and G. J. Wasserburg. 1986. ^{238}U, ^{234}U And ^{232}Th In Sea Water. Earth Planet. Sci. Lett., 80:241-251.

Coale K. H. and K W. Bruland. 1987. Oceanic Stratified Euphotic Zone as Elucidated by ^{234}Th:^{238}U Disequilibria Limmol. Oceanogr. 32(1): 189-200.

Cochran, J. K. 1992. The Oceanic Chemistry Of The Uranium- And Thorium-Series Nuclides. In: Uranium-Series Disequilibrium: Applications to Earth, Marine, and Environmental Sciences. 2nd ed. eds. M. Ivanovich and R.S. Harmon. Oxford University Press. New York, USA.

Cochran, K. K., A. Carey, E. R. Sholkovitz, and L. D. Surprenant. 1986. The Geochemistry Of Uranium And Thorium In Coastal Marine Sediments And Sediment Pore Waters. Geochim Cosmochim. Acta. 50:663-80.

Coggle, J. E. 1983. Some Properties Of Ionising Radiation. In:Biological effects of Radiation. 2nd ed. The Wykeham Science Series. London and Basingstoke.

Cooper, J. R., G. N. Stradling, H. Smith. 1982. The Behavior Of Uranium-233 Oxide And Uranyl-233 Nitrate In Rats. Int. J. Radiat. Biol. Relat. Stud. Phys. Chem. Med. 41:421-433.

Cowart, J. B. and W. C. Burnett. 1994. The Distribution Of Uranium And Thorium Decay-Series Radionuclides In The Environment - A Review. J. Environ. Qual. 23:651-662.

Cox, R. 1994 a. Molecular Mechanisms Of Radiation Oncogenesis. Int. J. Radiat. Biol. 65:57-64.

Cox, R. 1994b. Human Cancer Predisposition And The Implications For Radiological Protection. Int. J. Radiat. Biol. 66:643-647.

Crounse, R. G., W. J. Pories, J. T. Bray, and R. L. Mauger. 1983. Geochemistry And Man: Health And Disease 2. Elements Possibly Essential, Those Toxic And Others. In: Applied Environmental Geochemistry. Academic Press Geology Series, London.

Dang, H. S., and V. R. Pullat. 1993. Normal Concentration And Excretion Ratio Of Uranium In Serum Of Normal Individuals In India. Heath Phys. 65:303-305.

De Rey, B. M., H. E. Lanfranchi, R. L. Cabrini. 1984. Deposition Pattern And Toxicity Of Subcutaneously Implantedd Uranium Dioxide In Rats. Health Phys. 46:688-692.

De Rey, B. M., H. E. Lanfranchi, R. L. Cabrini. 1983. Percutaneous Absorption Of Uranium Compounds. Environ. Res. 30:480-491.

Doll, R., and R. Peto. 1981. The Causes Of Cancer: Quantitative Estimates Of Avoidable Risks Of Cancer In The United States Today. Jnci. 66(6):1193-1305.

Dounce, A. L. and J. F. Flagg. 1949. The Chemistry Of Uranium Compounds. In: The Pharmacology And Toxicology Of Uranium Compounds. National Nuclear Energy Series. Division IV. Vol 1 Parts 1 and 2. New York: Mc Graw-Hill Book Co. Inc.Voegtlin and Hodge.

Durbin, P. W. 1984. Metabolic Models For Uranium. In: Biokinetics and Analysis of Uranium in Man. Richland, WA: Hanford Environmental Health Foundation; USUR-05/HEHF-47 F-1-65.

Durbin, P. W. and M. E. Wrenn. 1975. Metabolism and Effects of Uranium in Animals. In: Proceedings of the Conference on Occupational Health Experience with Uranium, 28-30 April 1975, Arlington, VA. Washington, DC: Energy Research and Development Administration, Washington; ERDA-93; 175: pp 68-139. Available from: National Technical Information Service, Springfield, VA.

EPA. Environmental Protection Agency. 1993. Wildlife Exposure Factors Handbook. U.S. Environmental Protection Agency, Office of Radiation Standards, Washington, DC. EPA 520/1-85-010. NTIS PB86-244936/GAR.

EPA. Environmental Protection Agency. 1985. Radionuclides: Background Information Document-Standard For Radon-222 Emissions From Underground Uranium Mines. Report. U.S. Environmental Protection Agency, Washington, DC. December. EPA 600/187.

Filippova, L. G., A. P. Nifativ, E. R. Lyubchanskii. 1978. Some Of The Long-Term Sequelae Of Giving Rats Enriched Uranium. Radiobiology. 18:400-405.

Finkel, M. P. 1953. Relative Biological Effectiveness of Radium and Other Alpha Emitters In CF no 1 Female Mice. Proc. Soc. Exp. Biol. Med. 83:494-498.

Fisenne, I. M., and G. A. Welford. 1986. Natural U Concentrations In Soft Tissues And Bone Of New York City Residents. Health Phys. 50: 739-746.

Fisenne, I. M., P. M. Perry, G. A. Welford. 1980. Determination Of Uranium Isotopes In Human Bone Ash. Anal. Chem. 52:777-779.

Fisher, D. R., R. L. Kathren, and J. S. Swint. 1991. Modified Biokinetic Model for Uranium From Analysis of Acute Exposure to UF_2. Health Phys. 60(3):335-342.

