"A" students work (without solutions manual)
~ 10 problems/night.

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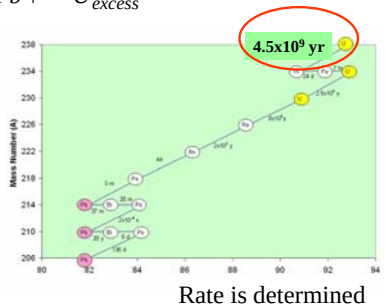
Office Hours Th&F 2-3:30 pm

Module #15
Applied Kinetics

Example 1: Geologic Age leading to a forensics example

A tricky example from geology and environmental science

$$^{238}U \rightarrow {}^{206}Pb + {}^{238}U_{excess}$$



Rate is determined By the slowest step

Example Calculation Applied Kinetics 1: A rock containing $^{238}_{92}$U and $^{206}_{82}$Pb had a ratio of Pb/U atoms of 0.115. Assuming no lead was originally present in the rock and that the half lives of the intermediate nuclides are negligible, calculate the age of the rock using the half-life of $^{238}_{92}$U as 4.5x10^9 years and assuming first order kinetics.

$$^{238}U \rightarrow {}^{206}Pb + {}^{238}U_{excess}$$

Know	Want	Red Herrings
$t_{1/2}$ = 4.5x10^9 yrs	t	
1st order reaction		
Pb/U=0.115 at t present		

$$kt = \ln\left\{\frac{[A_o]}{[A_t]}\right\} \qquad t_{1/2} = \frac{0.693}{k}$$

$$\left\{\frac{0.693}{t_{1/2}}\right\} t = \ln\left\{\frac{[A_o]}{[A_t]}\right\} \qquad k = \frac{0.693}{t_{1/2}}$$

Example Calculation Applied Kinetics 1: A rock containing $^{238}_{92}$U and $^{206}_{82}$Pb had a ratio of Pb/U atoms of 0.115. Assuming no lead was originally present in the rock and that the half lives of the intermediate nuclides are negligible, calculate the age of the rock using the half-life of $^{238}_{92}$U as 4.5x10^9 years and assuming first order kinetics.

$$^{238}U \rightarrow {}^{206}Pb + {}^{238}U_{excess}$$

No Pb at t_o
All Pb comes from U

Know	Want
$t_{1/2}$ = 4.5x10^9 yrs	t
1st order reaction	
Pb/U=0.115, at t present	

$$\left\{\frac{0.693}{t_{1/2}}\right\} t = \ln\left\{\frac{[A_o]}{[A_t]}\right\}$$

What information do we have That will allow us to get $[A_t]$ and $[A_o]$? Do we need both?

$$^{238}U \rightarrow {}^{206}Pb + {}^{238}U_{excess}$$

Mass balance equation

$$[U_0] = [Pb_t] + [U_t]$$

No Pb at t_o
All Pb comes from U

$$\frac{[Pb_t]}{[U_t]} = 0.115$$

Measured in the present, given in the problem

$$[Pb_t] = 0.115[U_t]$$

$$[U_0] = 0.115[U_t] + [U_t]$$

$$[U_0] = [U_t](0.115 + 1)$$

$$[U_0] = [U_t](1.115)$$

Example Calculation Applied Kinetics 1: A rock containing $^{238}_{92}$U and $^{206}_{82}$Pb had a ratio of Pb/U atoms of 0.115. Assuming no lead was originally present in the rock and that the half lives of the intermediate nuclides are negligible, calculate the age of the rock using the half-life of $^{238}_{92}$U as 4.5×10^9 years and assuming first order kinetics.

$$\left\{\frac{0.693}{t_{1/2}}\right\} t = \ln\left\{\frac{[A_o]}{[A_t]}\right\}$$

$$[U_0] = [U_t](1.115)$$

$$\left\{\frac{0.693}{4.5x10^9\ yr}\right\} t = \ln\left\{\frac{[U_t]1.115}{[U_t]}\right\}$$

$$\left(\frac{0.693}{4.5x10^9\ yr}\right) t = \ln(1.115)$$

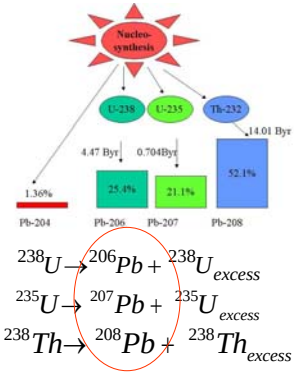
$$(1.54x10^{-10}\ yr^{-1})t = 0.10885$$

$$\frac{0.10885}{1.54x10^{-10}\ yr^{-1}} = t$$

$$7.068x10^8\ yr = t$$

CONTEXT Slide Geologic Ages of the U.S.:

CONTEXT Slide

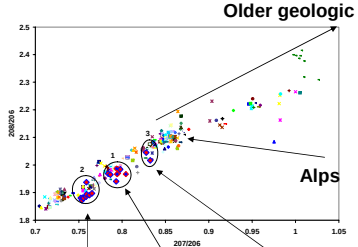


$$^{238}U \rightarrow {}^{206}Pb + {}^{238}U_{excess}$$

$$^{235}U \rightarrow {}^{207}Pb + {}^{235}U_{excess}$$

$$^{238}Th \rightarrow {}^{208}Pb + {}^{238}Th_{excess}$$

Older geologic age



Tooth lead from Grafton, Ill
Grave site = Diamonds

You Are What You Eat:

Your bones and teeth will reflect the background source of Pb isotopes

Forensic determinations based on body kinetics

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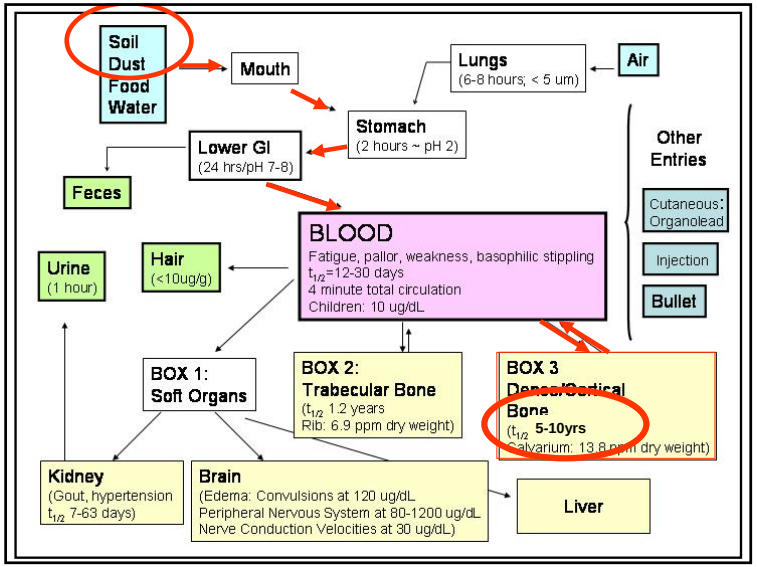
Module #15
Applied Kinetics

