SESSION 2 OVERHEAD 1

ICRP MODEL ASSUMPTIONS

1. Stress to organism proportional to DOSE = $\Delta E/\Delta M$ Joules/kg (swallowing a hot coal)
2. Target is cell nuclear DNA which at low dose involves one track only.
3. Outcome is fatal cancer or heritable genetic damage modeled as clinical change.
4. Cancer diagnosed inside 5 years of exposure is not caused by the exposure.
5. Response is linear with no threshold.
6. External model is applicable to internal exposures.
7. Risk from chronic internal exposure is based on linear extrapolation of Hiroshima survivors wh had very large acute external dose.

OMISSIONS AND ERRORS IN THE ICRPMODEL

1. BIOKINETIC

. Dose anisotropy at sub cell level due to affinity of Strontium, Plutonium etc for DNA.
. Movement or distribution of particles smaller than 1 micron not addressed.

2. DOSIMETRIC

. Organs modelled as 'bags of water' into which radiation energy is uniformly distributed;
. Isotopes only distinguished by affinity for organs: not organelles.
. No a1lowance for the state of the cell or its responses or repair systems.
. No allowance for hot particles.
. No consideration of transmutation e.g. Tritium-Helium.

3. PHILOSOPHICAL

Scientific method not used.
Clear evidence of ill health following internal exposure at low dose is routinely dismissed on basis of deductive application of External Risk model and Hiroshima results. This is DEDUCTION whereas science demands use of INDUCTION.

SESSION 2 OVERHEAD 2

The target is the cell and its genetic material

The overall probability of fixed genetic damage and then cancer following low dose exposure is the result of sequential binomial probabilities i.e several different things have to occur.

A fixed mutation depends upon:

1. A window of ionization density at the target, not too large, not too small.
2. Ionization location in the cell i.e the target organelle.
3. The state of the cell (phase) when it is hit.
4. The cell's DNA repair efficiency.
5. The effects of neighbour cells on the target cell.
6. The effect of the target cell on neighbour cells.

The final expression of cancer then may depend upon:

7. The acquisition of other critical mutations in the cell or its daughters.
8. The viability of the cell and its daughters
9. Damage to neighbouring cells.
10. Rate of replication of the damaged cell and daughters.
11. Immune surveillance.

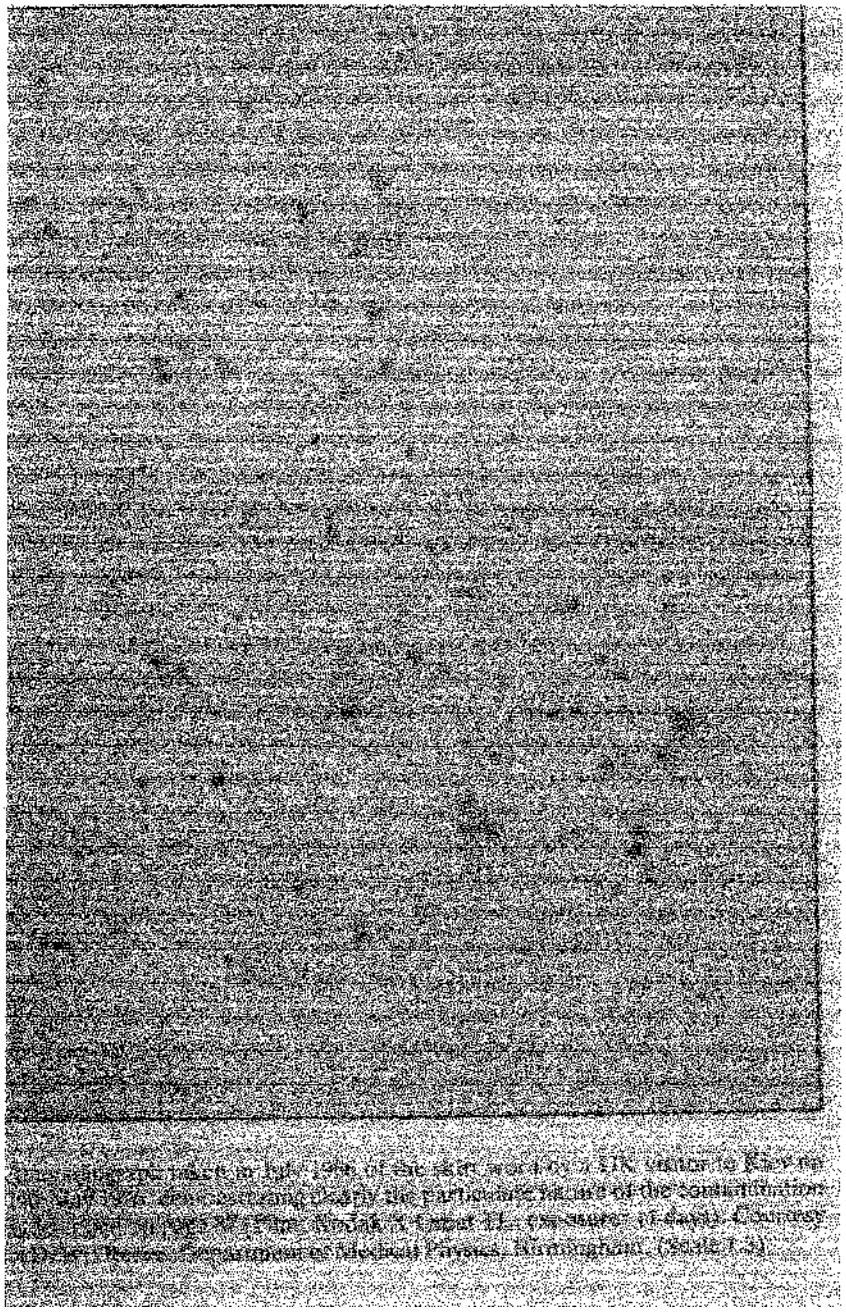
The ICRP model considers only No 1 in this list, ionization density, and even then, only in the case of RBE for alpha etc.