Gascoyne, M. 1992. Geochemistry Of The Actinides And Their Daughter. p. 34 -61. In: M. Ivanovich and R.S. Harmon (ed.) Uranium-Series disiquilibrium: Aplications to earth, marine, and environmental sciences. 2nd ed. Clarendon Press, Oxford.

Gottlieb,L. S., and L. A. Husen. 1982. Lung Cancer Among Navajo Uranium Miners. Chest. 81:449-452.

Grasshoff, Klaus. 1976. Methods Of Seawater Analysis. Chapter 1 Verlag Chemie., N.Y.

Grosch, D. S., and L. E. Hopwood. 1979. Biological Effects Of Radiations. Academic Press, Inc. 111 Fifth Avenue, New York, New York 10003

Guernsey, D. L., and C. Borek. 1980. Thyroid Hormone Modulation Of X Ray Induced In Vitro Neoplastic Transformation. Nature. 288:591-592.

Guedalia, D., A. Ntsila, A. Druilhert, and J. Fontan. 1980. Monitoring Of The Atmospheric Stability Above An Urban And Suburban Site Using Solar And Radon. J. Appl. Met. 19:833-848.

Guimond, R. J. 1977. The Radiological Aspects Of Fertilizer Utilization. Proc. Symp., Public Health Aspects Of Radioactivity In Consumer Products. Atlanta, GA. 2-4 Feb. 1977. FDA, NRC, EPA, Georgia Inst. of Technol., Atlanta, GA.

Guimond, R. J. 1976. Radiation and the Phosphate Industry-An Overview, In Natural Radiation in Man's Environment, Proc. Health Phys. Soc. 10th Midyear Topical Symposium Troy, Saratoga Springs, NY., pp. 13-28.

Hall, E. J. 1972. Radiation Dose-Rate: A Factor Of Importance In Radiobiology And Radiotheraphy. Brit. J. Radiol. 45:81-97.

Hall, E. J., and T. K. Hei. 1987. Oncogenic Transformation By Radiation And Chemicals. Pp. 507-512 in Proceedings of the 8th International Congress of Radiation Research, E. M. Fielden, J. F. Fowler, J. H. Hendry, and D. Scott. eds. Taylor and Francis.

Hall, E. J., and T. K. Hei. 1985. Oncogenic Transformation In Vitro By Radiations Of Varying LET. Radiat. Prot. Dosimetry 13:149-151.

Hamilton, E. I. 1972. The Concentration Of Uranium In Man And His Diet. Health Phys. 22(2):149-153.

Hamilton, E. I. 1971. The Concentration And Distribution Of Uranium In Human Skeletal Tissues. Calc. Tiss. Res. 7:150-162.

Hamilton, E. I. 1970. The Concentration Of Uranium In Air From Contrasted Natural Environments. Health Phys. 19:511-520.

Hanawalt, P. C., and A. Sarasin. 1986. Cancer-Prone Hereditary Diseases With DNA Processing Abnormalities. Trends Genet. 2:124-129.

Hanmin, Z. L. Shuqing, W. Fushou, S. Fuju, L. Qinglin and Y. Wenchun. 1991. Radioactivity in The Coastal Waters of The Bohai and Yellow Seas of China. J. Envirn. Radioactivity. 14: 193-209.

Hausback, B. 1984. Cenozoic Volcanic And Tectonic Evolution Of Baja California Sur, Mexico. In: Geology Of The Baja California Peninsula. Friezzell V.(editor) Pacific Section S.E.P.M. 39: 219-236.

Hei, T. K., D. J. Chen, D. J. Brenner, and E. J. Hall. 1988. Mutation Induction By Charged Particles Of Defined Linear Energy Transfer. Carcinogenesis 9:1233-1236.

Hieber, L., G. Ponsel, H. Roos, S. Senn, E. Fromke, and A. N. Kellerer. 1987. Absence Of Dose Rate Effect In The Transformation Of C3H10 1/2 Cells By Alpha Particles. Int. J. Rad. Biol. 52:859-869.

Hodge, H. C. 1973. A History Of Uranium Poisoning (1824-1942). In: U, Plutonium And The Transplutonic Elements; Handbook of Experimental Pharmacology Vol. 36 Springer-Verlag.

Hostetler, P. B., and R. M. Garrels. 1962. Transportation And Precipitation Of Uranium And Vanadium At Low Temperature, With Special Reference To Sandstone-Type Uranium Deposits. Econ. Geol. 57:137-167.

Howarth R. J. and Thornton. 1983. buscar titulo In: Regional Geochemical Mapping And Its Aplication To Environmental Studies In Applied Environmental Geochemistry. Academic Press, London.

Hueper, W. C., J. H. Zuefle, A. M. Link, and M. G. Jhonson. 1952. Experimental Studies In Metal Carcinogenesis. Ii. Experimental Uranium Cancer In Rats. J. National Cancer Institute 13: 291-301.

Hursh, J.B , and N.L. Spoor. 1973. Uranium Data On Man. In: U, Plutonium And The Transplutonic Elements; Handbook of Experimental Pharmacology Vol. 36 Springer-Verlag.

ICRP. International Commission on Radiological Protection. 1988. Individual Monitoring For Intakes Of Radionuclides By Workers: Design And Interpretation. Oxford: Pergamon Press; ICRP Publication 54; Ann. ICRP 19:1-3.

