ΑΡΧΕΣ ΧΗΜΕΙΑΣ

20. Πυρηνική Χημεία

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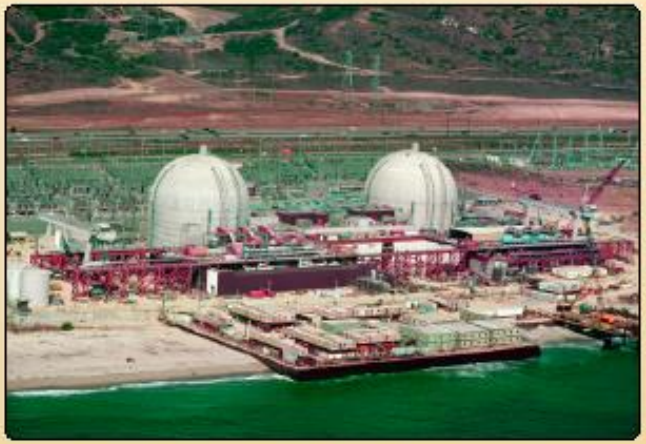
Γραφείο: Γ206

Πυρηνική Χημεία



Atomic power plants supply about 20% of the electricity generated in the United States. (Joe Azzara/Getty/The Image Bank)

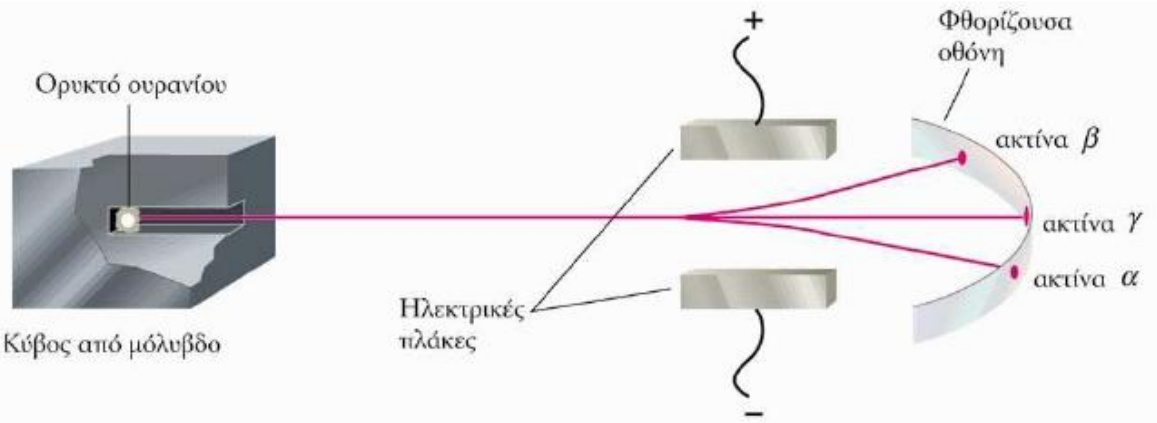
A patient inhales radioactive xenon, which is taken up and carried by the bloodstream throughout the body. The helmet on the patient's head detects gamma rays from the decay, providing a visualization of blood flow in the brain. (Will and Deni McIntyre/Photo Researchers, Inc.)

Treating foods with radiation kills pathogens and makes food safer. (MDS Nordian)

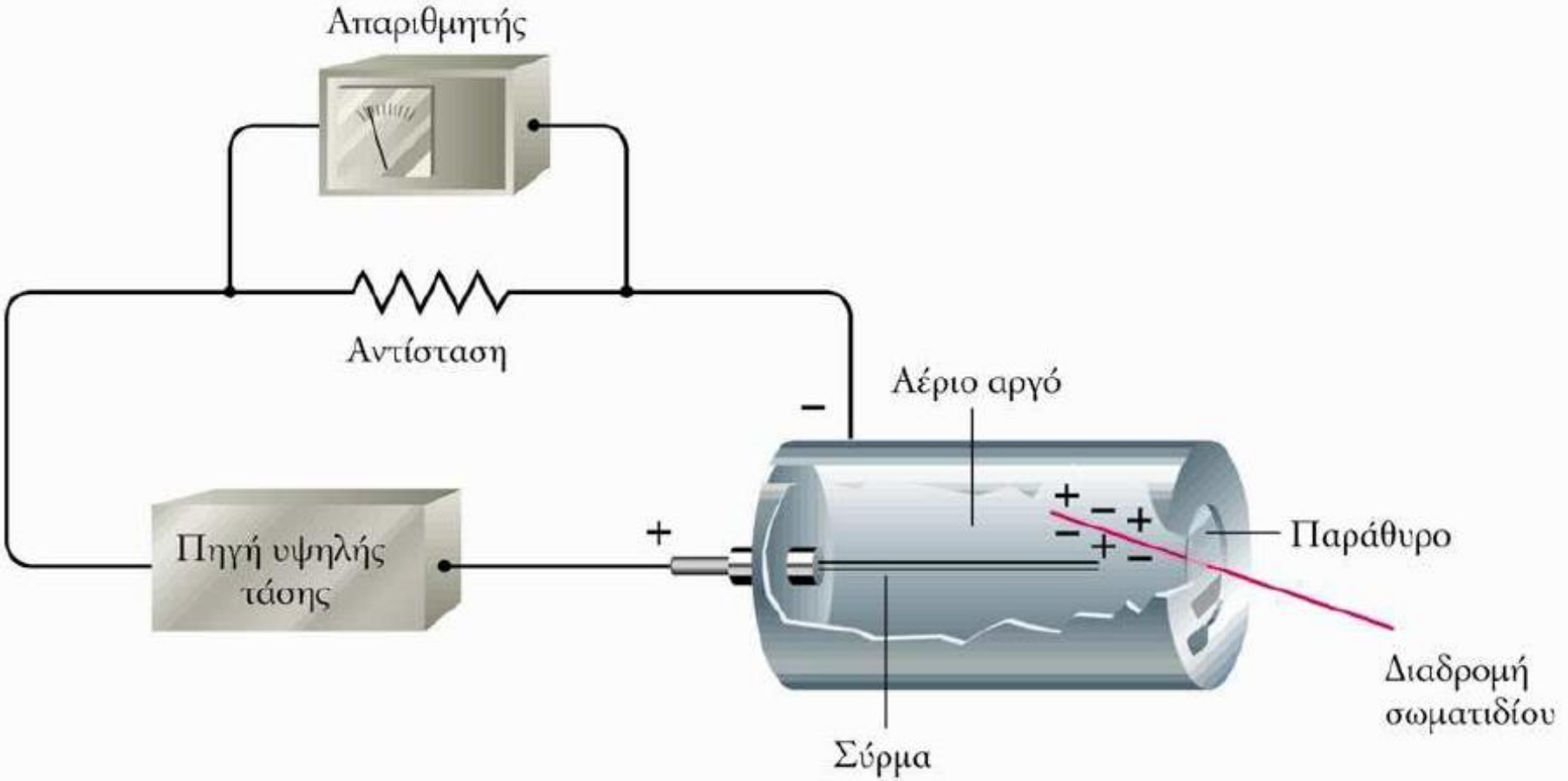
A household smoke detector uses radioactive americium-241. This alpha emitter has a half-life of 470 years. In a smoke detector the emission ionizes smoke particles to activate the alarm. (Charles D. Winters)

Ραδιενέργεια

- Το φαινόμενο της ραδιενέργειας ανακαλύφθηκε από τον **Antoine Henri Becquerel** το 1896
- Η **Marie Curie** (1876-1934) μαθήτρια του Antoine Becquerel (Nobel στη Φυσική το 1903) εντελώς τυχαία παρατήρησε ότι μια ένωση του ουρανίου προκαλούσε μαύρισμα σε φωτογραφικές πλάκες
- Η Marie Curie πρότεινε το όνομα ραδιενέργεια (η αυθόρμητη διάσπαση ενός ατόμου με εκπομπή σωματιδίων και/ή ακτινοβολίας)



Απαριθμητής Geiger-Muller



Σύγκριση μεταξύ Πυρηνικών και Χημικών αντιδράσεων

- Οι πυρηνικές αντιδράσεις αφορούν τον πυρήνα.
- Ο πυρήνας ανοίγει, και τα πρωτόνια και τα νετρόνια αναδιατάσσονται.
- Το άνοιγμα του πυρήνα απελευθερώνει ένα τεράστιο ποσό ενέργειας που κρατά τον πυρήνα μαζί - που ονομάζεται ενέργεια σύνδεσης (Binding Energy).
- Οι "Κανονικές" Χημικές Αντιδράσεις περιλαμβάνουν ηλεκτρόνια, όχι πρωτόνια και νετρόνια.

