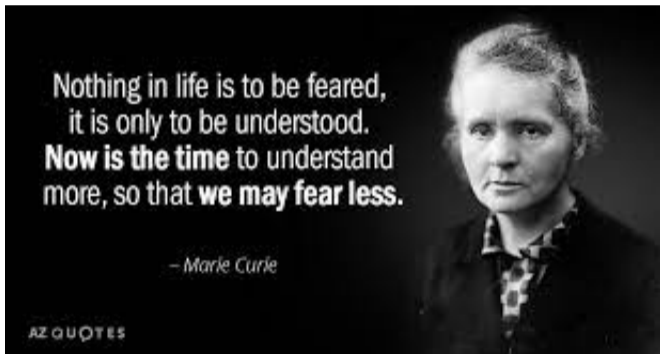


# Understanding Radiation Damage and Repair





Hand out dosimeter and tell people to pass it around.

Radiation is every where.

You are radioactive, your dog is radioactive.

Ask whoever has the dosimeter what s(he) is reading

Figure 0.1 uSv/h is  $1.2 \times 10^6$  gammas/min

In the last minute, as we are sitting here

our bodies have absorbed

$x$  (in tenths uSv/h) \* 1.2 million particles

with enough energy to produce cell damage.

Without radiation we would not be here.

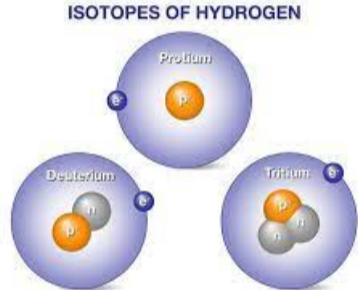
Nonsensical to call something radioactive.

The only question is how radioactive.

With radiation, we must get quantitative.

# ISOTOPES

- Ordinary matter is made up of about 100 elements.
- Each element is made up of a nucleus surrounded by a cloud of electrons.
- Each nucleus is made up of protons and neutrons.
- Each element determined by number of protons. Hydrogen 1; Helium 2; and so on.
- But the number of neutrons can vary. Hydrogen can have 0, 1, or 2 neutrons. A particular combination of protons and neutrons is an *isotope*.
- An isotope is denoted  ${}^n\text{X}_x$  (e.g.  ${}^2\text{H}$  or  ${}^4\text{He}$ )  
 $\text{X}_x$  tells us the element, the number of protons, and  $n$  is the total number of protons and neutrons.



## ISOTOPES

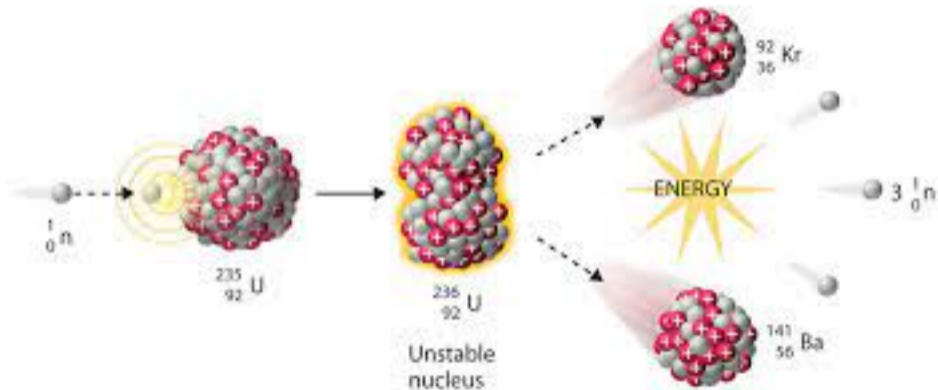
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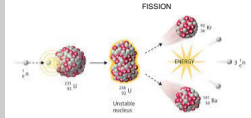


Read from Slide.

Over 3000 known isotopes, but most are very rare,  
and only a small handful are important in a NPP release.

# FISSION

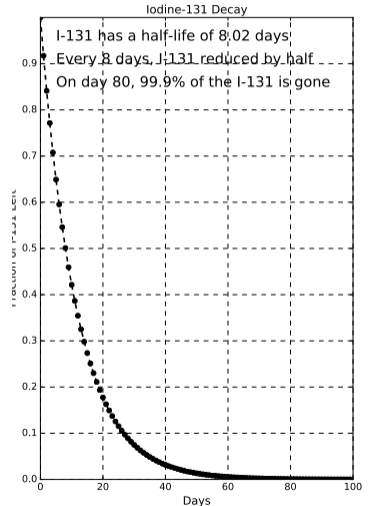




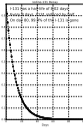
- A few isotopes will split in two when hit by a neutron.  
***Split or fission releases 50 million times as much energy as combining carbon and oxygen to CO<sub>2</sub>.***  
This energy density is why nuclear power should be cheap, very cheap.
- One such isotope is  ${}^{235}\text{U}$ , (92 protons, 143 neutrons).
- Fission also releases 2 or 3 neutrons.  
Under the right conditions, these neutrons can hit another nucleus, producing a self-sustaining *chain reaction*.
- A reactor's job is to maintain these conditions while capturing the energy released by the process.