ICRP. International Commission on Radiological Protection. 1979. Limits For Intakes Of Radionuclides By Workers. Oxford: Pergamon Press; ICRP Pubication 30, Part 1; Ann. ICRP 2:3-4.

Kalyani, V. D. M. L., M. V. S. Chandrasekhar Rao, G. Sree Krishna Murty, G. Satyanarayana, D. L. Sastry, S. G. Sahasrabhude, D. R. R. Babu and M. R. Iyer. 1990. Analysis Of ^{232}th And ^{238}U In The Beach Sands And The Ocean Sediments. Indian J. Environmental Protection. 10(12): 931-935.

Kathren R. L., J. F. McInroy, R. H. Moore, S. E. Dietert. 1989. Uranium In The Tissues Of An Occupationally Exposed Individual. Health Phys. 57:17-21.

Kathren, R. L. and R. H. Moore. 1986. Acute Accidental Inhalation Of U: A 38-Year Follow Up. Health Phys. 51:609-619.

Kathren, R L. 1984. Radioactivity In The Environment. Hardwood Academic Publishers.

Kennedy, A. R., G. Murphy, and J. B. Little. 1980. Effect Of Time And Duration Of Exposure To 12-O-Tetradecanoyl-Phorbol-13-Acetate Aon X-Ray Transformation Of C3H10t1/2 Cells. Cancer Res. 40:1915-1920.

Kennedy, A. R. and J. B. Little. 1981. Effects Of Protease Inhibitors On Radiation Transformation In Vitro. Cancer Res. 41:2103-2108.

Kim, K. H. and Burnett W. C. 1985. Ra In Phosphate Nodules From The Peru/Chile Sea Floor. Geochim. Cosmochim. Acta. 49:1073-1081.

Koch, J. and J. Tadmor. 1986. RADFOOD A Dynamic Model For Radioactivity Transfer Through The Human Food Chain. Health Phys. 50:721-737.

Kraft, G., W. Kraft-Weyrather, H. Meister, H. G. Miltenburger, R. Roots, and H. Wulf. 1982. The Influence Of Radiation Quality On The Biological Effectiveness Of Heavy Charged Particles. In :Radiation Protection, J. Booz and H. G. Ebert eds.

Ku, Teh-Lung, G. K. Knauss, and G. M. G Mathieu. 1977. Uranium In Open Ocean: Concentration And Isotopic Composition. Deep-Sea Research. 24:1005-1017.

Ku, Teh-Lung. 1965. An Evaluation Of The ^{234}U/^{238}U Method As A Tool For Dating Pelagic Sediments. Journal of Geophysical Research. 70(14):3457-3474.

Laconte, Gazz. 1854. Med. 9:488

Laiche, T. P, and L. M. Scott. 1991. A Radiological Evaluation Of Phosphogypsum. Health Phys. 60(5):691-693.

Langmuir, D. 1978. Uranium Solution-Mineral Equilibria At Low Temperatures With Applications To Sedimentary Ore Deposits. Geochim. Cosmochim. Acta. 42:547-569.

Lea, D. E. 1956. DEA: Actions Of Radiations On Living Cells. 2nd ed. Cambridge, England: Cambridge University Press.

Leach L. J., C. L. Yuile, H. C. Hodge, G. E. Sylvester, and H. B. Wilson. 1973. A Five-Year Inhalation Study With Natural Uranium Dioxide (UO_2) Dust. II. Postexposure Retention And Biologic Effects In The Monkey, Dog, And Rat. Health Phys. 25:239-258.

Leach, L. J., E. A. Maynard, H. C. Hodge, J. K. Scott, C. L. Yuile, G. E. Sylvester, and H. B. Wilson. 1970. A Five-Year Inhalation Study With Natural Uranium Dioxide (Uo_2) Dust. II. Postexposure Retention And Biologic Effects In The Monkey, Dog, And Rat. Health Phys. 18: 599-612.

LeBeau, M. M., and J. D. Rowley. 1984. Heritable Fragile sites in cancer. Nature. 308:607-608.

Leggett, R. W., and J. D. Harrison. 1995. Fractional Absorption Of Ingested Uranium In Humans. Health Phys. 68 (4): 484-498.

Leggett, R. W. 1989. The Behavior And Chemical Toxicity Of U In The Kidney: A Reassessment. Health Phys. 57 (3):365-383.

Linsalata, P. 1994. Uranium And Thorium Decay Series Radionuclides In Human And Animal Foodchains -A Review. Journal Of Environmental Quality. 23 (4):633-642.

Linsalata, P., R. S. Morse, H. Ford, M. Eisenbud, E. Penna Franca, M. B. de Castro, N. Lobao, I. Sachett, and M. Carlos. 1991. Th, U, Ra And Rare Earth Element Distributions In Farm Animal Tissues From An Elevated Natural Radiation Background Environment. J. Environ. Radioactivity. 14:233-257.

Linsalata, P. 1989. Exposure To Long-Lived Members Of The U And Th Decay Chains. Radiat. Phys. Chem. 34:241-250.

Linsalata, P., R. S. Morse, H. Ford, M. Eisenbud, E. Penna Franca, M. B. de Castro, N. Lobao, I. Sachett, and M. Carlos. 1989a. Transport Pathways Of Th, U, Ra And La From Soil To Cattle Tissues. J. Environ. Radioactivity 10:115-140.