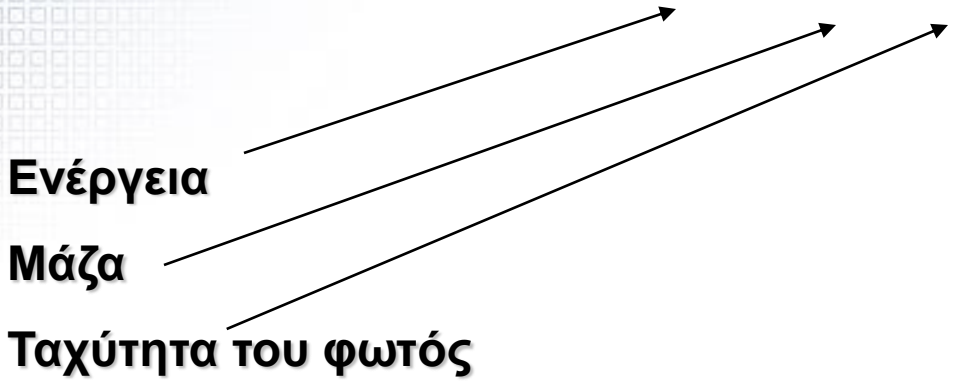
Σύγκριση μεταξύ Πυρηνικών και Χημικών αντιδράσεων

	Χημικές αντιδράσεις	Πυρηνικές αντιδράσεις
1.	Τα άτομα διευθετούνται με τη διάσπαση και τη δημιουργία χημικών δεσμών.	Τα στοιχεία (ή τα ισότοπα τους) μετατρέπονται μεταξύ τους.
2.	Μόνο ηλεκτρόνια σε ατομικά ή μοριακά τροχιακά σχετίζονται με τη διάσπαση και τη δημιουργία δεσμών.	Πρωτόνια, νετρόνια, ηλεκτρόνια, και άλλα στοιχειώδη σωματίδια μπορεί σχετίζονται.
3.	Οι αντιδράσεις συνοδεύονται από την απορρόφηση ή απελευθέρωση σχετικά μικρών ποσών ενέργειας.	Οι αντιδράσεις συνοδεύονται από την απορρόφηση ή απελευθέρωση τεράστιων ποσών ενέργειας.
4.	Οι ταχύτητες των αντιδράσεων επηρεάζονται από τη θερμοκρασία, πίεση, συγκέντρωση και καταλύτες.	Οι ταχύτητες των αντιδράσεων δεν επηρεάζονται από τη θερμοκρασία, πίεση, συγκέντρωση και καταλύτες.

Έλλειμμα μάζας

- Μέρος της μάζας μπορεί να μετατραπεί σε ενέργεια όπως φαίνεται από τη διάσημη εξίσωση!

$$E=mc^2$$



Πυρηνικά σωματίδια και τύποι ακτινοβολίας

- Ακτίνες α – ${}^{4}_{2}He$
- Ακτίνες β – ${}^{0}_{-1}e$
- Ακτίνες γ – καθαρή ενέργεια ${}^{0}_{0}\gamma$
- Νετρόνιο – ${}^{1}_{0}n$
- Ποζιτρόνιο – ${}^{0}_{+1}e$
- Πρωτόνιο – ${}^{1}_{1}H$

Τύποι ραδιενεργής διάσπασης

ΠΙΝΑΚΑΣ 20.2

Τύποι ραδιενεργού διάσπασης

Τύποι διάσπασης	Ακτινοβολία	Ισοδύναμη διαδικασία	Απορρέουσα μεταβολή του πυρήνα		Συνήθης κατάσταση πυρήνα
			Ατομικός αριθμός	Μαζικός αριθμός	
Εκπομπή άλφα (α)	${}_{2}^{4}\mathrm{He}$	—	−2	−4	$Z > 83$
Εκπομπή βήτα (β)	${}_{-1}^{0}\mathrm{e}$	${}_{0}^{1}\mathrm{n} \longrightarrow {}_{1}^{1}\mathrm{p} + {}_{-1}^{0}\mathrm{e}$	+1	0	N/Z πολύ μεγάλο
Εκπομπή ποζιτρονίου (β^+)	${}_{1}^{0}\mathrm{e}$	${}_{1}^{1}\mathrm{p} \longrightarrow {}_{0}^{1}\mathrm{n} + {}_{1}^{0}\mathrm{e}$	−1	0	N/Z πολύ μικρό
Σύλληψη ηλεκτρονίου (EC)	ακτίνες X	${}_{1}^{1}\mathrm{p} + {}_{-1}^{0}\mathrm{e} \longrightarrow {}_{0}^{1}\mathrm{n}$	−1	0	N/Z πολύ μικρό
Εκπομπή γάμμα (γ)	${}_{0}^{0}\gamma$	—	0	0	Διεγερμένη

Πυρηνικές αντιδράσεις
Balancing Nuclear Equations

1. Διατήρηση μαζικού αριθμού (A).

The sum of protons + neutrons in the products must equal the sum of protons + neutrons in the reactants.

$$^{235}_{92}U + ^{1}_{0}n \longrightarrow ^{138}_{55}Cs + ^{96}_{37}Rb + 2\ ^{1}_{0}n$$

235 + 1 = 138 + 96 + 2x1

2. Διατήρηση ατομικού αριθμού (Z) ή πυρηνικού φορτίου.

The sum of nuclear charges in the products must equal the sum of nuclear charges in the reactants.

$$^{235}_{92}U + ^{1}_{0}n \longrightarrow ^{138}_{55}Cs + ^{96}_{37}Rb + 2\ ^{1}_{0}n$$

92 + 0 = 55 + 37 + 2x0

Το ^{212}Po διασπάται με εκπομπή α-σωματιδίων.

α σωματίδιο - $^{4}_{2}He$ ή $^{4}_{2}\alpha$

$$^{212}_{84}Po \longrightarrow {}^{4}_{2}He + {}^{A}_{Z}X$$

$212 = 4 + A$ $\qquad$ $A = 208$

$84 = 2 + Z$ $\qquad$ $Z = 82$

$$^{212}_{84}Po \longrightarrow {}^{4}_{2}He + {}^{208}_{82}Pb$$

Τύποι ραδιενεργής διάσπασης

Εκπομπή β σωματιδίων

$^{14}_{6}C \longrightarrow {}^{14}_{7}N + {}^{0}_{-1}e + \bar{\nu}$ Μείωση # νετρονίων κατά 1

$^{40}_{19}K \longrightarrow {}^{40}_{20}Ca + {}^{0}_{-1}e + \bar{\nu}$ Αύξηση # πρωτονίων κατά 1

$$^{1}_{0}n \longrightarrow {}^{1}_{1}p + {}^{0}_{-1}e + \bar{\nu}$$

Εκπομπή ποζιτρονίων

$^{11}_{6}C \longrightarrow {}^{11}_{5}B + {}^{0}_{+1}e + \nu$ Αύξηση # νετρονίων κατά 1

$^{38}_{19}K \longrightarrow {}^{38}_{18}Ar + {}^{0}_{+1}e + \nu$ Μείωση # πρωτονίων κατά 1

$$^{1}_{1}p \longrightarrow {}^{1}_{0}n + {}^{0}_{+1}e + \nu$$

Τα ν και $\bar{\nu}=1/\lambda=c/\nu$ έχουν A = 0 και Z = 0

όπου c=ταχύτ. του φωτός

Τύποι ραδιενεργής διάσπασης

Σύλληψη ηλεκτρονίου

$^{37}_{18}Ar + {}^{0}_{-1}e \longrightarrow {}^{37}_{17}Cl + \nu$ Αύξηση # νετρονίων κατά 1

$^{55}_{26}Fe + {}^{0}_{-1}e \longrightarrow {}^{55}_{25}Mn + \nu$ Μείωση # πρωτονίων κατά 1

$$^{1}_{1}p + {}^{0}_{-1}e \longrightarrow {}^{1}_{0}n + \nu$$

Εκπομπή α σωματιδίων

$^{212}_{84}Po \longrightarrow {}^{4}_{2}He + {}^{208}_{82}Pb$

Μείωση # νετρονίων κατά 2

Μείωση # πρωτονίων κατά 2

Αυθόρμητη σχάση - Spontaneous fission

$^{252}_{98}Cf \longrightarrow 2{}^{125}_{49}In + 2{}^{1}_{0}n$

Παράδειγμα

Ποιο ραδιενεργό ισότοπο παράγεται στο βομβαρδισμό του βορίου;

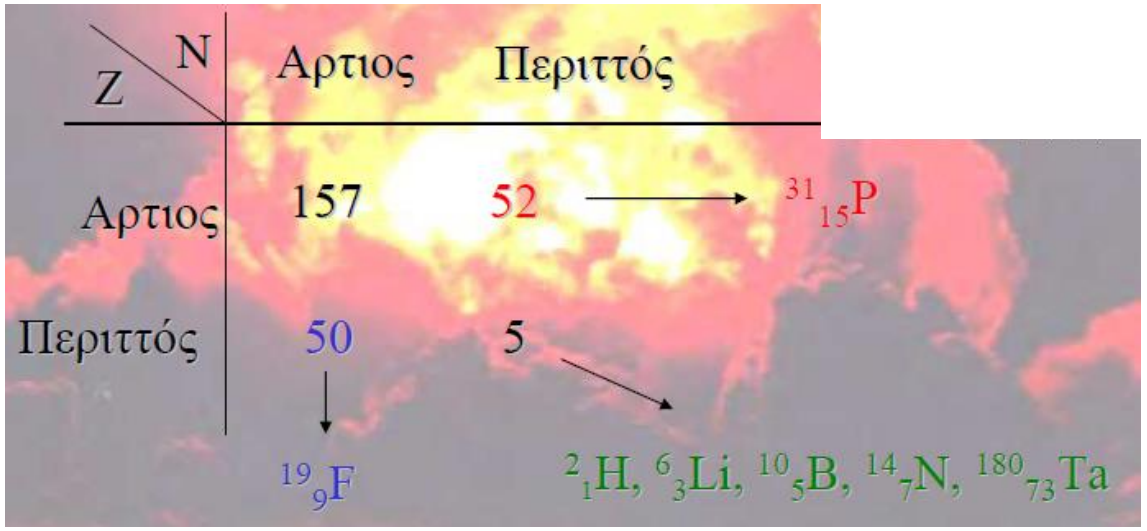
$$^{10}_{5}B + ^{4}_{2}He \longrightarrow ? + ^{1}_{0}n$$