The Linear No Threshold model is both theoretically and empirically wrong

SESSION 2 OVERHEAD 3

Barsby CERRIE

2:3



SESSION 2 OVERHEAD 4

OPENING LATOUR'S BLACK BOX

THE RADIATION RISK BOX

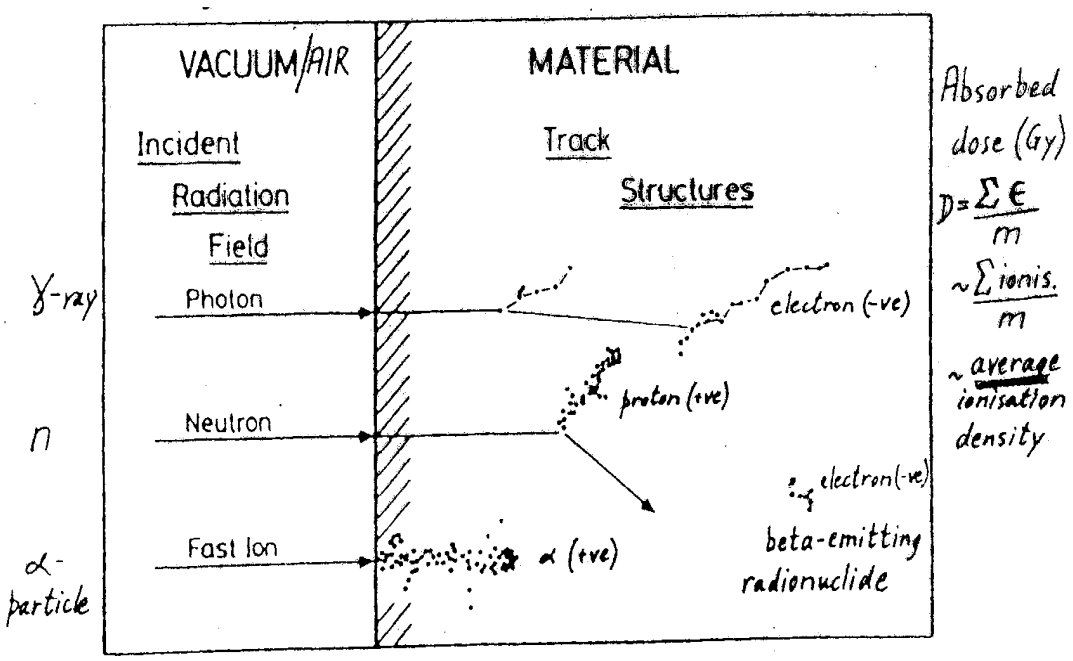
CONSTRUCTED 1945-1955 FROM PHYSICS
BASED BUILDING BLOCKS, ENERGY,
MASS, TARGETS, ABSORPTION
COEFFICIENTS ETC.