# FISSION PRODUCTS

- Fragments of split are called *fission products*.
- Some fission products are unstable isotopes. Combinations of protons and neutrons that cannot stay together for long.
- These unstable isotopes spontaneously *decay* to another isotope.
- Each such isotope decays at its own rate which is measured by the isotope's *half-life*
- When an isotope decays, it releases energy.



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Combinations of protons and neutrons that cannot stay together for long.

These unstable isotopes spontaneously *decay* to another isotope.

Each such isotope decays at its own rate

which is measured by the isotope's *half-life*

the time it takes for half of the isotope to change to something else.

One half life, down by a factor of 2, etc

10 half-lives down by a factor of 1000.

Half-lives can vary from a small fraction of a second to thousands of years.

Talk about I-131.

When an isotope decays, it releases energy.



## There are three kinds of decay energy

### **Alpha particle.**

2 protons, 2 neutrons bound together.

Almost no penetrating power.

Stopped by piece of paper or few cm of air.

### **Electron.**(Beta)

Similar to the electrons produced by old fashioned, cathode ray televisions.

Little penetrating power.

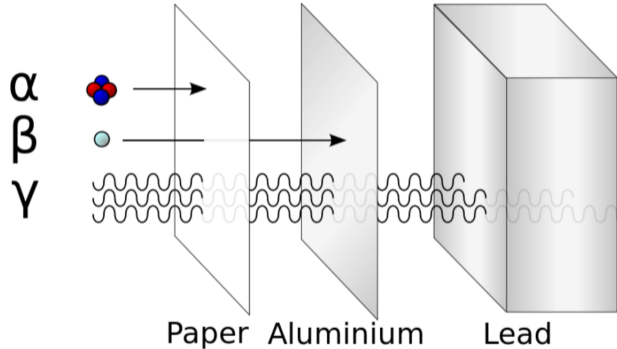
Most are stopped by outer layer of skin.

### **Photon.**(Gamma)

Same particle as sunshine, but much higher energy.

Lots of penetrating power.

50% can pass all the way through a human body,



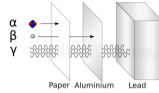
# Radiation Damage and Repair

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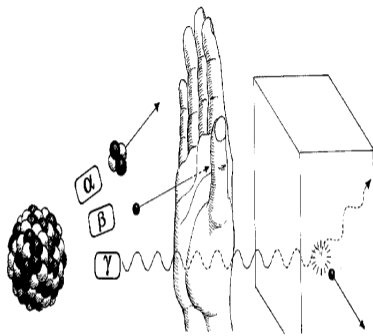
**Electron, (Beta)**  
Similar to the electrons produced by old fashioned cathode ray televisions.  
Little penetrating power.  
Most are stopped by outer layer of skin.

**Photons, (Gamma)**  
Same particle as sunshine, but much higher energy.  
Lots of penetrating power.  
50% can pass all the way through a human body.



The energy released by decay can take 3 very different forms.

Particle Symbol	Linear Energy Transfer	Charge	Relative mass	Tissue Penetration
Alpha Particle $\alpha$	High RBE=20	+2	7273	Nil. Must ingest or inhale to cause damage
Electron $\beta$	Low RBE=1	-1	1	Very weak. High energy can damage skin, else must ingest or inhale.
Photon $\gamma$	Low RBE=1	0	0	Very high



Only photons can hurt us from the outside

# Radiation Damage and Repair

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Only photons can hurt us from the outside

Alphas and electrons are charged particles.

They interact with the electromagnetic fields in our tissue.

This slows them down very quickly.

As a result they have very little penetrating power.

They must be ingested or inhaled in order to do real damage.

Photons have no charge and do not interact.

They have lots of penetrating power.

Only photons can hurt us externally.

Photon emitters tend to decay fairly rapidly.

In spent nuclear fuel, essentially all the photon emitters are gone in about 500 years.

After that spent fuel would need to be ingested to do any harm.

At that point, spent fuel becomes just another poison

Just don't eat it.