Example 2: Environmental Risk Assessment Models



Influence of bone resorption on the mobilization of lead from bone among middle-aged and elderly men: the normative aging study. Tsaih, Shirng-Wern; Korrick, Susan; Schwartz, Joel; Lee, Mei-Ling Ting; Amarasiriwardena, Chitra; Aro, Antonio; Sparrow, David; Hu, Howard. Occupational Health Program, Department of Environmental Health, Harvard School of Public Health, Boston, MA, USA. Environmental Health Perspectives (2001), 109(10), 995-999. Publisher: National Institute of Environmental Health Sciences, Abstract

Bone stores of lead accrued from environmental exposures and found in most of the general population have recently been linked to the development of hypertension, cognitive decrements, and adverse reproductive outcomes. The skeleton is the major endogenous source of lead in circulating blood, particularly under conditions of accelerated bone turnover and mineral loss, such as during pregnancy and in postmenopausal osteoporosis. We studied the influence of bone resorption rate on the release of lead from bone in 333 men, predominantly white, middle-aged and elderly (mostly retired) from the Boston area. We evaluated bone resorption by measuring cross-linked N-telopeptides of type I collagen (NTx) in 24-h urine samples with an ELISA. We used K-X-ray fluorescence to measure lead content in cortical (tibia) and trabecular (patella) bone; we used graphite furnace at. absorption spectroscopy and inductively coupled plasma mass spectroscopy to measure lead in blood and urine, resp. After adjustment for age and creatinine clearance, the pos. relation of patella lead to urinary lead was stronger among subjects in the upper two NTx tertiles (b for patella lead ^{3}0.015) than in the lowest NTx tertile (b for patella lead = 0.008; overall p-value for interactions = 0.06). In contrast, we found no statistically significant influence of NTx tertile on the relationship of blood lead to urinary lead. As expected, the magnitude of the relationship of bone lead to urinary lead diminished after adjustment for blood lead. Nevertheless, the pattern of the relationships of bone lead to urinary lead across NTx tertiles remained unchanged. Furthermore, after adjustment for age, the relation of patella lead to blood lead was significantly stronger in the upper two NTx tertiles (b for patella lead ^{3}0.125) than in the lowest NTx tertile (b for patella lead = 0.072). **The results provide evidence that bone resorption influences the release of bone lead stores (particularly patella lead) into the circulation**.

Lead poisoning secondary to hyperthyroidism: report of two cases. Klein, Marc; Barbe, Francoise; Pascal, Veronique; Weryha, Georges; Leclere, Jacques. Clinique Medicale et Endocrinologique, CHU de Nancy, Hopitaux de Brabois, Vandoeuvre-les-Nancy, Fr. European Journal of Endocrinology (1998), 138(2), 185-188. Publisher: BioScientifica, CODEN: EJOEEP ISSN: 0804-4643. Journal written in English. CAN 128:253919 AN 1998:162366 CAPLUS

Abstract

With long-term exposure to lead, lead accumulates in bone, where it is stored for years. These quiescent lead stores are mobilized when increased bone turnover occurs, and latent lead toxicity may then become symptomatic. **Although Graves' disease is a common cause of increased bone turnover, to date hyperthyroidism has been implicated in lead poisoning only twice. The authors describe herein two cases of hyperthyroidism, one caused by toxic multinodular thyroid enlargement, the second by Graves' disease, leading to lead poisoning**. Treatment of hyperthyroidism with radioactive iodine cured both hyperthyroidism and lead poisoning and no chelating agent therapy was necessary. Lead poisoning is an important environmental health problem, and physicians must be aware of the endocrine disorders such as hyperthyroidism and hyperparathyroidism that increase bone turnover, favoring lead mobilization. Atypical symptoms should draw the physician's attention to the possibility of lead poisoning, particularly in workers with occupational exposure to lead and in areas where lead poisoning is endemic.

Use of sequentially administered stable lead isotopes to investigate changes in blood lead during pregnancy in nonhuman primate (Macaca fascicularis). Franklin, C. A.; Inskip, M. J.; Baccanale, C. L.; Edwards, C. M.; Manton, W. I.; Edwards, E.; O'Flaherty, E. J. Pest Management Regulatory Agency, Health Canada, Ottawa, ON, Can. Fundamental and Applied Toxicology (1997), 39(2), 109-119. Publisher: Academic Press, CODEN: FAATDF ISSN: 0272-0590. Journal written in English. CAN 128:19604 AN 1997:741981 CAPLUS

Abstract

The effects of pregnancy on the flux of lead from maternal bone were investigated in five females from a unique colony of cynomolgus monkeys (Macaca fascicularis) which had been dosed orally with lead (approx. 1100-1300 mg Pb/kg body wt) throughout their lives (about 14 yr). Through the use of stable lead isotopes 204Pb, 206Pb, and 207Pb, it was possible to differentiate between the lead contributed to blood lead from the skeleton and the lead contributed from the current oral dose. Blood samples and bone biopsy samples taken before, during, and after pregnancy were analyzed for lead (total and stable isotope ratios) by thermal ionization mass spectrometry. Through the use of end-member unmixing equations, the contribution to blood of lead from material bone during pregnancy was estd. and compared to the contribution of lead from maternal bone before pregnancy. A 29 to 56% decrease in bone lead mobilization in the first trimester was followed by an increase in the second and third trimesters, up to 44% over baseline levels. In one monkey, the third-trimester increase did not reach baseline. In a single low-lead monkey, a similar decrease in the first trimester was followed by a 60% increase in the third trimester, indicating that a similar pattern of flux is seen over a wide range of lead concns. Anal. of maternal bone and fetal bone, brain, liver, and kidneys confirmed a substantial transplacental transfer of endogenous lead. Lead concns. in fetal bone often exceeded maternal bone lead concns. From **7 to 39% of the lead in the fetal skeleton originated from the maternal skeleton.**