Linsalata, P., R. S. Morse, H. Ford, M. Eisenbud, E. Penna Franca, M. B. de Castro, N. Lobao, I. Sachett, and M. Carlos. 1989b. An Assessment Of Soil-To-Plant Concentration Ratios For Some Natural Analogues Of Transuranic Elements. Health Phys. 56:33-46.

Linsata, P., M. Eisenbud, and E. Penna Franca. 1986. Ingestion Estimates Of Th And The Light Rare Earth Elements Based On Measurements Of Human Feces. Health Phys. 50:163-167

Liu, Hsueh-Hsuan, and Jiunn-Tzong Wu. 1993. Uptake And Recovery Of Americium And Uranium By Anacystis Biomass. J. Environ. Sci. Health. A28(2):491-594.

Lloyd, E. L., M. A. Gemmell, C. B. Henning, D. S. Gemmell, and B. J. Zabransky. 1979. Transformation Of Mammalian Cells By Alpha Particles. Int. J. Radia. Biol. 36:467-478.

LLobet J. M. J. J. Siervent, A. Ortega and J. L. Domingo. 1991. Influence Of Chronic Exposure To Uranium On Male Reproduction In Mice. Environmental and Applied Toxicology. 16:821-829.

Lopez, L. R., and R. R. Martinez. 1989. Evaluacion Del Contenido De Fluor En Rocas Fosforicas Mexicanas Por Medio Del Electrodo Especifico. Vet. Mex. 20:57-59

Lovley, D. R., E. J. P. Philips, Y. A. Gorby, and E. R. Landa. 1991. Microbial Reduction Of Uranium. Nature (London). 350:413-416.

Luckey T. B., B. Venugopal, and D. Hutcheson. 1975. Group III B Metals. In Heavy Metal Toxicity Safety And Hormology. Edited by Frederick Coulston Albany/N.Y. Supplement Vol. 1

Lundin, F. E., W. Lloyd, E. M. Smith. 1969. Mortality Of Uranium Miners In Relation To Radiation Exposure, Hard Rock Mining And Cigarette Smoking 1950 through September 1967. Health Phys. 16:571-578.

Luther, S. M., M. J. Dudas, and P. M. Rutherford. 1992. Radioactivity And Chemical Characteristics Of Alberta Phosphogypsum. Water, Air, And Soil Pollution. 69:277-290.

Macaskie L. E., R. M. Empson, A. K. Cheetham, C. P. Grey, and A. J. Skarnulis. 1992. Uranium Bioaccumulation By A Citrobacter Sp. As A Result Of Enzymically Mediated Growth Of Polycrystalline HUO_2PO_4. Science. 257 (7):782-784.

Maeda, M., and Winsdom, H. L. (1982). Behavior Of Uranium In Two Estuaries Of The Southeastern United States. Mar. Chem. 11:427-436.

Makweba, M. M. and E. Holm. 1993. The Natural Radioactivity Of The Rock Phosphates, Phosphatic Products And Their Environmental Implications. The Science of The Total Environment, 133:99-110.

Manyama, M. M. and E. Holm. 1993. The Natural Radioactivity Of The Rock Phosphates, Phosphatic Products And Their Environmental Implications. The Science of the Total Environment. 133:99-110.

Martin, J. M., V. N. Nijampurkar, and F. Salvadori. 1978. Uranium And Thorium Isotopes Behaviour In Estuarine Systems. In: Biogeochemistry Of Estuarine Sediments (ed. E.D. Goldberg), pp. 111-127. UNESCO, Paris.

Martinez-Aguirre, A., M. Garcia-Leon, and M. Ivanovich. 1994. Identification And Effects Of Anthropogenic Emissions Of U And Th On The Composition Of Sediments In A River/Extuarine System In Southern Spain. J. Environ. Radioactivity. 23:231-248.

Masuda, K. 1971a. Intake And Urinary Excretion Of Uranium In Non-Occupationally Exposed Persons. I. Recovery Of Uranium In The Daily Diet And Urine. Japanese J. Hyg. 26:434-437.

Masuda, K. 1971b. Intake And Urinary Excretion Of Uranium In Non-Occupationally Exposed Persons. II. Uranium In The Daily Diet. Japanese J. Hyg. 26:434-437.

Masuda, K. 1971c. Intake And Urinary Excretion Of Uranium In Non-Occupationally Exposed Persons. III. Uranium Excretion In Urine. Japanese J. Hyg. 26:434-437.

Masuda, K. 1971d. Intake And Urinary Excretion Of Uranium In Non-Occupationally Exposed Persons. IV. Discussions On Dietary Intake And Urinary Excretion Of Uranium. Japanese J. Hyg. 26:434-437.

Mays, C. W., Rowland R. E. and Stehney A. F. 1985. Cancer Risk From Lifetime Intake Of Radium And Uranium Isotopes. Health Phys. 48: 635-647.

Mays, C. W., and H. Speiss. 1984. Bone Sarcomas In Patients Given Radium-224. In: Boice, J. B.; Fraumeni, J. F., eds. Radiation Carcinogenesis. Epidemiology And Biological Significance. New York:Falta informacion 241-252.