1. DNA STRUCTURE NOT DISCOVERED

2. CELL BIOLOGY BARELY BEGUN. CELL DNA REPAIR NOT DISCOVERED
3. MODERN SPECTROSCOPIC AND OTHER ANALYTICAL TOOLS NOT DEVELOPED

Fig 4 The effect of ionizing radiation at the molecular and cell level

Insult to cells from ionizing radiations is <u>always</u> in the form of structured <u>tracks</u> from charged particle



SESSION 2 OVERHEAD 5

Official recognition of need to re-assess ICRP model

1. ASPIS (DG XV) Kos statement on cancer epidemic.
2. European Parliament Resolution May 2001
3. WHO conference on Chernobyl conclusions Kiev June 2001
4. Formation of UK Committee Examining Radiation Risk from Internal Emitters CERRIE
5. Formation of the UK MoD Depleted Uranium Oversight Board
6. European Committee on Radiation Risk ECRR 2002

SESSION 2 OVERHEAD 6

Calculating absorbed dose from a 1 micron diameter Plutonium Oxide Particle using (1) dose to the lymphatic system defined by NRPB in R-276 (2) lymphatic system defined by ICRP 23 Reference Man (3) actual dose to tissue in range of the 30 micron alpha decays

Table l. Mass of tissue into which decay energy is diluted (g).

	NRPB R-276	**ICRP 23 Reference Man	30 micron radius
Lymph nodes	250		
Liver	1800		
Spleen	180		
Kidneys	310		
Pancreas	100		
Uterus	100		
Thymus	20		
Thyroid	20		
Stomach	150		
Intestine and colon	1000		
Red bone marrow	1500		
Cells on bone surface	1000		
Total lymphatic system	6430	800	1.13 x 10 E-7

* NRPB R-276: *p* 86 *Doses to the lymphatic system: No comprehensive assessment of doses to the lymphatic system has' been undertaken by an international body such as UNSCEAR or ICRP, In this report these doses have been calculated as the mass weighted average dose to a combination of tissues which account for a substantial fraction of the total lymphatic system o f the body. The combination of tissues used for the calculation comprises the thoracic and extra thoracic lymph nodes, the liver, spleen, kidmeys, pancreas, uterus, thymus, thyroid, stomach, small intestine, upper large intestine, colon, red bone marrow and cells on the bone surfaces.*
** *ICRP* 23: *Report of the Task Group on Reference Man* 1975

PuO_2; density 11.46; particle diameter = 1μ; particle Pu mass = 5.2×10^{-12} g; activity = 0.0119Bq; 43 decays/hr; decay energy = 5.16MeV = 8.25×10^{-13} J

Table 2: Absorbed dose (Grays) to lymphatics in the three calculations above.

	NRPB R-276 and COMARE 4	ICRP Reference man	Dose to 30μ sphere of tissue in range of α-'s
Per decay	1.28 E-13	1.03 x E-12	7.3 E-3
In 10 hrs repair period	5.5 E-11	4.4 E-10	3.1

SESSION 2 OVERHEAD 7

HIROSHIMA STUDY

ERRORS INVOLVED IN USING THE HIROSHIMA SURVIVORS TO PREDICT RISKS FOR LOW-LEVEL INTERNAL EXPOSURE

(1) EXTRAPOLATION OF HIGH-DOSE TO LOW DOSE.
Cells are killed at high dose but are just damaged at low-dose so that cancer and other results of mutation are unlikely to be linear functions of dose over the wide range between the very high doses received by the exposed and low-dose exposure. Cells at the high-dose level received hundreds of tracks, whereas at the low dose level they receive only one track.