Relationship of blood and bone lead to menopause and bone mineral density among middle-age women in Mexico City. Latorre, Francisco Garrido; Hernandez-Avila, Mauricio; Orozco, Juan Tamayo; Medina, Carlos A. Albores; Aro, Antonio; Palazuelos, Eduardo; Hu, Howard. Instituto Nacional de Salud Publica, Morelos, Mex. Environmental Health Perspectives (2003), 111(4), 631-636. Publisher: U. S. Department of Health and Human Services, Public Health Services, CODEN: EVHPAZ ISSN: 0091-6765. Journal written in English. CAN 139:105305 AN 2003:336807 CAPLUS

Abstract

To describe the relationship of blood lead levels to menopause and bone lead levels, we conducted a cross-sectional study on 232 pre- or perimenopausal (PreM) and postmenopausal (PosM) women who participated in an osteoporosis-screening program in Mexico City, Mexico, during the first quarter of 1995. Information regarding reproductive characteristics and known risk factors for blood lead was obtained using a std. questionnaire by direct interview. The mean age of the population was 54.7 yr (SD = 9.8), with a mean blood lead level of 9.2 mg/dL (SD = 4.7/dL) and a range from 2.1 to 32.1 mg/dL. After adjusting for age and bone lead levels, the mean blood lead level was 1.98 mg/dL higher in PosM women than in PreM women (p = 0.024). The increase in mean blood lead levels peaked during the second year of amenorrhea with a level (10.35 mg/dL) that was 3.51 mg/dL higher than that of PreM women. Other important predictors of blood lead levels were use of lead-glazed ceramics, schooling, trabecular bone lead, body mass index, time of living in Mexico City, and use of hormone replacement therapy. Bone d. was not assocd. with blood lead levels. **These results support the hypothesis that release of bone lead stores increases during menopause and constitutes an internal source of exposure possibly assocd. with health effects in women in menopause transition.**

Bone remodeling increases substantially in the years after menopause and remains Increased in older osteoporosis patients

RECKER Robert (1) ; LAPPE Joan (1) ; DAVIES K. Michael (1) ; HEANEY Robert (1) ;
(1) Creighton Osteoporosis Research Center, Creighton University, Omaha, Nebraska, ETATS-UNIS

Résumé / Abstract

Introduction: Increased bone remodeling rates are associated with increased skeletal fragility independent of bone mass, partially accounting for the age-related increase in fracture risk in women that is independent of bone loss. We examined bone remodeling rates before and after menopause and in women with osteoporosis by measurements of activation frequency (Ac.f, #/year) in transilial bone biopsy specimens. Materials and Methods: We recruited 75 women, >46 years old, who had premenopausal estradiol and gonadotropin levels and regular menses. During 9.5 years of observation, 50 women experienced normal menopause and had 2 transilial bone biopsy specimens after tetracycline labeling, one at the beginning of observation and the second 12 months after the last menses, when serum follicle-stimulating hormone (FSH) was >75 mIU/ml and serum estradiol was <20 pg/ml. Ac.f was also computed for a group of older healthy postmenopausal women and a group of women with untreated osteoporosis studied earlier by the same biopsy (Bx) and labeling protocol. Results: Median Ac.f rose from 013/year to 0.24/year ($p < 0.001$) across menopause and was greater still in the older normals ($p < 0.009$) than in the second Bx. Ac.f was not significantly greater in the osteoporosis patients than in the older postmenopausal normals. **Conclusion: Bone remodeling rates double at menopause, triple 13 years later**, and remain elevated in osteoporosis. This change contributes to increases in age-related skeletal fragility in women.

Revue / Journal Title
Journal of bone and mineral research (J. bone miner. res.) ISSN 0884-0431 CODEN JBMREJ
Source / Source
2004, vol. 19, no10, pp. 1628-1633 [6 page(s) (article)] (26 ref.)

Applied Kinetics Example Calculation 2: If a woman was exposed early in life to lead and had a cortical bone lead concentration of 100 ppm, what amount of lead would remain in the cortical bone 10 years after exposure assuming that she was pre-menopausal? Assuming that she was post-menopausal? Assuming that she was elderly? Assume removal of lead from bone is first order reaction.

$$\left[A_o\right] = 100\,ppm = \frac{100\,parts}{1{,}000{,}000\,parts}$$

$$t = 10\,yrs$$

$$t_{1/2,pre-menopausal} = 7.5\,yrs \quad \textbf{From risk assessment model}$$

$$t_{1/2,post-menopausal} = \frac{7.5\,yrs}{2}$$

$$t_{1/2,elderly} = \frac{7.5\,yrs}{3} \quad \textbf{From article}$$

$$\left\{\frac{0.693}{t_{1/2}}\right\} t = \ln\left\{\frac{\left[A_o\right]}{\left[A_t\right]}\right\}$$

$$\left[A_t\right] = \left[A_o\right] e^{\left[-\left\{\frac{0.693}{t_{1/2}}\right\} t\right]} = 100\,ppm\left(e^{\frac{-6.93}{t_{1/2}}10\,years}\right)$$

Woman	Pb,ppm
Pre-menopausal	39.7
Post-menopausal	15.7
Elderly	6.2

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Module #15
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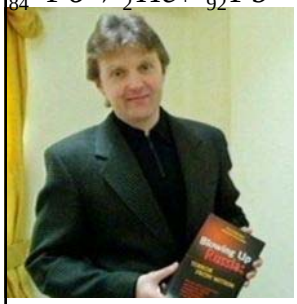
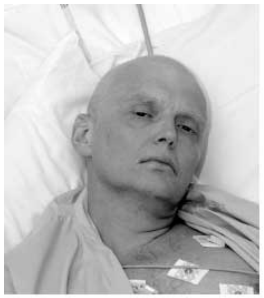
Example 3: ^{210}Po

An Example of rate constants in the real world: context and calculations

Toxicology of Radioactive Exposure

$$^{210}_{84}Po \rightarrow {}^{4}_{2}He + {}^{206}_{92}Pb$$





Alexander Litvinenko, former Russian KGB agent poisoned with Polonium on Nov. 1, died Nov. 23, 2006

Example: About how many grams of Polonium would be required to kill Mr. Litvinenko given the committed toxic dose of Po is 2.14×10^{-7} Sv/Bq, the half life of Po is 138 day, and that the toxic dose is 5 Sv? How long with the Po stay in the body?