Παράδειγμα

Ποιο ραδιενεργό ισότοπο παράγεται στο βομβαρδισμό του βορίου;

$$^{10}_{5}B + {}^{4}_{2}He \longrightarrow {}^{13}_{7}N + {}^{1}_{0}n$$

Πυρηνική σταθερότητα



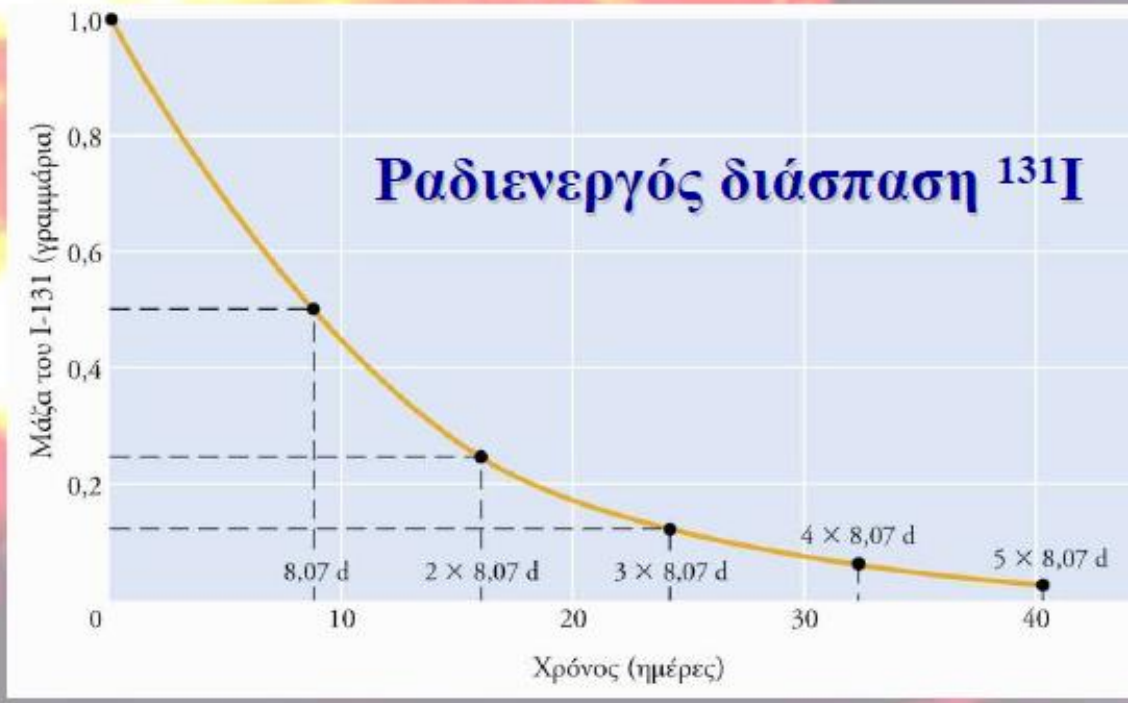
ΠΙΝΑΚΑΣ 20.1

Αριθμός σταθερών ισοτόπων με άρτιους και περιττούς αριθμούς πρωτονίων και νετρονίων

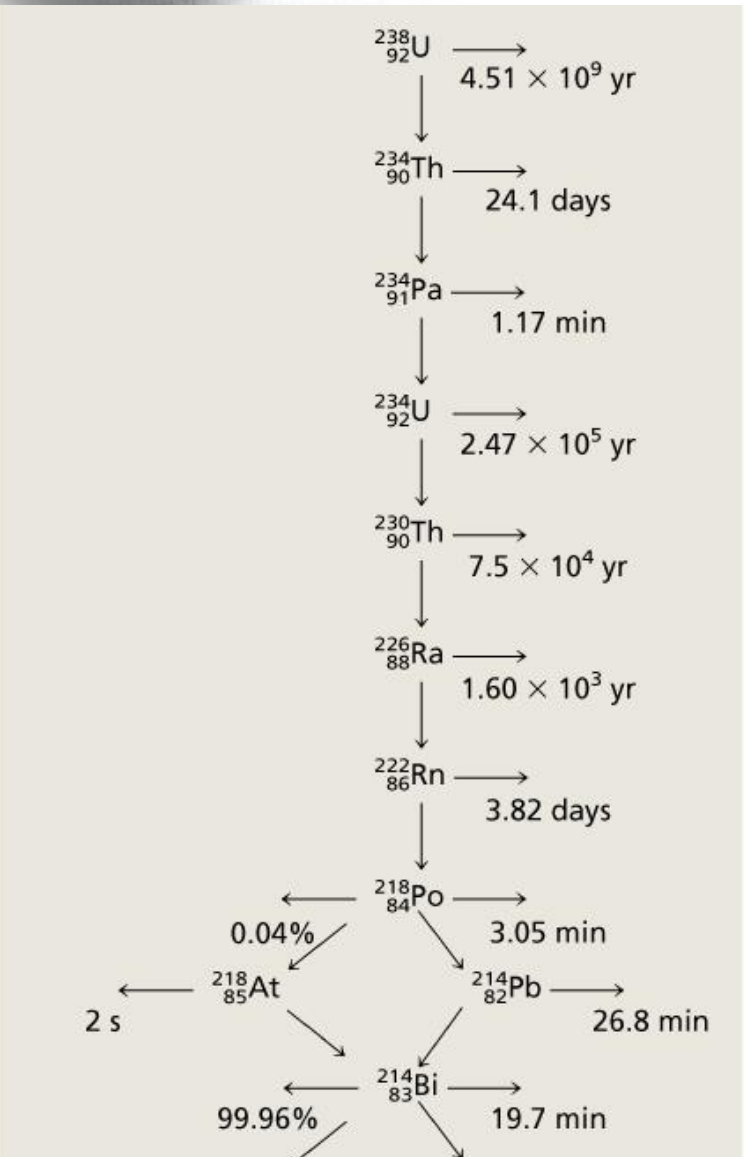
	Αριθμός σταθερών ισοτόπων			
	157	52	50	5
Αριθμός πρωτονίων	Άρτιος	Άρτιος	Περιττός	Περιττός
Αριθμός νετρονίων	Άρτιος	Περιττός	Άρτιος	Περιττός

Χρόνος ημιζωής

- **Χρόνος ημιζωής** είναι ο χρόνος που απαιτείται για να διασπαστούν οι μισοί πυρήνες σε ένα δείγμα.
- Η ταχύτητα της διάσπασης εξαρτάται μόνο από τη συγκέντρωση του ραδιενεργού δείγματος.