(2) EXTRAPOLATION FROM EXTERNAL TO INTERNAL
External radiation gives average homogenous dose to all the cells and can be treated by averaging techniques. With internal radiation there may be very high doses in the vicinity of the internal isotopes due to biochemical concentration effects or due to short range radiation types

(3) OTHER EXTRAPOLATIONS and F AUL TS
Control group was exposed to internal radiation.
Extrapolation of Acute to Chronic irradiation
Extrapolation of Japanese population to European
Extrapolation of War survivors to peacetime population

SESSION 2 OVERHEAD 8

Mechanisms of failure of Hiroshima risk

1. Averaging errors
2. Inappropriate control
3. Internal vs external

Biological mechanisms

1. Second Event
2. Hot particles
3. Variation in cell sensitivity
4. Genomic instability???

SESSION 2 OVERHEAD 9

HIROSHIMA STUDY

ERRORS INVOLVED IN USING THE **HIROSHIMA** SURVIVORS TO PREDICT RIS-KS FOR LOW-LEVEL INTERNAL EXPORSURE

(1) EXTRAPOLATION OF HIGH-DOSE TO LOW DOSE.

Cells are killed at high dose but are just damaged at 1ow-dose so that cancer and other results of mutation are unlikely to be linear functions of dose over the wide range between the very high doses received by the exposed and low-dose exposure. Cells at the high dose level received hundreds of tracks, whereas at the low dose level they receive only one track.

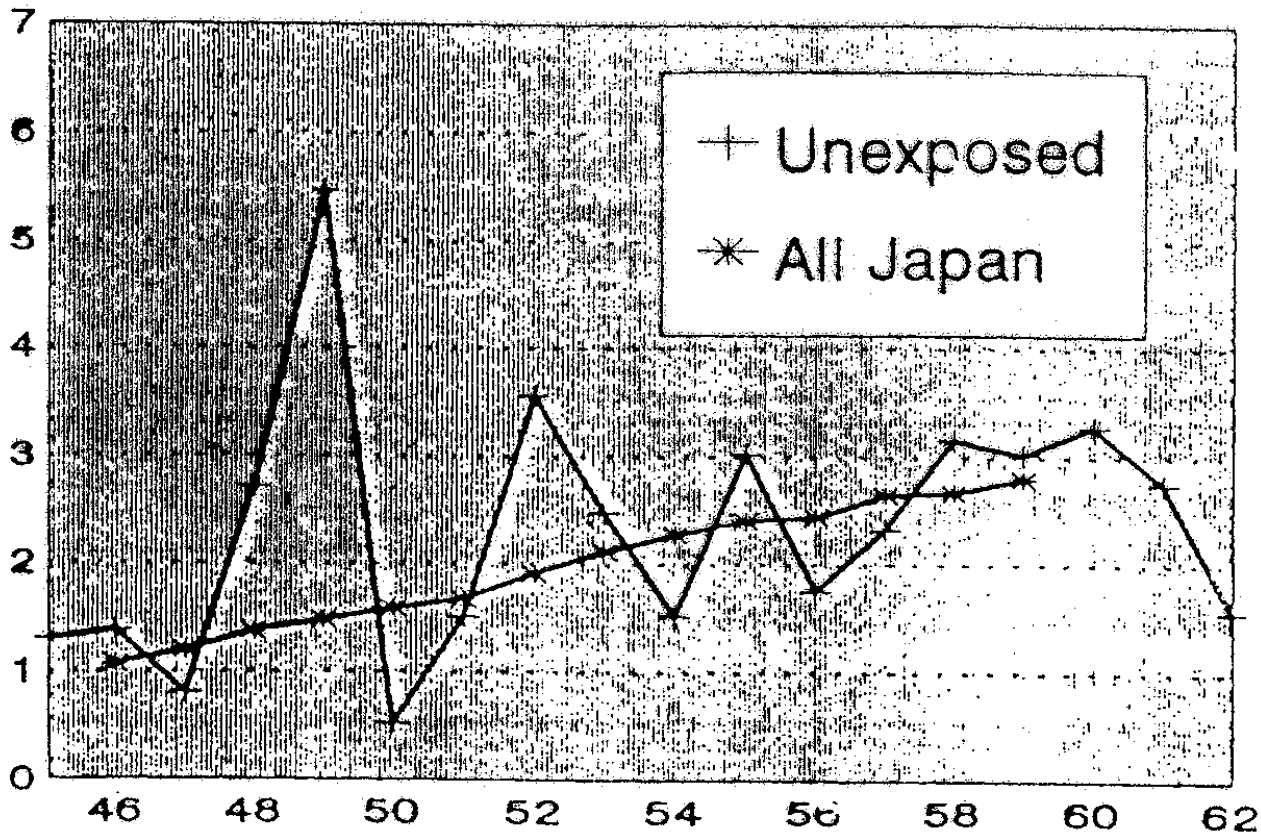
(2) EXTRAPOLATION FROM EXTERNAL TO INTERNAL