Toxicology of Radioactive Exposure

1. **Uptake, transport, and excretion in body (depends on chemistry)**
2. **Effect of radiation**
3. **Tissue damage scaled to energy**

Read Chapter 19.1, 19.2, p. 523 Of Masterton and Hurley,
Problems: 13-29 of (Chapter 19)

Or Read Brown et al 21.1 to 21.6; And then 21.9

To solve this problem we will need to use information from
a) Chemists (MM, molecular chemistry, bond strengths, free radicals)
b) Physicists (energy of expelled particles)
c) Geologists ($t_{1/2}$ of the atom)
d) Medical radiologists (types of tissue damage)
e) Toxicologists (physiological half lives)

For the phenomena each field has it's own
language
And reference states
%# conversions!!!!

How and where Po might go depends upon it's chemistry

1. Same family as O, S, an Se, Te

$$Po = [Xe]6s^2 4f^{14} 5d^{10} 6p^4$$

2. But with a smaller ionization energy

$$M \rightarrow M^+ + e$$

2. it does not form covalent bonds
E.N. =2.0 for Po vs. 2.55 for C and 3.44 for O

3. Forms ionic, soluble compounds
$PoCl_2$; $PoCl_4$, $PoBr_2$, $PoBr_4$, PoI_2, PoI_4, PoO_2,

4. Atomic radii similar to
Ga, Sb

Predict:

attaches to negatively charged sites of hemoglobin once pulled into the blood stream – (similar to lead)

will move to sites within the body which look for "junk" – liver;

will also have large impact on the kidneys and colon where excretion occurs.

Will be excreted faster than lead (body has Little need for 4+ species)

http://www.webelements.com/webelements/elements/text/Po/eneg.html

Example Calculation

Toxicology of Radioactive Exposure

How much Po remains in body to cause Problems after 24 hours?

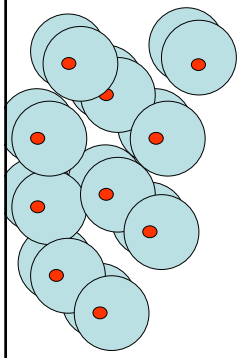
Pb half life = 7-63 days from kidney
5-10 years from bone

Po 50 day ½ life in body

First question: What order is the reaction?

What will happen in our Polonium experiment if we double the amount of Po, present?

$^{210}_{84}Po \rightarrow {}^{4}_{2}He + {}^{206}_{92}Pb$

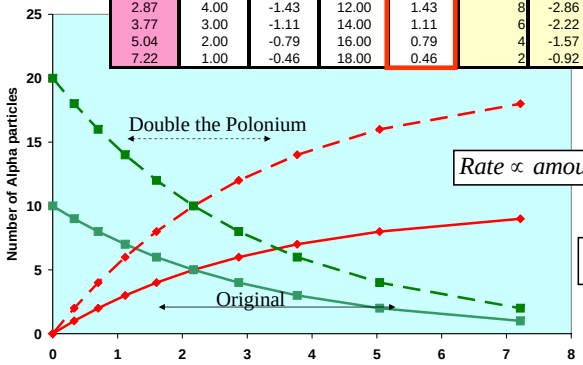


Time, s	total alpha particles	Double Time, s	total alpha particles
0.33	1	0.33	2
0.699	2	0.699	4
1.12	3	1.12	6
1.6	4	1.6	8
2.17	5	2.17	10
2.87	6	2.87	12
3.77	7	3.77	14
5.04	8	5.04	16
7.21	9	7.21	18

What do you observe?

Review Module 14

	Original Polonium				2XPolonium			
t,s	[Po]	average slope	[alpha]	average slope	[Po]	average slope	[alpha]	average slope
0.00	10.00		0.00		20		0	
0.33	9.00	-3.03	2.00	3.03	18	-6.06	2	6.06
0.70	8.00	-2.71	4.00	2.71	16	-5.42	4	5.42
1.12	7.00	-2.39	6.00	2.39	14	-4.78	6	4.78
1.60	6.00	-2.07	8.00	2.07	12	-4.14	8	4.14
2.17	5.00	-1.75	10.00	1.75	10	-3.50	10	3.50
2.87	4.00	-1.43	12.00	1.43	8	-2.86	12	2.86
3.77	3.00	-1.11	14.00	1.11	6	-2.22	14	2.22
5.04	2.00	-0.79	16.00	0.79	4	-1.57	16	1.57
7.22	1.00	-0.46	18.00	0.46	2	-0.92	18	0.92



What do you Observe?

$Rate \propto amount\ of\ polonium$

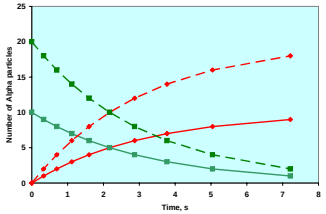
$Rate \propto \left[{}^{210}Po\right]^{1}$

Review Module 14

Visualization

Radioactive Decay Reactions Are ALL 1st Order

$^{210}_{84}Po \xrightarrow{speed} {}^{4}_{2}He + {}^{206}_{92}Pb$



Slope = time dependent

$$rate = \frac{\Delta[\alpha]_{f-i}}{\Delta t_{f-i}} = -\frac{\Delta\left[{}^{210}Po\right]_{f-i}}{\Delta t_{f-i}}$$

$Rate \propto \left[{}^{210}Po\right]^{1}$

$Rate \propto k_T$

Review Module 14

$aA \rightarrow cC + dD$ $\quad rate = k[A]^m$

Order, m	Rate expression	UNITS Of k	$t_{1/2}$	Concentration vs Time	Example
0	$rate = k[A]^0$	$\frac{M}{s}$	$t_{1/2} = \frac{[A_o]}{2k}$	$[A_t] = [A_0] - kt$	$H_2O_{\ell} \rightarrow H_2O_g$
1	$rate = k[A]^1$	$\frac{1}{s}$	$t_{1/2} = \frac{0.693}{k}$	$\ln[A_t] = \ln[A_o] - kt$	$^{210}_{84}Po \rightarrow {}^{4}_{2}He + {}^{206}_{92}Pb$
2	$rate = k[A]^2$	$\frac{1}{Ms}$	$t_{1/2} = \frac{1}{k[A_o]}$	$\frac{1}{[A]} - \frac{1}{[A_0]} = kt$	$2H_3O^+_{aq} \rightarrow H_{2,g} + H_2O$

$rate_{measured} = \frac{\Delta[C]}{c\Delta t} = \frac{\Delta[D]}{d\Delta t}$

$rate_{measured} = -\frac{\Delta[A]}{a\Delta t}$

Review Module 14

Example Calculation

Toxicology of Radioactive Exposure

How much Po remains in body to cause Problems after 24 hours?