For ingested material, uptake and biological half-life important.

- Uptake is fraction of emitter absorbed into our organs. Rest will be excreted in a day of two.
- Biological half-life is time for half of absorbed emitter to be eliminated in the normal course of events.

	Plutonium	Cesium	Iodine
Uptake	0.001	0.80	0.20
Biological Half-life	200 years	70 days	80 days
Main Organ	Bone	Soft Tissue	Thyroid

# Radiation Damage and Repair

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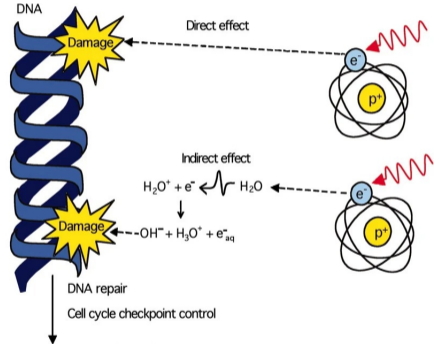
- Uptake is fraction of emitter absorbed into our organs. Most will be excreted in a day or two.
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99.9% of any Pu injected is eliminated in a day or two.  
 But bit that is absorbed goes to bones, and stays there for a life.  
 Most Pu isotopes decay very slowly.  
 Tell Galen Winsor story.  
 Tell Queen Elizabeth story.  
 Cesium apes potassium. It is readily absorbed and spreads uniformly in soft tissue.  
 The bio half-life is much shorter than the radioactive half-life, 2 and 30 years.  
 Iodine's biological half-life is much longer than the radioactive half-life (8 days or less), and concentrates in 10-15 gram thyroid gland, magnifying the dose to that organ by roughly a factor of 1000.

# IONIZING RADIATION

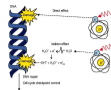
- Living tissue is made up of cells.
- Cells are mostly water.
- If a radioactive decay particle enters a cell, it transfers a portion of its energy to the cell mainly by breaking the bonds that hold the water molecule together. Particles which have enough energy to do this are called *ionizing radiation*.
- Ionization creates chemically active free radicals, called Reactive Oxygen Species (ROS), that can disrupt the cell's chemistry.
- Main concern is DNA damage.  
Can lead to cancer.
- Amount of energy deposited in tissue, the *dose*, is measured in joules per kg tissue (J/kg).
- The shorthand for J/kg is a gray (Gy).



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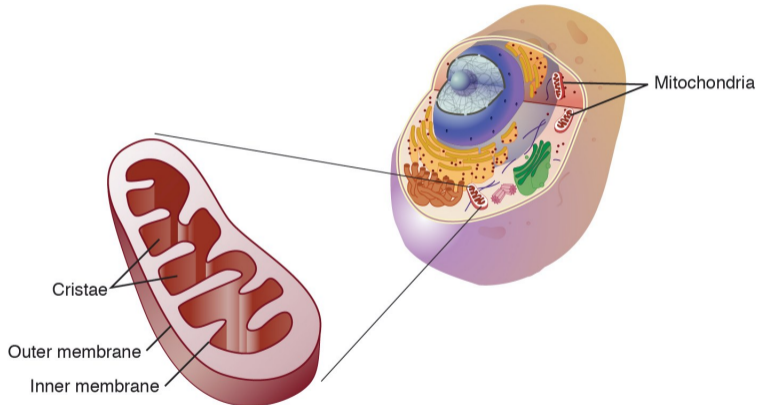


Read from slide.



## Why are we so good at repairing DNA damage?

- Answer: our oxygen based metabolism.
- Each of our cells contain up to 1000 or more mitochondria
- Tiny powerhouses use oxygen to release the energy we need.
- But also leak ROS into the rest of the cell.
- One billion ROS microbombs per cell per day.
- 1 in 20,000 of these damage our DNA.
- Nature had to come up with a repair system to handle this onslaught.



# Radiation Damage and Repair

Rad damage is not the only problem our DNA faces.

In fact, it's far from the biggest problem.

We use an oxygen based metabolism. Oxygen is dangerous stuff.

Oxygen oxidizes. In metabolism most O<sub>2</sub> ends up as CO<sub>2</sub>.

But about 5% converted to Reactive Oxygen Species.

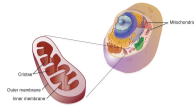
These reactive ions roam around screwing things up.

The info in our DNA must be preserved.

Nature had to come up with an amazingly accurate DNA repair system.