External radiation gives average homogenous dose to all the cells and can be treated by averaging techniques. With internal radiation there may be very high doses in the vicinity of the internal isotopes due to biochemical concentration effects or due to short range radiation types

(3) OTHER

Control group was exposed to internal radiation
Extrapolation of Acute to Chronic irradiation
Extrapolation of Japanese population to European
Extrapolation of War survivors to peacetime population

Fig 2 Leukemia in the Hiroshima Control Group



SESSION 2 OVERHEAD 10

Mechanism oversights for low dose region <10mSv:

Double hits to cell or cell component inside the repair replication period:

1. Double hit in space results in quadratic response to what is modeled as linear dose e.g. with hot particles

2. Double hit in time: Second Event.
Quiescent phase cells moved by first hit into high sensitivity repair replication phase and second hit inside ten hours damages cell with no chance of further repair. e.g. Sr-90/Y- 90 on chromosome or Plutonium hot particle.

Dia. (μ)	PuO2 Hits/d	PuO2 Sv/d	U3O8 Hits/d	U3O8 mmSv
0.2	8.3	1.2	3.3e-5	4e-3
1	1002	146	7.6e-3	0.9
2	8294	1220	0.03	3.7

. Hits per day and dose per day to sphere volume defined by the 30μ range of alpha decays from various common environmental particles

SESSION 2 OVERHEAD 11

Stage	Problem
Assume linear response	1. Assumes that at 1mGy, cell receives one hit per year. Not true for internal irradiation. 2. Empirical evidence shows non linear response at low dose.
Use external high-dose acute study to assess low-dose chronic internal	1. Theoretically invalid since internal exposure qualitatively different. 2. Empirical evidence shows higher cancer yield at lower dose. 3. Populations have different sensitivity. 4. Wartime Japanese survivors more healthy group. (Alice Stewart).
Check against internal studies.	Not adequate
Ignores non-cancer effects	e.g.non specific ageing, infant and perinatal mortality, sterility etc.

Conclusion

ICRP model may be approximately accurate for external irradiation but fails for internal irradiation, and as a consequence, through its misapplication, more than 50 million people will die worldwide as a result of nuclear pollution

SESSION 2 OVERHEAD 12

Risk of cancer following exposure Part II: recent evidence

11.1 *Nuclear sites and their proximity*
The period of major atmospheric weapons testing and fallout exposure ended in 1963 with the Kennedy-Kruschev test ban. During this period, no research papers linking fallout with cancer were published, and suggestions that the fallout had caused infant mortality were ridiculed and attacked. This may have been partly due to the secrecy and control associated with cold-war politics but may also have been a consequence of .the agreement made in 1958 between the World Health Organisation (WHO) and the International Atomic Energy Agency (IAEA) to leave such research to IAEA. The committee note that this agreement is still in force (though recent statements suggest that it is being reconsidered) and believe that accurate reports of the health consequences of the Chernobyl catastrophe may have been suppressed as a result of this. However, in 1983, a TV company discovered the first of the nuclear site childhood cancer and leukaemia clusters at Seascale near the nuclear fuel reprocessing plant Sellafield (earlier 'Windscale') in West Cumbria.

Following the confirmation of this by epidemiologists, and after a government enquiry, the UK government setup two new committees to (a) develop epidemiological surveillance methods for small areas and (b) investigate .the origin of the leukaemia excesses near nuclear sites. In the 15 years following the Sellafield leukaemia cluster, similar clusters were established near the other two reprocessing plants in Europe, Dounreay in Scotland and La Hague in northern France. In addition, childhood leukaemia clusters were reported for other nuclear sites which released radioisotopes to the environment, Aldermaston, Burghfield, Harwell and Hinkley Point in the UK, Kruemmel in Germany and Barsebeck in Sweden. The sites which have been studied are given in Table 11.1.