Κινητική ραδιενεργής διάσπασης



$$N \rightarrow \Pi$$

$$\text{ταχύτητα} = -\frac{\Delta N}{\Delta t}$$

$$N = N_0 e^{(-kt)}$$

$$\ln N = \ln N_0 - kt$$

Π = προϊόντα

N = άτομα τη χρονική στιγμή *t*

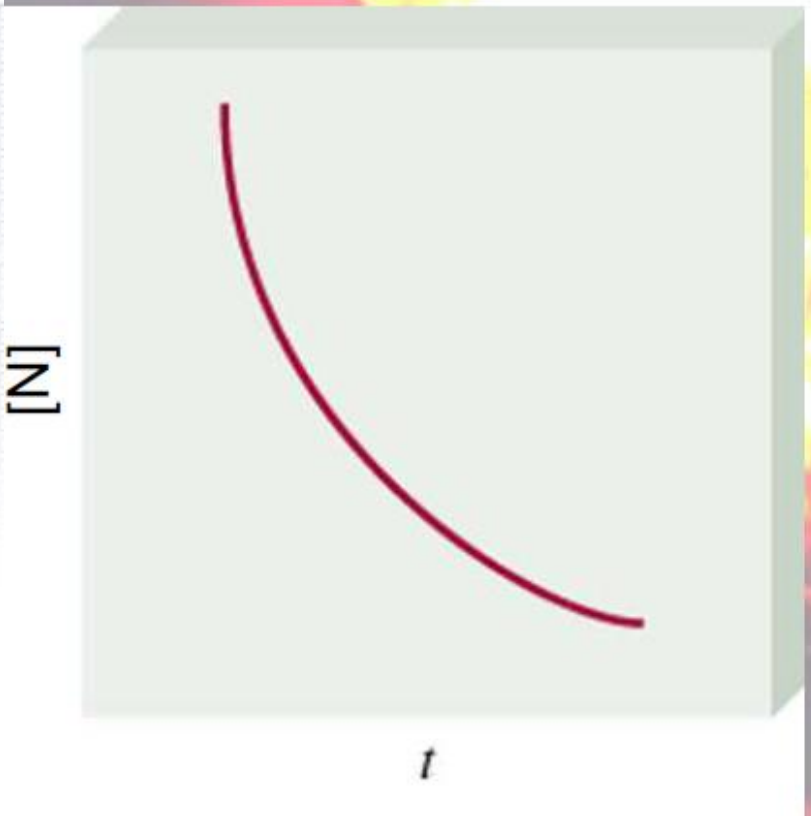
N_0 = άτομα τη χρονική στιγμή *t = 0*

k είναι η σταθερά ταχύτητας

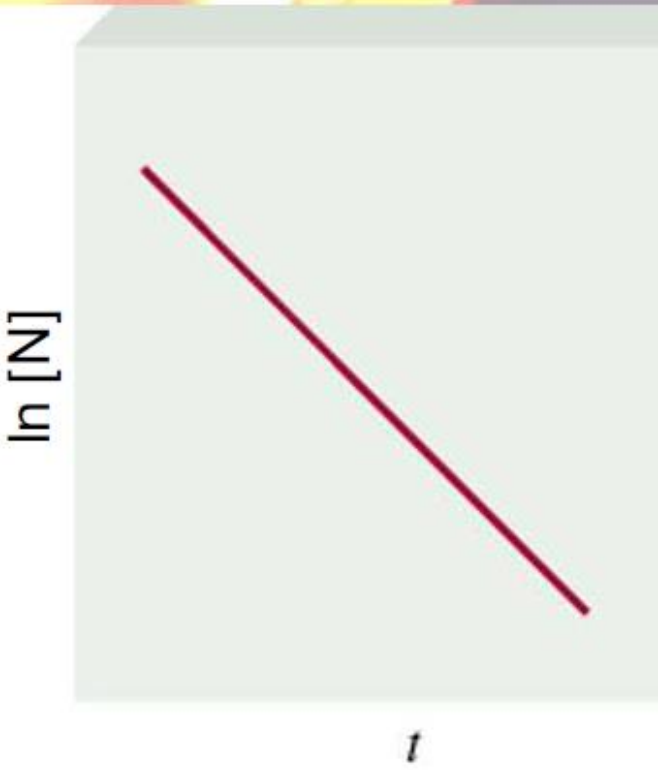
$$t_{½} = \frac{\text{Ln } 2}{k} \qquad t_{½} = \frac{0.693}{k}$$

Κινητική ραδιενεργής διάσπασης

$[N] = [N]_0 \exp(-kt)$



$\ln[N] = \ln[N]_0 - kt$



Ραδιοχρονολόγηση – Radiocarbon Dating

- Ο ραδιενεργός ^{14}C σχηματίζεται στην ανώτερη ατμόσφαιρα από τις πυρηνικές αντιδράσεις που προκαλούνται από τα νετρόνια της κοσμικής ακτινοβολίας.

$$^{14}_{7}N + ^{1}_{o}n \rightarrow ^{14}_{6}C + ^{1}_{1}H$$

- Ο ^{14}C οξειδώνεται προς CO_2, που κυκλοφορεί στην ατμόσφαιρα, οπότε οι ζώντες οργανισμοί διατηρούν μια σταθερή αναλογία ^{14}C / ^{12}C.
- Όταν όμως ένας οργανισμός πεθάνει, ο ^{14}C δεν αναπληρώνεται.
- Ο ^{14}C συνεχίζει να διασπάται με $t_{1/2}$ = 5730 χρόνια

$$^{14}_{6}C + ^{1}_{o}n \rightarrow ^{14}_{7}N + ^{0}_{-1}e$$

- Η ραδιενέργεια του δείγματος μπορεί να χρησιμοποιηθεί για να γίνει ραδιοχρονολόγηση.

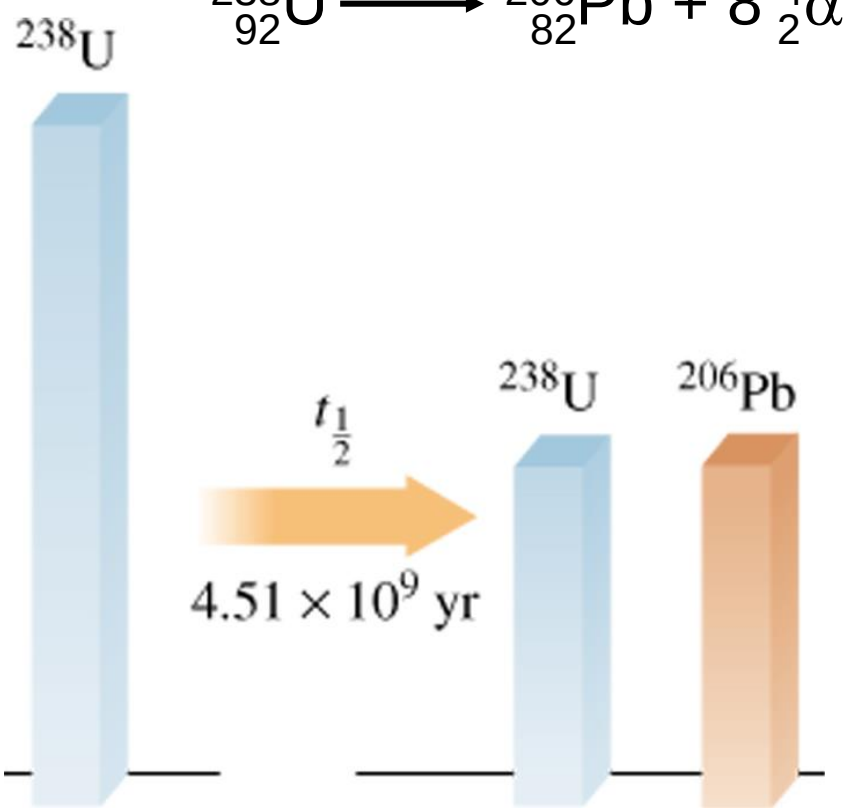
Ραδιοχρονολόγηση με $^{14}_{6}C$ - Radiocarbon Dating

$$^{14}_{7}N + ^{1}_{0}n \longrightarrow ^{14}_{6}C + ^{1}_{1}H$$

$$^{14}_{6}C \longrightarrow ^{14}_{7}N + ^{0}_{-1}e \qquad t_{½} = 5730 \text{ χρόνια}$$

Ραδιοχρονολόγηση με $^{238}_{92}U$ - Uranium-238 Dating

$$^{238}_{92}U \longrightarrow ^{206}_{82}Pb + 8\ ^{4}_{2}\alpha + 6\ ^{0}_{-1}e \qquad t_{½} = 4.51 \times 10^{9} \text{ χρόνια}$$



Ραδιοχρονολόγηση

- **Radiocarbon dating in 1988 by three independent teams of scientists yielded results published in *Nature** indicating that the shroud was made during the Middle Ages, approximately 1300 years after Jesus lived.**

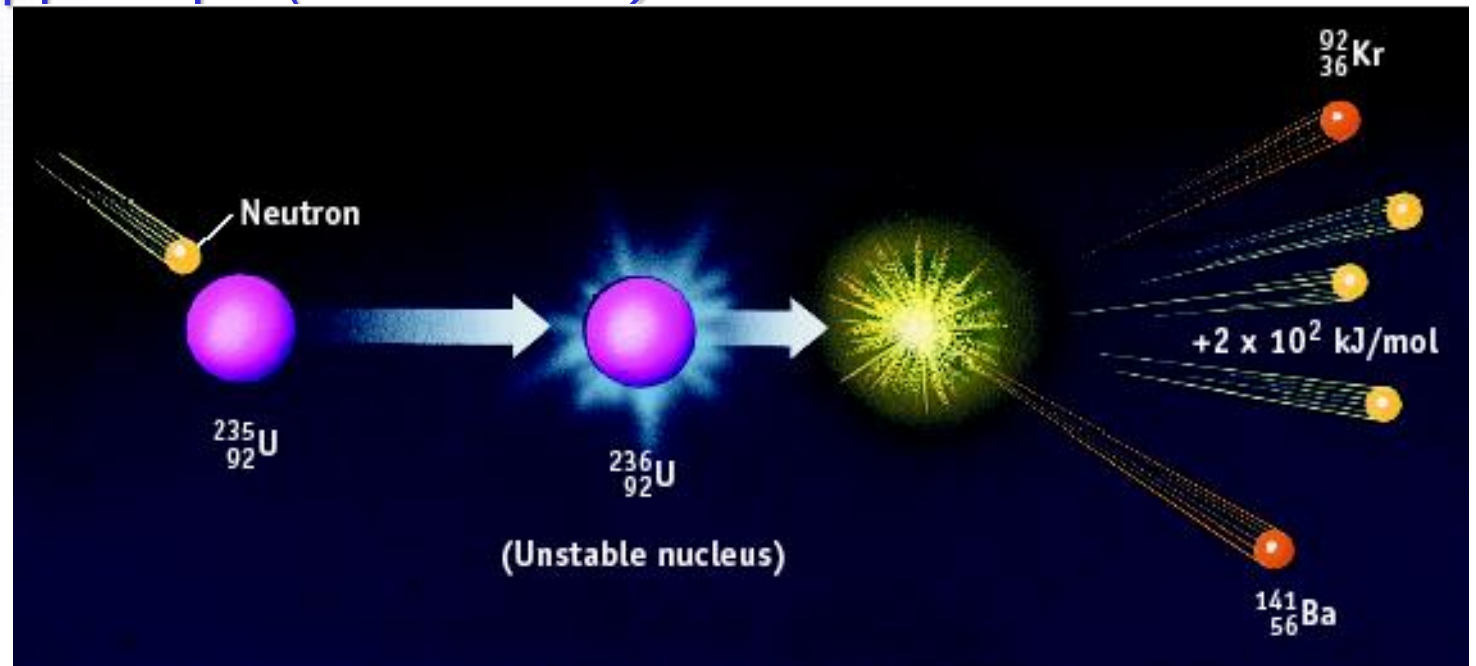
*Damon, P. E. et. al. (1989). "Radiocarbon dating of the Shroud of Turin" *Nature* 337 (6208): 611–615.