Mc. Kee, B. A., D. J. DeMaster, and C. A. Nittrouer. 1987. Uranium Geochemistry On The Amazon Shelf: Evidence For Uranium Release From Bottom Sediments. Geochim. Cosmochim. Acta. 51:27779-86.

Menzel, F. G. 1968. Uranium, Radium, And Thorium Content In Phosphate Rocks And Their Possible Radiation Hazards. Journal of Agriculture and Food Chemistry. 16(2):231-234.

Meyer, S., and Urlich, C. 1923. The Content Of Ionium-Thorium In The Uranium Pitchblende of St. Joachimsthal. Sitzber. Akad. Wiss. Wien. Naturw. Kl., 132 (IIA), 279-284.

Michaels, H. B., and J.W. Hunt. 1978. A Model For Radiation Damage In Cells By Direct Effect And By Indirect Effect: A Radiation Chemistry Approach. Radiat. Res. 74:23-24.

Miller, T. J. 1991. Development Of A Rapid, Economical And Sensitive Method For The Determination Of Excreted Uranium In Urine. Analytical Letters 24(4):657-664.

Miller, H. T. 1977. Radiation Exposures Associated With Surface Mining For Uranium. Health Phys. 32:523-528.

Minoru, Koide and E. D. Goldberg. 1965. Uranium-234/Uranium-238 Ratios In Sea Water. Progress In Oceanography Vol. 3 Pergamon Press Ltd.

Mitsunobu, Tatsumoto and E. D. Goldberg. 1959. Some Aspects Of The Marine Geochemistry Of Uranium. Geochimica Et Cosmochimica Acta. 17:201-208.

Miyake, Y., Y. Sugimura, and T. Uchida. 1966. Ratio U^{234}/U^{238} And The Uranium Concentration In Seawater In The Western North Pacific. Journal Of Geophysical Research. 71(12):3083-3087.

Moore, W. S 1992. Radionuclides Of The Uranium And Thorium Decay Series In The Estuarine Environment. In: Uranium-Series Disequilibrium: Applications to Earth, Marine, and Environmental Sciences. 2nd ed. eds. M. Ivanovich and R.S. Harmon. Oxford University Press. New York, USA.

Moore, W.S. 1967. Amazon And Mississipi River Concentrations Of Uranium, Thorium And Radium Isotopes. Earth Planet. Sci. Lett. 2:231-234.

Moorhead, P. S., P. C. Nowell, W. J. Mellman, D. M. Battips, D. A. Hungerford. 1960. Chromosome Preparations Of Leukocyte Cultures From Human Peripheral Blood. Exp. Cell. Res. 20:613-616.

Mottram, J. C. 1936. Factor Of Importance In Radiosensitivity Of Tumors. Br. J. Radiol. 9:606-614.

Mortvedt, J. J. 1994. Plant And Soil Relationships Of Uranium And Thorium Decay Series Radionuclides - A Review. J. Environ. Qual. 23:643-650.

Morrow, P. E. 1984. Biokinetics And Toxicology Of Uranium. In: Biokinetics And Analysis Of Uranium In Man. Richland, WA: Hanford Environmental Health Foundation; USUR-05/HEHF-47 E 1-27

Morrow, P. E., Gelein, R. M. Beiter, H. D. Scott, J. B.: Picano, J. J.; Yuile, C. L. 1982a. Inhalation and intravenous studies of UF_6/UO_2F_2 in dogs. Health Phys. 43:859-873

Morrow, P. E., L. J. Leach, F. A. Smith, R. M. Gelein, J. B. Scott, H. D. Beiter, F. J. Amato, J. J. Picano, C. L. Yuile, T. G. Consler. 1982b. Metabolic Fate And Evalution Of Injury In Rats And Dogs Following Exposure To The Hydrolysis Products Of Uranium Hexafluoride. Washington, D.C.: U.S. Nuclear Regulatory Commission; nureg/cr-2268

Moss, M. A., and R. F McCurdy. 1982. Clinical Study Of A Population Exposed To Excessive Levels Of Uranium In Drinking Water. Paper Presented At The 51st Annual Meeting Of The Royal College Of Physicians And Surgeons Of Canada, Quebec City, September 1982. Annals R.C.P.S.C. 15, 4 (June 1982) (abstract).

Murillo Jimenez, J. M. 1987. Algunas Caracteristicas Paleoceanograficas Y Cuerpos De Agua Inferidos A Partir Del Registro Micropaleontologico (Radiolaria), En La Bahia De La Paz, B.C.S. Mexico. Thesis De Licenciatura, Universidad Autonoma De Baja California Sur. 70p.

Narayana, Y., A. P. Radhakrishna, H. M. Somashekarappa, N. Karunakara, K. M. Balakrishana, and K. Siddappa. 1995. Distribution And Enrichment Of Radionuclides In The Newly Discovered High Background Area In Ullal On The Southwest Coast Of India. Health Phys. 69(2):178-186.

Neuman, W. F., C. Voegtlin, and H. C. Hodge. 1949. Deposition Of Uranium In Bone. Pp. 1911-1991 In National Nuclear Energy Series, Part 1 of Div. VI, Vol1, W. F. Neuman, ed. New York: McGraw-Hill.

Nomiyama, K., and Foulkes, E. C. 1968. Some Effects Of Uranyl Acetate On Proximal Tubular Functions In Rat Kidney. Toxicol. Appl. Pharmacol. 13. 89-98.