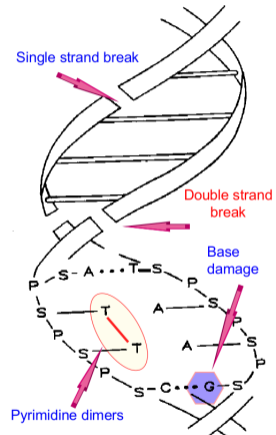
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# DNA Damage

- The concern is DNA damage which can lead to cancer.
- Damage can be Single Strand Break (SSB) which leaves one side intact or Double Strand Break (DSB), both sides broken.
- SSB's are repaired almost automatically using intact side as template.
- Repair is quick ( $\approx 30min$ ) and essentially error free,



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SSB's astonishingly frequent, but they are repaired almost automatically by the clever chemistry of the double helix.

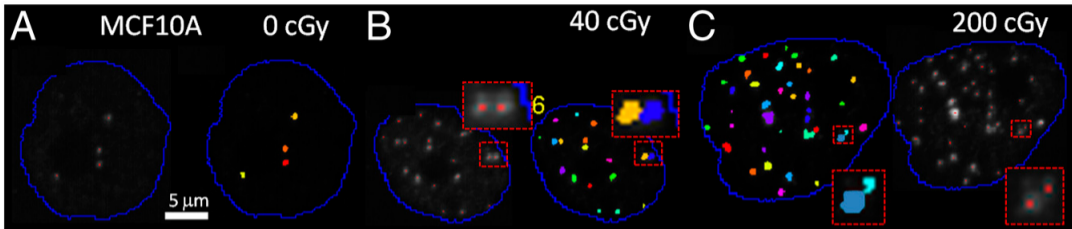
As long as one side is OK, we have a template.

2 mSv/d only increases SSB's by 1.0%, no problem.

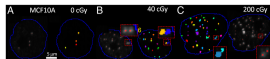
SSB's a non-problem.

In this context, harm is NOT damage.

Harm (eg cancer incidence) is related to viable UNREPAIRED damage.



- Our bodies can handle *isolated* DSB's.
- UC Berkeley has pictures of the DSB repair process.
- Bright spots are RIF's, Radiation Induced Foci, clusters of damage sensing/repair proteins.
- They glom on to the broken ends and rejoin them.



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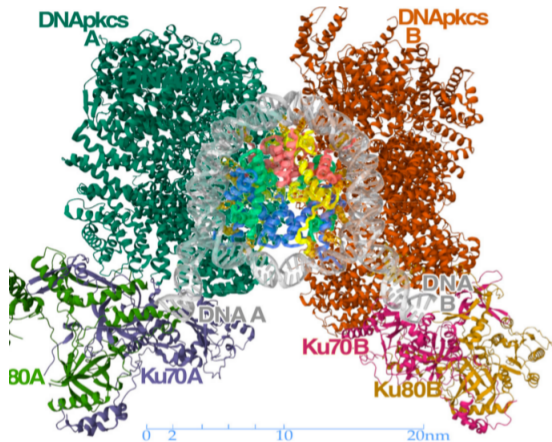
Amazingly Berkely has pictures of the DSB repair process.

Three screen shots, right to left,  
are for 0, 400 mGy, 2000 mGy acute to cell.

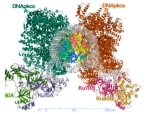
Each third shows two different imaging techniques.

Bright spots are clusters of damage sensing and repair proteins.  
called Radiation Induced Foci (RIF's).

- Real problem is closely spaced DSB's. (aka double double strand breaks or DDSB's)
- If DSB's are too close together, multiple RIF's don't have room to form.
- One RIF faced with multiple DSB's not good.
- Can end up rejoining the wrong ends.
- Rothkamm et al, 2001:  
80 Gy in 3.5 minutes, 50% misrejoins.  
80 Gy in 14 days no detectable misrepairs.



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Are bodies are quite good at handling isolated DSBs.

Molecules call Ku's (coo's) are always drifting around in the cell waiting for a DSB to happen.

When one occurs, they quickly — in a matter of seconds — clamp on to the broken ends.

The Ku's then call is a large complex of proteins to do the rejoining.

The real problem is closely spaced DSB's,

called *double double strand breaks (DDSB)*, a rare case of the acronym being better than the name.

The RIF's are larger in size than the portions of the DNA they are attempting to repair.

If the DSB's are too close together,

multiple RIF's simply do not have room to form.

Slide shows two DSB's label A and B (DNA is translucent gray)

Ku's have moved in and called in two DNAPkcs complexes, who are fighting for position.