Πυρηνική σχάση - Nuclear Fission

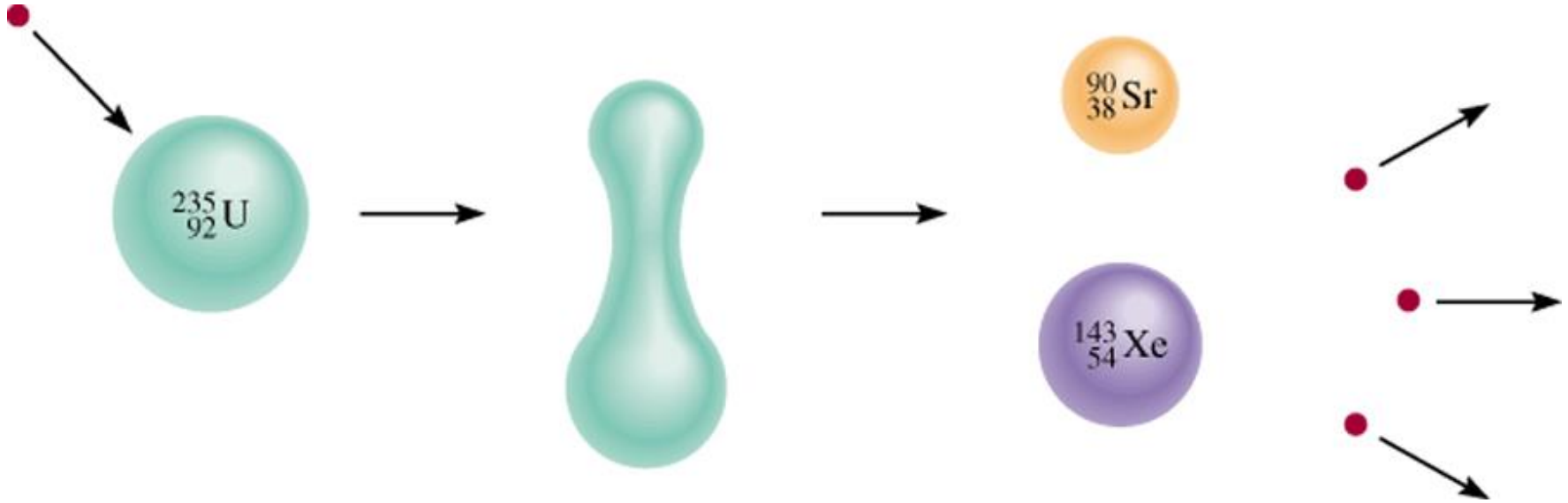
Σχάση είναι η διάσπαση των ατόμων

Διακρίνουμε 3 βασικά στάδια:

1. Εκκίνηση (*Initiation*). (π.χ., ^{235}U + neutron)
2. Διάδοση (*Propagation*). (Η σχάση του ^{236}U απελευθερώνει νετρόνια που ξεκινούν νέες σχάσεις)
3. Τερματισμό (*Termination*).



Πυρηνική σχάση - Nuclear Fission



$$^{235}_{92}U + {}^{1}_{0}n \longrightarrow {}^{90}_{38}Sr + {}^{143}_{54}Xe + 3{}^{1}_{0}n + \text{Ενέργεια}$$

Ενέργεια = [μάζα ^{235}U + μάζα n – (μάζα ^{90}Sr + μάζα ^{143}Xe + 3 x μάζα n)] x c^2

Ενέργεια = 3.3 x 10^{-11} J / ^{235}U

= 2.0 x 10^{13} J / mole ^{235}U

Καύση 1 τον λιθάνθρακα = 5 x 10^{7} J

Πυρηνική ενέργεια σύνδεσης (Nuclear binding energy-BE) είναι το απαιτούμενο ποσό ενέργειας για τη διάσπαση ενός πυρήνα σε πρωτόνια και νετρόνια.

$$BE + {}^{19}_{9}F \longrightarrow 9\,{}^{1}_{1}p + 10\,{}^{1}_{0}n$$

$$E = mc^2$$

$$BE = 9 \times (\text{μάζα p}) + 10 \times (\text{μάζα n}) - \text{μάζα } {}^{19}F$$

$$BE\ (amu) = 9 \times 1.007825 + 10 \times 1.008665 - 18.9984$$

$$BE = 0.1587\ amu$$

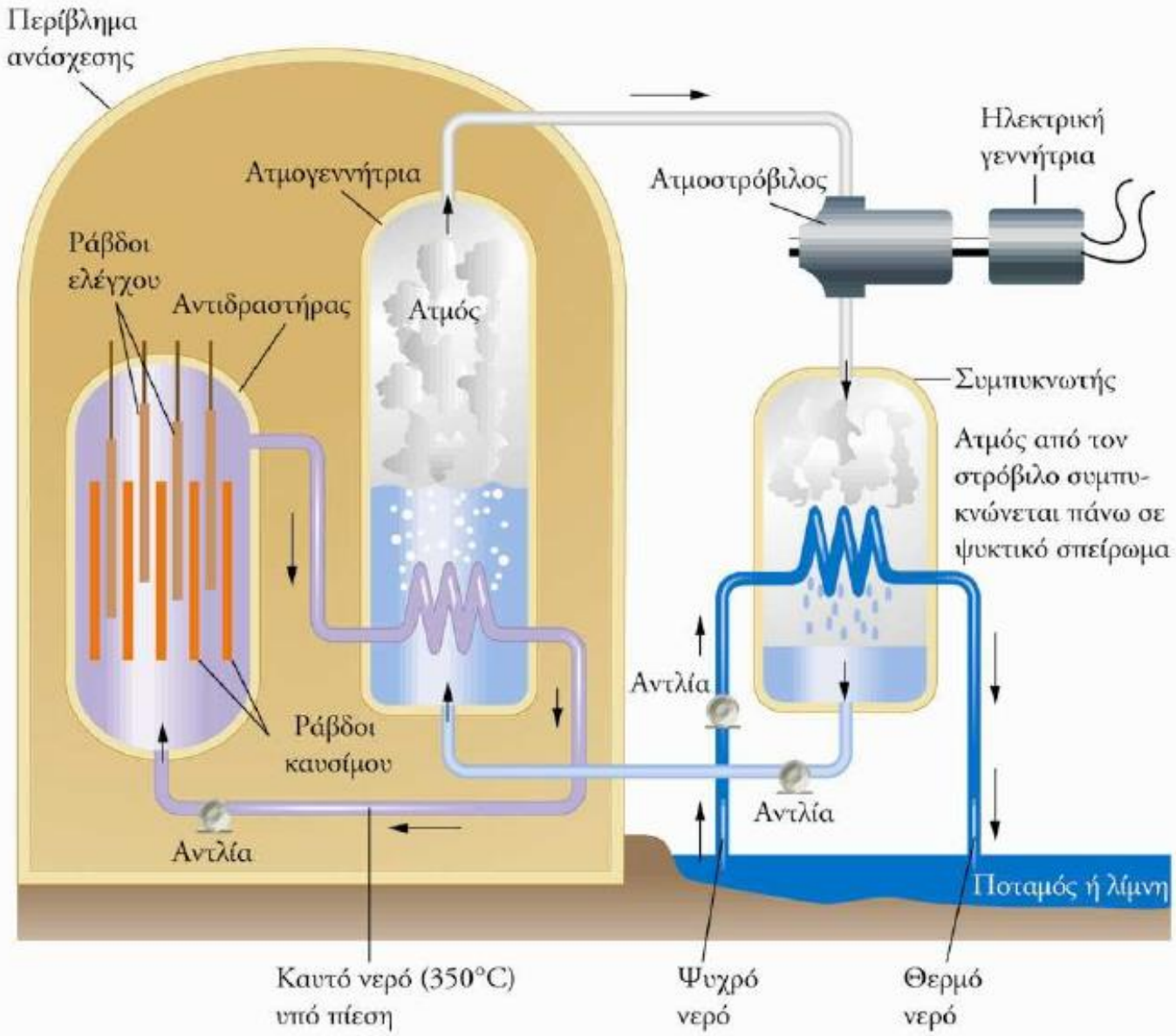
$$1\ amu = 1.49 \times 10^{-10}\ J$$

$$BE = 2.37 \times 10^{-11} J$$

$$BE / \text{νουκλεόνιο} = \frac{BE}{\text{Αριθμό νουκλεονίων}}$$

$$= \frac{2.37 \times 10^{-11}\ J}{19\ \text{νουκλεόνια}} = 1.25 \times 10^{-12}\ J / \text{νουκλεόνιο}$$