The body excretes immediately ~45%
After that body $t_{1/2}$ = 50 days

$$k = \frac{0.693}{t_{\frac{1}{2}}} = \frac{0.693}{(50days)\left(\frac{24hrs}{day}\right)} = \frac{5.775x10^{-4}}{hr}$$

Assume 24 hours is of interest

$$\ln[A_t] = \ln[A_o] - kt$$

$$\ln[A_t] - \ln[A_o] = -kt$$

$$\ln\left(\frac{[A_t]}{[A_o]}\right) = -kt = \left(\frac{-5.775x10^{-4}}{hr}\right)(24hr) = -0.0138$$

$$\left(\frac{[A_t]}{[A_o]}\right) = e^{-0.0138} = 0.986$$

$$[A_t] = 0.986[A_o]$$

Example Calculation **What is the specific activity of Po?**

Chemist (first order reaction)

$^{210}_{84}Po \rightarrow {}^{4}_{2}He + {}^{206}_{92}Pb \qquad t_{1/2} = 138.39days$

$specific\ activity = A = \frac{Bq}{g} = k\left(\frac{N_A}{MM}\right)$

$k = \frac{0.693}{t_{\frac{1}{2}}}$ **Atoms**

g

$$k = \frac{0.693}{t_{\frac{1}{2}}} = \frac{0.693}{(138.39days)\left(\frac{24hrs}{day}\right)\left(\frac{60\,\text{min}}{day}\right)\left(\frac{60s}{\text{min}}\right)} = \frac{5.795x10^{-8}}{s}$$

$$A = \frac{kN}{MM} = \left(\frac{5.795x10^{-8}}{s}\right)\left(\frac{1mol}{209g}\right)\left(\frac{6.022x10^{23}atoms}{mol}\right)\left(\frac{1emission}{1atom}\right) = \frac{1.669x10^{14}emissions}{g_{^{210}Po} \cdot s}$$

Radiation Specialists Express this in Curies or Bequerels

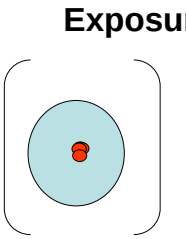
$1Bq = \frac{1emission}{s} \qquad 1Ci = 37{,}000\,MBq$

$$\frac{1.665x10^{14}emissions}{s \cdot g_{^{210}Po}}\left(\frac{1Bq}{1emission}{s}\right)\left(\frac{1Ci}{37{,}000x10^{6}Bq}\right) = \left(\frac{4{,}500Ci}{g}\right)$$

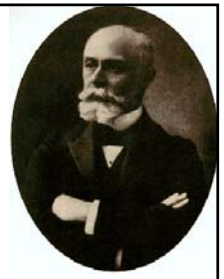
$specific\ activity\ of\ ^{210}Po = \frac{4{,}500Ci}{g}$

Definitions

Toxicology of Radioactive Exposure



Marie Curie
1867-1934



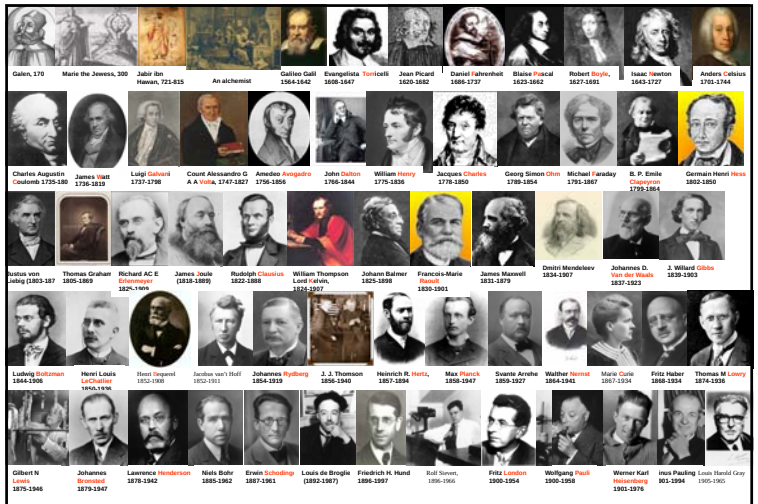
Henri Bequerel
1852-1908, shared the Nobel prize with his students Marie and Pierre Curie

1. **Radiation Source**

a) # emissions/time (Curies vs Bequerels)

$Bq = \frac{emissions}{s}$ — Chemists express this differently

$1Ci = 37GBq = 37x10^9\ Bq$



Fitch Rule G3: Science is Referential

Toxicology of Radioactive Exposure

Energy of emissions

Mass of expelled particle
Kinetic energy of expelled particle

$$E_k = \tfrac{1}{2}mv^2 = 1\frac{kg - m^2}{s^2} \equiv J$$

Expelled Material	Symbol	Mass (kg)	relative mass
Radiation	(	-	
Electron	$	$9.109x10^{-31}$	0.00055
Neutron	n	$1.6749285x10^{-27}$	1.00867
Proton	p	$1.6726231x10^{-27}$	1.00728
Helium nucleus	"	$6.6951032x10^{-27}$	4.0319

Speed of 1 e
Moving in a 1 V
Field is $5.92x10^5$ m/s

$$E_k = \tfrac{1}{2}mv^2 = \tfrac{1}{2}\left(9.109x10^{-31}\,kg\right)\left(5.92x10^5\,\frac{m}{s}\right)^2 = 1.6x10^{-19}\,J$$

What happens if an ejected alpha particle is moving at 1/20 speed of light? (Radon daughter)

$$E_k = \tfrac{1}{2}mv^2 = \tfrac{1}{2}\left(6.695x10^{-27}\,kg\right)\left(\frac{2.99x10^8}{20}\,\frac{m}{s}\right)^2 = 7.48x10^{-13}\,J$$

What is energy on kJ/mole basis?

$$\left(\frac{7.48x10^{-13}\,J}{emission}\right)\left(\frac{6.02x10^{23}\,emissions}{mole\,emissions}\right) = \frac{4.5x10^8\,kJ}{mole}$$