When a single RIF is faced with multiple DSB's,

all sorts of bad things can happen,

probably the worst of which is rejoining the wrong ends

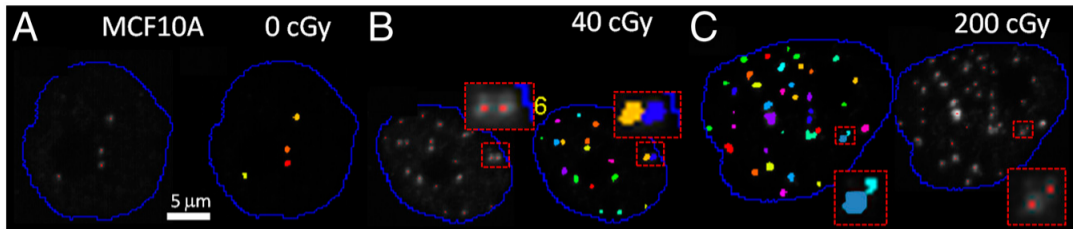
creating an uncorrectable misrepair.

A few of these mutations will be viable,

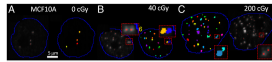
a few of the viable mutations will escape our immune system,

and a few of those could become cancerous.





- If RIF faced with 1 DSB, repair accurate. If not, error rate skyrockets.
- RIF/Gy decreases with dose. 100 mGy: 73 RIF's/Gy. 1000 mGy: 28 RIF/Gy.
- Expect 25-40 DSB/Gy.
- Do the DSB/RIF math. 100mGy: 4.0/7.3, OK. 1000 mGy: 40/28, not good.
- **Process is non-linear; dose rate driven.**
- Cell repair process takes minutes to  $\approx$  10 hours depending on dose.  $\approx$  2 hrs for 100 mGy



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If a RIF is faced with a single DSB, repair is almost always correct.

If a RIF is faced with more than one DSB, error rate skyrockets.

Number of RIF's increases less than linearly with dose.

At 100 mGy, they saw 73 RIF's per gray.

At 1000 mGy, they saw 28 RIF's per gray.

Expect 25-40 DSB/Gy. Do the math. 40/73 DSB/RIF, OK. 40/28, not good.

This repair process is both non-linear and dose rate driven.

If damage rate is substantially less than repair rate, all good.

If not, trouble.

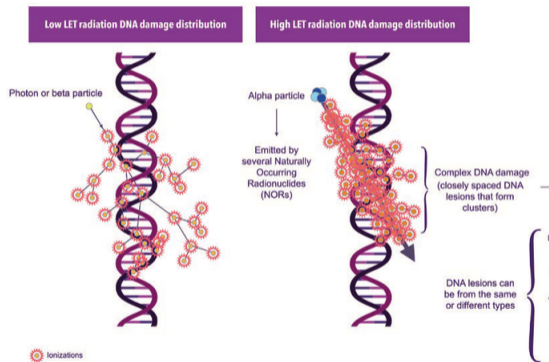
These experiments also showed intra-cellular repair process

took between 1 and 10 hours depending on dose.

About 2 hrs for 100 mGy.

## Low/High LET and Sieverts

- Alpha particle damage is highly localized along particle's short, straight track. Called High LET (Linear Energy Transfer) damage.
- Photon, electron damage much more spread out. Called Low LET damage.
- High LET much more likely to create DSB's and DDSB's than Low LET for same energy.
- In NNP release, almost all public harm is caused by Low LET.
- To handle difference, a unit called sievert (Sv) was concocted.
- Sievert is gray times a factor (RBE).
- RBE for alphas is 20.  
RBE for photons and electrons is 1.0.



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Read from slide.

## Endogenous ROS Damage vs Radiation Damage

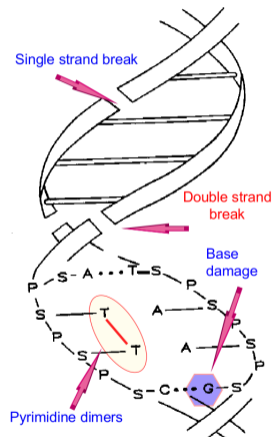
- Some estimates of endogenous damage.

Source	SSB/cell-day	DSB/cell-day
Vilenchik-2003	20,000	10 - 50
Bouwman-2016	20,000	10 - 50
Lieber-2010		about 10
Lees-Miller	60,000	10
Costes-2021		10 - 50
Henriksen-2013	50,000	8

- Some estimates of radiation damage.

Source	DSB/mSv
Vilenchik-2003	0.03
White-2016	0.01 - 0.05
Neumaier-2011	0.025- 0.04

- At low end 10 endogenous DSB/cell-day and high end 0.04 DSB/mSv, need 250 mSv/d to match endo ROS.
- 2 mSv/d won't stress system. 20 mSv/d might.