Διάγραμμα ενός πυρηνικού εργοστασίου

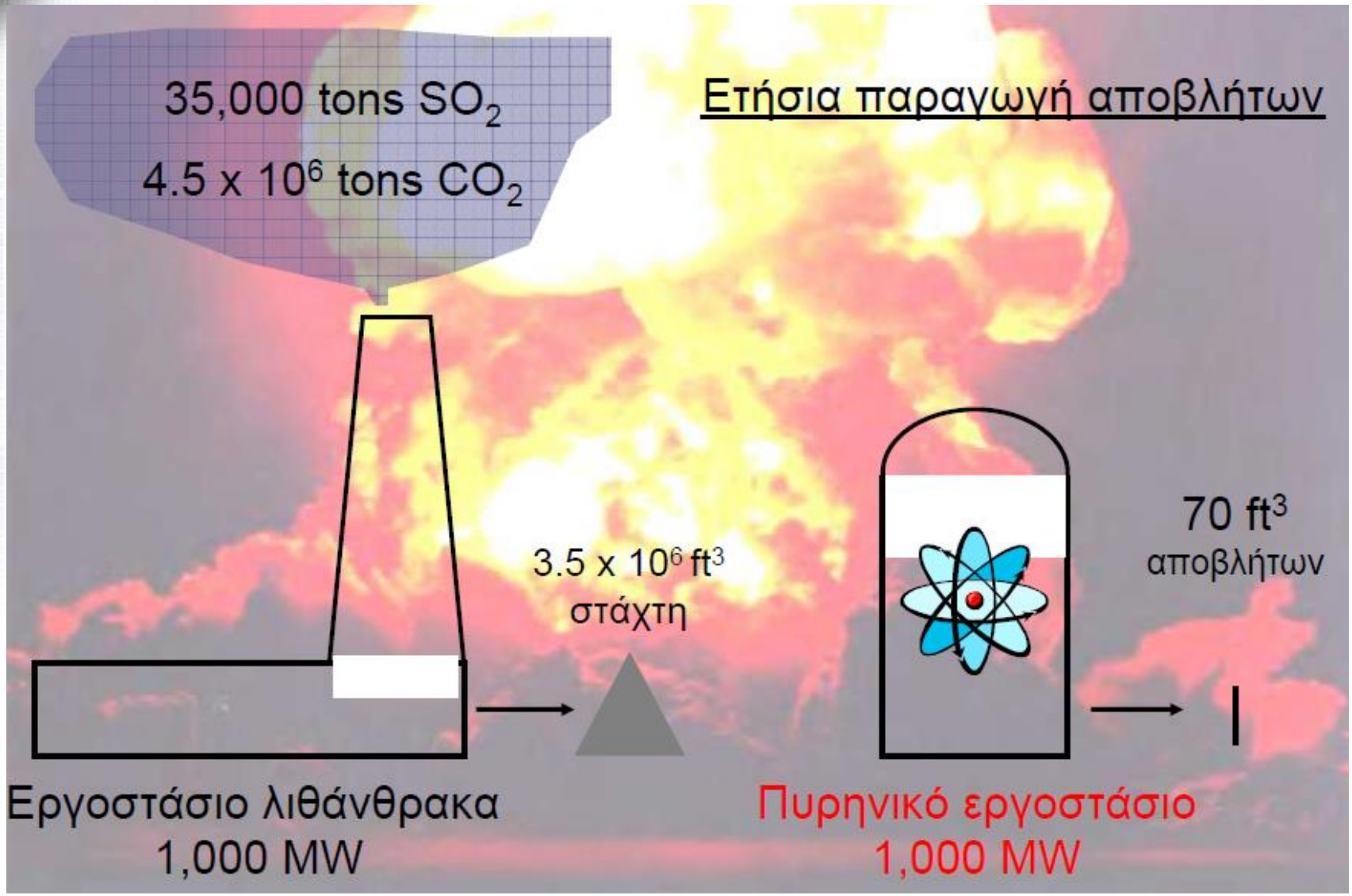


Πυρηνική ενέργεια

- Λειτουργούν περίπου 103 πυρηνικά εργοστάσια στις Η.Π.Α. και περίπου 435 διεθνώς.
- Το 17% της παγκόσμιας παραγωγής ηλεκτρικής ενέργειας προέρχεται από πυρηνικά εργοστάσια.

Country (rank)	Total power from nuclear energy (%)
1. France	75.0
2. Lithuania	73.1
3. Belgium	57.7
4. Bulgaria	47.1
5. Slovak Republic	47.0
6. Sweden	46.8
. . .	
19. United States	19.9
20. Russia	14.4
21. Canada	12.7

Διάγραμμα ενός πυρηνικού εργοστασίου



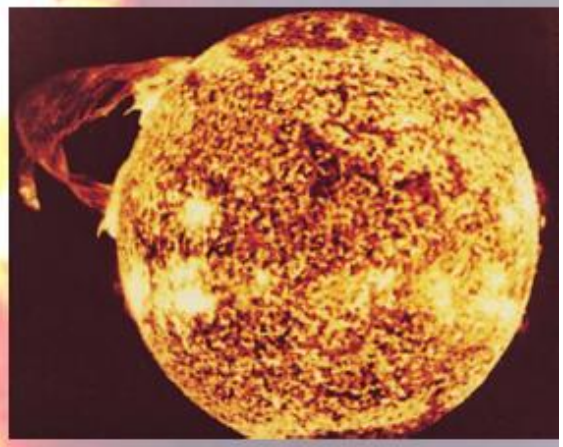
Πυρηνική σύντηξη - Nuclear Fusion

μικροί πυρήνες συνδιάζονται

$$^{2}_{1}H + {}^{3}_{1}H \rightarrow {}^{4}_{2}He + {}^{1}_{0}n + \text{Ενέργεια}$$

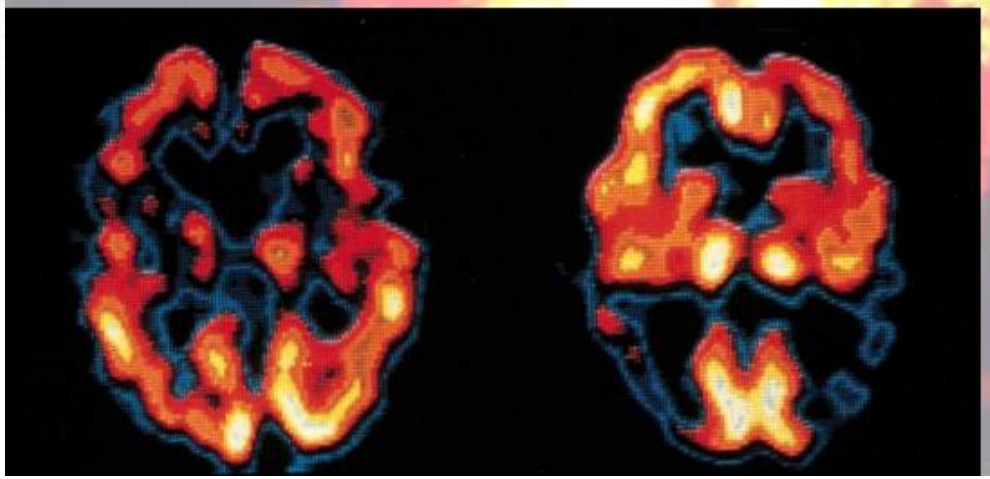
Συμβαίνει στον ήλιο και άλλα αστέρια

Αντίδραση σύντηξης	Ενέργεια
$^{2}_{1}H + {}^{2}_{1}H \longrightarrow {}^{3}_{1}H + {}^{1}_{1}H$	6.3×10^{-13} J
$^{2}_{1}H + {}^{3}_{1}H \longrightarrow {}^{4}_{2}He + {}^{1}_{0}n$	2.8×10^{-12} J
$^{6}_{3}Li + {}^{2}_{1}H \longrightarrow 2\ {}^{4}_{2}He$	3.6×10^{-12} J



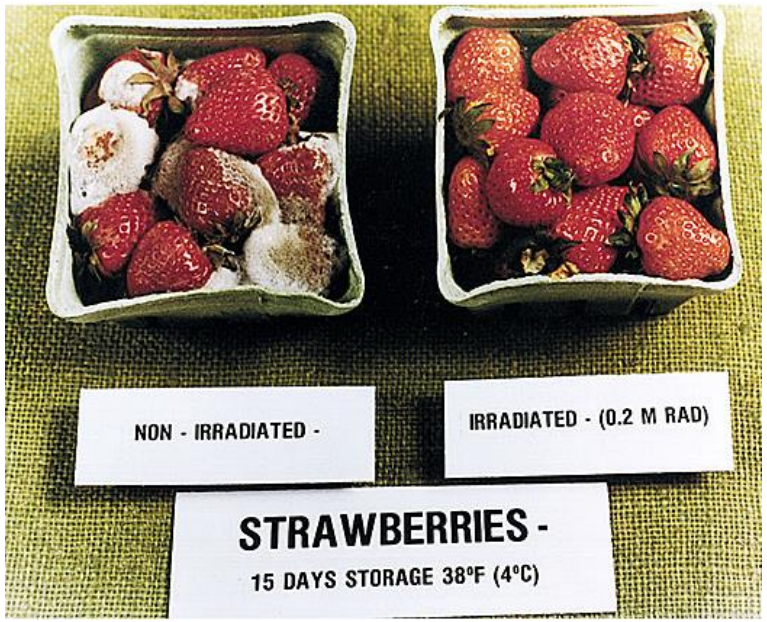
Ραδιοϊσότοπα στην Ιατρική

- Στατιστικά 1 στα 3 άτομα θα υποστούν μια εξέταση με ραδιοισότοπα
- ^{24}Na, $t_{1/2}$ = 14.8 hr, β emitter, blood-flow tracer
- ^{131}I, $t_{1/2}$ = 14.8 hr, β emitter, thyroid gland activity
- ^{123}I, $t_{1/2}$ = 13.3 hr, γ–ray emitter, brain imaging
- ^{18}F, $t_{1/2}$ = 1.8 hr, β^+ emitter, positron emission tomography PET
- ^{99m}Tc, $t_{1/2}$ = 6 hr, γ–ray emitter, imaging agent



Brain images with ^{123}I-labeled compound

Ακτινοβόληση τροφίμων - Food Irradiation



Dosage	Effect
Up to 100 kilorad	Inhibits sprouting of potatoes, onions, garlics. Inactivates trichinae in pork. Kills or prevents insects from reproducing in grains, fruits, and vegetables.
100 – 1000 kilorads	Delays spoilage of meat poultry and fish. Reduces salmonella. Extends shelf life of some fruit.
1000 to 10,000 kilorads	Sterilizes meat, poultry and fish. Kills insects and microorganisms in spices and seasoning.