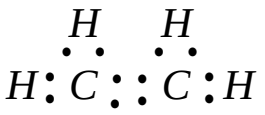
Context Slide # Toxicology of Radioactive Exposure

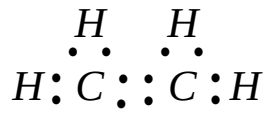
Alpha particles lose energy rapidly
Can not penetrate skin
But, if ingested, can deliver ionizing energy to susceptible tissue
Ionization energy of C atom= 10864. kJ/mol.

$$\frac{4.5x10^8\,kJ}{mole\,alpha} >>>>> \frac{11x10^3\,kJ}{mole\,C}$$

"

Large energy, and large mass of " particle implies
a) Path is linear (linear energy transfer)
b) Can deliver energy to multiple electrons



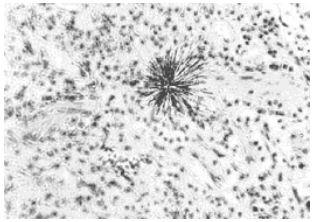


A free radical is left

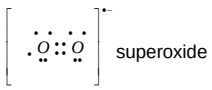
$$C_{([He]2s^2 2p^2)} \xrightarrow{\alpha,\beta,n,p} C_{([He]2s^2 2p^1)}$$

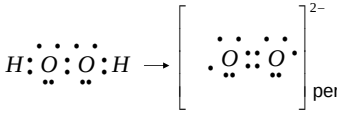
This unstable species attacks other
Electron rich areas, such as DNA strands

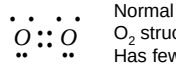
Context Slide



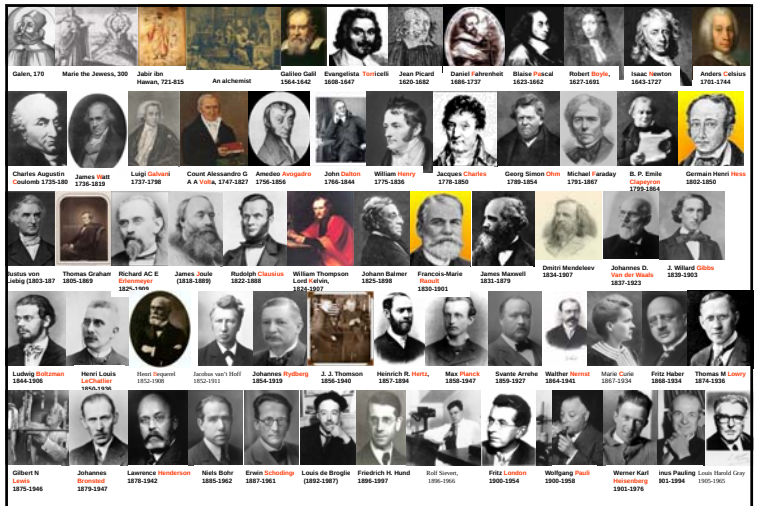
1. Cell death by alpha radiation
2. Linear Energy Transfer (LET) caused by the alpha particle can cause DNA mutation which can exist up to 50 cell cycles, resulting in tumor growth
3. Formation of reactive oxidative species
   a) H_2O_2 hydrogen peroxide
   b O_2^{-2} superoxide
   The O can act as electron donors


superoxide


peroxide


Normal
O_2 structure
Has fewer e

http://enhs.umn.edu/hazards/hazardssite/radon/radonmolaction.html



Fitch Rule G3: Science is Referential

Biological dose

$$1Gy = 1gray = \frac{1J}{kg \cdot tissue}$$

$$Sv = sievert = (1Gy)\left(Q_{uality\ factor\ related\ type\ of\ radiation}\right)\left(N_{factor\ related\ to\ type\ of\ tissue}\right)$$

Radiation Weighting Factor (RWF)
importance of the organ
number of electron acceptors in organ
ability of element to be embedded in organ

Particle	KeV	kJ/mol $\frac{1.602x10^{-19}\ J}{ev}$	Radiation Weighting Factor (RWF)	Organ	N
Photon			1	Bone surface, Skin	0.01
Electron			1	Bladder, brain, breast, kidney, liver	0.05
Neutron	< 10	964,404	5	Colon, lung, stomach	0.12
	>10 -100		10		
	100-2000		20		
	2000-20000		10		
	>20000		5		
Proton	>2000		5		
Alpha			20		

>5 Sv	Risk of death within days or weeks
4.5Sv	Acute exposure
1 Sv	Risk of cancer later in life (5 in 100)
100 mSv	Risk of cancer later in life (5 in 1000)
50 mSv	TLV for annual dose for radiation workers in any one year
20 mSv	TLV for annual average dose, averaged over five years

Biological dose

$$1Gy = 1gray = \frac{1J}{kg \cdot tissue}$$

$$Sv = sievert = (1Gy)\left(Q_{uality\ factor\ related\ type\ of\ radiation}\right)\left(N_{factor\ related\ to\ type\ of\ tissue}\right)$$

$$\left.\frac{Sv}{Bq} = \frac{J \cdot s}{emission}\right|_{type\ of\ emission,\ type\ of\ tissue}$$

← **Established By radiation specialists**

Total power delivered to tissue

To calculate dose:

$$(A)\left(\frac{Sv}{Bq}\right)(g) = Sv$$

$$\left(\frac{emissions}{g \cdot s}\right)\left(\frac{Sv}{\left(\frac{emissions}{s}\right)}\right)(g) = Sv$$