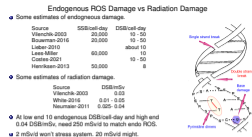


## Radiation Damage and Repair

Read from slide

If we use high endo, low rad,  
need 5000 mSv/d to match endo damage.

How does all this jibe with real world data?



Group	Size	Period	Cumulative dose mSv	Dose rate mSv/day	Result
<b>Atom bomb survivors</b>					
Leuk 5-150	33,459	seconds	5 to 150	5 to 150	Insignificant decrease in leukemia
Solid 5-20	14,555	seconds	5 to 20	5 to 20	Insignificant decrease in solid cancers
Solid 20-40	6,411	seconds	20 to 40	20 to 40	Solid cancers same as control
Solid 40-125	10,970	seconds	40 to 125	40 to 125	Insignificant increase in solid cancers.
Leuk 150-300	5,463	seconds	150 to 300	150 - 300	Insignificant increase in leukemia.
Solid 125+	16,166	seconds	125+	125+	Significant increase in solid cancers.
Leuk 300+	6,793	seconds	300-5000+	300-5000+	Significant increase in leukemia.
Goiania	≈46	hrs or less	1000-6000	1000-6000	50% mortality abv 4000 mSv
Thai scrap	≈10	hrs or less	1000-6000	1000-6000	100% mortality abv 6000 mSv
Chern 1st responders	134	<2 hrs	1000-16000	1000-16000	Sigmoid mortality, 50% mortality at 6000 mSv.
H. Daghlian	1	seconds	5900	5900	Died in 25 days
Tokaimura	3	seconds	3000-17000	3000-17000	> 10,000 mSv died
Louis Slotin	1	seconds	21000	21000	Died in 9 days
Norway tech	1	< hour	38500	38500	Died in 13 days
R. Peabody	1	seconds	>100000	100000+	Died in 2 days
Chernobyl liquidators	220,000	2 min to 90 days	1-1500	nil to 1500	Low/high dose rate mushed together. 6% incre
Litvenko	1	3 weeks	96,000	4,000	Died in 23 days
Belarus kids	13,127	2-3 weeks	ave 780 max 48k	39-2400	45 thyroid cancer, eventual 50? deaths
Ukraine kids	11,611	2-3 weeks	ave 560 max 33k	28-1600	87 thyroid cancer, eventual 50? deaths
Eben Byers	1	2 years	366,000	300	Horrible bone cancer. Died in 3 years.
Evans radium hi	127	10 years	>80000	80+	Cancers. Hi mortality >200 mSv/d
Dial painters hi	273	10 to 60 yrs	190000-440000	20+	96 bone cancers, 1 below 20
Evans radium mid	17	10 years	20000-80000	20 to 80	Abnormalities. Nil clinical symptoms.
Dial painters lo	2,110	10 to 60 yrs	200 - 160000	up to 20	Zero bone cancers.
Evans radium lo	59	10 years	up to 20000	max 20	Nil abnormalities.
Albert Stevens	1	20 years	61,000	8	Died at age 79 of heart failure.
UPPU Club	26	≈10y	up to 7200	0.03-2	Lower mortality than coworkers.
Taipei Apt hi	1,100	18 years	up to 4000	up to 3	Decrease in cancer, maybe non-rad.
Taipei Apt mid	900	18 years	ave 420	up to .160	Decrease in cancer, maybe non-rad.
Taipei Apt low	8,000	18 years	ave 120	up to .050	Decrease in cancer, maybe non-rad.
Keralans	69,956	10-15 yrs	50-650	.016 to .160	Insignificant decrease in cancer



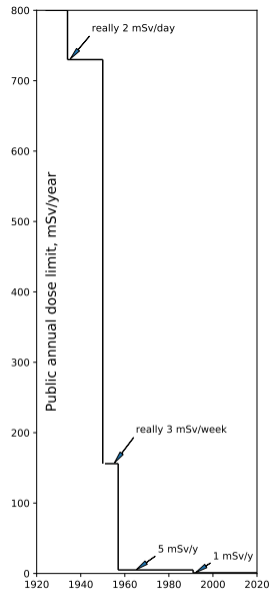


- To get out of the green, we need at least 20 mSv/d, even if that dose rate is received for many years.
- Prior to 1950, ICRP/NCRP *tolerance dose [rate]* was 2 mSv/d. Renamed Maximum Permissible Dose in 1940's.
- Better than a factor of 10 margin.