Nuclear Site	Year established	Defined ICRP Risk Multiplier	Notes
[a]Sellafield/ Windscale	1983	100-300	Well studied by COMARE: high level of discharge to atmosphere and sea
[a]Dounreay	1986	100-1000	Well studied by COMARE: particle discharges to atmosphere and sea.
[a]La Hague	1993	100-1000	Particle discharges to atmosphere and sea: ecological and case control studies
[c]Aldermaston/ Burghfield	1987	200-1000	Well studied by COMARE: particle discharges to atmosphere and rivers
[b]Hinkley Point	1988	200-1000	Discharges to offshore mud bank
[d]Harwell	1997	200-1000	Discharges to atmosphere and river
[b]Kruemmel, Germany	1992	200-1000	Discharges to atmosphere and river
[d]Julich, Germany	1996	200-1000	Discharges to atmosphere and river
[b]Barsebaeck, Sweden	1998	200-1000	Discharges to atmosphere and sea

[a]Reprocessing plants discharging to sea; [b]Nuclear power station discharging to sea or river; [c]Atomic weapon and nuclear material fabrication plants; [d]Atomic research with discharges to local rivers

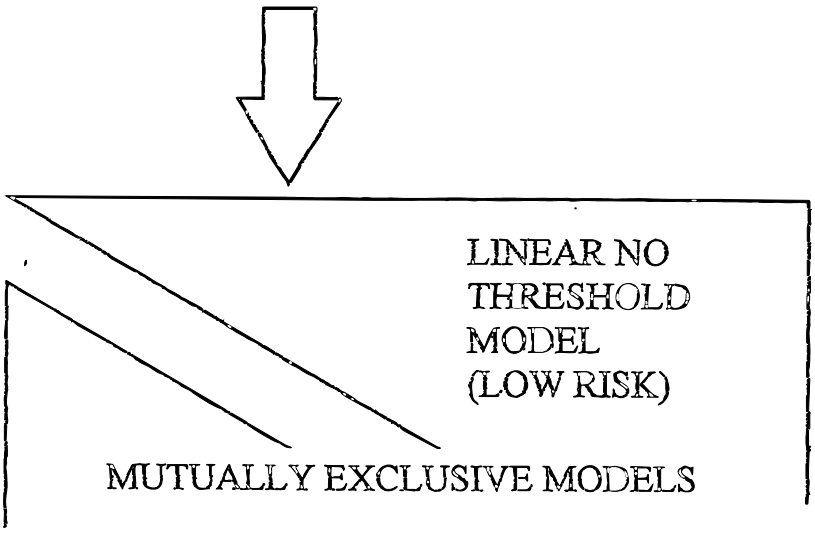
Table 11.1 Studies establishing excess leukaemia and cancer risk in children living near nuclear sites.

SESSION 2 OVERHEAD 13

A-BOME SURVIVORS
(HIGH DOSE
EXTERNAL

ACUTE)PHYSICS
AVERAGING
THEORETICAL

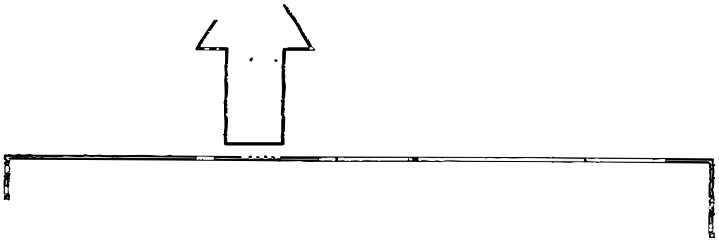
SCIENTIFIC
DEDUCTION



BIPHASIC
CELL RESPONSE MODEL
HIGH RISK)

BIOLOGY
EPIDEMIOLOGY
EMPIRICAL

SCIENTIFIC
INDUCTION



NUCLEAR SI1'E LEUKEMIAS (SELLAflELD)
IRISH SEA COAST EFFECT
CHERNOBYL INFANTS
MINISATELLITE MUTATI ONS
WEAPONS FALLOUT CANCERS
DU GULF VETERANS
IRAQI CHILDREN
(INTERNAL CHRONIC ISOTOPIC)

They were deliberating amongst themselves as to how they could give wings to Death, so that it could, in a moment, penetrate everywhere, both near and far.