Example Calculate the number of grams of ^{210}Po necessary to achieve a toxic dose of 5 Sv, given that the Sv/Bq for ^{210}Po daughter alpha particle is $2.14x10^{-7}$ Sv/Bq. The half life for the radioactive decay of ^{210}Po is $5.7954x10^{-8}$ 1/s

$$k = \frac{0.693}{t_{\frac{1}{2}}}$$

$$specific\ activity = A = \frac{Bq}{g} = k\left(\frac{N_A}{MM}\right)$$

$$(A)\left(\frac{Sv}{Bq}\right)(g) = Sv$$

We did this about 3 slides ago

$$k = \frac{0.693}{t_{\frac{1}{2}}} = \frac{0.693}{(138.39days)\left(\frac{24hrs}{day}\right)\left(\frac{60\min}{day}\right)\left(\frac{60s}{\min}\right)} = \frac{5.795x10^{-8}}{s}$$

$$A = \frac{kN}{MM} = \left(\frac{5.795x10^{-8}}{s}\right)\left(\frac{1mol}{209g}\right)\left(\frac{6.022x10^{23}atoms}{mol}\right)\left(\frac{1emission}{1atom}\right) = \frac{1.669x10^{14}\,emissions}{g_{^{210}Po} \cdot s}$$

$$\left(\frac{1.699x10^{14}\,Bq}{g_{^{210}Po}}\right)\left(2.14x10^{-7}\,\frac{Sv}{Bq}\right)(g)_{\alpha_{Po}} = 5Sv$$

$$(g)_{\alpha_{Po}} = \frac{5Sv}{\left(\frac{1.699x10^{14}\,Bq}{g_{^{210}Po}}\right)\left(2.14x10^{-7}\,\frac{Sv}{Bq}\right)} = 1.37x10^{9}\,g$$

Context Slide

Source of 210Po

$$^{1}_{0}n + {}^{209}_{83}Bi \rightarrow {}^{210}_{83}Bi \rightarrow {}^{0}_{-1}e + {}^{210}_{84}Po$$

Need a neutron source

Could use same technology as in a nuclear reactor

$$^{238}_{94}Pu \rightarrow {}^{4}_{2}He + {}^{234}_{92}U$$

$$^{4}_{2}He + {}^{9}_{4}Be \rightarrow {}^{12}_{6}C + {}^{1}_{0}n$$

$$^{1}_{0}n + {}^{209}_{83}Bi \rightarrow {}^{210}_{83}Bi \rightarrow {}^{0}_{-1}e + {}^{210}_{84}Po$$

Context Slide

Could he have been saved?
–or how could you remove Po? Or Pb? Will pick up this topic in about three chapters

Alexander Litvinenko, former Russian KGB agent poisoned with Polonium on Nov. 1, died Nov. 23, 2006

Cost of the poison. From Oak Ridge National Labs

$$\left(\frac{\$3200}{\mu Ci}\right)\left(\frac{1\mu Ci}{10^{-6}Ci}\right)\left(\frac{1Ci}{37x10^{9}}\right)\left(\frac{1.66x10^{14}\,Bq}{g}\right)141x10^{-9}\,g = \$2\,Million U.S.$$

From Scientific Supply:

$$\left(\frac{\$69.00}{0.1\mu Ci}\right)\left(\frac{1\mu Ci}{10^{-6}Ci}\right)\left(\frac{1Ci}{37x10^{9}}\right)\left(\frac{1.66x10^{14}\,Bq}{g}\right)141x10^{-9}\,g = \$4.3\,Million U.S.$$

Somebody REALLY wanted Mr. Litvinenko dead

FOR the exam

Read Chapter 19.1, 19.2, p. 523 Of Masterton and Hurley,
Problems: 13-29 of (Chapter 19)

Or Read Brown et al 21.1 to 21.6; And then 21.9

Know: alpha beta particles; neutrons
Know: their relative physiologic effect rationalized from
a) mass
b) kinetic energy $E_k = \frac{1}{2}mv^2 = 1\frac{kg - m^2}{s^2} \equiv J$

Know that radioactive decay reactions are 1st order

$$rate = k[A]^1 \qquad \ln[A_t] = \ln[A_o] - kt \qquad k = \frac{0.693}{t_{\frac{1}{2}}}$$

a) calculate time to a specific decayed product
b) specific activity given the half life

$$specific\ activity = A = \frac{Bq}{g} = k\left(\frac{N_A}{MM}\right)$$

c) toxic dose if given Sv/Bq, knowing 5Sv is lethal

$$(A)\left(\frac{Sv}{Bq}\right)(g) = Sv$$

"A" students work (without solutions manual)
~ 10 problems/night.

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Module #15
Applied Kinetics

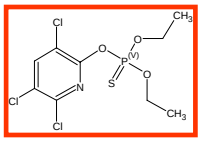
Example 4: Drugs and Environmental Contaminants

Example 1

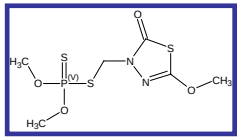
The Pesticide Manual, 8th Ed.

Compound	Use	mg/kg LD_{50}(rats)	$t_{1/2}$
Chlorpyrifos	Insecticide	135	1.5-100d
Chlorfenvinphos	Insecticide	9.7	1.3-700hr
Dioxacarb	Cockroaches	72	85hr
Formothion	aphids	365	≥1d
Methidathion	sucking bugs	25	30min

LD_{50}= Lethal Dose which kills 50% of test subjects



What might account For difference in t½?



Possible trade war with Europe over
REACH (Registration Evaluation and Authorization of Chemicals)
Based on the "precautionary principle" not "risk assessment"
Will regulate based on half lives, not on "risk"
"Risk" supposedly balances "projected harm" vs economic benefit

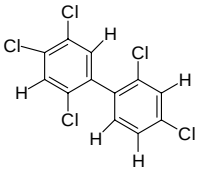
	Persistent	Very Persistent	
Marine water	60d	>60d	
Fresh, estuarine water	40d	>60d	2m-1y PCB
Marine sediment	180d	>180d	
Fresh,estuarine sediment	120d	>180d	
Soil	120d	>180d	

Bottom line: does not consider "real" harm
does not consider economic benefit
Based on idea: world ecosystem is too complex
to predict, therefore err on side of caution

Example 3

PCB: polychlorinated biphenyl
one of 209 congeners

Manufactured 1929-1977
Peak production 100,000 tons, 1970



Excellent properties as a power transformer coolant
A. low vapor pressure (WHY?) (Henry's Law)
doesn't build up and explode
B. Non-conducting (WHY?)
C. Chemically stable (WHY?)
good for manufacturing

Bad for environment
a. Chemically stable (long $t_{1/2}$)
b. Soluble in water? Why? How much?
More soluble in non-water (lipids, fats, body tissues)
bioaccumulators

Bind to the Ah receptor which is present in all mammalian species. This receptor interacts with cell's DNA, one effect of which is to induce cytochrome P450 enzyme

c. Long $t_{1/2}$ in estuarine sediments =solubility in black organic muck

Chicago Tribune Jan. 9, 2004

Pacific Salmon Farming

Chicago Tribune
Jan. 9, 2004

Original *Science* article indicates that there is a 10 year half life for PCBs in tissue

"A" students work (without solutions manual)
~ 10 problems/night.