No one has been identifiably injured by radiation while working within the numerical standards [2 mSv/d] first set by the NCRP, then the ICRP in 1934.[Lauriston Taylor, 1980]

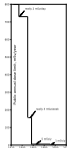
Still true.

- In 1951 NCRP went to 3 mSv/week.  
In 1957 went to 5 mSv/year for public.
- NCRP explained. The changes in the accumulated Maximum Permissible Dose are not the result of positive evidence of damage due to use of earlier permissible dose levels but rather are based on the desire to bring the MPD into accord with the trends in scientific opinion.  
Opinion trends not based on data are hardly scientific.



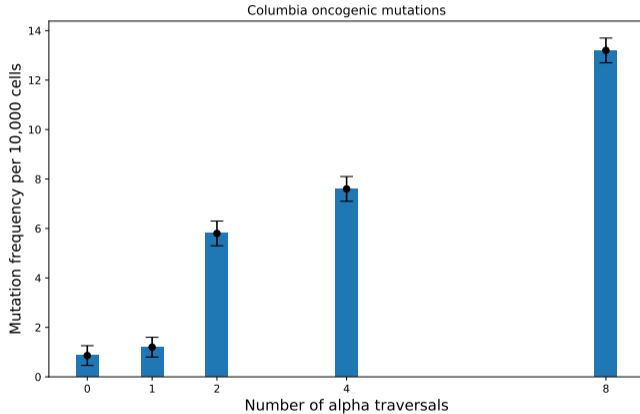
# Radiation Damage and Repair

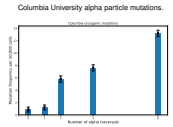
- To get out of the green, we need at least 20 mSv/d, even if that dose rate is received for many years.
- Prior to 1950, ICRP/NCRP tolerance dose (rate) was 2 mSv/d. Renamed Maximum Permissible Dose in 1940's.
- Better than a factor of 10 margin.  
No one has been identified injured by radiation while working within the numerical standards (2 mSv/d) first set by the NCRP then the ICRP in 1934 (Lauriston Taylor, 1960).  
Still true.
- In 1951 NCRP went to 3 mSv/week.  
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Read from slide.

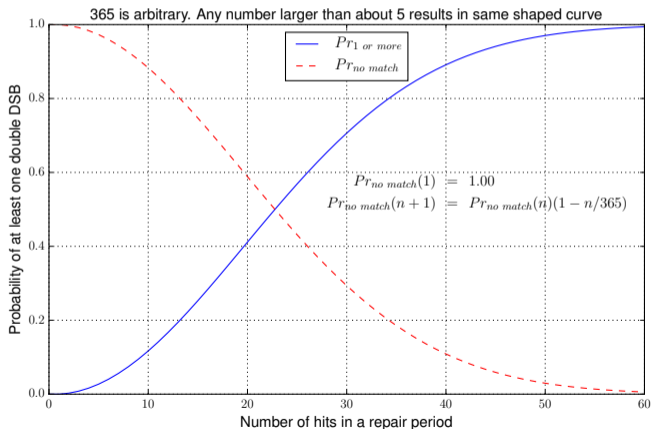
# Columbia University alpha particle mutations.





- Many studies support the importance of DDSB's.
- One example. Columbia was able to hit a cell with exactly 1, 2, 4, or 8 alphas.
- Measured number of oncogenic transmutations which resulted.
- Number of mutations for a single hit was not statistically different from sham control (zero hit) results.
- But if a cell was hit twice, the number of mutations jumped by a factor of six.
- These results match the double DSB theory nicely.

- Divide DNA into N (say 365) target zones.
- If DDSB's are real problem, we need more than one hit in the same target area.
- This is a version of the same-birthdays hustle.
- End up with an S-shaped dose response curve.



# Radiation Damage and Repair

If double DSB's are the real problem, harmful damage is highly non-linear in the number of unrepaired hits. In fact, this kind of damage is S-shaped in the number of currently unrepaired DSB's. Stay with me.

If it takes two closely spaced DSB's to cause harm, we are dealing with a version of the same birthdays problem.

This is an old bar hustle, in which the hustler bets even money that among the 30 patrons in the bar, there is at least one duplicate birthday. People do the numbers in their head.

There's one chance in 365 that somebody's birthday is on any given day of the year. Therefore, the hustler's chance of winning the bet is 30 times  $1/365$  or less than 10%.

They take the bet. 70% of the time they lose.

If the number of patrons is 35, they lose 81% of the time.

What does this have to do with double DSB's?

Define a hit as something that causes a DSB.

Divide the DNA into 365 target zones.

(You can substitute any other number for 365. The shape of the curve will be the same.)