Jan Amos Komensky (Comenius)
last Bishop Unitas Fratrus Bohemorum.
From: The Labyrinth of the Worlds.(Czech 1623)

SESSION 2 OVERHEAD 15

Evidence of error in Hiroshima risk analysis

1

Early evidence: Alice Stewart, Sternglass

2

Nuclear site cancer clusters:
Sellafield, Dounreay, La Hague, Aldermaston, Harwell

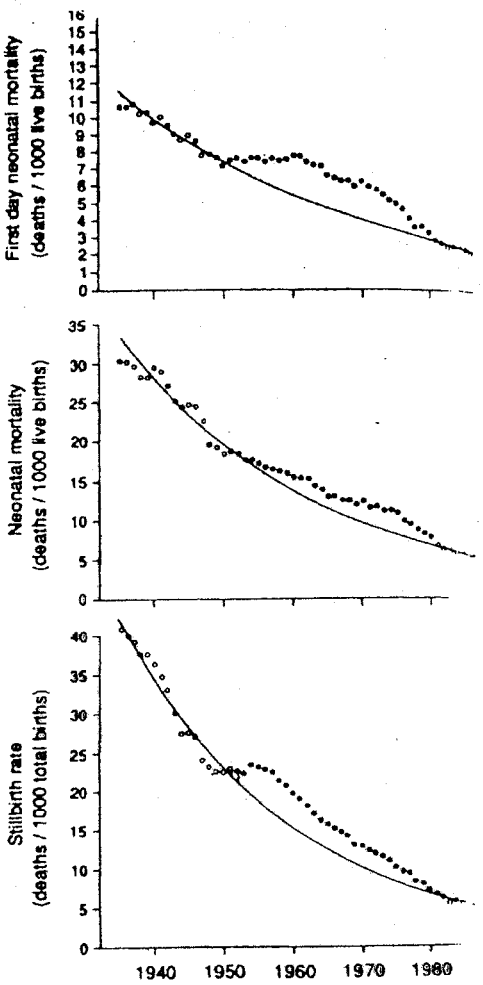
3.

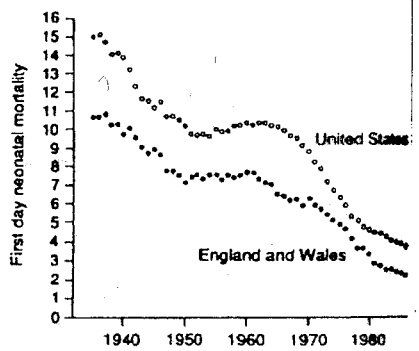
Post Chemobyl evidence:
Disputed effects in ex Soviet Union
Infant mortality effects
Genetic fingerprints in exposed children
Infant leukemia in Greece and US

4

Wales:
Weapons fallout effects: infant mortality, cancer increases
Post Chernobyl infant leukemia and low birthweight
Sellafield effects on the coast