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Module #15
Kinetics Applied to Biology

END

Context Slide

Toxicology of Radioactive Exposure

$$k = \left[\frac{0.693}{t_{\frac{1}{2}}}\right]$$

A toxic scale

$$\left[\frac{Sv}{Bq}\right]\left[\frac{Bq}{g}\right] = \frac{Sv}{g}$$

Atom	Mass	t1/2	Unit	k (s-1)	A Bq/g	daughter particle	Sv/Bq	5 Sv, g lethal dose	Sv/g
U	238	45,000,000,000	yr	4.88331E-19	1235.6	alpha	2.58E-08	1.57E+05	3.19E-05
Th	234	24	day	3.34201E-07	8.6E+14	beta	5.30E-09	1.10E-06	4.56E+06
U	234	250,000	yr	8.78995E-14	2.26E+08	alpha	2.82E-08	7.84E-01	6.38E+00
Th	230	80,000	yr	3.78341E-13	9.91E+08	alpha	7.75E-08	6.51E-02	7.68E+01
Ra	226	1,600	yr	1.37343E-11	3.66E+10	alpha	2.25E-07	6.07E-04	8.23E+03
Pb	214	27	min	0.00043097	1.21E+18	beta	1.54E-10	2.68E-08	1.87E+08
Bi	214	20	min	0.000580402	1.63E+18	beta	1.07E-10	2.86E-08	1.75E+08
Pb	210	22	yr	9.85421E-10	2.83E+12	beta	8.02E-07	2.21E-06	2.27E+06
Bi	210	5	day	1.60096E-06	4.59E+15	beta	[illegible]	[illegible]	8.86E+06
Po	210	138	day	5.7954E-08	1.66E+14	alpha	2.14E-07	1.41E-07	3.56E+07

Given the information In the table calculate The lethal dose of Po

$$A = \frac{Bq}{g} = kN$$

$$= k\left[\frac{mole}{g}\right]\left[\frac{6.022x10^{23}\,atoms}{mole}\right]$$

Established by Radiation specialists

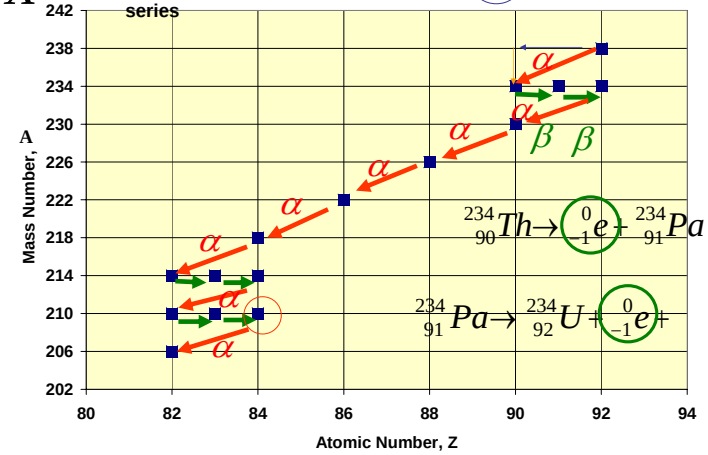
5 Sv, is a lethal dose

$$(5Sv)\left(\frac{Bq}{2.14x10^{-7}\,Sv}\right)\left(\frac{1g}{166x10^{12}\,Bq}\right) = 1.4x10^{-7}\,g = 140ng$$

$^{A}_{Z}X$

Look at Biological Assessment of the 238-U series

$^{238}_{92}U \rightarrow {}^{4}_{2}He + {}^{234}_{90}Th$



$^{234}_{90}Th \rightarrow {}^{0}_{-1}e + {}^{234}_{91}Pa$

$^{234}_{91}Pa \rightarrow {}^{234}_{92}U + {}^{0}_{-1}e +$

$^{210}_{84}Po \rightarrow {}^{4}_{2}He + {}^{206}_{92}Pb$

Context Slide

Toxicology of Radioactive Exposure

$$(A)\left(\frac{Sv}{Bq}\right)(g) = Sv \qquad k = \left[\frac{0.693}{t_{\frac{1}{2}}}\right]$$

Atom	Mass	t1/2	Unit	k (s-1)	A Bq/g	daughter particle	Sv/Bq	5 Sv, g lethal dose
U	238	45,000,000,000	yr	4.88331E-19	1235.6	alpha	2.58E-08	1.57E+05
Th	234	24	day	3.34201E-07	8.6E+14	beta	5.30E-09	1.10E-06
U	234	250,000	yr	8.78995E-14	2.26E+08	alpha	2.82E-08	7.84E-01
Th	230	80,000	yr	3.78341E-13	9.91E+08	alpha	7.75E-08	6.51E-02
Ra	226	1,600	yr	1.37343E-11	3.66E+10	alpha	2.25E-07	6.07E-04
Pb	214	27	min	0.00043097	1.21E+18	beta	1.54E-10	2.68E-08
Bi	214	20	min	0.000580402	1.63E+18	beta	1.07E-10	2.86E-08
Pb	210	22	yr	9.85421E-10	2.83E+12	beta	8.02E-07	2.21E-06
Bi	210	5	day	1.60096E-06	4.59E+15	beta	1.93E-09	5.64E-07
Po	210	138	day	5.7954E-08	1.66E+14	alpha	2.14E-07	1.41E-07

Given the information
In the table calculate
The lethal dose of Po

$$A = \frac{Bq}{g} = \frac{kN_A}{MM}$$

$$= k\left[\frac{mole}{g}\right]\left[\frac{6.022x10^{23}\,atoms}{mole}\right]$$

Established by Radiation specialists

5 Sv, is a lethal dose

$$(5Sv)\left(\frac{Bq}{2.14x10^{-7}\,Sv}\right)\left(\frac{1g}{166x10^{12}\,Bq}\right) = 1.4x10^{-7}\,g = 140ng$$

Element	Ionization Energy kJ/mol	Calc. atomic radii pm	Radii atomic pm	Ion	Ionic radii Pm octahedral
O	1313.9	48	60	O(-II)	22
S	999.6	88	100	S(+IV)	51
Se	941	103	115	Se(+IV)	64
Te	869.3	123	140	Te(+IV)	111
Po	812	135	190	Po(IV)	108

Most similar atomic Radii: diagonal rule
Ga= 136pm
Sb=133pm

Not atoms used by body

Can skip this