Βιολογικές επιδράσεις

0–25	No effect observed
26–50	Small decrease in white blood cell count
51–100	Significant decrease in white blood cell count, lesions
101–200	Loss of hair, nausea
201–500	Hemorrhaging, ulcers, death in 50% of population
>500	Death

Βιολογικές επιδράσεις

Radiation ***a***bsorbed ***d***ose (***rad***)

1 rad = 1 x 10^{-5} J/g of material

Roentgen ***E***quivalent for ***M***an (***rem***)

Source	Dose (mrem/yr)*
Cosmic rays	20–50
Ground and surroundings	25
Human body†	26
Medical and dental X rays	50–75
Air travel	5
Fallout from weapons tests	5
Nuclear waste	2
Total	133–188

*1 mrem = 1 millirem = 1×10^{-3} rem.

†The radioactivity in the body comes from food and air.

1 rem = 1 rad x ***Q***

<u>***Q***uality Factor</u>

γ-ray = 1

β = 1

α = 20

* Mettler FA, et al. "**Effective Doses in Radiology and Diagnostic Nuclear Medicine: A Catalog**," *Radiology* (July 2008), Vol. 248, 254–63.

Imaging procedures and their approximate effective radiation doses*

Procedure	Average effective dose (mSv)	Range reported in th
Bone density test+	0.001	0.00–0.035
X-ray, arm or leg	0.001	0.0002–0.1
X-ray, panoramic dental	0.01	0.007–0.09
X-ray, chest	0.1	0.05–0.24
X-ray, abdominal	0.7	0.04–1.1
Mammogram	0.4	0.10–0.6
X-ray, lumbar spine	1.5	0.5–1.8
CT, head	2	0.9–4

The sievert - **1 Sv = 1 joule/kilogram** - a biological effect.
The sievert represents the equivalent biological effect of the deposit of a joule of radiation energy in a kilogram of human tissue. .

Available online at www.sciencedirect.com
ScienceDirect
Food and Chemical Toxicology 45 (2007) 2581–2591
Food and Chemical Toxicology
www.elsevier.com/locate/foodchemtox

Toxicological potential of 2-alkylcyclobutanones – specific radiolytic products in irradiated fat-containing food – in bacteria and human cell lines

Abstract

Food irradiation has been considered as a safe processing technology to improve food safety and preservation, eliminating efficiently bacterial pathogens, parasites and insects. This study aims to characterize the toxicological potential of 2-alkylcyclobutanones (2-ACBs), radiolytic derivatives of triglycerides, formed uniquely upon irradiation of fat-containing food. In irradiated food they are generated proportionally to fat content and absorbed radiation dose.

The cyto- and genotoxic potentials of various highly pure synthetic 2-ACBs were studied in bacteria and human cell lines. While pronounced cytotoxicity was evident in bacteria, no mutagenic activity has been revealed by the Ames test in *Salmonella* strains TA 97, TA 98 and TA 100. In mammalian cells genotoxicity was demonstrated mainly by the induction of DNA base lesions recognized by the Fpg protein as determined by both the Comet Assay and the Alkaline Unwinding procedure. Formation of DNA strand breaks was observed by the Alkaline Unwinding procedure but not by the Comet Assay. The extent of cytotoxicity and genotoxicity were dependent on chain length and degree of unsaturation of the fatty acid chain. Further studies will have to clarify mechanisms of action and potential relevance for human exposure situation.

Keywords: Food irradiation; 2-Alkylcyclobutanones; Cytotoxicity; Genotoxicity; Mutagenicity; Oxidative DNA lesions

Display Settings: Summary, Sorted by Link Ranking

Results: 5

DNA double-strand breaks induced by mammographic screening procedures in human mammary epithelial cells.

1. Colin C, Devic C, Noël A, Rabilloud M, Zabot MT, Pinet-Isaac S, Giraud S, Riche B, Valette PJ, Rodriguez-Lafrasse C, Foray N.
Int J Radiat Biol. 2011 Nov;87(11):1103-12. Epub 2011 Sep 19.
PMID: 21797809 [PubMed - in process]
Related citations

Use of γH2AX and other biomarkers of double-strand breaks during radiotherapy.

2. Sak A, Stuschke M.
Semin Radiat Oncol. 2010 Oct;20(4):223-31. Review.
PMID: 20832014 [PubMed - indexed for MEDLINE]
Related citations

Mammography-oncogenecity at low doses.

3. Heyes GJ, Mill AJ, Charles MW.
J Radiol Prot. 2009 Jun;29(2A):A123-32. Epub 2009 May 19. Review.
PMID: 19454801 [PubMed - indexed for MEDLINE]
Related citations

Does gammaH2AX foci formation depend on the presence of DNA double strand breaks?

4. Takahashi A, Ohnishi T.
Cancer Lett. 2005 Nov 18;229(2):171-9. Epub 2005 Aug 29. Review. Erratum in: Cancer Lett. 2006 May 8;236(1):155-6.
PMID: 16129552 [PubMed - indexed for MEDLINE]
Related citations

[Very low-dose hyper-radiosensitivity: impact for radiotherapy of micrometastases].

5. Thomas C, Fertil B, Foray N.

Int J Radiat Biol. 2011 Nov;87(11):1103-12. Epub 2011 Sep 19.

DNA double-strand breaks induced by mammographic screening procedures in human mammary epithelial cells.

Colin C, Devic C, Noël A, Rabilloud M, Zabot MT, Pinet-Isaac S, Giraud S, Riche B, Valette PJ, Rodriguez-Lafrasse C, Foray N.

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Abstract

Abstract Purpose: To assess in vitro mammographic radiation-induced DNA damage in mammary epithelial cells from 30 patients with low (LR) or high (HR) family risk of breast cancer. Materials and methods: Spontaneous and radiation-induced DNA double-strand breaks (DSB) were quantified by using immunofluorescence of the phosphorylated H2AX histone (γH2AX) in different conditions of mammography irradiation (2, 4, 2 + 2 mGy). Results: HR patients showed significantly more spontaneous γH2AX foci than LR patients (p = 0.014). A significant dose-effect was observed, with an exacerbation in HR patients (p = 0.01). The dose repetition (2 + 2 mGy) provided more induced and more unrepaired DSB than 2 mGy and 4 mGy, and was exacerbated in HR (p = 0.006). Conclusions: This study highlights the existence of DSB induced by mammography and revealed by γH2AX assay with two major radiobiological effects occurring: A low-dose effect, and a LOw and Repeated Dose (LORD) effect. All these effects were exacerbated in HR patients. These findings may lead us to re-evaluate the number of views performed in screening using a single view (oblique) in women whose mammographic benefit has not properly been proved such as HR patients.

The Neoplastic Transformation Potential of Mammography X Rays and Atomic Bomb Spectrum Radiation

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Radiation Biophysics Group, School of Physics and Astronomy, The University of Birmingham, Birmingham B15 2TT, United Kingdom

Heyes, G. H. and Mill, A. J. The Neoplastic Transformation Potential of Mammography X Rays and Atomic Bomb Spectrum Radiation. *Radiat. Res.* 162, 120–127 (2004).

Considerable controversy currently exists regarding the biological effectiveness of 29 kVp X rays which are used for mammography screening. This issue must be resolved to enable proper evaluation of radiation risks from breast screening. Here a definitive assessment of the biological effectiveness of 29 kVp X rays compared to the quality of radiation to which the atomic bomb survivors were exposed is presented for the first time. The standard radiation sources used were (a) an atomic bomb simulation spectrum and (b) 2.2 MeV electrons from a strontium-90/yttrium-90 (^{90}Sr/^{90}Y) radioactive source. The biological end point used was neoplastic transformation *in vitro* in CGL1 (HeLa × human fibroblast hybrid) cells. No significant difference was observed for the biological effectiveness of the two high-energy sources for neoplastic transformation. A limiting relative biological effectiveness (RBE_M) of 4.42 ± 2.02 was observed for neoplastic transformation by 29 kVp X rays compared to these two sources. This compares with values of 4.67 ± 3.93 calculated from previously published data and 3.58 ± 1.77 when the reference radiation was 200 and 220 kVp X rays. This suggests that the risks associated with mammography screening may be approximately five times higher than previously assumed and that the risk–benefit relationship of mammography exposures may need to be re-examined.

tional X rays relative to hard X rays, but its recommendation is to "attribute the same w_R (i.e. 1) for γ rays, X-rays and electrons as a matter of practicability in the absence of definitive information".

In this paper we present a definitive study of the oncogenicity of mammography X rays compared to high-energy X-ray and high-energy electron sources. The marker for oncogenicity used was neoplastic transformation in the nontumorigenic HeLa × skin fibroblast cell line, CGL1. The high-energy X-ray spectrum we have used matches that experienced by survivors 1500 m from the epicenter of the Nagasaki atomic bomb. This, in combination with a high-energy ^{90}Sr/^{90}Y electron source, has been used as our standard reference source, in effect matching the energy range upon which the epidemiological evaluations of radiation risks are based.

A clinical mammography X-ray set provided the low-energy X rays (29 kVp) identical to those used in breast cancer screening. Difficulties in recent studies raised by other groups have been addressed, and we present a rigorous dosimetric assessment of each source and a defense of the culture conditions used and demonstrate biological uncertainties lower than those published previously.