NRC. National Research Council. 1990. Health Effects Of Exposure To Low Levels Of Ionizing Radition (Beir V). National Academy Of Sciences, Washington, DC.

NRC. National Research Council. 1988. Health Risks Of Radon And Other Internally Deposited Alpha-Emitters. (Beir Iv).National Academy Of Sciences, Washington, DC.

NCRP. National Council on Radiation Protecion and Measurements. 1984. Exposure From The Uranium Series With Emphasis On Radon And Its Daughter. NCRP report No. 77. Bethesdas, MD

NCRP. National Council on Radiation Protecion and Measurements. 1959. Maximum Permissible Body Burdens And Maximum Permissible Concentrtions Of Radionuclides In Air And Water For Occupational Exposure. U.S. Department of Commerce,National Bureu of Standard Handbook 69, Washington, D.C.

Obeso Nieblas, M. 1986. Propagacion De La Constituyente M2 De La Marea En La Bahia De La Paz, B.C.S. Mexico: Mediante Un Modelo Bidmensional Hidrodinamico Numerico. Tesis De Maestria. (CICIMAR) Inst. Pol. Nal., 123p.

O'Brien, G. W., A. R. Milnes, and D. J. Cullen. 1987. Sea-Floor Weathering Of Phosphate Nodules Off East Australia: Its Effects On Uranium Oxidation State And Isotopic Composition. Geochim. Cosmochim. Acta. 51:2051-2064.

Orcutt, J. A. 1949. The Toxicology Of Compounds Of Uranium Following Application to the skin. In: Voegtlin C. Hodge H. C., eds. Pharmacology and Toxicology of Uranium Compounds. Vols 3 and 4. New York, NY: McGraw Hill Book Co.

Osburn, W. S. 1965. Primordial Radionuclide: Their Distribution, Movement, And Possible Effect Within Terrestrial Ecosystems. Health Phys. 11:1275-1295

Palcic, B., and L. D. Skarsgard. 1984. Reduced Oxygen Enhancement Ratio At Low Doses If Ionizing Radiation. Radiat. Res. 100:328-339.

Parker, H. M., and S. T. Cantril. 1986. The Tolerance Dose. In: Herbert M. Parker, Publications And Other Contributions To Radiological And Health Physics, Part IV: Radiation Protection: The Manhattan District And Beyond.

Piispanen, R. 1991. Correlation Of Cancer Incidence With Groundwater Geochemisatry In Northern Finland. Environmental Geochemistry and Health. 13(2):66-69.

Piispanen, R. 1989. Geochemical Interpretation Of Cancer Maps Of Finland. Environmental Geochemistry and Health. 11:145-147.

Pimpl, M., B. Yoo, and I. Yordanova. 1992. Optimization Of Radioanalytical Procedure For The Determination Of Uranium Isotopes In Environmental Samples. Journal Of Radioanalytical And Nuclear Chemistry, Articles. 161 (2):437-441.

Potten, C. S. 1985. Radiation And The Skin. Taylor And Francis Inc.

Priest, N. D., G.R. Howells, D. Green, and J. W. Haines. 1982. Uranium In Bone; Metabolic And Autoradiographic Studies In The Rat. human toxicol. 1:97-114.

Rayno, D. 1983. Estimated Dose To Man From Uranium Milling Via The Beef/Milk Food Chain Pathway. Sci. Total Environ. 31:219-241.

Reif, R H., J.B. Turner, and D.S. Carlson. 1992. Uranium In Vitro Bioassay Action Level Used To Screen Workers For Chronic Inhalation Intakes Of Uranium Mill Tailings. Health Phys. 63(4):398-401.

Riss, W. 1924. The Composition Of Bröggerite And The Genetic Connection Between Thorium And Uranium. sitzber. akad. wiss. wien. naturw. kl. 132 (IIA), 91-100.

Robertson, J. B., A. Koehler, J. George, and J. B. Little. 1983. Oncogenic Transformation Of Mouse Balb/3t3 Cells By Plutonium-238 Alpha Particles. Radiat. Res. 96:261-274.

Rodean H. C. 1991. A Structure For Models Of Hazardous Materials With Complex Behavior. atmospheric environment 25a(5/6):885-898.

Rodriguez, A., E. L. Alpen, P. Powers-Risius. 1992. The RBE-LET Relationship For Rodent Intestinal Crypt Cell Survival, Testes Weight Loss, and Multicellular Spheroid Cell Survival after Heavy-Ion Irradiation. Radiation Research 132: 184-192

ROFOMEX. Roca Fosforica Mexicana. Instruction Booklet. Roca Fosforica Mexicana S.A. de C.V. Calle Puesta del Sol No. 150 Colonia Puesta del Sol APDO. Postal No. 335 C.P 23090 La Paz, B. C. S. Mexico.

Rogers, J. J. W., and J. A. S. Adams. 1969. Uranium. P. 92-A-1 To 92-G-7. In: Handbook Of Geochemistry. K.H. Wedepohl (Ed) Springer-Verlag, New York.
Rona, E. 1956. Uranium Determination In Seawater. Transactions American Geophysical Union. 37(6):697-701.

Rowland, R. E., and J. E. Farnham. 1969. The Deposition Of Uranium In Bone. Health Phys. 17:139-144.