session 2 overhead 16



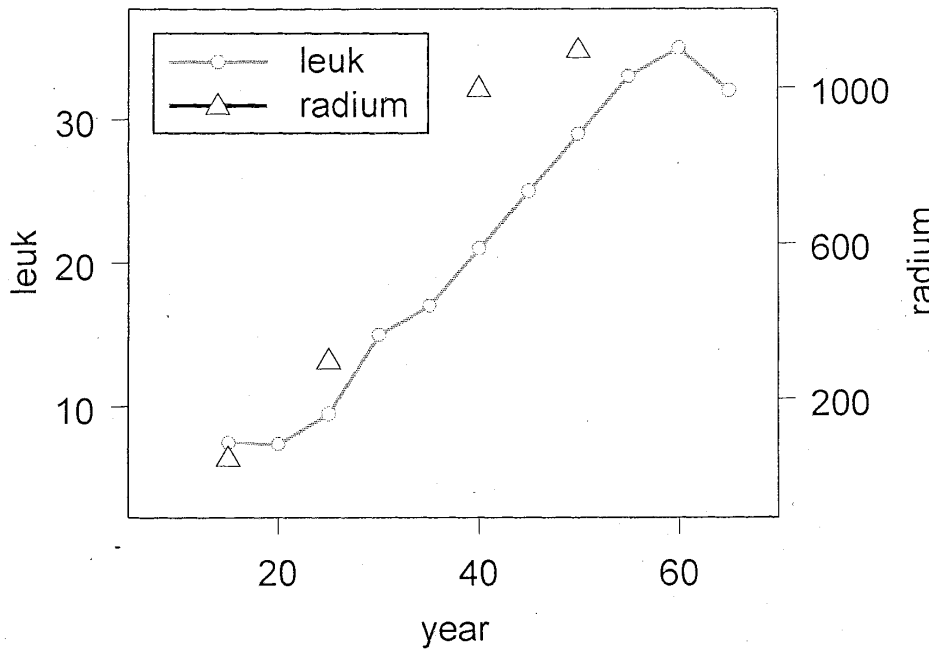


First day neonatal mortality. Deaths per 1000 live births for England and Wales and the United States by calendar years. Source: Whyte, 1992.

First day mortality, neonatal (0-28days) mortality, and stillbirth rates for England and Wales. Lines of best fit interpolated from data for conforming years 1935-50 and 1981-87. Solis circles correspond to deviant years. Source: Whyte, 1992.

Figure 11. Infant mortality effects in England and Wales and in the US (from Whyte- see text)

Session 2 overhead 17



SESSION 2 OVERHEAD 18

Main Shortcomings of Hiroshima and Nagasaki studies

"exposed" group = people who were in the open at the tine of thee explosion

Their exposure was

- . single
- . massive
- . acute
- . external
- . gamma rays

" control" group = people who were elsewhere at the time or were shielded.

Both groups lived in the bombed cities, and were therefore exposed to ingesting, inhaling and absorbing fallout.

The studies are therefore silent on internal radiation and the very different types of exposure involved:-

- chronic
- low dose
- **low** dose rate
- **internal**
- **alpha and beta** emitters.

SESSION 2 OVERHEAD 19

The system of theories which Freud has generally developed is so consistent that when one is entrenched in them it is difficult to make observations unbiased by his way of thinking.

Horney (1939)
New ways of psychoanalysis.

They reason excellently in the idiom of their beliefs, but they cannot reason outside, or against their beliefs, because they have no other idiom in which to express their thoughts.
. . . The contradiction between experience and one mystical notion is explained by reference to other mystical notions.

Evans-Pritchard (1937)
Witchcraft, Oracles and Magic among the Azande

[F or] the stability of the naturalistic system we currently accept, instead, rests on the same logical structure as Azande witchcraft beliefs. Any contradiction between a particular scientific notion and the facts of experience will be explained by other scientific notions. There is a ready reserve of possible scientific hypotheses available to explain any conceivable event. Secured by its circularity and defended by its epicyclical reserves science may deny or at least cast aside as of no scientific interest whole ranges of experience which to the unscientific mind appear both massive and vital.

M.Polanyi, FRS (1958)
Personal Knowledge

SESSION 2 OVERHEAD 20

Scientific Method

The classical exposition of the inductive method (originally due to William of Occam) is as what are now called Mill's Canons, the two most important of which are:

- The *Canon of Agreement,* which states that whatever there is in common between the antecedent conditions of a phenomenon can be supposed to be the cause or related to the cause of the phenomenon.
- The *Canon of Difference,* which states that the differences in the conditions under which an effect occurs and those under which it does not must be the cause or related to the cause of that effect.

In addition, the method relies upon the *Principle of Accumulation,* which states that scientific knowledge grows additively by the discovery of indendent laws, and the *Principle of Instance Confirmation,* that the degree of belief in the truth of a law is proportional to the number of favourable instances of the law.
In addition to the inductive method outlined above, the scientific method includes the range of analytical methods subsumed within Popper's *Doctrine of Falsifiability.* This regards science as moving forward through the experimental falsification of existing belief structures. Finally to the methods of inductive reasoning we must add considerations of
Plausibility of Mechanism. ,
These are the methods of science (Mill, 1879; Popper, 1962; Harre, 1985; Papineau, 1996