MATERIALS AND METHODS

Radiation Dose Associated With Common Computed Tomography Examinations and the Associated Lifetime Attributable Risk of Cancer

Rebecca Smith-Bindman, MD; Jafi Lipson, MD; Ralph Marcus, BA; Kwang-Pyo Kim, PhD; Mahadevappa Mahesh, MS, PhD; Robert Gould, ScD; Amy Berrington de Gonzalez, DPhil; Diana L. Miglioretti, PhD

Background: Use of computed tomography (CT) for diagnostic evaluation has increased dramatically over the past 2 decades. Even though CT is associated with substantially higher radiation exposure than conventional radiography, typical doses are not known. We sought to estimate the radiation dose associated with common CT studies in clinical practice and quantify the potential cancer risk associated with these examinations.

Methods: We conducted a retrospective cross-sectional study describing radiation dose associated with the 11 most common types of diagnostic CT studies performed on 1119 consecutive adult patients at 4 San Francisco Bay Area institutions in California between January 1 and May 30, 2008. We estimated lifetime attributable risks of cancer by study type from these measured doses.

Results: Radiation doses varied significantly between the different types of CT studies. The overall median effective doses ranged from 2 millisieverts (mSv) for a routine head CT scan to 31 mSv for a multiphase abdomen and pelvis CT scan. Within each type of CT study, effective dose varied significantly within and across institutions, with a mean 13-fold variation between the highest and lowest dose for each study type. The estimated number of CT scans that will lead to the development of a cancer varied widely depending on the specific type of CT examination and the patient's age and sex. An estimated 1 in 270 women who underwent CT coronary angiography at age 40 years will develop cancer from that CT scan (1 in 600 men), compared with an estimated 1 in 8100 women who had a routine head CT scan at the same age (1 in 11 080 men). For 20-year-old patients, the risks were approximately doubled, and for 60-year-old patients, they were approximately 50% lower.

Conclusion: Radiation doses from commonly performed diagnostic CT examinations are higher and more variable than generally quoted, highlighting the need for greater standardization across institutions.

Arch Intern Med. 2009;169(22):2078-2086

Rethinking Screening for Breast Cancer and Prostate Cancer

Laura Esserman, MD, MBA

Yiwey Shieh, AB

Ian Thompson, MD

After 20 years of screening for breast and prostate cancer, several observations can be made. First, the incidence of these cancers increased after the introduction of screening but has never returned to prescreening levels. Second, the increase in the relative fraction of early stage cancers has increased. Third, the incidence of regional cancers has not decreased at a commensurate rate. One possible explanation is that screening may be increasing the burden of low-risk cancers without significantly reducing the burden of more aggressively growing cancers and therefore not resulting in the anticipated reduction in cancer mortality. To reduce morbidity and mortality from prostate cancer and breast cancer, new approaches for screening, early detection, and prevention for both diseases should be considered.

JAMA. 2009;302(15):1685-1692 www.jama.com

BREAST CANCER AND PROSTATE cancer account for 26% of all cancers in the United States, with an estimated 386 560 patients diagnosed annually: 194 280 for breast cancer and 192 280 for prostate cancer[1] For both, there are remarkable differences between outcomes of localized vs advanced disease (breast cancer: 5-year relative survival rates of 98.1% vs 27.1%; prostate cancer: 100% vs 31.7%).[2] As a result, screening for both cancers has been promoted on the assumption that early detection and treatment is the best way to reduce disease-associated morbidity and mortality.

diagnosed with breast cancer, like prostate cancer, has almost doubled as well.

The increase in early cancers as a fraction of total cancers detected is not necessarily beneficial. The introduction of an optimal screening test should be fol-

cer and prostate cancer (FIGURE 2) resemble the intermediate-case scenario at best. The incidence of invasive breast cancer (excluding in situ lesions) has increased substantially and remains higher than prescreening rates.

Screening for Breast Cancer: U.S. Preventive Services Task Force Recommendation Statement

U.S. Preventive Services Task Force*

Description: Update of the 2002 U.S. Preventive Services Task Force (USPSTF) recommendation statement on screening for breast cancer in the general population.

Methods: The USPSTF examined the evidence on the efficacy of 5 screening modalities in reducing mortality from breast cancer: film mammography, clinical breast examination, breast self-examination, digital mammography, and magnetic resonance imaging in order to update the 2002 recommendation. To accomplish this update, the USPSTF commissioned 2 studies: 1) a targeted systematic evidence review of 6 selected questions relating to benefits and harms of screening, and 2) a decision analysis that used population modeling techniques to compare the expected health outcomes and resource requirements of starting and ending mammography screening at different ages and using annual versus biennial screening intervals.

Recommendations: The USPSTF recommends against routine screening mammography in women aged 40 to 49 years. The decision to start regular, biennial screening mammography before the age of 50 years should be an individual one and take into account patient context, including the patient's values regarding specific benefits and harms. (Grade C recommendation)

The USPSTF recommends biennial screening mammography for women between the ages of 50 and 74 years. (Grade B recommendation)

The USPSTF concludes that the current evidence is insufficient to assess the additional benefits and harms of screening mammography in women 75 years or older. (I statement)

The USPSTF concludes that the current evidence is insufficient to assess the additional benefits and harms of clinical breast examination beyond screening mammography in women 40 years or older. (I statement)

The USPSTF recommends against clinicians teaching women how to perform breast self-examination. (Grade D recommendation)

The USPSTF concludes that the current evidence is insufficient to assess additional benefits and harms of either digital mammography or magnetic resonance imaging instead of film mammography as screening modalities for breast cancer. (I statement)

Ann Intern Med. 2009;151:716-726. www.annals.org

For author affiliation, see end of text.

* For a list of the members of the USPSTF, see the **Appendix** (available at www.annals.org).

ELSEVIER

Journal of Nutritional Biochemistry xx (2011) xxx–xxx

Pomegranate extract demonstrate a selective estrogen receptor modulator profile in human tumor cell lines and in vivo models of estrogen deprivation

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Abstract

Selective estrogen receptor modulators (SERMs) are estrogen receptor (ER) ligands exhibiting tissue-specific agonistic or antagonistic biocharacter and are used in the hormonal therapy for estrogen-dependent breast cancers. Pomegranate fruit has been shown to exert antiproliferative effects on human breast cancer cells in vitro. In this study, we investigated the tissue-specific estrogenic/antiestrogenic activity of methanol extract of pericarp of pomegranate (PME). PME was evaluated for antiproliferative activity at 20–320 μg/ml on human breast (MCF-7, MDA MB-231) endometrial (HEC-1A), cervical (SiHa, HeLa), ovarian (SKOV3) carcinoma and normal breast fibroblast (MCF-10A) cells. Competitive radioactive binding studies were carried out to ascertain whether PME interacts with ER. The reporter gene assay measured the estrogenic/antiestrogenic activity of PME in MCF-7 and MDA MB-231 cells transiently transfected with plasmids coding estrogen response elements with a reporter gene (pG5-ERE-luc) and wild-type ERα (hEG0-ER). PME inhibited the binding of [^{3}H] estradiol to ER and suppressed the growth and proliferation of ER-positive breast cancer cells. PME binds ER and down-regulated the transcription of estrogen-responsive reporter gene transfected into breast cancer cells. The expressions of selected estrogen-responsive genes were down-regulated by PME. Unlike 17β—estradiol [1 mg/kg body weight (BW)] and tamoxifen (10 mg/kg BW), PME (50 and 100 mg/kg BW) did not increase the uterine weight and proliferation in ovariectomized mice and its cardioprotective effects were comparable to that of 17β-estradiol. In conclusion, our findings suggest that PME displays a SERM profile and may have the potential for prevention of estrogen-dependent breast cancers with beneficial effects in other hormone-dependent tissues.

Keywords: Breast cancer; Estrogen; Estrogen receptors; Pomegranate; Selective estrogen receptor modulator (SERM); Uterotrophic assay

Breast Cancer RESEARCH

EDITORIAL

Is the tide turning against breast screening?

Karsten Juhl Jørgensen*

See related research by Nederend *et al.*, http://breast-cancer-research.com/content/14/1/R10

Abstract

Herein I argue that mammographic screening has not delivered on its fundamental premise: to reduce the incidence of advanced breast cancer. Indeed, achieving this goal is required if screening is to reduce breast cancer mortality or mastectomy use. Rather, screening has caused substantial increases in the incidence of *in situ* and early invasive cancers. Moreover, evidence indicates that these screen-detected cancers are unlikely to be cases that were 'caught early', but instead represent women who would not have been diagnosed in the absence of screening and who, as a result, have received harmful, unnecessary treatment. If true, these observations raise the specter that screening creates breast cancer patients and that this practice carries little or no benefit.

A claimed reduction in breast cancer mortality [4-6] as well as a reduction in the use of mastectomies [7,8] have also been called into doubt in studies of population-based breast screening. In addition, the detection of cancers that would otherwise not have developed into clinical, symptomatic disease (overdiagnosis) is now recognised as an important harm, also for invasive breast cancer [9,10].

A recent systematic review of incidence trends in seven countries with at least seven years of screening [2] found that breast screening has not fulfilled its promise of fewer advanced breast cancers. It included The Netherlands, but not data from before organised screening was introduced in the late 1980s. Including data from 1980 to 2008 is a strength of the new study, as it allows reliable estimates of both pre- and post-screening incidence trends of advanced breast cancer. If the background incidence was increasing prior to screening, but stable