Rubery, E. D. 1989. Radionuclides In Food: A Neglected Branch Of Toxicology. Human Toxicology. 8:79-86.

Saccomanno, G., C. Yale, W. Dixon, O. Averbach and G. C. Huth. 1986. An Epidemiological Analysis Of The Relationship Between exposure to Radon Progeny, Smoking and Bronchogenic Carcinoma In The Uranium-mining Population of The Colorado Plateu (USA): 1960-1980. Health Phys. 50:605-618.

Sakaguchi, T., T. Horikoshi, and A. Nakajima. 1978. Uptake Of Uranium From Sea Water By Microalgae. J. Ferment. Technol. 56:561-565.

Samet, J. M. , D. M. Kutvirt, R. J. Waxweiler, et al., 1984. Uranium Mining And Lung Cancer In Navaho Men. N. Eng. J. Med. 310:1481-1484.

Sankaranarayanan, K., and R. Chakraborty. 1995. Cancer Predisposition, Radiosensitivity And The Risk Of Radiation-Induced Cancers. 1 Background. Radiation Research. 143:121-143.

Simon, S. L., and S. A. Ibrahim. 1988. Biological Uptake Of Radium By Terrestrial Plants: A review. Radium In The Enviroment. Atomic Energy Agency. Vienna, Austria.

Simon, S. L., and S. A. Ibrahim. 1987. The Plant/Soil Concentration Ratio For Calcium, Radium, Lead And Polonium: Evidence For Non-Linearity With Reference To Substrate Concentration. J. Environ. Radioact. 5:123-142.

Sinclair, W. K., and R. A. Morton. 1966. X Ray Sensitivity Diring The Cell Generation Cycle Of Cultured Chinese Hamster Cells. Radiat. Research. 29:450-474.

Spalding, R. F., and W. M. Sackett. 1972. Uranium In Runoff From The Gulf Of Mexico Distributive Province: Anomalous Concentratrions. Science. 175:629-631.

Spencer, H., D. Osis, I. M. Fisenne, P. M. Perry, N. H. Harley. 1990. Measured Intake And Excretion Patterns Of Naturally Occurring ^{234}U, ^{238}U, And Calcium In Humans. Radiat. Res. 124:90-95.

Stevens, W, F. W. Bruenger, D. R. Atherton. 1980. The Distribution And Retention Of Hexavalent Uranium-233 In The Beagle. Radiat. Res. 83:109-126.

Sutter, G. W. 1993. Ecological Risk Assessment. Lewis Publisher, Boca Raton, Fl.

Tandy, B. C. 1974 . New Radioactive Nodule And Reduction Occurrance In The Littleham-Larksbeare Area Of Devon. In: Radioactive and Metalliferous Minerals Unit. Report No. 316 Inst. Geological Sciences

Tandy, B. C. 1973. A Radiometric And Geochemical Reconnaissance Of The Permian Outcrop And Adjacent Areas In South-West England. In: Radioactive and Metalliferous Minerals Unit. Report No. 315 Inst. Geological Sciences.

Tannenbaum, A., H. Silverstone, and J. Koziol. 1951. Tracer Studies Of The Distribution And The Excretion Of U In Mice, Rats, And Dogs. In: Toxicology of Uranium. A. Tannenbaum ed. National Nuclear Energy Series. Division VI. 23:128-181. NY: Mc Graw-Hill Book Co. Inc.

Terasima, T., and L. J. Tolmach. 1963. Variations In Several Responses Of Hela Cells To X-Irradiation During The Division Cycle. Biophys. J. 3:11-33.

Thurber, D. L. 1962. Anomalous ^{234}U/^{238}U and an investigation of the potential of ^{234}U for Pleistocene chronology. PhD. thesis, Columbia University, New York.

Toole, J., Baxter, M. S. and Thomson, J. 1987. The Behaviour Of Uranium Isotopes With Salinity Change In Three U.K. Estuaries. Estuar. Coast. Shelf. Sci. 25:283-297.

Toole, J., M. S. Baxter, and J. Thomson. 1988. Some Aspects Of The Marine Geochemistry Of Uranium. In Radionuclides: A Tool For Oceanography (ed. J.C. Guary, P. Guegueniat, and R. J. Pentreath) pp. 183-194. Elsevier, London.

Turekian, K. K., and L. H. Chan. 1971. The Marine Geochemistry Of The Uranium Isotopes, Th-230 And Pa-231. In: Activation Analysis In Geochemistry And Cosmochemistry (ed. A.O. Brunfelt and E. Steinnes), pp 311-320. Universite forlaget, Oslo.

UNSCEAR. United Nations Scientific Committee on the Effects of Atomic Radiation. 1988. Sources, Effects, And Risks Of Ionizing Radiation. Unscear 1988 Report To Un General Assembly, With Annexes, United Nations.

UNSCEAR. United Nations Scientific Committee on the Effects of Atomic Radiation. 1982. Ionizingh Radiation: Sources And Biological Effects . Report To The General Assembly, With Annexes: Thirty-Seventh Session, Supplement 45 (A/37/45). United Nations, New York.

UNSCEAR. United Nations Scientific Committee on the Effects of Atomic Radiation. 1977. Sources And Effects Of Ionizingh Radiation. Report To The General Assembly, With Annexes: Thirty-Second Session, Supplement 40 (A/32/40). United Nations, New York.