Two hits in the same target zone cause a DDSB.

A single hit has no chance of causing a double DSB.

The second hit only has one chance in 365 of hitting the same target zone and causing a double DSB.

But if it doesn't, the third hit has two chances in 365 of creating a DDSB.

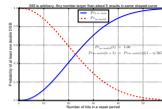
If it doesn't, the fourth hit has three chances. And so on.

This builds up pretty quickly.

In the hustler's case, if there has been no duplicate by the time he gets to patron 30, there is an 8% chance that number 30 has the same birthday as one of the other 29.

Go thru equation.

- Divide DNA into  $N$  (say 365) target zones.
- If DDSB's are real problem, we need more than one hit in the same target area.
- This is a version of the same-birthdays hustle.
- End up with an S-shaped dose response curve.



## The All Important Time Dimension

- Repair takes time:  $\approx 30$  minutes to  $\approx$ half-day
- Dose is experienced over an exposure period.
- Exposure period can be as short as 0.1 seconds (Xray)
- Or as long as a life time (background radiation)
- Dose rate relative to repair time is critically important.
- Number of unrepaired DSB's sets the number of target zones, in which a hit produces a DDSB.
- If dose rate larger than repair rate, inventory of DSB's builds up. Big problem.
- What counts is the dose received within the repair period.

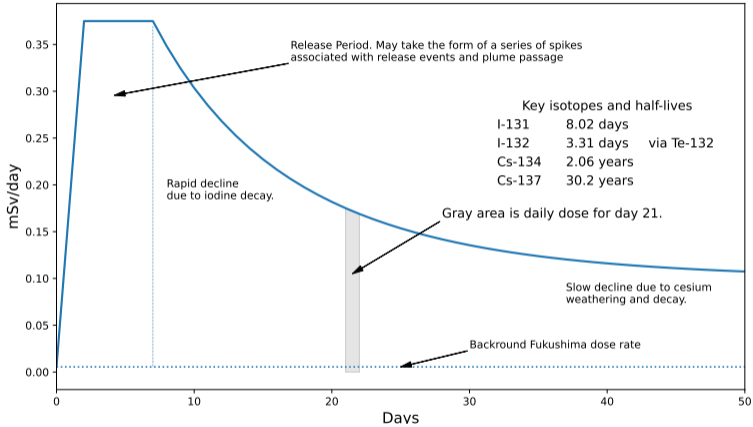
- Repair takes time: ~20 minutes to ~half-day
- Dose is experienced over an exposure period.
- Exposure period can be as short as 0.1 seconds (Xray)
- Or as long as a life time (background radiation)
- Dose rate relative to repair time is critically important.
- Number of unrepaired DSB's sets the number of target zones, in which a hit produces a DGSB.
- If dose rate larger than repair rate, inventory of DGSB's builds up. Big problem.
- What counts is the dose received within the repair period.

Bar scene was static situation.  
Number of patrons did not change.  
In radiation damage, hits are always happening.  
DSB's are continually being repaired.  
The time dimension becomes all important.  
Stating a dose without an exposure time  
is like saying "I got a hundred points"  
without saying whether that was for a game or for a career.

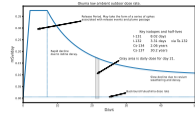
But we are very sloppy in our use of "dose"  
Sometimes we mean dose rate. mSv/d  
Sometimes we mean dose amount. mSv.  
If we are serious about radiation,  
that is not acceptable.



Okuma low ambient outdoor dose rate.



# Radiation Damage and Repair



- Since the time dimension is crucial, to assess harm, we must know an individual's dose rate profile, the dose rate (s)he gets hit with thru time.
- Here's an idealized profile for someone in Okuma after Fukushima
- In NPP release, nearby exposed individuals, see
  1. a rapid rise,
  2. a period of high, spikey dose rates until release stopped
  3. rapid decline due to decay of radioiodine
  4. far slower decline due to decay of cesium. Latter period can last decades.
- Whatever the profile, the daily dose is the area under the curve for that day.

Depending on the audience, explain key isotopes.

# Understanding Radiation Damage and Repair

- We are very good at DNA repair.
- Endogenous ROS  $\approx$  250 - 1000 mSv/d in DSB generation
- Expect any harm from 2 mSv/d to be undetectable
- We see no harm in humans unless dose rates above 20 mSv/d.
- Double DSB's one thing our repair systems have trouble with.
- Means dose rate and repair period all important.
- Must keep damage rate below repair rate lest inventory of unrepaired DSB's build.
- Need individual's dose rate thru time to figure out harm.

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