Veeh, H. Herbert. 1968. U^{234}/U^{238} In The East Pacific Sector Of The Antartic Ocean And In The Red Sea. geochimica et cosmochimica acta, 32: 117-119.

Voegtlin, C., and H. C.Hodge (Eds). 1953. The Pharmacology And Toxicology Of Uranium Compounds. National Nuclear Energy Series. Division VI. Vol 1 Parts 1 and 2. New York: Mc Graw-Hill Book Co. Inc.

Voegtlin, C., and H. C.Hodge (Eds). 1949. The Pharmacology And Toxicology Of Uranium Compounds. National Nuclear Energy Series. Division IV. Vol 1 Parts 1 and 2. New York: Mc Graw-Hill Book Co. Inc.

Vogel, F. 1992. Risk Calculations For Hereditary Effects Of Ionizing Radiation In Humans. A Review. Hum. Genet. 89:127-146.

Wang, C. H., D. L. Willis, W. D. Loveland. 1975. Radiotracer Methodology In The Biological Environmental, And Physical Sciences. Prentice-Hall Biological Science Series. p 46

Waxweiler, R. J., V. E. Archer, R. J. Roscoe, A. Watanabe, and J. J. Thun. 1983. Mortality Patterns Among A Retrospective Cohort of Uranium Mill Workers. Presented at Epidimiology Applied to Health Physics. Pp. 428-435. In: Proceedings Of The Health Physics Society.

Welford, G. A., R. Baird., and I.M. Fisenne. 1976. Concentrations Of Natural Uranium In The Human Body. In: Proc. of the 10th Midyear Topical Symp. of the Health Physics Society. pp 239-244 (Troy, NY: Rensselaer Polytechnic Institute).

Welford, G. A., and R. Baird. 1967. Uranium Levels In Human Diet And Biological Materials. Health Phys. 13:1321-1324.

Welford, G. A., J. S., Aiercio, R. S. Morse. 1960. Urinary Uranium Levels In Non-Exposed Individuals. Am. Ind. Hyg. Assoc. J. 21: 68-70.

West, C. M, L. M. Scott, and N. B. Schultz. 1979. 16 Years Of Uranium Personnel Monitoring Experience-Retrospec. Health Physics. 36:665-669.

Williams, Ross W.. 1988. Uranium And Thorium Decay Series-Disequilibria In Young Volcanic Rock. Ph. D. Thesis, University of California, Santa Cruz.

Williams, G. M. 1977. Detection Of Chemical Carcinogens By Unscheduled Dna Synthesis In Rat Liver Primary Cell Cultures. Cancer Res. 37:1845-1851.

Willis, A. E., and T. Lindahal. 1987. DNA Ligase I Defficiency In Bloom's Syndrome. Nature. 325:355-357.

Wilson, J. D., R. K. Webster, G. W. C. Milner, G. A. Barnett, and A. A. Smales. 1960. A Comparison Of Three Methods Of Determining The Concentration Of Uranium In Sea Water. Anal. Chim. Acta. 23: 505-514.

Wrenn, M. E., H. Ruth, D. Burleigh, N. P. Singh. 1992. Background Levels Of Uranium In Human Urine. J. Radioanalyt. Nucl. Chem. 156:407-412.

Wrenn M. E., J. Lipsztein, L. Bertelli. 1989. Pharmacokinetic Models Relevant To Toxicity And Metabolism For Uranium In Humans And Animals. Radiat. Prot. Dosim. 26:243-248.

Wrenn, M. E., P. W. Durbin, B. Howard, J. Lipsztein, J. Rundo, E.T. Still and D.L. Willis. 1985. Metabolism Of Ingested U And Ra. Health Phys. 48 (5)(May):601-633.

Wrenn, M. E., P. E. Morrow, and J. B. Hursh. 1984. Personal Communication To Union Carbide Corporation (Published In Report On Toxicological Studies Concerning Exposures To Uf6 And Uf6 Hydrolysis Products (Edited by R. A. Just), KID-5573, Rev. 1, Martin Marietta Energy Systems, Inc., Oak Ridge, TN.

Wrenn, M. E., N. P. Singh, N. Cohen, S. A. Ibrahim, and G. Saccomanno. 1981. Th In Human Tissues. U.S. Nuclear Regulatory Commission. NUREG/CR-1227 RH. NTIS, Springfield, VA.

Yammamoto, T, E. Yunoki, M. Yamakawa, M. Shimizu, and K. Nukada. 1974. Studies On Environmental Contamination By Uranium. J. Radiat. Res. 15:156-162.

Yammamoto T, K. Masuda, K. Nukada. 1971. Studies On Environmental Contamination By Uranium. 6. Uranium In Total Diet And Human Urine From Nonoccupationall Exposed Persons In Okayama Prefecture (1969-1971) [abstract]. J Radiat. Res .13(1):5.

Yang, T. C. H., L. M. Craise, M. T. Mei, and C. A. Tobias. 1985. Neoplastic Cell Transformation By Heavy Charged Particles. Radiat. Res. 104:S177-178.

Yuile, C.L. 1973. Animal Experiments. In: Handbook Of Experimental Pharmacology. Hodge HC., JN Stannard and J.B.Hursh. eds. Berlin: Springer-Verlag.